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Original Article

Neuroprotective Effects of Resveratrol in STZ-induced Diabetic Neuropathy in Rats by Behavioral and Biochemical Effects

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ABSTRACT

Objectives: Diabetic neuropathy is one of the major complications, which is characterized by chronic neuropathic pain, oxidative stress, and neuroinflammation related to diabetes. The current research is designed to assess the anti-neuropathic ability of resveratrol in Streptozotocin (STZ)-induced diabetic rats. **Materials and Methods:** Male Wistar rats (220-280 g) were made diabetic by a single intraperitoneal injection of STZ (50 mg/kg). Rats (confirmed with hyperglycaemia) were fed resveratrol (10, 20, and 40 mg/kg p.o) for a period of four weeks. Flexion and heat plate tests were used to measure neuropathic pain in tail immersion. Fasting blood glucose was measured using a glucometer after overnight fasting. Biochemical parameters such as TNF-α in serum and brain nitrite levels are found to measure the responses of inflammatory and oxidative stress. **Results:** The administration of STZ had caused a great hyperglycaemia, body weight decreases, lowered nociceptive threshold, and also an increase in serum TNF-α. The treatment of resveratrol had a great effect in halting these modifications in a dose-dependent fashion. The maximum dose (40 mg/kg) had a significant positive effect on pain threshold, decreased the signs of inflammatory and oxidative stress, and partially normalised glucose levels by normal standards. **Conclusion:** Resveratrol proves to have a superior protective effect against diabetic neuropathy by acting as a regulator of oxidative stress and inhibition of pro-inflammatory cytokines. This indicates that resveratrol has potential as a therapeutic candidate to be used in controlling diabetic neuropathic pain.

Keywords Diabetic Neuropathy, Neuropathic Pain, Nitric Oxide, Oxidative Stress, Resveratrol, TNF-α.

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INTRODUCTION

Neuropathic pain is a chronic condition that significantly impairs the lives of millions of people worldwide. Unlike nociceptive pain, which results from direct tissue damage, neuropathic pain stems from abnormalities in the nervous system and presents as spontaneous pain, hyperalgesia, and allodynia (Finnerup *et al*., 2021). Conditions like diabetes, chemotherapy-induced neuropathy, spinal cord injury, multiple sclerosis, and postherpetic neuralgia are associated with PN (Kumar *et al*., 2023). The complex mechanisms underlying neuropathic pain make it challenging to treat, and existing pharmacological treatments are often insufficient and can lead to severe side effects (Petroianu *et al*., 2023). Neuropathic pain is widely recognized as a prevalent complication associated with diabetes, affecting both types of diabetes equally. It is primarily characterized by pain that may arise spontaneously or in response to mild stimuli, a phenomenon known as hyperalgesia (Pop-Busui *et al*., 2022). Although changes in pain perception have been linked to neuronal loss or alterations in neurotransmitter levels, the precise causes remain unclear (Quiroz-Aldave *et al*., 2023). Various new medications, including opioids, antidepressants, anticonvulsants, and nonsteroidal anti-inflammatory medications, are being explored for the management of diabetic neuropathic pain (Sloan *et al*., 2022). However, the use of these medications is prohibited because of their limited effectiveness and potential toxicity (Singh *et al*., 2020). A significant variable is oxidative stress, a pathogenic factor in the onset of neuropathic pain, resulting from a conflict between the body's defenses against antioxidants and its creation of Reactive Oxygen Species (ROS) (Stojanovic *et al*., 2025). An overabundance of ROS can lead to lipid peroxidation, mitochondrial damage, and neuronal death, all of which intensify pain perception (Trofin *et al*., 2025). Additionally, oxidative stress is crucial in neuroinflammation, which is triggered by immune activation and inflammatory mediators (Anwar *et al*., 2025).

