Comparative Nephroprotective Effect of Turmeric Essential Oil (Curcuma longa) and Losartan on Gentamicin-Induced Nephrotoxic Experimental Rats

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ABSTRACT

Background: Gentamicin is a commonly used aminoglycoside antibiotic, but its use is limited because it can cause kidney damage. This condition is marked by increased serum creatinine and blood urea nitrogen (BUN) levels along with tubular necrosis. Curcuma longa, or turmeric, essential oil contains strong antioxidant and anti-inflammatory compounds that may help protect the kidneys. This study aimed to assess and compare the kidney protective effects of turmeric essential oil (TEO) at two doses with losartan in rats with gentamicin-induced kidney injury. **Methods & Materials:** The study was carried out at the Department of Pharmacology and Therapeutics, Sir Salimullah Medical College, Dhaka, from July 2019 to June 2020. Gentamicin-induced nephrotoxicity was used to study the efficacy of TEO (50 and 100 mg/kg) and losartan (10 mg/kg). Statistical analysis of serum creatinine, BUN, and histopathological studies were done by using SPSS version 26. **Results:** Group B showed a significant increase in serum creatinine (1.38±0.05 vs 0.89±0.045 mg/dl) and BUN (47.00±2.16 vs 14.2±1.44 mg/dl) compared to Group A. Both doses of TEO (Groups C and D) significantly lowered these markers compared to Group B ($p<0.05$). Group D (100 mg/kg) achieved a greater reduction (creatinine: 1.10±0.03; BUN: 20.34±2.35 mg/dl), which was similar to Group C ($p>0.05$). Group E (losartan) showed some reduction but was significantly less effective than both TEO doses ($p<0.05$). Histopathologically, Group B had severe tubular necrosis and inflammatory infiltration. The TEO-treated groups showed only focal degeneration. **Conclusion:** Both turmeric essential oil and losartan show a dose-dependent ability to protect the kidneys. Turmeric essential oil was more effective than losartan in reducing gentamicin-induced kidney damage in rats. This finding calls for more research to translate these results.

Keywords: *Turmeric essential oil, Nephrotoxicity, Gentamicin, Losartan.*

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INTRODUCTION

Acute kidney injury (AKI) caused by drug-induced nephrotoxicity is a major challenge for both healthcare and public health. It accounts for a large portion of hospital-acquired kidney problems worldwide [1]. Among the nephrotoxic agents, gentamicin, an aminoglycoside antibiotic widely used against serious gram-negative infections, is one of the most common drugs involved. It can cause nephrotoxicity in up to 25% of patients treated, depending on the dose and duration [2]. The mechanisms behind gentamicin-induced nephrotoxicity are complex. They include the buildup of the drug in proximal tubular epithelial cells, the production of reactive oxygen species (ROS), mitochondrial dysfunction, and eventual cell death in the tubular cells. This leads to increased levels of serum creatinine and blood urea nitrogen (BUN) [3]. Conventional management strategies for gentamicin nephrotoxicity, including dose adjustment and the use of renin-angiotensin-aldosterone system (RAAS) blockers like losartan, offer only partial protection [4]. Losartan, which is an angiotensin II receptor blocker (ARB), helps reduce changes in renal blood flow and lowers tubular inflammation. However, its ability to protect the kidneys from oxidative stress caused by aminoglycoside injury is still limited [5]. This has led to significant interest in natural plant-based compounds that have antioxidant and anti-inflammatory properties as potential alternative or supplementary nephroprotective agents. Curcuma longa, commonly known as turmeric, is a plant from the Zingiberaceae family that has been used for centuries in traditional South Asian medicine due to its wide range of medicinal properties. While curcumin, the main polyphenolic compound, has attracted the most research attention, the essential oil fraction of Curcuma longa (TEO) contains various sesquiterpenes, monoterpenes, and aromatic compounds such as ar-turmerone, alpha-turmerone, and beta-turmerone [6]. These volatile compounds show strong abilities to scavenge free radicals, reduce inflammation, and stabilize cell membranes, which may be especially useful in addressing oxidative injury to the kidneys [7]. Preclinical evidence shows that Curcuma longa extracts can affect pro-inflammatory cytokines, block NF-κB signaling pathways, and lower lipid peroxidation in the kidney. This helps maintain tubular cell integrity [8]. The essential oil fraction has a different chemical makeup and bioavailability than curcumin. It has not been studied as thoroughly in terms of kidney protection, even though it has a promising antioxidant profile [9]. Additionally, limited experimental data compare TEO directly with established drugs like losartan in the published research. Given the increasing global issue of drug-induced AKI and the need for effective, low-cost kidney protection methods, it makes sense and is timely to evaluate plant-based oils in detailed in vivo studies [10]. Therefore, this study aimed to assess the kidney-protective effects of Curcuma longa essential oil at two dose levels (50 mg/kg and 100 mg/kg) and losartan against gentamicin-induced kidney damage in Wistar albino rats.

