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Original Article

Targeting AGE-RAGE Signaling in Neuroprotective Effects of Antrocin in Streptozotocin-Induced Diabetic Neuropathy: An *in vivo* Study

Zhongjiang Niu[1], Fei Xiao[2,*], Alok S Tripathi[3]

[1]Department of Neurology, Harbin 242 Hospital, Harbin, CHINA.
[2]Department of Neurology, Baoji Hospital of Traditional Chinese Medicine, Baoji, CHINA.
[3]ERA College of Pharmacy, ERA University, Lucknow, Uttar Pradesh, INDIA.

ABSTRACT

Background: Diabetic Neuropathy (DN) is a common and debilitating complication of diabetes, characterized by neuronal dysfunction driven partly by oxidative stress, inflammation, and Advanced Glycation End Product (AGE)-mediated signaling. Antrocin, a bioactive compound with reported pharmacological properties, was investigated for its neuroprotective efficacy in an experimental model of DN. Objectives: This study aimed to examine the effect of antrocin on behavioral, biochemical, and molecular alterations associated with Streptozotocin (STZ)-induced diabetic neuropathy in rats, with particular focus on the AGE-RAGE signaling axis. **Materials and Methods:** Diabetes-associated neuropathy was induced using STZ in rats. Antrocin was administered orally at doses of 15 and 30 mg/kg for four weeks following diabetes induction. Neuromuscular coordination and nociceptive responses were evaluated to assess neuropathic manifestations. Serum biochemical indices, including glucose, insulin, lipid parameters, and glycated hemoglobin (HbA_{1C}), were quantified. Oxidative stress markers and pro-inflammatory cytokines were analyzed, and the protein expression levels of AGE and its receptor RAGE were assessed using Western blotting. **Results:** Antrocin treatment significantly improved behavioral outcomes related to neuropathic pain and motor coordination. Normalization of hyperglycemia, dyslipidemia, and elevated HbA_{1C} levels was observed, along with restoration of serum insulin levels. Antrocin also attenuated oxidative stress and inflammatory responses in diabetic rats. Molecular analysis demonstrated downregulation of AGE and RAGE protein expression in antrocin-treated groups compared with untreated diabetic controls. **Conclusion:** Antrocin confers significant neuroprotection in STZ-induced diabetic neuropathy. Its effects appear to be mediated through suppression of AGE-RAGE signaling, reducing oxidative damage and inflammation, thereby improving neuronal function in diabetic conditions.

Keywords: AGE-RAGE signaling, Antrocin, Diabetic neuropathy, Streptozotocin, Neuroinflammation, Oxidative damage.

Correspondence:
Dr. Fei Xiao
Department of Neurology, Baoji Hospital of Traditional Chinese Medicine, Baoji-721000, CHINA.
Email: 2024newhome@sina.com
ORCID: 0009-0004-7326-8101

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INTRODUCTION

Diabetic Neuropathy (DN) is one of the major chronic complications of diabetes that occurs due to uncontrolled hyperglycemia. It affects more than 1.8 % of type II diabetic patients, which shows clinical features like burning feeling, dysesthesia, allodynia, and hyperalgesia.[1] Neurotropic, metabolic, and vascular disorders are also causes of DN, which can cause changes in electrophysiology, altered perception of sensory nerves, and neuronal injury.[2] There are several pathogenic pathways, including oxidative stress and inflammation, as well as the AGE-RAGE signaling pathway.[3] Uncontrolled hyperglycemia dysregulates several metabolic pathways, such as the inducible nitric oxide synthase pathway, the mitogen-activated protein kinase pathway, and the polyol pathway, and stimulates the formation of AGEs.[4] Production of reactive oxygen species increases in hyperglycemia, promoting nonenzymatic metabolism of carbohydrates, proteins, and lipids with nonreducing glucose, thereby enhancing the formation of AGEs.[5] AGEs bind to their receptor, RAGE, which dysregulates several signaling pathways, including calcium, MAPK, and JAK-STAT.[6] It is commonly observed that these pathways are activated by AGE-RAGE, which enhances the production of Reactive Oxygen Species (ROS), thereby stimulating the apoptosis pathway.[7] Moreover, AGE-RAGE alters cellular function by promoting the production of inflammatory cytokines.[8] These changes contribute to the development of several complications,

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including DN. Regulating the AGE-RAGE signaling pathway or reducing AGE production could be used to manage DN.

