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In-vitro antioxidant, anti-lipid peroxidation, and anti-inflammatory activities of isorhamnetin: Relevance to myocardial infarction

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ABSTRACT

Myocardial infarction is a major cardiovascular disorder characterized by excessive oxidative stress and inflammatory responses that contribute to progressive myocardial injury. Natural flavonoids have attracted considerable attention as potential cardioprotective agents because of their antioxidant and anti-inflammatory properties. The current work used complementary biochemical assays to assess isorhamnetin's *in vitro* antioxidant and anti-inflammatory capabilities. Ferric reducing antioxidant power, nitric oxide scavenging, superoxide radical scavenging, phosphomolybdenum total antioxidant capacity, and the inhibition of linoleic acid oxidation assay were used to measure antioxidant activity. Assays for protein denaturation and bovine serum albumin (BSA) denaturation inhibition were used to assess anti-inflammatory efficacy. In every test, isorhamnetin demonstrated strong concentration-dependent antioxidant activity (6-30 µM). Although its activity was still lower than that of ascorbic acid, the molecule showed high ferric reducing ability, effective prevention of lipid peroxidation, improved overall antioxidant capacity, and efficient scavenging of nitric oxide and superoxide radicals. Furthermore, isorhamnetin significantly inhibited heat-induced protein and BSA denaturation in a concentration-dependent manner, indicating notable protein-stabilizing and anti-inflammatory properties when compared with the standard drug. Collectively, these findings demonstrate that isorhamnetin possesses considerable antioxidant and anti-inflammatory activities, suggesting its potential to protect against oxidative stress- and inflammation-mediated myocardial injury. Further *in vivo* and mechanistic investigations are warranted to validate its cardioprotective efficacy and explore its therapeutic application in the management of cardiovascular diseases.

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INTRODUCTION

Myocardial infarction (MI) is one of the most severe clinical presentations of cardiovascular diseases (CVDs), which continue to be the leading cause of death globally. MI occurs due to prolonged interruption of coronary blood flow, resulting in irreversible myocardial injury and impaired cardiac function (Roth *et al.*, 2020). According to Frangogiannis (2014) and Bhattacharjee *et al.* (2022), oxidative stress and inflammation are important pathological events during MI. Excessive production of reactive oxygen species (ROS) causes lipid peroxidation, protein oxidation, and the activation of inflammatory mediators, all of which accelerate myocardial damage. Therefore, compounds capable of reducing oxidative stress and inflammatory responses have attracted considerable attention as potential cardioprotective agents.

Flavonoids are polyphenolic substances that occur naturally and are well known for their anti-inflammatory and antioxidant properties. Among these, isorhamnetin, a methylated flavonol found in a number of therapeutic plants and food sources, has shown a variety of pharmacological characteristics, such as the ability to scavenge free radicals, prevent oxidative damage, and alter inflammatory signaling pathways (Panche *et al.*, 2016; Gong *et al.*, 2020).

According to earlier research, isorhamnetin's anti-inflammatory and antioxidant properties may help prevent cardiovascular diseases and shield against oxidative stress-induced cellular damage (Li *et al.*, 2016). Thus, utilizing complementary biochemical tests, the current work was conducted to examine isorhamnetin's in vitro antioxidant and anti-inflammatory properties.

Protein denaturation and bovine serum albumin (BSA) denaturation inhibition assays were used to measure anti-inflammatory activity, while ferric reducing antioxidant power (FRAP), nitric oxide scavenging, superoxide radical scavenging, phosphomolybdenum assay, and inhibition of linoleic acid oxidation were used to measure antioxidant activity. These investigations provide preliminary evidence supporting the potential cardioprotective relevance of isorhamnetin through its ability to attenuate oxidative stress and inflammation.

MATERIALS AND METHODS

Antioxidant activity assays

Ferric-reducing antioxidant power assay

Ferric reducing antioxidant activity of isorhamnetin was assessed as per method described by Vijayalakshmi and Ruckmani (2016) with a few modifications. The different concentrations of isorhamnetin (6, 12, 18, 24, and 30µM) and the positive control (ascorbic acid) were formulated individually. Then 250 µL of 0.2 M phosphate buffer (pH 6.6) and 250 µL of 1% potassium ferricyanide were added to each 100 µL of sample or standard. The reactions were allowed to proceed for 30 min at 50°C and then stopped by adding 250 µL of 10% trichloroacetic acid.