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This polyphenolic compound is naturally found in berries, red wine, and grapes, and there is now growing interest in the compound due to its possible neuroprotective activity (Zaa *et al*., 2023). It has been known to have antioxidant, anti-inflammatory, and analgesic therapeutic effects. Various previous studies have established that Resveratrol has the capacity to counter the effects of free radicals, affect inflammatory signal transduction, and improve the performance of mitochondria (Zhao *et al*., 2020). Resveratrol suppresses the processing of TNF-α, Interleukin-8 (IL-8), and Interleukin-1β (IL-1β) by lipopolysaccharide-stimulated monocytes and alveolar macrophages (Sławińska and Olas, 2022). Research has shown that low levels of Resveratrol can also reduce the synthesis of nitric oxide, as indicated by the nitrite levels in the testing culture medium (Wang *et al*., 2024). The recent research aimed to enhance the effects of resveratrol on diabetes induced neuropathic pain. Additionally, the study aims to examine the roles of NO and TNF-α in the pain-relieving effects of resveratrol.

MATERIALS AND METHODS

Animals

The selected weighing range for Wistar Albino male rats was between 220 and 280 g, acquired from the Central Animal House of Teerthanker Mahaveer College of Pharmacy, Delhi Road (NH-24), Moradabad, Uttar Pradesh (Registration No: 1205/PO/Re/S/08/CPCSEA. The animals were kept in a well-maintained laboratory environment with a natural light-dark cycle, with free access to standard nutrition and water. The overall experimental protocol was conducted over 8 weeks. Diabetes was induced by streptozotocin administration, followed by 4 weeks for the development of diabetic neuropathy. Resveratrol treatment was initiated at Week 4 and continued until the end of Week 8. The Institutional Animal Ethics Committee approved the research protocol (registration number CCSEA/1205/2024/17), and the study followed the ethical guidelines for animal research dated 13/08/2024.

Drugs and Reagents

The chemicals STZ and resveratrol used in the experiments were obtained from Yucca Pharmaceuticals, Mumbai, India. The Central Chemical Store of Teerthanker Mahaveer University provided Sulphanilamide, Phosphoric Acid, and N-(1-naphthyl) ethylenediamine dihydrochloride. The source of a Glucose Oxidase-Peroxidase (GOD-POD) enzyme diagnostic kit was Sigma (USA). Tumor Necrosis Factor-Alpha (TNF-α) testing kit was provided by Sigma-Aldrich, India, and Enzyme-Linked Immunosorbent Assay (ELISA) kits were provided by the same company.

Induction of Diabetes

Experimental diabetes was induced by a single intraperitoneal administration of streptozotocin (50 mg/kg), freshly prepared in sterile 0.9% normal saline. Following 48 hr of streptozotocin administration, diabetes was confirmed (Sharma *et al*., 2023). Blood samples were collected by tail vein puncture using standard procedures for blood collection in small laboratory animals and plasma glucose levels were measured using the glucose oxidase-peroxidase diagnostic kit method. Animals with plasma glucose levels greater than 13.8 mmol/L were selected for further study (Parasuraman *et al*., 2010).

Experimental scheme

Four weeks after streptozotocin administration, diabetic neuropathy was confirmed by behavioural assessment. The animals were then randomly divided into five groups (n=6 per group). Group I served as the normal control, Group II as the diabetic control, and Groups III-V received resveratrol orally at doses of 10, 20, and 40 mg/kg/day, respectively. Treatment was continued for 4 weeks (Weeks 4 to 8). Control and diabetic control animals received an equal volume of vehicle (0.5% CMC). And all doses were prepared before administration, with every dose being given orally in a fixed volume of 1 mL/ 100 g body mass.

