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Nephroprotective Potential of Selenium and Quercetin Nanoparticles Against Acrylamide-Induced Renal Toxicity in Male Albino Wistar Rats

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Abstract
Acrylamide, a toxic compound formed during high-temperature cooking, is widely known for its neurotoxic and carcinogenic effects, while its nephrotoxic impact remains less defined. This study evaluates acrylamide-induced renal toxicity in male Albino Wistar rats and examines the therapeutic efficacy of selenium nanoparticles (SeNPs) and quercetin nanoparticles (QNPs). Acrylamide exposure significantly impaired renal function, elevating serum creatinine (1.85 ± 0.22 mg/dL vs. 0.47 ± 0.08 in control) and urea (61.00 ± 4.58 mg/dL vs. 23.33 ± 6.02) while reducing albumin levels (2.25 ± 0.58 g/dL vs. 5.33 ± 0.25). Oxidative stress was evident through decreased GSH (3.25 ± 2.7 vs. 6.35 ± 2.01 mg/g tissue) and SOD (13.23 ± 4.01 vs. 28.66 ± 5.20 U/g tissue), alongside elevated MDA (36.10 ± 0.24 vs. 15.05 ± 0.28 nmol/g). Treatment with SeNPs and QNPs significantly ameliorated these alterations. SeNPs improved creatinine, urea, and albumin to 1.40 ± 0.04, 47.66 ± 3.51, and 3.26 ± 0.37, respectively, while QNPs showed greater normalization (1.30 ± 0.08; 35.20 ± 0.02; 3.40 ± 0.10). Oxidative markers also improved markedly, particularly in QNP-treated rats. Histopathological observations confirmed near-normal renal architecture following QNP treatment. These findings demonstrate the strong nephroprotective potential of SeNPs and especially QNPs, supporting their future application in mitigating acrylamide-induced renal damage and enhancing human kidney health.

Keywords: Antioxidative Enzymes; Toxicity; In vivo; Histopathology, Oxidative stress

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Introduction
Acrylamide is a chemical compound that forms in carbohydrate-rich foods during high-temperature cooking processes exceeding 120°C, such as frying, roasting, or baking. It is primarily produced through the Maillard reaction between the amino acid asparagine and reducing sugars **[1].** While acrylamide is widely recognized for its neurotoxic and carcinogenic properties, its nephrotoxic effects have garnered increasing attention in recent years. Acrylamide induces nephrotoxicity through several mechanisms, including oxidative stress, mitochondrial dysfunction, and inflammation. After ingestion, acrylamide is metabolized into glycidamide by cytochrome P450 2E1 (CYP2E1), which generates reactive oxygen species (ROS) **[2].** These ROS disrupt cellular redox homeostasis in renal tissues, leading to lipid peroxidation, protein carbonylation, and DNA damage **[3].** This oxidative stress damages kidney cells, compromising their structure and function. In animal models, acrylamide exposure has been shown to significantly reduce antioxidant defenses, including glutathione (GSH) levels and superoxide dismutase (SOD) activity **[4]**. At the same time, acrylamide increases markers of renal oxidative stress, such as malondialdehyde (MDA) **[5]**. These changes contribute to renal cell apoptosis and functional impairment. Inflammation also plays a central role in acrylamide-induced nephrotoxicity. Studies indicate that acrylamide exposure triggers the activation of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which amplify renal damage **[6].** Chronic inflammation in the kidney can result in tubular atrophy, glomerular damage, and interstitial fibrosis **[7]**. Histopathological analyses in experimental studies have revealed glomerular atrophy, tubular degeneration, and interstitial fibrosis in kidneys exposed to acrylamide **[8].** Functional impairments are further evidenced by elevated levels of blood urea nitrogen (BUN) and serum creatinine, which are key markers of renal dysfunction **[9].** Long-term acrylamide exposure has also been associated with decreased glomerular filtration rate (GFR), an early indicator of chronic kidney disease **[10].** To counteract acrylamide-induced nephrotoxicity, antioxidant therapies have shown promise. Quercetin, a natural flavonoid with potent antioxidative and anti-inflammatory properties, has been shown to mitigate oxidative stress and preserve renal function **[11]**. Selenium nanoparticles (SeNPs), due to their high biocompatibility and selective targeting, have also been effective in reducing

oxidative and inflammatory damage to kidney tissues **[12].** The objective of this study is to explore the use of nanoparticle-based natural molecules, such as quercetin and selenium nanoparticles, to counteract the nephrotoxic effects of acrylamide. This investigation aims to elucidate the mechanisms by which these nanoparticles protect renal tissue and restore kidney function following acrylamide exposure. By understanding these mechanisms, we can develop effective strategies to mitigate acrylamide's harmful effects and contribute to the production of safer, health-conscious food products.