Later, 250 µL of the reaction mixture was added to an equal volume of distilled water and then the freshly prepared 0.1% solution of ferric chloride was added. The absorbance was measured at 700nm after incubation at room temperature for 10min by a UV–Visible spectrophotometer. The higher absorbance values reflected the higher Ferric reducing antioxidant activity. Each experiment was repeated three times and results are presented as mean ± S.D.

Phosphomolybdenum assay

The Total Antioxidant Capacity (TAC) was measured by the phosphomolybdenum method developed by Khan *et al.*, (2012) with some modification. In brief, the phosphomolybdate reagent consisted of freshly made solution of 0.6 M of sulfuric acid, 28 mM of sodium phosphate, and 4 mM of ammonium molybdate. The isorhamnetin in 5 different concentrations (6, 12, 18, 24, and 30 µM) and the positive control (ascorbic acid) were each diluted with 150 µL of the phosphomolybdate reagent. All the reaction mixtures were heated at 95°C for 90 min and cooled at room temperature. The absorbance was measured at 695 nm using a UV–Visible spectrophotometer against an appropriate blank. The higher absorbance was related to a higher total antioxidant activity. All experiments were performed

in triplicate, and the results were expressed as mean ± standard deviation (Khan *et al.*, 2012).

Nitric oxide radical scavenging assay

Nitric oxide radical scavenging activity of isorhamnetin was determined using the Sreejayan and Rao, 1997 method with slight modifications. Briefly, 5 mM sodium nitroprusside prepared in phosphate-buffered saline (PBS, pH 7.4) was mixed with different concentrations of isorhamnetin. The reaction mixture was incubated at 25°C for 150 min under normal laboratory light conditions to facilitate nitric oxide generation. Following incubation, 0.5 mL of the reaction mixture was combined with an equal volume of freshly prepared Griess reagent containing 1% sulphanilamide, 2% phosphoric acid, and 0.1% N-(1-naphthyl)ethylenediamine dihydrochloride.

The mixture was allowed to stand for 10 min at room temperature to permit chromophore formation, and the absorbance was measured at 546 nm using a UV–Visible spectrophotometer. A control containing all reagents except isorhamnetin was prepared under identical conditions.

Superoxide free radicals scavenging assay

This method was done Liu *et al.*, 1997 with slight modification. Isorhamnetin at different concentrations was added to 3.0 ml of Tris-HCl buffer (16 mM, pH 8.0), which contained 78 pM P-nicotinamide adenine dinucleotide (reduced form, NADH), 50 pM nitroblue tetrazolium (NBT), and 10 pM phenazin methosulfate (PMS), to generate superoxide radicals. The color reaction of NBT with superoxide radicals was observed at OD 560 nm. Ascorbic acid were used as the control.

Antilipid peroxidation assay

Inhibition of linoleic acid oxidation

The inhibitory activity against the peroxidation of linoleic acid was measured by the thiobarbituric acid reactive substances (TBARS) method of Osawa and Namiki (1985), modified slightly. Briefly, five different concentrations of isorhamnetin (6, 12, 18, 24 and 30 µM), and the positive control (ascorbic acid) were separately dissolved in a linoleic acid emulsion made in phosphate buffer (0.2 M, pH 7.0). The incubation of the reaction mixtures at 37°C, in the dark, allowed the promotion of lipid peroxidation. The amount of each reaction mixture after incubation was transferred to new test tubes, thiobarbituric acid (TBA) reagent was then added to each test tube along with trichloroacetic acid (TCA) solution. The mixture was cooked at boiling water bath (95–100°C) for 15 min to enable the pink-colored thiobarbituric acid reactive substances (TBARS) to form. The tubes were then quickly cooled to room temperature and centrifuged at 3000 rpm for 10 minutes to collect any protein precipitates or insoluble residues. The absorbance of the Supernatant was measured at 530 nm in UV–Visible spectrophotometer with suitable blank.