Evaluation of Thermal Nociception

Warm water tail immersion assay

The tail immersion trial was done according to a modified protocol by using the methodology outlined by (Sharma *et al*., 2023). During the process, the tail of the rats was dipped with its distal portion in a water bath adjusted to a temperature of 52.5±0.5°C. It was recorded as the latency to respond either through tail flicking or an indication of discomfort through struggling. To avoid the injury, a cut-off time of 12 sec was imposed (Anjaneyulu and Chopra, 2003)

Estimation of TNF-α

TNF-alpha in the homogenate of the sciatic nerve tissue was quantified by a commercially available ELISA kit by following the manufacturer's guidelines. Briefly, sciatic nerve tissues were homogenized in Phosphate-Buffered Saline (PBS) containing a protease inhibitor. The homogenate was centrifuged at 12,000 rpm for 15 min at 4°C, and the supernatant was collected and transferred to a 96-well plate pre-coated with anti-TNF-α antibodies. Colour development was achieved using TMB substrate, and absorbance was measured at 450 nm. TNF-α levels were quantified from a standard curve and expressed as pg/mg protein (Chen *et al*., 2022).

Statistical analysis

Nociceptive threshold, which was indicated by the latency (seconds) in response to a thermal stimulus, was determined as the mean +/- Standard Error of the Mean (S.E.M.). One-way Analysis of Variance (ANOVA) was used to compare the statistical

significance between two or more groups, with Tukey's *post hoc* test as a subsequent test to make group comparisons.

RESULTS

Effect of Resveratrol on Blood Glucose Levels (mg/dL) in STZ-Induced Diabetic Rats

The experimental groups had no significant differences in the level of blood glucose at baseline (Day 0). After STZ administration, the blood glucose of the diabetic control group had a dramatic increase during the course of the study. By Week III, blood glucose levels in the STZ group were found to be much higher than those in the normal group

The resveratrol (10, 20, and 40 mg/kg) treatment had a dose-dependent effect of reducing the rise in blood glucose levels caused by the STZ. The greatest dose of resveratrol (40 mg/kg) reduced the level of blood glucose most significantly by Week III, nearly matching the values of normal levels in comparison with the diabetic control group (Table 1 and Figure 1).

Impact of Resveratrol on Body Weight in STZ-Induced Diabetic Rats

The changes in body weight were observed in the course of the experiment. When administered with STZ, the diabetic control group experienced a gradual reduction in body weight, which signalled the change in metabolism that came with experimental diabetes. Conversely, resveratrol therapy did a great job in countering the phenomenon of body weight loss in diabetic rats. The protective action was larger in those animals that were given increased doses of resveratrol (20 and 40 mg/kg). With 40 mg/kg of resveratrol, rats showed a significant reduction in body weight when compared to the control group (STZ Week III) (Table 2 and Figure 2).

Impact of Resveratrol on Nociceptive Threshold in Diabetic Rats

At baseline (Day 0), there were no significant differences in the nociceptive threshold among all groups, indicating uniformity before STZ induction. By Week 1, the diabetic control (STZ) group demonstrated a marked reduction in nociceptive threshold (3.520±0.120), confirming the development of diabetic neuropathy characterized by thermal hyperalgesia. This decline continued progressively through Week 3 (2.510±0.100). Chronic administration of resveratrol significantly ameliorated the STZ-induced decrease in nociceptive threshold in a dose-dependent manner. The low dose (10 mg/kg) of resveratrol produced only a modest improvement by Week 3 (3.120±0.110), whereas the medium (20 mg/kg) and high (40 mg/kg) doses resulted in more pronounced protective effects, with Week 3 values of 3.550±0.120 and 3.810±0.130, respectively. The high-dose group approached near-normal values, suggesting substantial neuroprotective activity. The statistical analysis (one-way ANOVA with Tukey's *post hoc* test) confirmed that all resveratrol-treated groups exhibited significantly higher nociceptive thresholds compared to the diabetic control group ($p<0.05$) (Table 3). These findings indicate that resveratrol mitigates STZ-induced neuropathic pain, likely through its antioxidant and anti-inflammatory mechanisms (Figure 3).

