

Hepatoprotective and Nephroprotective Effects of Empagliflozin and Ethanolic-leaf extract of *Mentha piperita* against Aluminium chloride and D-galactose-Induced Oxidative Stress in the Liver and Kidney of Wistar Rats

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Abstract

Background: Aluminium chloride (AlCl₃) and D-galactose induce systemic oxidative toxicity with documented accumulation in the liver and kidney. This study evaluated the hepatoprotective and nephroprotective effects of empagliflozin (EMPG) and ethanolic leaf extract of *Mentha piperita* (ELMP) against AlCl₃ and D-galactose-induced oxidative stress in liver and kidney tissues of male Wistar rats.

Methods: Thirty-six male Wistar rats were divided into six groups (n=6): normal control (A), disease control (B: AlCl₃ 150 mg/kg plus D-galactose 50 mg/kg for 6 weeks), and four treated groups (C and D: ELMP 200 or 400 mg/kg; E: EMPG 10 mg/kg; F: ELMP 400 mg/kg plus EMPG 10 mg/kg) given for 4 weeks alongside AlCl₃ and D-galactose. Tissues were assayed for malondialdehyde (MDA), superoxide dismutase (SOD), reduced glutathione (GSH), and catalase. Data were analysed by one-way ANOVA with post hoc LSD comparison at p less than or equal to 0.05.

Results: Group B showed significantly elevated hepatic and renal MDA alongside severely depleted SOD, GSH, and catalase compared to Group A. ELMP produced dose-dependent restoration of antioxidant enzymes. Group F achieved the most complete restoration, approaching normal control values in both organs. F-ratios confirmed significant overall group differences across all parameters (kidney MDA: 56.125; liver MDA: 49.366).

Conclusion: AlCl₃ and D-galactose induce significant hepatic and renal oxidative stress. ELMP and EMPG, particularly in combination, demonstrate potent hepatoprotective and nephroprotective effects through multi-target antioxidant mechanisms, supporting their therapeutic potential as integrated strategies for peripheral organ protection in aluminium-induced toxicity.

Keywords: *Mentha piperita*; Empagliflozin; Hepatoprotection; Nephroprotection; Oxidative stress; Aluminium chloride

1. Introduction

Aluminium is among the most abundant metallic elements in the earth's crust and constitutes a major component of human environmental exposure through food additives, drinking water, pharmaceutical products, and occupational contact. While aluminium in its inert form poses limited biological risk, its ionic form, particularly trivalent aluminium

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(Al³⁺), demonstrates significant pro-oxidant activity upon systemic absorption. Aluminium chloride (AlCl₃) has been extensively employed as an experimental model of aluminium toxicity in rodents owing to its consistent ability to induce oxidative stress, inflammatory responses, and organ damage across multiple biological compartments [1,2].

D-galactose is a reducing sugar that, upon chronic systemic administration, undergoes metabolic conversion to galactitol and reactive oxygen species (ROS) via an aldose reductase-mediated pathway. This process accelerates cellular senescence, promotes mitochondrial dysfunction, and induces a systemic ageing phenotype characterised by elevated oxidative damage markers across multiple organs [3,4]. The combined administration of AlCl₃ and D-galactose therefore creates a compounded model of oxidative toxicity that simultaneously targets the central nervous system and peripheral organs, including the liver and kidney.

The liver and kidney are primary sites of aluminium biotransformation and excretion, rendering them particularly susceptible to AlCl₃-induced oxidative injury. Hepatocytes and renal tubular cells are rich in polyunsaturated fatty acids and operate under high metabolic demands, making them highly vulnerable to reactive oxygen species-mediated lipid peroxidation [1]. Elevated malondialdehyde (MDA), a terminal product of lipid peroxidation, alongside depletion of enzymatic antioxidants, namely superoxide dismutase (SOD), reduced glutathione (GSH), and catalase, are established hallmarks of hepatic and renal oxidative toxicity in aluminium exposure models.

Mentha piperita L. (peppermint) is a widely studied medicinal plant with documented antioxidant, anti-inflammatory, and hepatoprotective properties attributable to its diverse phytochemical profile. GC-FID analysis of its ethanolic leaf extract (ELMP) has identified limonene (68.62%), citral (6.62%), myricetin (6.60%), flavone (5.91%), nobiletin (5.89%), epicatechin (4.88%), and resveratrol (4.82%) as major constituents, all possessing well characterised antioxidant and organ-protective properties [5,6]. Empagliflozin (EMPG), a sodium-glucose cotransporter-2 (SGLT2) inhibitor, has demonstrated antioxidant and anti-inflammatory properties beyond its primary antidiabetic mechanism, with emerging evidence of hepatoprotective and nephroprotective effects through AMPK pathway activation and mitochondrial protection [7,8].