Anti-inflammatory assays

Protein denaturation inhibition assay

Isorhamnetin's protein denaturation inhibitory activity was determined using the method described by Nawaz *et al.* (2023) with minor changes. In brief, fresh egg albumin was mixed with phosphate buffer (pH 6.4) and Tween 80 (1% of the total volume) added. Three mL of freshly prepared egg albumin solution were mixed with 2 mL of various concentrations of isorhamnetin (6–30 µM). The reference standard (positive control) used was diclofenac sodium.

The mixtures were then incubated at 37 ± 2 °C for 15 min and then heated at 70 °C for 5 min to denature the protein. Reaction mixtures were cooled down to room temperature and absorbance was determined at 660 nm with distilled water as blank.

Bovine serum albumin denaturation inhibition assay

Anti-inflammatory activity of isorhamnetin was studied by its capacity to inhibit the heat-induced denaturation of bovine serum albumin (BSA) as described by Williams *et al.* (2008) with some modifications. Briefly, a 0.4% (w/v) BSA solution was prepared in Tris-buffered saline (TBS). The pH of the solution was brought to 6.4 by the addition of glacial acetic acid. A stock solution of isorhamnetin was made using appropriate solvent and various concentrations of isorhamnetin (6, 12, 18, 24 and 30 µM) were prepared. The respective aliquots of isorhamnetin

solutions were added to test tubes containing 1 mL of 0.4% (w/v) BSA solution. The anti-inflammatory drug (positive control) used was diclofenac sodium while the negative control was the corresponding solvent without isorhamnetin. Reaction mixtures were incubated in a water bath at 72°C for 10 min and then allowed to cool at room temperature for 20 min. The extent of protein denaturation was judged by the turbidity of the reaction mixture at 660 nm as measured in a Spectrophotometer. The experiment was repeated three times and the results are presented as mean ± standard deviation (SD).

Statistical analysis

Data are expressed as mean ± SD of three independent experiments. Statistical analysis was performed using one-way ANOVA followed by Duncan's Multiple Range Test (DMRT). Differences were considered statistically significant at $p<0.05$.

RESULTS

Ferric-reducing antioxidant power assay

Isorhamnetin exhibited significant ferric reducing antioxidant activity in a concentration-dependent manner (Fig. 1). The reducing activity increased progressively from 24.3 ± 1.4% at 6 µM to 86.2 ± 2.3% at 30 µM. Although the activity remained lower than that of the standard antioxidant ascorbic acid (38.5 ± 1.3% to 95.4 ± 1.8%), isorhamnetin demonstrated considerable electron-donating capacity throughout the tested concentration range.

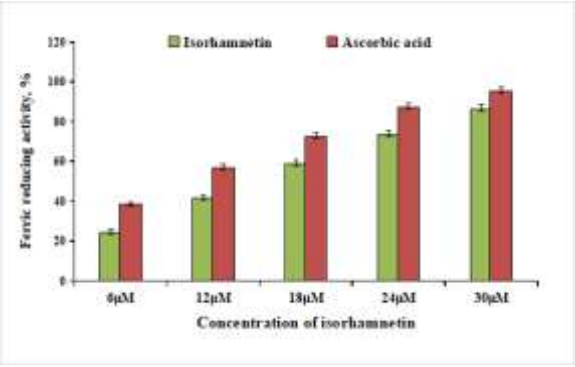

Fig. 1. FRAP of isorhamnetin at different concentrations (6–30 µM) compared with ascorbic acid

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

Nitric oxide radical scavenging assay

The nitric oxide scavenging activity of isorhamnetin increased significantly with increasing concentration (Fig. 2). The percentage inhibition increased from 19.8 ± 1.3% at 6 µM to 82.4 ± 2.2% at 30 µM. Ascorbic acid showed higher scavenging activity at all concentrations, with inhibition ranging from 31.5 ± 1.4% to 92.1 ± 1.8%.