Naturally bioactive compounds have shown promising effects against the management of several chronic disorders, including diabetic complications like DN. Antrocin is chemically a sesquiterpene lactone isolated from fungus (*Antrodia cinnamomea*) used traditionally for medicinal purposes like anti-diabetic and abdominal pain in Asia.[9] *Antrodia cinnamomea* contains several chemical constituents, including antrocin, known for its medicinal properties.[10] Antrocin has been reported to have strong antioxidant and anticancer properties.[9] Moreover, antrocin suppresses cancer growth by regulating the JAK/STAT3 and Akt/mTOR/GSK-3β/NF-κB signaling pathways.[11] Thus, the present study determines the beneficial effect of antrocin against diabetic neuropathy.

MATERIALS AND METHODS

Experimental animal and induction of diabetic neuropathy

Male Wistar rats(250-300g), which were maintained at the following conditions: temperature: (24 ± 1)°C, dark/light cycle: 12:12 hr; relative humidity: 45%-55%, standard pellet chow, and*ad libitum* water. The study was approved by the institutional ethical committee (2002/PO/Re/S/18/CPCSEA/28). The research was conducted in 2023.

Diabetic neuropathy was induced by the administration of Streptozotocin (STZ) in rats as per the reported method. Diabetes was induced by intraperitoneal administration of STZ (55 mg/kg) and the blood glucose level was determined to confirm it after 48 h of administration of STZ. Rats with glucose> 200 mg/dL should be considered for further study; keep the animals as such for 4 weeks. All the rats were separated into four groups: Normal; DN group, which received STZ (55 mg/kg, i.p.); and Antrocin 15 and 30 mg/kg, p.o., for four weeks after the four weeks of STZ administration. Body weight was determined at the end of each week, and blood glucose was estimated from the serum of all groups of rats.

Behavioural study

Determination of hyperalgesia

Hyperalgesia was assessed using the tail-immersion test as reported in the study.[12] Tail withdrawal time was determined before and after the protocol. Moreover, the hot-plate method was also used, and paw latency was measured before and after drug administration at different time intervals. Mechanical hyperalgesia was determined in antrocin-treated DN rats at the end of the protocol using a Randall-Selitto analgesiometer.

Determination of motor coordination

Motor function was assessed by estimating grip strength using a rota-rod apparatus. Fall time was observed to estimate the grip strength for each rat.

Determination of biochemical parameters

Blood was withdrawn from each rat using the retro-orbital puncture technique and homogenized to separate the serum for biochemical parameter estimation. The lipid profile was assessed in serum as per the manufacturer's instructions. Moreover, the levels of insulin and glycosylated hemoglobin were determined in serum using ELISA kits.

Determination of oxidative stress parameters, inflammatory cytokines and AGEs

The sciatic nerve was isolated from each rat after euthanizing them with cervical dislocation and homogenized in Tris-HCl buffer (0.1 M, and pH 7.4). Centrifuge it to separate the supernatant, and estimate the parameters of oxidative stress in the tissue homogenate. The level of MDA and activity of SOD were estimated in the tissue homogenate of each rat with ELISA kits. Inflammatory cytokines, including IL-6, IL-1β, and TNF-α, and AGEs were also measured in the tissue homogenate of each rat using an ELISA kit.

Determination of membrane-bound inorganic phosphate

The membrane-bound enzyme (Na^+-K^+-ATPase) was detected in the tissue homogenate as described in the reported method. Enzyme activity is expressed as μM of inorganic phosphorus liberated/mg protein/h.

Determination of the expression of AGE/RAGE protein

Radioimmunoprecipitation Assay (RIPA) buffer was used to prepare the Sciatic nerve tissue homogenate, and the BCA assay was used to assess total protein concentration. A PVDF membrane was used to filter the protein isolated from the tissue, and SDS-PAGE analyzed the protein. Further, antibodies like anti-RAGE, anti-NF-κB, and anti-β-actin were incubated with the membrane overnight at 4°C, followed by incubation with secondary antibodies for 1 hr at room temperature. ImageJ software was used to determine the optical density of protein bands, which are visualized with Tanon 5200 chemiluminescence imaging system.

Statistical analysis

SPSS version 10 was used to analyse the statistical significance of the data obtained. All the experiments were run in triplicate, and the values are provided as Mean±SEM. One-way ANOVA was used for the statistical analysis and Dunnet's multiple comparison

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test was performed to compare the significance between the treated and control groups. $p<0.05$ is considered significant.