Impact of resveratrol on the serum TNF-α levels

The levels of serum TNF- cytokine in the STZ-treated diabetes patients were high (significantly higher than in the normal levels

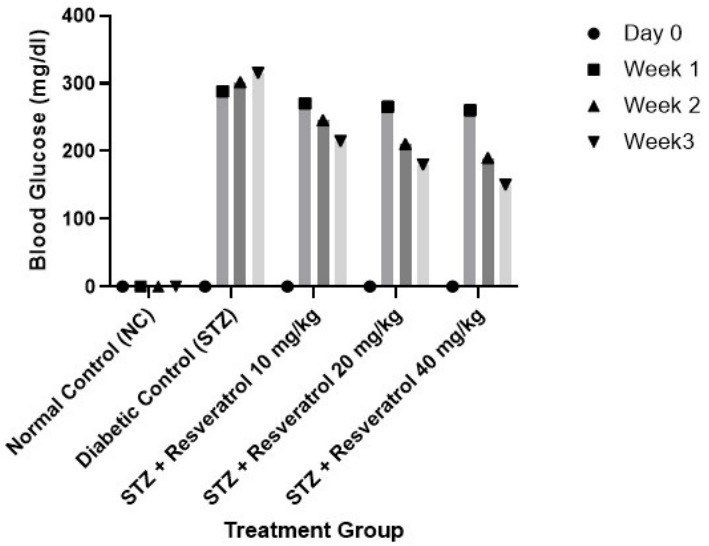


Figure 1: Impact of Resveratrol on plasma glucose levels (mg/dL) in Streptozotocin-treated diabetic Rats. $p<0.05$ as compared to the Streptozotocin (STZ)-treated diabetic group.

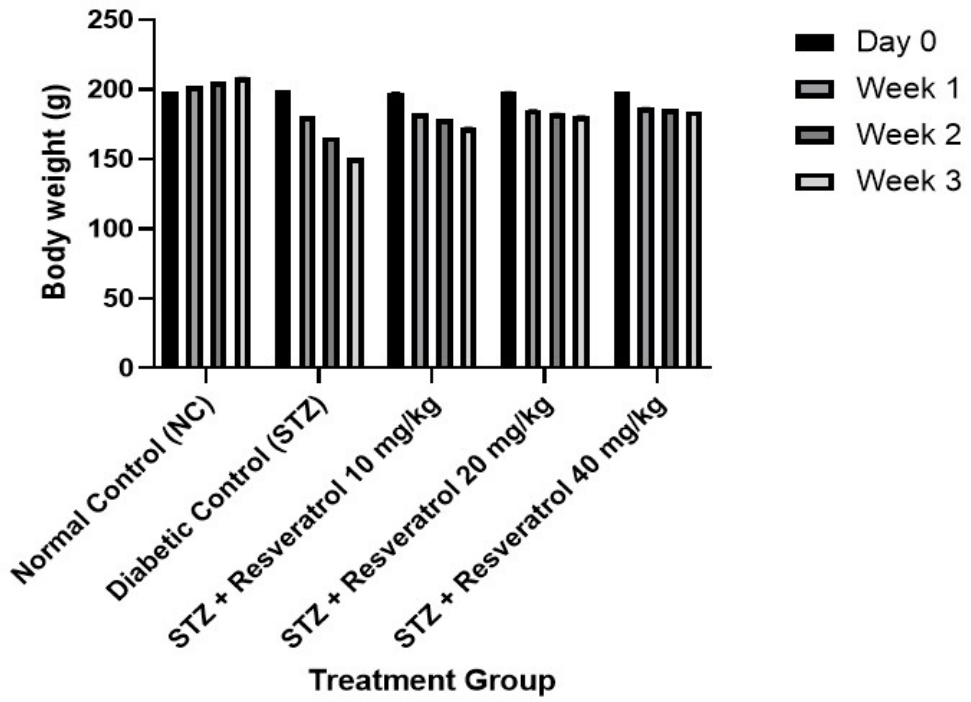


Figure 2: Effect of Resveratrol on body mass (g) in STZ-Induced Diabetic Rats. $p<0.05$ as compared to the control group. $p<0.05$ as compared to the STZ-treated diabetic group.

Table 1: Impact of Resveratrol on Plasma Glucose Concentration (mg/dL) in Streptozotocin-treated diabetic Rats.