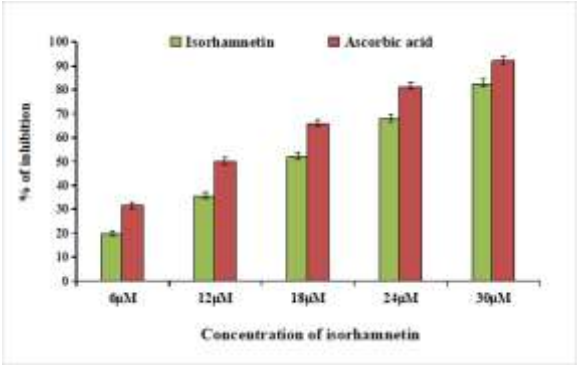

Fig. 2. Nitric oxide radical scavenging activity of isorhamnetin at different concentrations (6–30 µM) compared with ascorbic acid

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

Superoxide radical scavenging assay

Isorhamnetin effectively scavenged superoxide radicals in a concentration-dependent manner (Fig. 3). The scavenging activity increased from 18.6 ± 1.2% at 6 µM to 81.5 ± 2.2% at 30 µM.

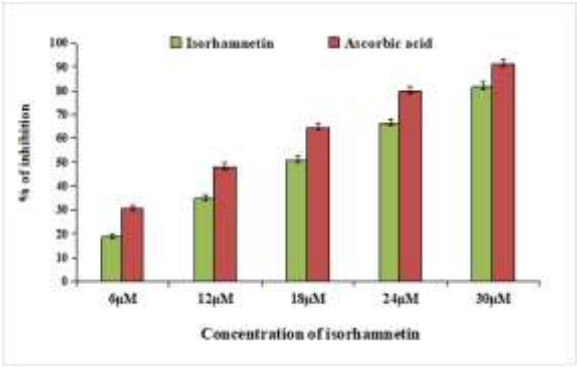

Fig. 3. Superoxide radical scavenging activity of isorhamnetin at different concentrations (6–30 µM) compared with ascorbic acid

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

The activity was slightly lower than that of vitamin C, which exhibited inhibition values between 30.4 ± 1.3% and 91.2 ± 2.0%.

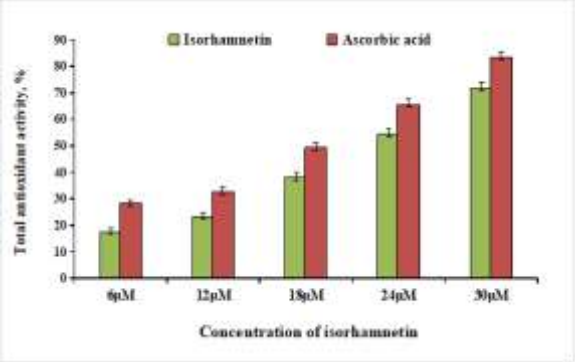

Fig. 4. Total antioxidant activity of isorhamnetin determined by the phosphomolybdenum assay at different concentrations (6–30 µM) compared with ascorbic acid

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

Phosphomolybdenum assay

The total antioxidant capacity determined by the phosphomolybdenum assay increased significantly with increasing concentrations of isorhamnetin (Fig. 4). The antioxidant activity increased from 17.5 ± 1.4% to 71.8 ± 2.3%, whereas ascorbic acid exhibited higher antioxidant activity ranging from 28.3 ± 1.3% to 83.6 ± 1.8%.

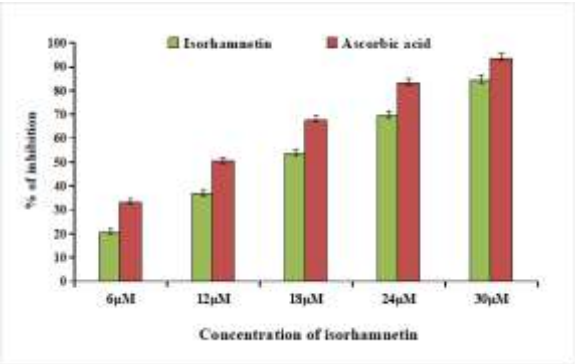

Fig. 5. Inhibitory effect of isorhamnetin on linoleic acid oxidation determined by the TBARS assay at different concentrations (6–30 µM) compared with ascorbic acid