METHODS & MATERIALS

This experimental study took place at the Department of Pharmacology and Therapeutics, Sir Salimullah Medical College in Dhaka. It was done in collaboration with the Institute of Nutrition and Food Science at the University of Dhaka from July 2019 to June 2020. 25 healthy adult male Wistar albino rats, aged 10 to 12 weeks and weighing between 150

and 200 grams, were obtained and kept individually under standardized laboratory conditions. They experienced a 12-hour light/dark cycle, a controlled room temperature between 22 and 25 degrees Celsius, and had unrestricted access to standard rat chow and water. The animals were acclimatized for one week before the intervention. The sample size was calculated using the resource equation method for one-way ANOVA, resulting in (E = N – n = 20), which fell within the acceptable range of 10 to 20. This ensured enough statistical power. After acclimatization, rats were randomly divided into five equal groups (n=5 per group). Group A, the normal control, received normal saline via intraperitoneal injection throughout the study. Group B, the nephrotoxic control, received gentamicin at a dose of 80 mg/kg/day intraperitoneally from days 8 to 14. Groups C and D received Curcuma longa essential oil at doses of 50 mg/kg/day and 100 mg/kg/day orally, respectively, for 14 days, with gentamicin also administered during the final seven days. Group E received losartan at a dose of 10 mg/kg/day orally for 14 days, plus gentamicin during the last seven days. Turmeric essential oil was made in-house by hydrodistilling fresh Curcuma longa rhizomes at 105 degrees Celsius for four hours using a Clevenger-type apparatus. On day 15, the animals were euthanized under light anesthesia. Blood samples were taken via cardiac puncture for serum creatinine and blood urea nitrogen (BUN) measurement using the Jaffe and urease-coupled colorimetric methods, respectively. Both kidneys were collected and preserved in 10% neutral buffered formalin for histopathological examination. Tissue sections were stained with hematoxylin and eosin (H&E) and evaluated for glomerular integrity, tubular necrosis, vacuolation, inflammatory cell infiltration, and vascular congestion using a validated semi-quantitative scoring system. Data were entered and analyzed using SPSS version 26.0. Continuous variables were presented as mean ± standard deviation (SD). One-way ANOVA was used for comparing multiple groups, followed by unpaired t-tests for pairwise comparisons. A p-value of less than 0.05 was considered significant.

RESULTS

Table I shows the comparison of serum creatinine and BUN in all five groups. Gentamicin treatment in Group B caused a significant rise in both biomarkers compared to the normal control Group A. This confirms that nephrotoxicity was successfully induced. Turmeric essential oil at both doses in Groups C and D significantly lowered serum creatinine and BUN compared to Group B, with Group D having the lowest levels. Group E (losartan) also showed reduced levels compared to Group B, but remained higher than both TEO-treated groups. All differences between the groups were statistically significant ($p<0.05$), indicating a clear dose-response and ranking order.

Table I

Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels in experimental rats of different groups. (*n*=25).