Groups	Day 0	Week I	Week II	Week III
Normal Control (NC)	096.2±2.4	097.5±2.6	098.1±2.7	097.4±2.5
Diabetic Control (STZ)	095.8±2.1	288.3±5.4	301.7±6.2	315.5±7.0
STZ + Resveratrol 10 mg/kg	096.4±2.3	270.5±5.0	245.8±4.8	215.3±4.1
STZ + Resveratrol 20 mg/kg	095.9±2.2	265.7±5.2	210.9±4.3	180.4±3.9
STZ + Resveratrol 40 mg/kg	096.1±2.4	260.4±5.0	190.3±3.9	150.8±3.4

Table 2: Impact of Resveratrol on Body Weights (g) in Streptozotocin-Induced diabetic Rats.

Groups	Day 0	Week I	Week II	Week III
Normal Control (NC)	198.4±3.2	202.1±3.5	205.3±3.7	208.6±3.9
Diabetic Control (STZ)	199.2±3.4	180.5±3.0	165.8±2.9	150.4±2.7
STZ + Resveratrol 10 mg/kg	197.9±3.3	182.9±3.2	178.4±3.1	172.6±2.9
STZ + Resveratrol 20 mg/kg	198.1±3.3	185.3±3.2	182.7±3.0	180.9±2.9
STZ + Resveratrol 40 mg/kg	198.0±3.2	186.9±3.3	185.6±3.1	184.3±3.0

Table 3: Effect of Resveratrol (10, 20, and 40 mg/kg, p.o.) on the pain threshold values in STZ-injected diabetic rats subjected to tail immersion in warm water (52.5±0.5°C).

Group	Day 0 (Baseline)	Week I	Week II	Week III
Normal Control (NC)	4.800±0.150	4.850±0.140	4.870±0.160	4.890±0.130
Diabetic Control (STZ)	4.790±0.140	3.520±0.120	2.980±0.110	2.510±0.100
STZ + RES 10 mg/kg	4.810±0.130	3.650±0.130	3.410±0.120	3.120±0.110
STZ + RES 20 mg/kg	4.820±0.140	3.880±0.120	3.700±0.130	3.550±0.120
STZ + RES 40 mg/kg	4.800±0.150	4.050±0.140	3.920±0.120	3.810±0.130

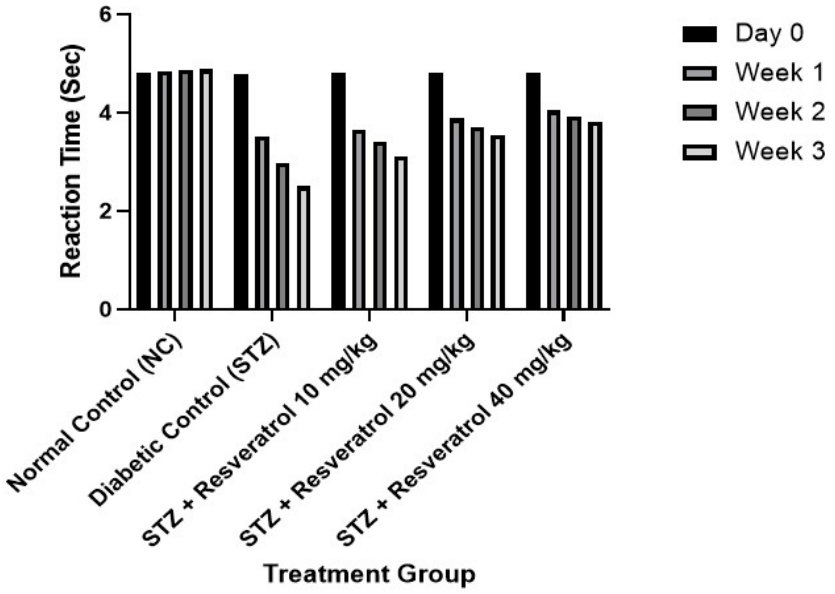


Figure 3: Impact of resveratrol (10, 20, and 40 mg/kg, p.o.) on pain threshold values (% control) in control and diabetic rats. Values are expressed as mean±S.E.M. (*n*=6 in each group). a $p<0.05$ as compared to the control group. b $p<0.05$ as compared to the Streptozotocin (STZ)-treated diabetic group.