Despite growing evidence of peripheral organ toxicity in AlCl₃ and D-galactose models, studies simultaneously evaluating hepatic and renal oxidative stress responses to combined phytochemical and pharmacological intervention remain limited. This study therefore aimed to evaluate the hepatoprotective and nephroprotective effects of ELMP and EMPG against AlCl₃ and D-galactose-induced oxidative stress in the liver and kidney of Wistar rats.

2. Materials and methods

2.1. Experimental animals

Thirty-six adult male Wistar rats (180 to 200 g) were obtained from the Animal House Facility of the Department of Physiology, Faculty of Basic Medical Sciences, College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus. The animals were maintained under standard laboratory conditions (12-hour light/dark cycle, temperature 22±2°C, relative humidity 50 to 60%) with free access to standard rat chow and clean drinking water throughout the experimental period. All procedures were conducted in accordance with internationally accepted principles for laboratory animal use and care.

2.2. Acute toxicity and sample size determination

Acute toxicity (LD₅₀) of ELMP was determined using Lorke's method [9]. The LD₅₀ was established as greater than 5000 mg/kg, confirming the safety of the extract for experimental use. Sample size was determined using the one-way ANOVA formula as described by Arifin and Wan Mohammad [10], yielding a minimum of 6 animals per group. An additional buffer of 8 animals was included, giving a total of 36 rats across 6 groups.

2.3. Experimental groups and drug administration

Animals were randomly assigned to six groups of 6 rats each:

- Group A (normal control): received feed and water ad libitum.
- Group B (disease control): AlCl₃ 150 mg/kg plus D-galactose 50 mg/kg orally for 6 weeks.
- Group C: AlCl₃ plus D-galactose for 6 weeks plus ELMP 200 mg/kg orally for 4 weeks.
- Group D: AlCl₃ plus D-galactose for 6 weeks plus ELMP 400 mg/kg orally for 4 weeks.
- Group E: AlCl₃ plus D-galactose for 6 weeks plus EMPG 10 mg/kg orally for 4 weeks.
- Group F: AlCl₃ plus D-galactose for 6 weeks plus ELMP 400 mg/kg plus EMPG 10 mg/kg for 4 weeks.

2.4. Tissue harvest and biochemical assays

At the end of the experimental period, animals were anaesthetised using mild chloroform and sacrificed. The liver and kidneys were excised, rinsed in ice-cold phosphate-buffered saline (PBS, 0.01 M, pH 7.4), weighed, and homogenised in PBS at a 1:9 tissue-to-buffer ratio using a glass homogeniser on ice. Homogenates were centrifuged at 5000×g for 10 minutes at 2 to 8°C and the resulting supernatants were used for biochemical assays.

Malondialdehyde (MDA) levels were determined by the thiobarbituric acid reactive substances (TBARS) method as described by Aguilar Diaz De Leon and Borges [11]. Superoxide dismutase (SOD) activity was assessed by the method of Yalcin et al. [12], based on inhibition of epinephrine auto-oxidation at 480 nm. Reduced glutathione (GSH) was quantified using the DTNB (5,5' dithiobis-2-nitrobenzoic acid) method at 412 nm. Catalase activity was assessed by incubating enzyme samples in hydrogen peroxide substrate in sodium-potassium phosphate buffer (pH 7.4) at 37°C for 3 minutes, stopped with ammonium molybdate, and read at 374 nm.

2.5. Statistical analysis

Data are expressed as mean±SEM. Statistical analysis was performed using one-way ANOVA followed by post hoc LSD comparison. Significance was set at p less than or equal to 0.05. An asterisk (*) denotes significance compared to Group A; a hash (#) denotes significance compared to Group B.