RESULTS

Antrocin attenuates body weight and blood glucose level

Body weight and blood glucose levels were estimated in the antrocin-treated DN rats at the end of each week of the protocol (Figures 1A, 1B). A significant decrease in body weight was observed in the DN group compared with the normal group of rats. The treated groups have shown significant improvement in body weight compared to the DN group of rats from the 4th to 8th week of the protocol (Figure 1A). Moreover, the DN group showed a significant increase ($p<0.001$) in blood glucose levels compared with the normal group of rats. Treatment with antrocin attenuates the elevated glucose levels in the serum of DN rats (Figure 1B).

Antrocin attenuates behavioural changes and motor function

Behavioural changes and motor function were assessed in antrocin-treated DN rats as shown in Figures 2A-2D. There was a significant decrease in tail withdrawal latency (Tail immersion test), Paw withdrawal latency (Hot plate method), and Paw withdrawal threshold (Randall-Selitto analgesiometer) in the DN group compared with the normal group of rats. Treatment with antrocin attenuates the behavioral alterations in DN rats (Figures 2A-2C). Moreover, motor function was assessed by measuring fall time using a rota rod apparatus. There was a significant decrease in fall time in the DN group compared with the normal group of rats, which was reversed in the antrocin-treated group (Figure 2D).

Antrocin attenuates biochemical parameters

The effect of antrocin was determined on the levels of insulin, HbA_{1C}, and lipid profile in the serum of DN rats. There was a significant decrease in insulin and an increase in HbA_{1C} (%) in the serum of the DN group compared with the normal group of rats, which was reversed in the antrocin-treated group in a dose-dependent manner (Figures 3A, 3B). The levels of triglycerides, total cholesterol, and LDL were significantly ($p<0.001$) elevated in the serum of the DN group compared with the normal group of rats, whereas HDL levels were reduced; this was ameliorated in the antrocin-treated group (Figure 3C).

Antrocin attenuates inflammatory mediators and parameters of oxidative stress

Oxidative stress parameters like activity of SOD and level of MDA were determined in antrocin-treated DN rats as shown in Figures 4A, 4B. Activity of SOD was observed to be significantly reduced ($p<0.001$) and level of MDA enhanced significantly in the tissue homogenate of the DN group than the normal group of rats, which was reversed in the antrocin-treated group. Further, inflammatory cytokines such as IL-1β, IL-6 and TNF-α were significantly elevated ($p<0.001$) in the DN group compared with the normal group, and this effect was reversed in the antrocin-treated group (Figure 4C).

Antrocin attenuates AGE/RAGE pathway

The effect of antrocin on the AGE/RAGE pathway was determined in DN rats. The level of AGE was significantly ($p<0.001$) elevated and the activity of Na+-K+-ATPase was reduced in the DN group compared with the normal group of rats, which was attenuated in the antrocin-treated group (Figures 5A, 5B). Expression of RAGE and NF-κB proteins was observed in the sciatic nerve of DN rats by Western blot assay (Figure 5C). There was a significant enhancement of RAGE and NF-κB protein expression in the DN group compared with the normal group of rats, which was reversed in the antrocin-treated group.

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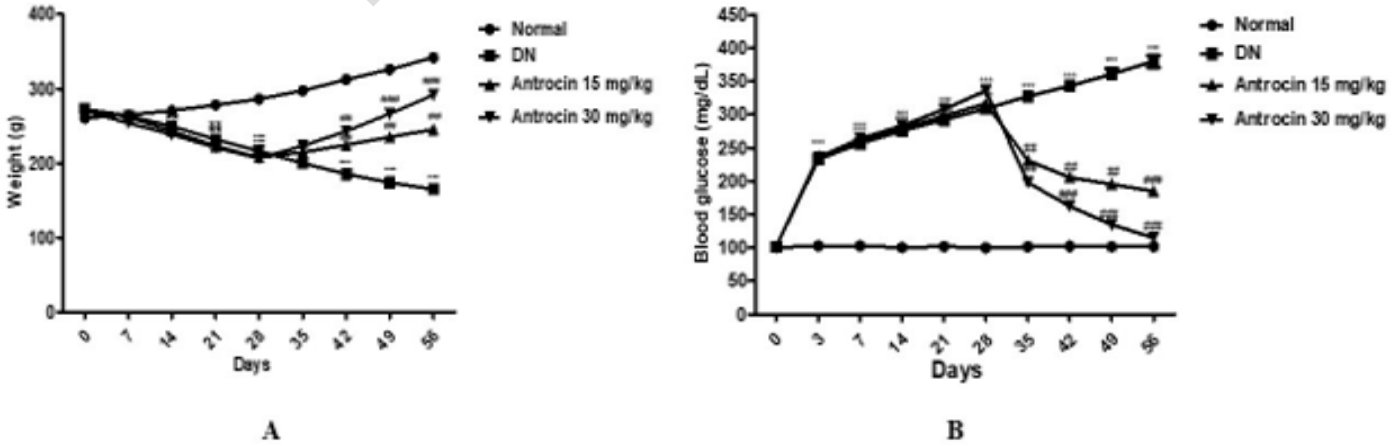