Materials and methods

MATERIAL

CHEMICALS:

Acrylamide dry crystals were obtained from sigma chemicals Co, USA. Quercetin and selenium nanoparticles were synthesized and characterized in laboratory using green synthesis method **[13,14]**. All chemicals were of commercially grade and kept at 4° C.

EXPERIMENTAL ANIMALS:

Twenty adult male Albino Wistar rats, weighing between 160 and 220 grams, were housed in appropriate cages under controlled environmental conditions. They were fed a standard pellet diet and provided free access to water.

METHODS

The experimental design included four groups of animals (5 per group) treated for 14 days via oral gavage. Group 1 (control) received 1 ml of physiological saline daily. Group 2 (Acrylamide) was administered acrylamide at a dose of 3 mg/kg per day. Group 3 (Acrylamide + Q) received acrylamide (3 mg/kg per day) along with quercetin nanoparticles (QNP). Group 4 (Acrylamide + S) was treated with acrylamide (3 mg/kg per day) combined with selenium nanoparticles (SeNP). This design aimed to evaluate the protective effects of QNP and SeNP against acrylamide-induced toxicity.

Kidney samples: Ether was used anesthetized after that the animals were sacrificed. The examination and dissection of kidney were done under light microscope to study the histopathological changes; the tissues were fixed in 10% formalin.

BIOCHEMICAL ANALYSIS:

ANTIOXIDANT ENZYMATIC BIOMARKERS:

Kidney tissue homogenate was used to analyze the antioxidants such as MDA, GSH and SOD. (nmoL/g tissue) was determined in by a colorimetric assay **[15]**. GSH (mg/g tissue) was assayed according to the method of Moron developed in 1979 **[16]** Superoxide dismutase (SOD) was evaluated according to Marklund & Marklund developed in 1974 **[17]**.

KIDNEY FUNCTION ANALYSIS:

Kidney function was analyzed by determining the urea, Creatinine and Albumin, the urea level was determined according to method of Diacetylmonoxine of Patton; 1977 **[18]**, creatinine was analyzed according to method developed in 1974 **[19]**, while albumin was determined by using the method developed in 1971 **[20]**. The total protein was determined by Biuret method **[21]**.

HISTOPATHOLOGICAL INVESTIGATIONS

Kideny tissue slices embedded in paraffin (5 µm) were cut using a sliding microtome (Leica RM2135 Rotary Microtome, Wichita, KS, USA) and stained with hematoxylin and eosin (H&E) stain for a later light microscope histological analysis sing the light microscope 46.

Statistical analysis*:* Statistical analyses (mean ± SE) were carried out by using one-way ANOVA. The significance level was at a $P<0.05$.

Ethics approval of research

The study was conducted in accordance with ethical guidelines and principles for the care and use of laboratory animals. All experimental procedures involving the use of male Albino Wistar rats were approved by Egyptian Network of Research Ethics Committees. The study adhered to the guidelines set by ENREC. Efforts were made to minimize animal suffering and ensure humane handling throughout the course of the experiment.

Results

Biochemical analysis:

Oxidative stress biomarkers:

The Acrylamide treatment causes significant change in the GSH and SOD mean ± SE (3.25 ±2.7 and 13.23 ±4.01respectively) as compared with the normal control group (6.35 ±2.01and 28.66 ±5.20 respectively), which indicates that the acrylamide is causing

the oxidative stress on kidney. The Acrylamide combined with selenium nanoparticles and/or quercetin nanoparticles induced marked decrease in oxidative damage as compared with the Acrylamide treated group, MDA, which is the clear marker of nephrotoxicity showed the significant elevation in the acrylamide treated group (36.10 ±0.24) in comparison to control group (15.05 ±0.28), while the SeNP and QNP group showed the promising results by leveling the enzymes approximately to the normal level **(Table I).**