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

Inhibition of linoleic acid oxidation

Isorhamnetin exhibited significant inhibition of linoleic acid oxidation in a concentration-dependent manner (Fig. 5). The percentage inhibition increased progressively from 20.6 ± 1.3% at 6 µM to 84.3 ± 2.3% at 30 µM, indicating its ability to suppress lipid peroxidation. The highest inhibitory activity was observed at 30 µM, demonstrating the protective effect of isorhamnetin against oxidative degradation of linoleic acid. The positive control, ascorbic acid, exhibited higher inhibitory activity at all tested concentrations, with inhibition values ranging from 33.2 ± 1.2% at 6 µM to 93.6 ± 2.1% at 30 µM. Although the activity of isorhamnetin was lower than that of the standard antioxidant, it demonstrated considerable protection against lipid peroxidation throughout the tested concentration range.

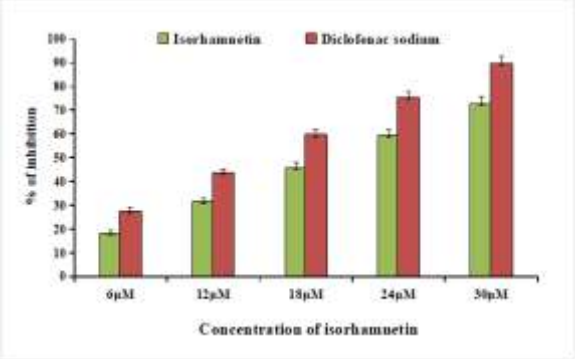

Fig. 6. Inhibitory effect of isorhamnetin on heat-induced protein denaturation at different concentrations (6–30 µM) compared with diclofenac sodium

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

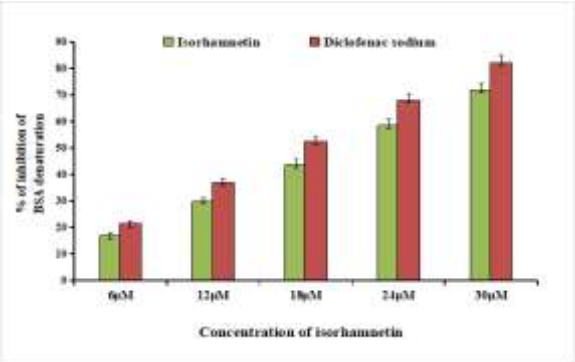

Fig. 7. Inhibitory effect of isorhamnetin on BSA denaturation at different concentrations (6–30 µM) compared with diclofenac sodium

Values are expressed as mean ± SD of experiments performed in triplicate (n = 3).

Bovine serum albumin denaturation inhibition assay

Isorhamnetin inhibited heat-induced bovine serum albumin (BSA) denaturation in a concentration-

dependent manner (Fig. 7). The percentage inhibition increased from 16.84 ± 1.12% at 6 µM to 71.65 ± 2.74% at 30 µM, indicating effective stabilization of albumin against thermal denaturation. The standard (diclofenac sodium) demonstrated comparatively higher inhibition at all tested concentrations, ranging from 21.35 ± 1.26% at 6 µM to 82.15 ± 2.89% at 30 µM. Nevertheless, isorhamnetin exhibited substantial inhibition of BSA denaturation, suggesting promising anti-inflammatory activity.

DISCUSSION

Excessive ROS and RNS production is a primary cause of pathogenesis of myocardial infarction, leading to lipid peroxidation, protein oxidation, mitochondrial dysfunction, and cardiomyocyte death (Halliwell and Gutteridge, 2015; Frangogiannis, 2014). Thus, drugs with both antioxidant and anti-inflammatory properties could protect against myocardial damage. In the present study, isorhamnetin exhibited significant concentration-dependent antioxidant and anti-inflammatory activities in all in vitro assays performed, suggesting its potential as a natural cardioprotective agent. The FRAP assay demonstrated that isorhamnetin possesses strong ferric reducing ability through electron donation, indicating its capacity to neutralize free radicals and interrupt oxidative chain reactions (Apak *et al.*, 2016). This reducing activity is mainly due to the presence of phenolic hydroxyl groups in the structure of flavonoids of isorhamnetin, which help to protect the intermediates of the reaction, and also to increase the antioxidant capacity of living cells (Heim *et al.*, 2002; Li *et al.*, 2016). In keeping with these results, isorhamnetin also showed remarkable activities against nitric oxide and superoxide radicals. During myocardial infarction, both nitric oxide and superoxide are produced in excess and can generate highly reactive oxidants like peroxynitrite which causes endothelial dysfunction, lipid peroxidation and damage to affected cells (Forstermann and Sessa, 2012; Pacher *et al.*, 2007). The radical scavenging properties of isorhamnetin indicate its protective effects against cardiovascular diseases through its ability to combat oxidative and nitrosative stress. The findings are consistent with other reports that flavonoids exhibit antioxidant and free radical scavenging effect (Gong *et al.* 2020; Panche *et al.* 2016). By showing its concentration-dependent reducing potential through the reduction of molybdenum(VI) to molybdenum(V), the phosphomolybdenum assay further validated isorhamnetin's overall antioxidant ability (Prieto *et al.*, 1999).