Figure 1: Antrocin ameliorates the altered body weight and level of blood glucose in STZ induced diabetic neuropathic rat model at the end of each week of protocol; A: Body weight; B: Blood glucose level. Mean±SEM (n=6); $^{**}p<0.01$, $^{***}p<0.001$ vs normal group; $^{\#\#}p<0.01$, $^{\#\#\#}p<0.001$ vs DN group.

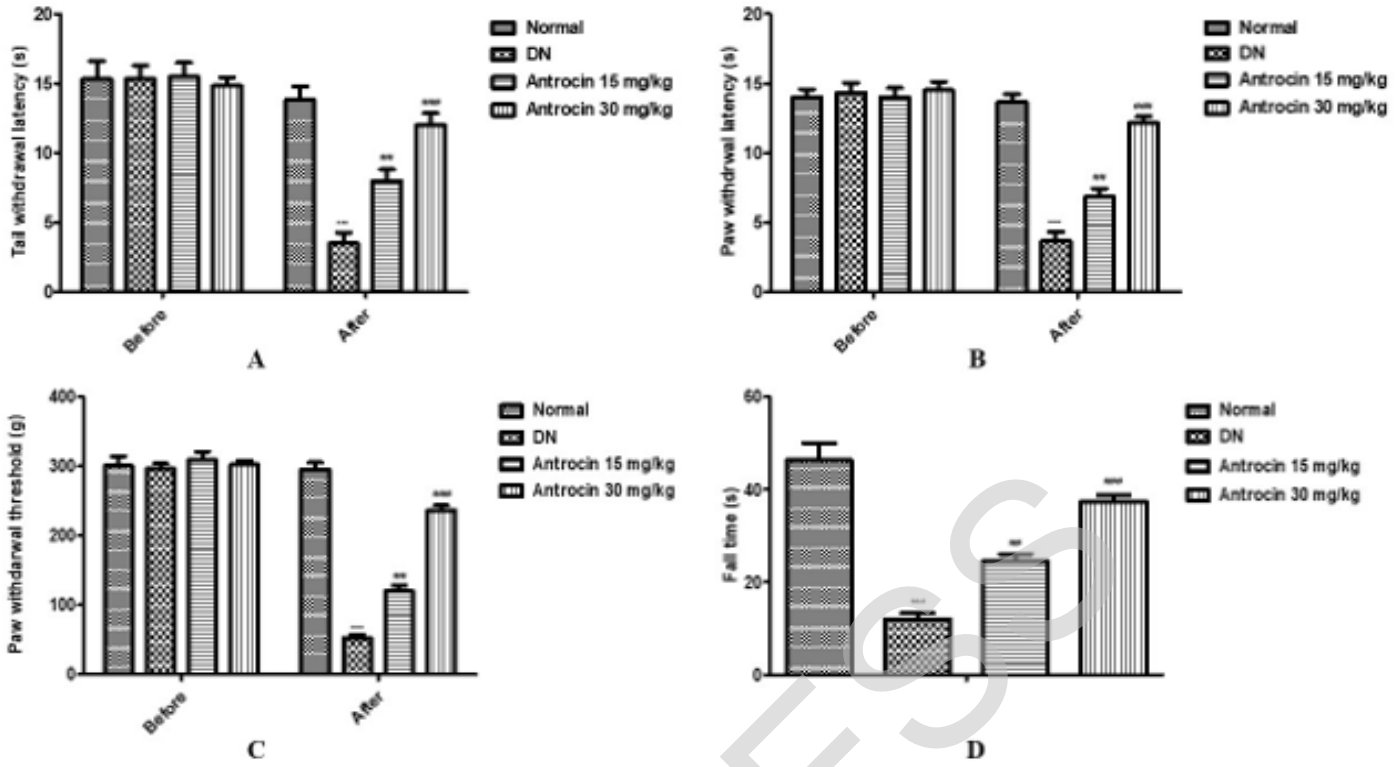


Figure 2: Antrocin ameliorates the altered behavioural changes and motor function in STZ-induced diabetic neuropathic rat model; A: Tail withdrawal latency (Tail immersion test); B: Paw withdrawal latency (Hot plate method); C: Paw withdrawal threshold (Randall-Selitto analgesiometer); Motor function by rota rod apparatus. Mean±SEM (n=6); *** p<0.001 vs normal group; $^{###}$ p<0.001 vs DN group.