Renal markers:

Acrylamide treatment to male Albino Wistar Rats significantly (P>0.05) increases the serum creatinine and urea levels mean ± SE (1.85 ±0.22 and 61.00 ±4.58 respectively) in comparison to control group (0.47 ±0.08 and 23.33±6.02 respectively), However, the total albumin concentrations of Acrylamide induced group showed a significant decrease (P<0.05) (2.25 ±0.58) compared to control group (5.33 ±0.25). While the animal groups treated with Acrylamide followed by either selenium or quercetin nanoparticles restore significantly (P<0.05) these parameters to its normal values (1.40 ±0.04, 47.66 ±3.51, and 3.26 ±0.37) and (1.30 ±0.08, 35.20 ±0.02, and 3.40 ±0.10) respectively **(Table II).**

Kidney Histopathological observations:

The control group had normal tubules and glomeruli. Renal sections from positive control group showing severely congested blood vessels (red arrows), diffuse marked tubular dilation with cast formation (black arrow). Selenium treated group showed multifocal areas of moderate tubular dilation with cast formation (black arrow) and congested blood vessels (red arrows) with gradual decrease of severity. Quercetin treated group has showing normal tubules and glomeruli **(Figure 1).** Renal sections from treated groups 2 (SeNPs treated), 3 (QNPs treated), showing multifocal areas of moderate tubular dilation with cast formation (black arrow) and congested blood vessels (red arrows) with gradual decrease of severity.

Table 1. Effect of SeNPs and QNPs on renal markers and oxidative enzymes in acrylamide induced nephrotoxicity

	Parameters		
Groups	**GSH**	**SOD**	**MDA**
Control	6.35 ±2.01	28.66 ±5.20	15.05 ±0.28
Acrylamide	3.25 ±2.7	13.23 ±4.01	36.10 ±0.24
SeNPs	6.96 ±0.05	25.03 ±3.10	18.05 ±0.15
QNPs	7.13 ±1.01	26.33 ±4.50	17.32 ±0.04

Table 2. Effect of SeNPs and QNPs on renal markers in acrylamide induced nephrotoxicity

Groups	Parameters		
	Creatinine	Urea	Albumin
Control	0.47 ±0.08	23.33 ±6.02	5.33 ±0.25
Acrylamide	1.85 ±0.22	61.00 ±4.58	2.25 ±0.58
Selenium nanoparticles	1.40 ±0.04	47.66 ±3.51	3.26 ±0.37
Quercetin nanoparticles	1.30 ±0.08	35.20 ±0.02	3.40 ±0.10