Likewise, the inhibition of linoleic acid oxidation indicated that isorhamnetin effectively suppressed lipid peroxidation, an important mechanism underlying oxidative damage to cellular membranes during myocardial infarction (Osawa and Namiki, 1985; Halliwell and Gutteridge, 2015). Isorhamnetin's capacity to contribute hydrogen atoms or electrons, which stops lipid peroxidation chain reactions and maintains membrane integrity, may be responsible for the suppression of lipid oxidation (Heim *et al.*, 2002). These antioxidant properties collectively suggest that isorhamnetin may protect cardiac tissues by limiting oxidative damage and maintaining cellular redox homeostasis.

Inflammation is another critical event in myocardial injury that contributes to cardiomyocyte loss and adverse cardiac remodeling (Frangogiannis, 2014). Heat-induced protein denaturation and BSA denaturation assays are widely accepted in vitro methods for evaluating anti-inflammatory activity because protein denaturation is closely associated with inflammatory responses (Williams *et al.*, 2008; Nawaz *et al.*, 2023). In the present study, isorhamnetin significantly inhibited both protein and BSA denaturation in a concentration-dependent manner, indicating effective protein-stabilizing activity. These findings are consistent with previous reports demonstrating that flavonoids exert anti-inflammatory effects by reducing oxidative stress and modulating inflammatory signaling pathways (Panche *et al.*, 2016; Gong *et al.*, 2020). By preventing protein denaturation and reducing oxidative injury, isorhamnetin may contribute to protecting cardiac tissues from inflammation-mediated damage.

Overall, the results of this study show that isorhamnetin has significant anti-inflammatory and antioxidant properties through a number of complementary mechanisms, such as stabilizing proteins, enhancing reducing power, inhibiting lipid peroxidation, and scavenging free radicals.

Although its activity was lower than that of the standard compounds, the concentration-dependent responses observed across all assays provide strong evidence supporting the therapeutic potential of isorhamnetin. These results suggest that isorhamnetin may serve as a promising natural cardioprotective agent for mitigating oxidative stress- and inflammation-associated myocardial injury. Further in vivo and mechanistic studies are warranted to confirm these protective effects and elucidate the underlying molecular mechanisms.

CONCLUSION

The present study demonstrated that isorhamnetin possesses significant *in-vitro* antioxidant, anti-lipid peroxidation, and anti-inflammatory activities in a concentration-dependent manner. The compound exhibited effective ferric reducing ability, free radical scavenging activity, inhibition of lipid peroxidation, and protein-stabilizing properties, indicating its ability to attenuate oxidative stress and inflammatory responses. Although its activity was lower than that of the respective standard compounds, isorhamnetin showed considerable biological potential across all assays evaluated. These findings suggest that isorhamnetin is a promising natural bioactive compound with potential relevance for the prevention of oxidative stress- and inflammation-associated myocardial injury.

RECOMMENDATION(S)

Further *in-vivo* studies are recommended to validate the cardioprotective efficacy of isorhamnetin in experimental models of myocardial infarction. In addition, molecular investigations are needed to elucidate the underlying mechanisms and signaling pathways responsible for its antioxidant and anti-inflammatory effects. Future preclinical and clinical studies are also warranted to establish its therapeutic potential and safety for cardiovascular disease management.

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