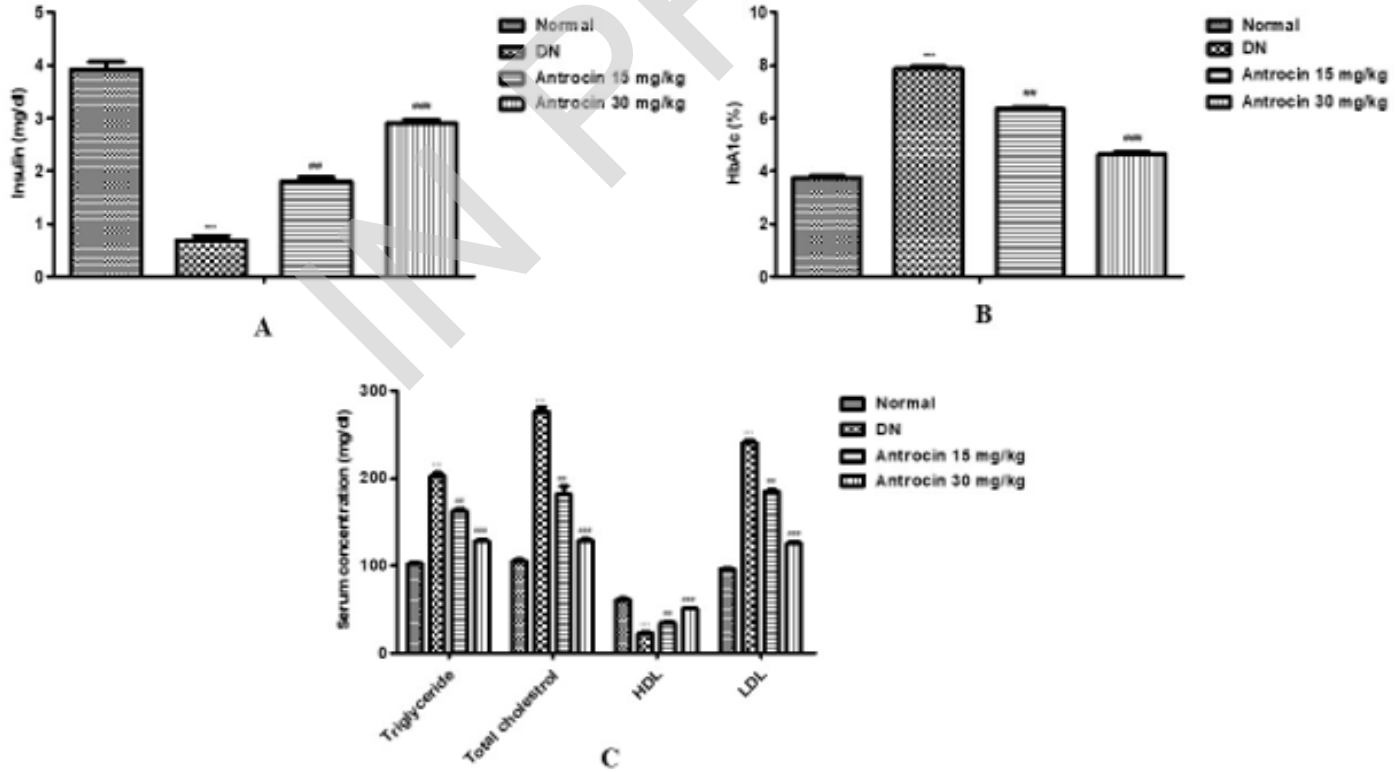


Figure 3: Antrocin ameliorates the altered biochemical parameters in the serum of STZ induced diabetic neuropathic rat model; A: Insulin; B: HbA_{1C} (%); C: Lipid profile. Mean±SEM (n=6); $^{***}p$<0.001 vs normal group; $^{###}p$<0.001 vs DN group.