3. Results

3.1. Effect on kidney oxidative stress markers

Table 1 Effect of ethanolic leaf extract of *Mentha piperita* and empagliflozin on oxidative stress markers and antioxidant enzymes in kidney tissue of aluminium chloride and D-galactose-induced rats

Group	MDA (nmol/mg)	SOD (U/mg)	GSH (μmol/mg)	Catalase (U/mg)
Group A (control)	1.10±0.07	40.2±2.0	33.8±1.8	26.9±1.4
Group B (AlCl ₃ + D-galactose, 6 weeks)	4.10±0.24*	16.5±1.3*	11.2±1.0*	9.60±0.8*
Group C (AlCl ₃ + D-galactose, 6 weeks; ELMP 200 mg/kg, 4 weeks)	2.60±0.15*#	28.4±1.7*#	23.1±1.4*#	18.7±1.1*#
Group D (AlCl ₃ + D-galactose, 6 weeks; ELMP 400 mg/kg, 4 weeks)	2.00±0.12*#	34.9±1.9#	29.0±1.7#	23.2±1.3#
Group E (AlCl ₃ + D-galactose, 6 weeks; EMPG 10 mg/kg, 4 weeks)	2.20±0.13*#	27.1±1.6*#	20.8±1.3*#	15.0±0.9*#
Group F (AlCl ₃ + D-galactose, 6 weeks; ELMP 400 mg/kg + EMPG 10 mg/kg, 4 weeks)	1.35±0.08#	38.6±2.1#	32.4±1.8#	25.5±1.4#
F-ratio	56.125	24.414	30.802	32.321

AlCl₃: aluminium chloride; ELMP: ethanolic leaf extract of *Mentha piperita*; EMPG: empagliflozin; MDA: malondialdehyde; SOD: superoxide dismutase; GSH: reduced glutathione. Data were analysed using ANOVA followed by post hoc LSD comparison and values were considered significant at p less than or equal to 0.05. *: significant compared to Group A; #: significant compared to Group B.

Table 1 reveals that renal MDA levels were significantly elevated in Group B (4.10±0.24 nmol/mg) compared to Group A (1.10±0.07 nmol/mg), confirming significant lipid peroxidation induced by AlCl₃ and D-galactose. Renal SOD, GSH, and catalase activities were markedly depleted in Group B (16.5±1.3, 11.2±1.0, and 9.60±0.8 U/mg respectively) compared to Group A (40.2±2.0, 33.8±1.8, and 26.9±1.4 U/mg respectively). Treatment with ELMP produced dose-dependent reductions in renal MDA alongside dose-dependent restoration of SOD, GSH, and catalase activities. Group D (ELMP 400 mg/kg) achieved the highest individual-treatment restoration of all three antioxidant enzymes (SOD: 34.9±1.9; GSH: 29.0±1.7; catalase: 23.2±1.3 U/mg). Group E (EMPG) also improved renal antioxidant markers. The combination treatment (Group F) achieved the lowest renal MDA (1.35±0.08 nmol/mg) and the highest SOD (38.6±2.1), GSH (32.4±1.8), and catalase (25.5±1.4 U/mg) among all treated groups, approaching normal control values. The high F-ratios for all parameters (MDA: 56.125; SOD: 24.414; GSH: 30.802; catalase: 32.321) confirm statistically significant overall group differences.

3.2. Effect on liver oxidative stress markers

Table 2 Effect of ethanolic leaf extract of *Mentha piperita* and empagliflozin on oxidative stress markers and antioxidant enzymes in liver tissue of aluminium chloride and D-galactose-induced rats

Group	MDA (nmol/mg)	SOD (U/mg)	GSH (μmol/mg)	Catalase (U/mg)
Group A (control)	1.20±0.08	42.5±2.1	35.4±1.9	28.6±1.5
Group B (AlCl ₃ + D-galactose, 6 weeks)	3.85±0.21*	18.3±1.4*	12.6±1.1*	10.4±0.9*
Group C (AlCl ₃ + D-galactose, 6 weeks; ELMP 200 mg/kg, 4 weeks)	2.40±0.14*#	30.2±1.8*#	24.8±1.5*#	20.1±1.2*#
Group D (AlCl ₃ + D-galactose, 6 weeks; ELMP 400 mg/kg, 4 weeks)	1.90±0.11*#	36.7±2.0#	30.5±1.8#	24.8±1.4#
Group E (AlCl ₃ + D-galactose, 6 weeks; EMPG 10 mg/kg, 4 weeks)	2.10±0.13*#	28.9±1.7*#	22.3±1.4*#	16.5±1.0*#
Group F (AlCl ₃ + D-galactose, 6 weeks; ELMP 400 mg/kg + EMPG 10 mg/kg, 4 weeks)	1.45±0.09#	40.1±2.2#	33.9±1.9#	26.9±1.5#
F-ratio	49.366	22.201	27.481	29.678

AlCl₃: aluminium chloride; ELMP: ethanolic leaf extract of *Mentha piperita*; EMPG: empagliflozin; MDA: malondialdehyde; SOD: superoxide dismutase; GSH: reduced glutathione. Data were analysed using ANOVA followed by post hoc LSD comparison and values were considered significant at p less than or equal to 0.05. *: significant compared to Group A; #: significant compared to Group B.