Parameters (mg/dl)	Groups					p-value
	A (n=5)	B (n=5)	C (n=5)	D (n=5)	E (n=5)	
Serum Creatinine	0.89±0.04	1.38±0.05	1.13±0.06	1.10±0.03	1.22±0.03	<0.05
Blood Urea Nitrogen (BUN)	14.2±1.44	47.00±2.16	21.4±1.6	20.34±2.35	30.93±1.41	<0.05

Group A: *Normal control group.* ***Group B:*** *Gentamicin-treated group.* ***Group C:*** *Turmeric essential oil (50 mg/kg orally) treated and Gentamicin-treated experimental group.* ***Group D:*** *Turmeric essential oil (100 mg/kg orally) treated and Gentamicin-treated experimental group.* ***Group E:*** *Losartan (10 mg/kg orally) pretreated and Gentamicin-treated experimental group.*

Table II shows that gentamicin treatment significantly raised serum creatinine from 0.89±0.045 mg/dl to 1.38±0.054 mg/dl and BUN from 14.20±1.44 to 47.00±2.16 mg/dl ($p<0.05$ for both) when compared to the normal control group. This notable increase in both renal function markers supports the model of gentamicin-induced nephrotoxicity. It confirms that Group B serves as a dependable baseline for testing future nephroprotective treatments (*Table II*).

Table II

Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels between group A (n=5) and group B (*n*=5).

Kidney function parameters (mg/dl)	Group A	Group B	*p*-value
Serum creatinine	0.89±0.045	1.38±0.054	<0.05
Blood Urea Nitrogen (BUN)	14.20±1.44	47.00±2.16	<0.05

Table III shows that pre-treatment with turmeric essential oil at 50 mg/kg/day (Group C) significantly lowered serum creatinine to 1.13±0.064 mg/dl and BUN to 21.4±1.6 mg/dl. In comparison, the gentamicin-only group (Group B) had values of 1.38±0.054 and 47.00±2.16 mg/dl, respectively ($p<0.05$ for both parameters). This important reduction highlights the protective effect of the lower dose of turmeric essential oil. It demonstrates its ability to lessen gentamicin-related oxidative and inflammatory kidney damage.

Table III

Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels between group B (n=5) and group C (*n*=5).

Kidney function parameters (mg/dl)	Group B	Group C	*p*-value
Serum creatinine	1.38±0.054	1.13±0.064	<0.05
Blood Urea Nitrogen (BUN)	47.00±2.16	21.4±1.6	<0.05

Table IV shows that the higher dose of turmeric essential oil (100 mg/kg/day, Group D) provided better protection for the kidneys. Serum creatinine levels dropped to 1.10±0.034 mg/dl and BUN to 20.09±2.35 mg/dl. Both of these values were significantly lower than those in Group B ($p<0.05$). The slightly better reduction in kidney markers at this dose compared to Group C suggests a dose-dependent protective effect of Curcuma longa essential oil. This effect may be linked to increased antioxidant enzyme activity at higher levels of its active turmerone compounds.

Table IV
Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels between group B (n=5) and group D (*n*=5).

Kidney function parameters (mg/dl)	Group B	Group D	*p*-value
Serum creatinine	1.38±0.054	1.10±0.034	<0.05
Blood Urea Nitrogen (BUN)	47.00±2.16	20.09±2.35	<0.05

Table V shows that losartan (10 mg/kg/day, Group E) provided a significant but modest level of nephroprotection against gentamicin-induced injury. It reduced serum creatinine to 1.22±0.034 mg/dl and BUN to 30.93±1.41 mg/dl compared to Group B ($p<0.05$). Losartan's partial protective effect likely comes from its ability to reduce angiotensin II-mediated intrarenal vasoconstriction and inflammation. However, the remaining increase in renal biomarkers compared to TEO-treated groups indicates a more limited effect against nephrotoxicity caused by oxidative stress.

Table V
Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels between group B (n=5) and group E (*n*=5).

Kidney function parameters (mg/dl)	Group B	Group E	*p*-value
Serum creatinine	1.38±0.054	1.22±0.034	<0.05
Blood Urea Nitrogen (BUN)	47.00±2.16	30.93±1.41	<0.05

Table VI shows that when comparing the two doses of turmeric essential oil (50 mg/kg in Group C and 100 mg/kg in Group D), the differences in serum creatinine (1.13±0.064 vs. 1.10±0.034 mg/dl) and BUN (21.4±1.6 vs. 20.09±2.35 mg/dl) were not statistically significant ($p>0.05$ for both). This finding suggests that both doses effectively reduce gentamicin-induced nephrotoxicity. The 50 mg/kg dose already provides nearly maximum protection for the kidneys, with only a slight and statistically similar benefit at the 100 mg/kg dose.