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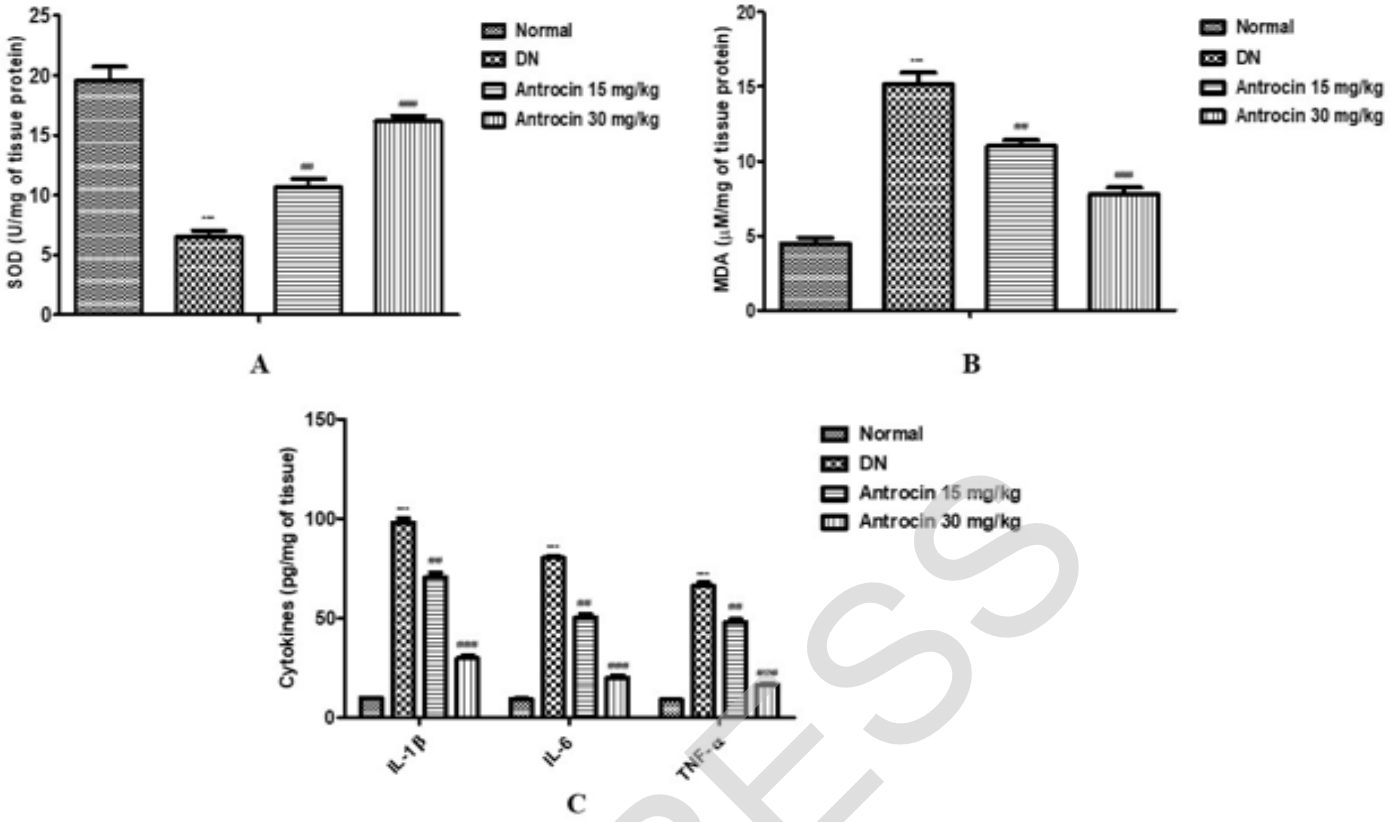


Figure 4: Antrocin ameliorates the altered level of parameters of oxidative stress and inflammatory cytokines in STZ-induced diabetic neuropathic rat model; A: Activity of SOD; B: Level of MDA; C: Level of inflammatory cytokines (IL-1β, IL-6 and TNF-α). Mean±SEM (n=6); $^{***}p<0.001$ vs normal group, $^{\#\#}p<0.01$, $^{\#\#\#}p<0.001$ vs DN group.

DISCUSSION

Diabetic neuropathy is one of the most common chronic complications associated with diabetes, characterized by altered neuronal function. STZ is isolated from the bacterium, which damages DNA and induces cellular death in the pancreas, leading to the development of diabetes.[13] Literature suggested that uncontrolled diabetes for a long duration leads to the development of chronic complications, including diabetic neuropathy 3. Evidence reports that DN occurs after 4 weeks of diabetes induction in rats,[14] and data from the investigation also support this. Weight loss is a common feature of diabetes, and its improvement has a beneficial effect on its management. There was a significant reduction in body weight in the DN group compared with the normal group, which was reversed in the antrocin-treated group.