Table 4: Serum TNF-α Levels (pg/mL) in Different Experimental Groups at Various Time Points.

Groups	Day 0	Week I	Week II	Week III
Normal Control (NC)	25.3±2.1	25.7±2.4	26.0±2.3	26.2±2.1
Diabetic Control (STZ)	25.6±2.0	55.4±3.5	72.8±4.2	89.5±4.7
STZ + Resveratrol 10 mg/kg	25.4±1.9	49.5±3.2	60.3±3.7	70.4±3.9
STZ + Resveratrol 20 mg/kg	25.7±2.1	45.2±3.0	52.7±3.5	60.2±3.4
STZ + Resveratrol 40 mg/kg	25.5±2.0	40.3±2.8	45.6±3.2	25.6±3.1

of the negative control group), which demonstrated the existence of systemic inflammation linked with diabetic neuropathy.

Resveratrol significantly lowered serum TNF-α levels of diabetic rats in a dose- dependent fashion. They found the largest change in the group of 40 mg/kg resveratrol, which revealed a considerable change in the levels of TNF-α until Week III compared to the diabetic control group (Table 4 and Figure 4).

DISCUSSION

The current research assessed the anti-neuropathic pain effect of resveratrol on STZ-induced diabetic neuropathy of rats. Streptozotocin administration induced hyperglycemia, reduced body weight, ameliorated inflammatory mediators, and augmented oxidative stress - typical traits of experimental diabetic neuropathy. Diabetic rats treated with STZ showed increased blood glucose and decreased body weight. The 40 mg/kg dose showed the most significant effect, indicating resveratrol's benefit in glycemic control through antioxidant effects and metabolic regulation. Diabetic rats displayed reduced nociceptive threshold in tail immersion tests, indicating thermal hyperalgesia. The study showed elevated serum TNF-α levels in diabetic rats, indicating systemic inflammation. TNF- α is associated with neuronal injury and neuropathic pain. Resveratrol treatment decreased TNF-α levels dose-dependently, demonstrating anti-inflammatory effects by regulating inflammatory cytokines and neuroinflammation pathways. Resveratrol treatment reduced nitrite concentrations, especially at higher doses, demonstrating its antioxidant effect and ability to protect neuronal tissues. The results prove resveratrol's protective capacity against streptozotocin-induced diabetic neuropathy through its hypoglycemic, anti-inflammatory, antioxidant, and neuroprotective actions. Although the present study demonstrates promising neuroprotective effects of resveratrol, certain limitations should be acknowledged. The study evaluated only TNF-α as an inflammatory biomarker, and additional oxidative stress markers, histopathological assessment, and molecular investigations were not performed. Future studies should explore these parameters to elucidate further the precise mechanisms underlying the neuroprotective effects of resveratrol and facilitate its potential clinical translation.