Table VI
Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels between group C (n=5) and group D (n=5).

Kidney function parameters (mg/dl)	Group C	Group D	*p*-value
Serum creatinine	1.13±0.064	1.10±0.034	>0.05
Blood Urea Nitrogen (BUN)	21.4±1.6	20.09±2.35	>0.05

Table VII shows that turmeric essential oil at 50 mg/kg (Group C) is more effective than losartan (Group E) in lowering serum creatinine levels (1.13±0.064 vs. 1.22±0.034 mg/dl) and BUN (21.4±1.6 vs. 30.93±1.41 mg/dl) ($p<0.05$). This result is important because it suggests that even a lower dose of Curcuma longa essential oil provides better kidney protection than the standard drug, losartan. This may be due to TEO's wider antioxidant and anti-inflammatory effects, which go beyond just regulating the RAAS.

Table VII
Comparison of Serum creatinine and Blood Urea Nitrogen (BUN) levels between group C (n=5) and group E (*n*=5).

Kidney function parameters (mg/dl)	Group C	Group E	*p*-value
Serum creatinine	1.13±0.064	1.22±0.034	<0.05
Blood Urea Nitrogen (BUN)	21.4±1.6	30.93±1.41	<0.05

Tables VIII a and b show the histopathological assessment of renal tissue using a reliable semi-quantitative scoring system. Group A kidneys were mostly normal with slight baseline vascular congestion. Group B had the most severe damage, with widespread inflammatory cell infiltration (grade 4), significant vascular congestion (grade 3), and necrosis affecting more than half of the proximal tubules. This group had the highest average damage score of 3. All treatment groups (C, D, and E) had much lower histopathological injury, each achieving an average score of 1. They only showed focal tubular degeneration, mild infiltration, and signs of regenerative activity, which confirmed the biochemical findings at the tissue level.

Table VIII (a)
Scores of the histopathological renal damage of rats of different treatments (*n*=25).

Histopathological renal damage of rats	Group-A n=5	Group-B n=5	Group-C n=5	Group-D n=5	Group-E n=5
Glomerular & Interstitial infiltration of inflammatory cells.	-	++++	++	+	+
Congestion of blood vessels	+	+++	+	+	+
Desquamation of necrosis of proximal tubules	-	>½ of proximal tubules	Focal tubular degeneration	Focal tubular degeneration	Focal tubular degeneration
Focal regeneration of tubular epithelium	-	-	+	+	+
Vacuolation of the interstitium	-	++	+	+	+
Average score	0	3	1	1	1

Table VIII (b)
Semi-Quantitative Grading System.

Symbol	Meaning	Severity Level	Approximate Interpretation
-	Absent	None	No detectable lesion
+	Mild	Grade 1	Minimal change, focal involvement
++	Moderate	Grade 2	Noticeable lesion, limited distribution
+++	Marked	Grade 3	Extensive involvement
++++	Severe	Grade 4	Diffuse, intense lesion affecting the majority of the tissue

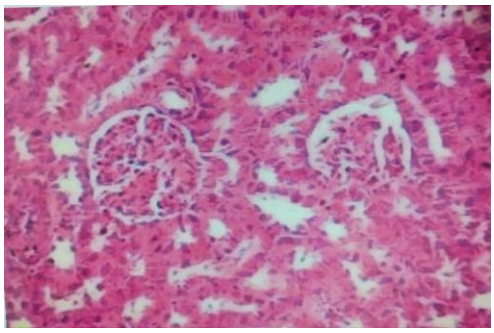

Figure 1 Photomicrograph of kidney sections of Control group (Group A).

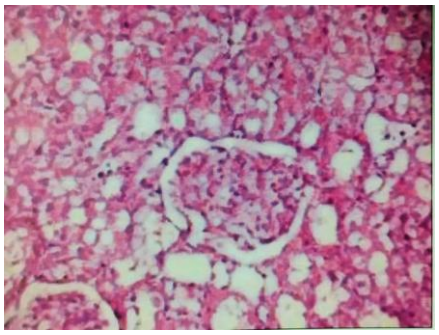

Figure 2 Photomicrograph of kidney sections from the Gentamicin-treated group (Group B).