Table 2 shows that hepatic MDA levels were markedly elevated in Group B (3.85±0.21 nmol/mg) compared to Group A (1.20±0.08 nmol/mg), confirming significant hepatic lipid peroxidation. Hepatic SOD, GSH, and catalase activities were severely depleted in Group B (18.3±1.4, 12.6±1.1, and 10.4±0.9 U/mg respectively) relative to Group A (42.5±2.1, 35.4±1.9, and 28.6±1.5 U/mg respectively). Treatment groups C, D, E, and F all demonstrated progressive reduction in hepatic MDA and progressive restoration of antioxidant enzyme activities. Group D (ELMP 400 mg/kg) achieved the greatest antioxidant restoration among monotherapy groups. The combination treatment (Group F) produced the most complete hepatic antioxidant restoration, with MDA reduced to 1.45±0.09 nmol/mg and SOD, GSH, and catalase reaching 40.1±2.2, 33.9±1.9, and 26.9±1.5 U/mg respectively, values closely matching normal control. F-ratios confirmed statistically significant group differences across all hepatic parameters (MDA: 49.366; SOD: 22.201; GSH: 27.481; catalase: 29.678).

4. Discussion

4.1. AlCl₃ and D-galactose-induced hepatic and renal oxidative stress

The significant elevation of MDA levels in both liver (3.85±0.21 nmol/mg) and kidney (4.10±0.24 nmol/mg) tissues of Group B rats, alongside parallel depletion of SOD, GSH, and catalase activities, provides robust biochemical confirmation of AlCl₃ and D-galactose-induced oxidative toxicity in peripheral organs. These findings are consistent with the established mechanisms of aluminium-mediated peripheral organ injury, wherein Al³⁺ ions readily accumulate in hepatic and renal tissues following systemic absorption and generate ROS through Fenton-type reactions, lipid peroxidation chain propagation, and impairment of mitochondrial electron transport [1,2]. Malondialdehyde, as a terminal aldehyde product of polyunsaturated fatty acid peroxidation, reflects the cumulative burden of oxidative membrane damage in these metabolically active tissues.

D-galactose potentiates this oxidative injury through a distinct mechanism involving its enzymatic conversion to galactitol and hydrogen peroxide by aldose reductase, generating an additional endogenous source of ROS that depletes GSH stores and impairs catalase function. The compounding of AlCl₃-induced pro-oxidant activity with D-galactose-induced ROS generation therefore explains the particularly severe antioxidant depletion observed in Group B, characterised by GSH reductions of approximately 66% in both liver and kidney compared to normal control. The high F-ratios across all four oxidative stress parameters in both organs (ranging from 22.201 to 56.125) confirm the statistical robustness of these toxicity findings and validate the experimental model for studying peripheral organ oxidative protection.

4.2. Nephroprotective mechanisms of ELMP and EMPG

The dose-dependent restoration of renal antioxidant status by ELMP is mechanistically attributable to the diverse phytochemical constituents identified in its GC-FID profile. Limonene, the dominant constituent (68.62%), activates the Nrf2/HO-1 antioxidant signalling pathway, inducing transcriptional upregulation of endogenous antioxidant enzymes including SOD and catalase, thereby explaining the observed dose-dependent increases in renal enzyme activities in Groups C and D [1]. Citral (6.62%) contributes additional ROS scavenging through direct superoxide anion neutralisation and inhibition of NADPH oxidase, reducing the upstream pro-oxidant burden in renal tubular cells. Myricetin (6.60%) chelates aluminium ions directly, reducing renal aluminium accumulation and limiting Al³⁺-mediated catalytic ROS generation, while also activating glutathione synthesis pathways that support GSH restoration [13].

Empagliflozin (EMPG) demonstrated independent nephroprotective activity, improving all four renal oxidative stress parameters in Group E relative to Group B. This effect is consistent with the established nephroprotective properties of SGLT2 inhibitors, which reduce renal tubular oxidative stress through multiple mechanisms including suppression of NADPH oxidase 4 (NOX4) activity, activation of AMPK-mediated mitochondrial biogenesis, reduction of hypoxia-inducible factor-1 α (HIF-1 α) expression, and promotion of hydrogen sulphide (H₂S) signalling that enhances antioxidant enzyme expression [7,8]. The combined treatment (Group F) achieved nephroprotective outcomes superior to either monotherapy, with renal MDA virtually normalised (1.35 ± 0.08 nmol/mg vs Group A: 1.10 ± 0.07) and all three antioxidant enzymes restored to near-normal levels. This synergistic nephroprotection likely reflects the complementary targeting of distinct oxidative pathways, with ELMP phytochemicals providing direct radical scavenging, metal chelation, and Nrf2 activation, while EMPG contributes AMPK activation and NOX4 suppression.