Hyperglycemia is a key feature of diabetes; prolongation of its development leads to complications. Pancreatic cells were injured, leading to a reduction in insulin levels, a hormone that enhances the utilization of glucose in the cell.[15] Clinical features of diabetes have shown increased glucose and Hb1Ac levels and reduced serum insulin,[16] and data from our investigation have shown the same in the DN group. HbA_{1C} represents the average level of glucose in the blood over 3-6 months and is observed to be higher among patients with DN. Drugs shown to have a promising effect in the management of diabetes, control glucose levels and HbA_{1C}, and improve insulin levels.[17] Our study has shown that treatment with antrocin ameliorates the altered level of glucose, insulin and Hb1Ac in the serum of DN rats. The lipid profile was also observed to be altered in diabetic patients, as glucose utilization decreases, altering lipid metabolism for energy production.[18] These changes alter the lipid levels in the serum and the lipid profile of the individual. Our study shows that the lipid profile was altered in the DN group compared with the normal group of rats.

Chronic complications associated with diabetes include DN, which shows the dysregulation of neurological functions like altered neuropathic pain and muscle coordination.[19] It has been shown that pain-bearing capacity and muscle coordination are lost among individuals suffering from DN, and the data from the given study support this. Several molecules that improve these factors have shown beneficial effects in the management of DN.[20] Antrocin treatment in the given study also shows improvement in neuronal function in DN rats.

Hyperglycemia enhances the production of reactive oxygen species, altering the parameters of oxidative stress in the cell. Superoxide anion is metabolized to H_2O_2 by superoxide dismutase, and reduced activity of superoxide dismutase is associated with increased production of reactive oxygen species.[21] These changes

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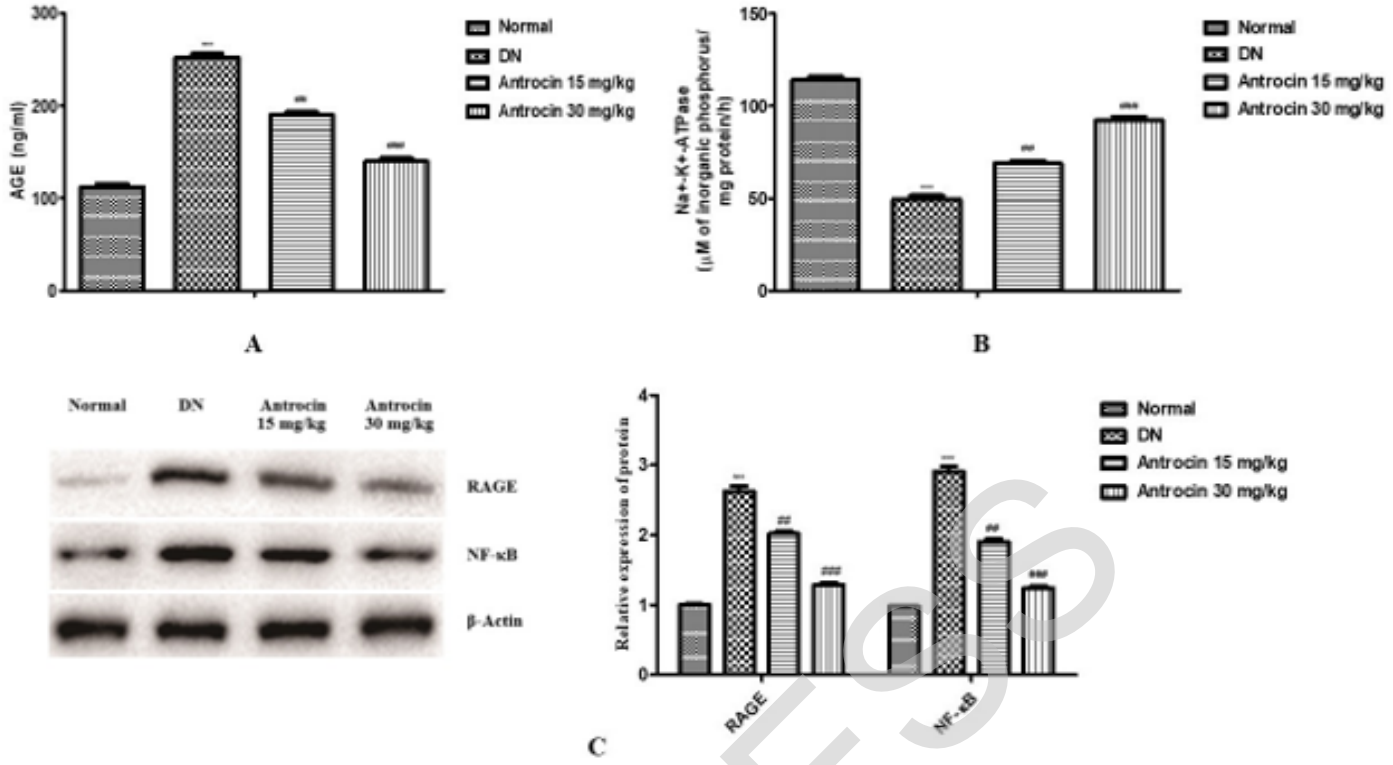