Figure 1 showing normal glomerular structure & interstitium and no inflammatory cell infiltration (100 X, H &E). *Figure 2* showing inflammatory cell (lymphocyte) infiltration, severe tubular necrosis, vacuolation of the interstitium, and congestion of blood vessels (100 X, H & E).

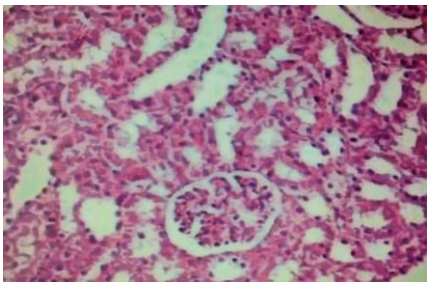

Figure 3 Photomicrograph of kidney sections treated with turmeric essential oil (50 mg/kg) (group C).

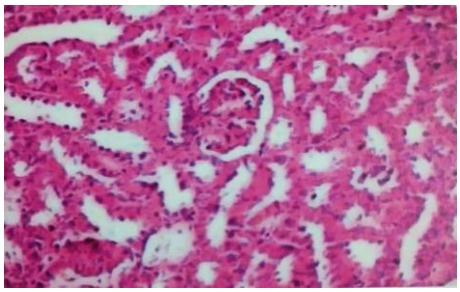

Figure 4 Photomicrograph of kidney sections treated with turmeric essential oil (100 mg/kg) (group D).

Figure 3 showing dilated tubule and degenerated tubular epithelium, mild necrosis and vacuolation, less inflammatory cell infiltration, and blood vessel congestion (100X, H &E). *Figure 4* showing dilated tubule and degenerated tubular epithelium, mild necrosis and vacuolation, few inflammatory cell infiltration and blood vessels congestion (100X, H &E).

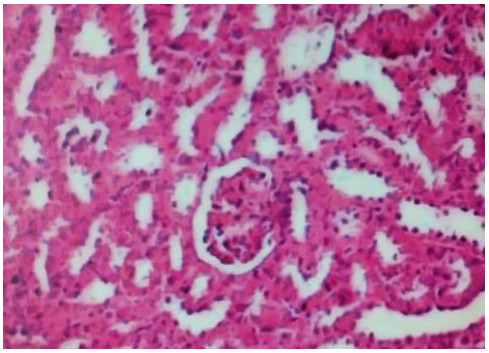

Figure 5 Photomicrograph of kidney sections treated with Losartan (10 mg/kg) (group E)

Figure 5 showing dilated tubule and degenerated tubular epithelium, moderate necrosis and vacuolation, inflammatory cell infiltration, and blood vessel congestion (100X, H &E).