4.3. Hepatoprotective mechanisms of ELMP and EMPG

The pattern of hepatic oxidative stress restoration closely mirrored the renal findings, reflecting the shared vulnerability of both organs to AlCl₃ and D-galactose-induced ROS generation. ELMP-treated groups demonstrated progressive, dose-dependent restoration of hepatic antioxidant status. The hepatoprotective activity of ELMP is primarily attributable to myricetin and flavone, which suppress NF- κ B-mediated transcription of pro-oxidant enzymes, inhibit cytochrome P450 2E1 (CYP2E1)-mediated hepatic ROS generation, and enhance glutathione S-transferase (GST) activity to promote hepatic detoxification of reactive aldehyde metabolites including MDA [13]. Epicatechin (4.88%) and resveratrol (4.82%), both identified in the GC-FID profile, exert hepatoprotective effects through SIRT1/Nrf2 pathway activation, directly upregulating antioxidant gene expression and promoting autophagy-mediated clearance of oxidatively damaged hepatic proteins.

Nobiletin (5.89%), a polymethoxylated flavone, contributes to hepatic protection through inhibition of hepatic stellate cell activation and suppression of inflammatory cytokine-driven oxidative amplification loops, mechanisms documented in previous hepatoprotection studies with structurally related polymethoxylated flavones. Empagliflozin demonstrated meaningful hepatoprotective activity in Group E, consistent with emerging evidence that SGLT2 inhibitors reduce hepatic steatosis and oxidative stress through AMPK-mediated inhibition of lipogenic pathways and reduction of hepatic fat accumulation, thereby limiting substrate-driven ROS generation in hepatocytes. The combination treatment (Group F) produced the most complete hepatic antioxidant restoration across all four parameters, with hepatic MDA nearly identical to normal control (1.45 ± 0.09 vs 1.20 ± 0.08 nmol/mg), confirming a synergistic hepatoprotective interaction between ELMP phytochemicals and empagliflozin.

4.4. Comparative hepatic versus renal response

A noteworthy comparative observation is that baseline (Group B) MDA elevation was proportionally greater in the kidney (4.10 ± 0.24 nmol/mg; 273% increase over control) than in the liver (3.85 ± 0.21 nmol/mg; 221% increase), suggesting that renal tubular cells may be more acutely susceptible to AlCl₃ and D-galactose-induced lipid peroxidation. This may reflect the higher aluminium concentration gradient in proximal renal tubular cells due to their role in aluminium filtration and reabsorption, as well as the thinner antioxidant buffer capacity of renal tissue compared to the metabolically more versatile hepatocyte. However, the antioxidant depletion pattern was comparable across both organs, with approximately 59% SOD depletion, 64% GSH depletion, and 64% catalase depletion in the kidney, versus 57%, 64%, and 64% respectively in the liver. The combination treatment achieved near-complete restoration in both organs, underscoring the broad-spectrum peripheral organ protective efficacy of combined ELMP and EMPG therapy.

5. Conclusion

This study demonstrates that AlCl₃ and D-galactose co-administration induces significant oxidative stress in both liver and kidney tissues, characterised by elevated MDA and severe depletion of SOD, GSH, and catalase. The ethanolic leaf extract of *Mentha piperita* and empagliflozin, particularly in combination, effectively attenuated these oxidative changes through complementary and synergistic antioxidant mechanisms. ELMP exerts hepatoprotection and nephroprotection primarily through its dominant phytoconstituents, namely limonene, citral, myricetin, flavone, nobiletin, epicatechin, and resveratrol, which collectively target Nrf2 activation, direct radical scavenging, aluminium chelation, and antioxidant enzyme upregulation. Empagliflozin independently contributes through AMPK activation, NOX4 suppression, and mitochondrial protection. The superior outcomes of combination therapy support the integration of phytochemical and pharmacological antioxidant strategies as a multi-target approach to peripheral organ protection in aluminium toxicity. These findings warrant further investigation in clinical settings, particularly in populations with occupational or environmental aluminium exposure.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The authors declare that no conflict of interest exists.

Statement of ethical approval

All animal procedures were conducted in accordance with internationally accepted principles for laboratory animal use and care and were approved by the appropriate institutional ethics committee.

Statement of informed consent

This study did not involve human participants; therefore, informed consent was not applicable.

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