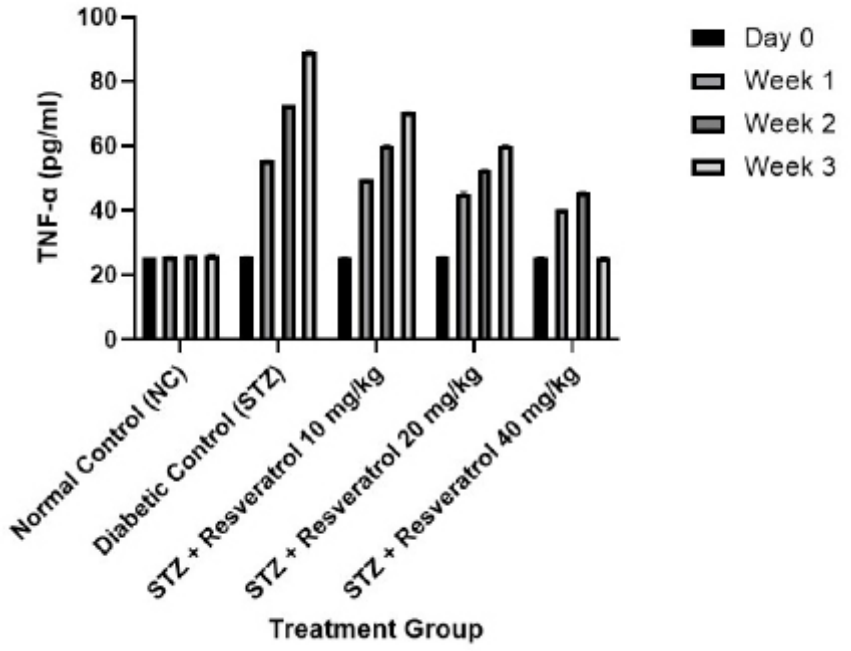


Figure 4: Effect of resveratrol (10, 20, and 40 mg/kg, p.o.) on Serum TNF-α (pg/mL) levels (% control) in control and diabetic rats. Values (n=6 per group) are shown as mean±S.E.M, with a $p<0.05$ in contrast to the control cohort. In comparison to the group that received Streptozotocin (STZ), $p<0.05$.

CONCLUSION

In the current study, a rigorous scientific process demonstrating that resveratrol has significant anti-nerve pain efficacy in streptozotocin-induced diabetic rats was presented. Resveratrol treatment resulted in significant changes in several key parameters, including a decrease in blood glucose levels, an increase in body weight, and a significant reduction in thermal hyperalgesia. Also, the application of resveratrol significantly reduced the quantity of biochemical enzyme indicators of oxidative stress and inflammation, including Tumour Necrosis Factor-Alpha (TNF-α). These therapeutic effects were identified as dose-dependent, with the greatest effect observed at the maximum dose (40 mg/kg). These findings support the neuroprotective potential of resveratrol, likely mediated through its antioxidant and anti-inflammatory properties. Further studies should investigate its effects on molecular pathways such as NF-κB, TLR4, and mitochondrial biogenesis, along with long-term evaluation in chronic diabetic models and clinical trials to confirm efficacy and safety. The potential synergistic use of resveratrol with other phytochemicals or conventional neuropathic pain therapies also warrants exploration.

ABBREVIATIONS

STZ: Streptozotocin; **TNF-α:** Tumor Necrosis Factor-Alpha; **ROS:** Reactive Oxygen Species; **IL-8:** Interleukin-8; **IL-1β:** Interleukin-1β; **NO:** Nitric Oxide; **GOD-POD:** Glucose Oxidase-Peroxidase; **ELISA:** Enzyme-Linked Immunosorbent Assay; **PBS:** Phosphate-Buffered Saline; **S.E.M.:** Standard Error of the Mean; **ANOVA:** Analysis of Variance.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

REFERENCES

Anjaneyulu, M. and K. Chopra (2003). "Quercetin, a bioflavonoid, attenuates thermal hyperalgesia in a mouse model of diabetic neuropathic pain." Progress in neuro-psychopharmacology and biological psychiatry 27(6): 1001-1005. DOI: 10.1016/S0278-5846(03)00160-X

Anwar, S., Sarwar, T., Khan, A. A., & Rahmani, A. H. (2025). Therapeutic applications and mechanisms of superoxide dismutase (SOD) in different pathogenesis. *Biomolecules*, *15*(8), 1130. https://doi.org/10.3390/biom15081130

Chen, S.-M., *et al.* (2022). "Neuroprotective effect of l-theanine in a rat model of chronic constriction injury of sciatic nerve-induced neuropathic pain." Journal of the Formosan Medical Association 121(4): 802-814. https://doi.org/10.1016/j.jfma.2021.08.023

Finnerup, N. B., Kuner, R., & Jensen, T. S. (2021). Neuropathic pain: from mechanisms to treatment. *Physiological Reviews*, *101*(1), 259-301. https://doi.org/10.1152/physrev.00045.2019

Kumar Sharma, K., Fatima, N., Ali, Z., Moshin, M., Chandra, P., *et al.* (2023). Neuropathy, its profile and experimental nerve injury neuropathic pain models: a review. *Current Pharmaceutical Design*, *29*(42), 3343-3356. https://doi.org/10.2174/0113816128274200231128065425

Parasuraman S, Raveendran R, Kesavan R. Blood sample collection in small laboratory animals. J Pharmacol Pharmacother. 2010;1(2):87–93. doi:10.4103/0976-500X.72350.