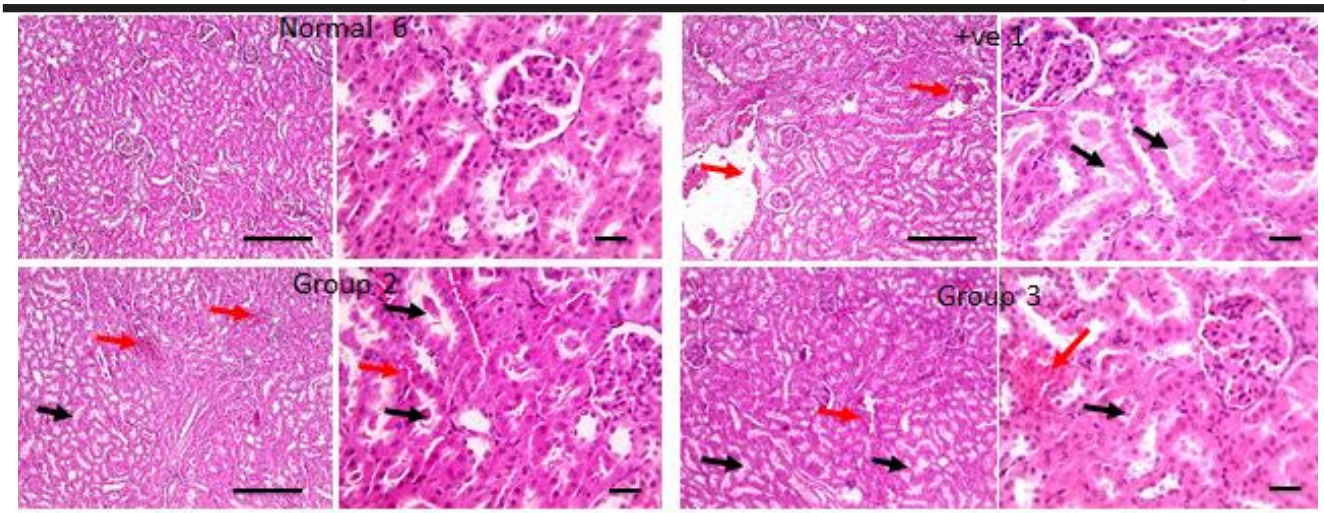

Fig. 1. Histopathological examination of rat kidney tissue (Normal (C) to group 3) normal (Normal (C) histological appearance including normal tubules and glomeruli, Renal sections from + control group (Acrylamide treated) showing severely congested blood vessels (red arrows), diffuse marked tubular dilation with cast formation (black arrow). Group 2 (SeNPs treated) indicates partial damage, but maybe less severe than positive control. but improved compared to the +ve control. Group 3 Seems to show moderate recovery

Discussion

The findings of this study demonstrate the nephrotoxic effects of acrylamide exposure, primarily mediated through oxidative stress, inflammation, and structural damage to renal tissues. Elevated levels of malondialdehyde (MDA), reduced glutathione (GSH), and decreased superoxide dismutase (SOD) activity in the acrylamide-treated group indicate a significant oxidative stress burden. This aligns with prior research highlighting acrylamide's role in disrupting redox homeostasis and promoting lipid peroxidation in renal tissues **[7, 22, 23]**. Furthermore, the elevated serum creatinine and urea levels in acrylamide-exposed rats are consistent with impaired renal function and structural damage, as evidenced by histopathological changes such as tubular dilation, cast formation, and glomerular damage **[24, 25].** The administration of selenium nanoparticles (SeNPs) and quercetin nanoparticles (QNPs) offered significant nephroprotective effects. Both SeNPs and QNPs restored antioxidant enzyme levels closer to normal, as indicated by improved GSH and SOD activity and reduced MDA levels. These findings suggest that the antioxidative and anti-inflammatory properties of these nanoparticles counteracted acrylamide-induced renal oxidative stress and inflammation **[26, 27].** Notably, quercetin-treated groups exhibited near-complete normalization of renal histology, indicating its potent therapeutic potential. The effectiveness of SeNPs in mitigating oxidative damage further underscores their role in selective targeting of reactive oxygen species while maintaining biocompatibility **[28, 29].** Histopathological analysis corroborated the biochemical findings, with treated groups showing progressively less severe tubular damage and vascular congestion compared to the acrylamide-only group. The observed improvements in renal markers, including creatinine and albumin levels, further validate the protective effects of SeNPs and QNPs against acrylamide-induced nephrotoxicity. These results align with previous studies that have demonstrated the antioxidative and cytoprotective roles of selenium and quercetin in mitigating chemical-induced organ damage **[30].**

Conclusions

This study highlights the nephrotoxic potential of acrylamide and its detrimental impact on renal oxidative status, inflammatory processes, and structural integrity. The protective effects of selenium and quercetin nanoparticles against acrylamide-induced renal damage emphasize their therapeutic potential. Both SeNPs and QNPs effectively mitigated oxidative stress, restored antioxidant defences, and preserved renal function. These findings pave the way for future investigations into nanoparticle-based therapies as a means of counteracting acrylamide toxicity and improving public health outcomes. Further research on the dose-dependent effects and long-term safety of these interventions is warranted to explore their potential applications in mitigating nephrotoxicity in humans.

USE OF ARTIFICIAL INTELLIGENCE (AI)-ASSISTED TECHNOLOGY

The authors declare that they have not used artificial intelligence (AI)- tools for writing and editing of the manuscript, and no images were manipulated using AI.

ETHICAL STATEMENT:

The study was conducted in accordance with ethical guidelines and principles for the care and use of laboratory animals. All experimental procedures involving the use of male Albino Wistar rats were approved by Egyptian Network of Research Ethics Committees. The study adhered to the guidelines set by ENREC. Efforts were made to minimize animal suffering and ensure humane

handling throughout the course of the experiment.

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Conflict of interest
None

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