Figure 5: Antrocin ameliorates the altered AGE/RAGE pathway in STZ induced diabetic neuropathic rat model; A: Level of AGE; B: Activity of Na^+-K^+-ATPase; C: Expression of RAGE and NF-κB protein. Mean±SEM (n=6); $^{***}p$<0.001 vs normal group, $^{\#\#}p$<0.01, $^{\#\#\#}p$<0.001 vs DN group.

may lead to lipid peroxidation of cell membrane lipids, which is further enhanced by oxidative stress. Moreover, cell membrane injury promotes the production of inflammatory cytokines, which are further responsible for cellular injury.[22] A drug used for the management of hyperglycemia, it also reduces oxidative stress and inflammatory mediators,[23] and our study shows that treatment with antrocin attenuates altered inflammatory cytokines and oxidative stress parameters in DN rats.

There are several pathogenic factors, including AGE-RAGE, that are involved in the development of DN. The literature reports that deposition of AGEs occurs in diabetes, which interacts with RAGE to promote the production of ROS, enhance oxidative stress, and increase inflammatory cytokines.[24] These factors contribute to neuronal damage in DN, and our study supports this. Treatment with antrocin attenuates the altered expression of AGE/RAGE in DN rats.

CONCLUSION

In conclusion, data reveal that treatment with antrocin ameliorates the altered neuronal function in STZ-induced DN rats. It reduces AGE-RAGE expression, thereby decreasing inflammatory cytokines and oxidative stress parameters in DN rats.

ACKNOWLEDGEMENT

None.

ABBREVIATIONS

CREC: China Resources Enterprise Corporation; **ERA:** ERA University; **ORCID:** Open Researcher and Contributor ID; **GSK:** Glycogen Synthase Kinase; **PO:** Per Os (oral administration); **CPCSEA:** Committee for the Purpose of Control and Supervision of Experiments on Animals; **MDA:** Malondialdehyde; **SOD**: Superoxide Dismutase; **RIPA:** Radioimmunoprecipitation Assay; **SDS:** Sodium Dodecyl Sulfate; **PAGE:** Polyacrylamide Gel Electrophoresis; **LDL:** Low-Density Lipoprotein; **HDL:** High-Density Lipoprotein; **AKT:** Protein Kinase B; **EXCLI:** Experimental and Clinical Sciences Journal; **DN:** Diabetic neuropathy; **STZ:** Streptozotocin; **AGE:** Advanced glycation end product; **RAGE:** Receptor for advanced glycation end products; **HbA_{1C}:** Glycated Hemoglobin; **ROS**: Reactive oxygen species; **MAPK:** Mitogen-activated protein kinase; **JAK-STAT:** Janus kinase-signal transducer and activator of transcription; **ELISA:** Enzyme-linked immunosorbent assay; **IL-6:** Interleukin-6; **IL-1β:** Interleukin-1 beta; **TNF-α:** Tumor necrosis factor-alpha; **NF-κB:** Nuclear factor kappa-light-chain-enhancer of activated B cells; **PVDF:** Polyvinylidene fluoride; **SEM:** Standard error of the mean; **i.p.:** Intraperitoneal; **BCA:** Bicinchoninic acid.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this article.

FUNDING

No funding received.

AUTHORS CONTRIBUTION

ZN, FX: Conceptualization, Methodology; FX, AST: Investigation, Writing-Original Draft; ZN, AST: Supervision, Review & Editing. All authors read and approved the final manuscript.

SUMMARY

This study investigates the neuroprotective effects of antrocin, a bioactive compound from Antrodia cinnamomea, in Streptozotocin (STZ)-induced diabetic neuropathy in rats. Antrocin was administered orally at 15 and 30 mg/kg for four weeks following STZ induction. Treatment significantly improved body weight, glucose, insulin, lipid profile, and HbA_{1C} levels. It also restored motor coordination and reduced neuropathic pain. Biochemical assays showed decreased oxidative stress and inflammatory cytokines (IL-1β, IL-6, TNF-α). Western blot analysis revealed reduced AGE-RAGE and NF-κB expression. Overall, antrocin ameliorates diabetic neuropathy by regulating AGE-RAGE signaling, reducing oxidative stress, and improving neuronal function.

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