DISCUSSION

This study demonstrates that administering gentamicin at 80 mg/kg/day for seven days reliably causes kidney damage in Wistar albino rats. This is indicated by significantly higher serum creatinine (1.38±0.05 mg/dl) and BUN (47.00±2.16 mg/dl). There was also severe kidney damage in Group B compared to the normal control ($p<0.05$). These results match previous models of aminoglycoside kidney damage. In these models, gentamicin uptake in proximal tubules triggers oxidative processes. This leads to mitochondrial dysfunction, tubular cell death, and impaired glomerular filtration [11]. A key finding of this study is the significant and dose-dependent protective effect of Curcuma longa essential oil. Both the 50 mg/kg (Group C) and 100 mg/kg (Group D) doses significantly lowered serum creatinine and BUN compared to the nephrotoxic control ($p<0.05$). The therapeutic potential of this result is backed by the known active compounds in turmeric essential oil, especially ar-turmerone and alpha-turmerone, which have strong free-radical scavenging and membrane-stabilizing effects [12]. Oxidative stress plays a major role in gentamicin nephrotoxicity. Therefore, the antioxidant activity of turmeric essential oil likely disrupts the process of tubular cell death and damage caused by reactive oxygen species, helping to maintain kidney filtration capacity [13]. Direct comparison between the two TEO doses showed no statistically significant difference in renoprotection ($p>0.05$). This means that 50 mg/kg essentially reaches a limit when it comes to lowering creatinine and BUN in this model. This plateau might indicate saturation of antioxidant enzyme induction pathways, particularly superoxide dismutase and catalase, at the lower dose. A previous study by Luo et al. on curcuminoid-rich fractions has also shown threshold effects in reducing oxidative biomarkers [14]. The lack of a dose-response gradient between Groups C and D suggests that even the lower concentration of TEO is enough to significantly counteract gentamicin-induced oxidative damage in rat kidneys. The comparison between TEO-treated groups and losartan (Group E) is particularly informative. Losartan, which is an angiotensin II type-1 receptor blocker, offered partial renal protection (creatinine: 1.22±0.034 mg/dl; BUN: 30.93±1.41 mg/dl). This is in line with a report by Rovin et al. showing that ARBs can reduce renal inflammation and hemodynamic stress [15]. However, both TEO doses were significantly better than losartan ($p<0.05$). This advantage likely comes from TEO's multiple actions, including antioxidant, anti-inflammatory, and possibly anti-apoptotic pathways, while losartan mainly works through RAAS blockade without directly targeting the generation of reactive oxygen species [16]. The histopathological findings support the biochemical results. Group B kidneys showed widespread inflammation, significant vascular congestion, and damage to more than half the proximal tubules. All intervention groups (C, D, and E) had noticeably lower pathological scores, averaging 1 compared to 3. They displayed only minor tubular degeneration and early signs of regeneration [17]. This suggests that TEO and losartan both help maintain tubular structure, though they work through different mechanisms. The occurrence of focal epithelial regeneration in treated groups signals active protection rather than just slowing down injury progression. The bioactive sesquiterpene compounds in TEO are known to reduce the transcription of pro-inflammatory cytokines like TNF-α, IL-6, and IL-1β, which play a key role in sustaining tubular inflammation in gentamicin nephrotoxicity [18]. Furthermore, turmerones might bind to iron and lessen the production of hydroxyl radicals in renal tubular cells, which is a different mechanism from that of losartan [19]. The combined antioxidant and anti-inflammatory effects may explain the overall kidney protection seen in TEO-treated groups, even at the lower dose. Turmeric essential oil is a natural product that is easily accessible and low-cost. It is widely grown in South and Southeast Asia. If further studies confirm its effectiveness, it could serve as an affordable option to protect the kidneys for patients using potentially harmful aminoglycosides. This is especially important in healthcare settings with limited resources [20]. However, this study only used a rodent model. To apply these findings to humans, we need well-designed phase I and II clinical trials to confirm safety, pharmacokinetics, and proper dosing [21]. This study shows strong experimental evidence that Curcuma longa essential oil provides better protection for the kidneys compared to losartan in a rat model of acute kidney injury induced by gentamicin. The safety, accessibility, and diverse mechanisms of turmeric essential oil suggest it has good potential as a natural kidney protector, which deserves further clinical research.

LIMITATIONS

This study focuses on an experimental rodent model. The results related to kidney protection may not directly apply to humans without more research on how the drug moves in the body and its safety. Also, the small number of subjects in each group, along with the lack of specific biomarkers like oxidative stress enzymes, inflammatory cytokines, and kidney injury markers, limits how deeply we can interpret the findings.

CONCLUSION

Both Turmeric essential oil and losartan demonstrated significant nephroprotective effects against gentamicin-induced renal injury, reflected by reduced serum creatinine and BUN levels and improved renal histology. Both 50 mg/kg and 100 mg/kg doses of TEO provided comparable protection, suggesting that the lower dose may achieve near-maximal efficacy. The nephroprotective effect of TEO was superior to that of losartan. These findings support the potential of turmeric essential oil as a promising candidate for further pharmacological and clinical evaluation in nephroprotection.

RECOMMENDATIONS

Future studies should explore the specific molecular mechanisms that enable TEO nephroprotection. This includes looking at how the NF-κB pathway is modified and how antioxidant enzymes are produced. Eventually, well-controlled phase I and II clinical trials in humans are needed to evaluate safety, bioavailability, and effectiveness in patients receiving aminoglycoside therapy.

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CONFLICT OF INTEREST

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