Petroianu, G. A., Aloum, L., & Adem, A. (2023). Neuropathic pain: Mechanisms and therapeutic strategies. Frontiers in Cell and Developmental Biology, 11, 1072629. https://doi.org/10.3389/fcell.2023.1072629

Pop-Busui, R., Ang, L., Boulton, A. J., Feldman, E. L., Marcus, R. L., Mizokami-Stout, K., Singleton, J. R., & Ziegler, D. (2022). Diagnosis and treatment of painful diabetic peripheral neuropathy. https://doi.org/10.2337/db2022-01

Quiroz-Aldave, J., Durand-Vásquez, M., Gamarra-Osorio, E., Suárez-Rojas, J., Roseboom, P. J., *et al.* (2023). Diabetic neuropathy: Past, present, and future. *Caspian journal of internal medicine*, *14*(2), 153. https://doi.org/10.22088/cjim.14.2.153

Sharma M, Chan HK, Lavilla CA Jr, Uy MM, Froemming GRA, Okechukwu PN. Induction of a single dose of streptozotocin (50 mg) in rat model causes insulin resistance with type 2 diabetes mellitus. Fundam Clin Pharmacol. 2023;37(4):769–778. doi:10.1111/fcp.12892.

Singh, R., Rao, H. K., & Singh, T. G. (2020). Neuropathic pain in diabetes mellitus: Challenges and future trends. Obesity Medicine, 18, 100215. https://doi.org/10.1016/j.obmed.2020.100215

Sławińska, N., & Olas, B. (2022). Selected seeds as sources of bioactive compounds with diverse biological activities. *Nutrients*, *15*(1), 187. https://doi.org/10.3390/nu15010187

Sloan, G., Alam, U., Selvarajah, D., & Tesfaye, S. (2022). The treatment of painful diabetic neuropathy. *Current diabetes reviews*, *18*(5), 42-96. https://doi.org/10.2174/1573399817666210707112413

Stojanovic, B., Milivojcevic Bevc, I., Dimitrijevic Stojanovic, M., Stojanovic, B. S., Lazarevic, T., *et al.* (2025). Oxidative stress, inflammation, and cellular senescence in neuropathic pain: mechanistic crosstalk. *Antioxidants*, *14*(10), 1166. https://doi.org/10.3390/antiox14101166

Trofin, D.-M., Sardaru, D.-P., Trofin, D., Onu, I., Tutu, A., *et al*. Onu, A., Onită, C., Galaction, A. I., & Matei, D. V. (2025). Oxidative stress in brain function. *Antioxidants*, *14*(3), 297. https://doi.org/10.3390/antiox14030297

Wang, B., Jiang, H. M., Qi, L. M., Li, X., Huang, Q., *et al.* (2024). Deciphering resveratrol's role in modulating pathological pain: From molecular mechanisms to clinical relevance. *Phytotherapy Research*, *38*(1), 59-73. https://doi.org/10.1002/ptr.8021

Zaa, C. A., Marcelo, Á. J., An, Z., Medina-Franco, J. L., & Velasco-Velázquez, M. A. (2023). Anthocyanins: molecular aspects on their neuroprotective activity. *Biomolecules*, *13*(11), 1598. https://doi.org/10.3390/biom13111598

Zhao, D., Simon, J. E., & Wu, Q. (2020). A critical review on grape polyphenols for neuroprotection: Strategies to enhance bioefficacy. *Critical Reviews in Food Science and Nutrition*, *60*(4), 597-625. https://doi.org/10.1080/10408398.2018.1546668

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