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Research Article

Dietary Fiber from Mentawai Taro (*Colocasia esculenta* var. Mentawai) Ameliorates Diabetic Neuropathy in Alloxan-Induced Mice

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ABSTRACT
Diabetic neuropathy is a severe complication of diabetes mellitus, and conventional synthetic drugs used for its management are often associated with undesirable side effects. This study evaluated the potential of Mentawai taro (Colocasia esculenta var. Mentawai*) corm as a functional food for the management of diabetic neuropathy. Adult male mice were divided into five groups: a control group (healthy mice fed a standard diet), a diabetic group (alloxan-induced diabetic mice fed a standard diet), and three diabetic groups fed diets supplemented with 15% Mentawai taro corm whole flour, fiber, or starch. After 28 days of dietary intervention, blood glucose levels, sensory and motor functions, malondialdehyde (MDA) levels, and cerebellar histopathology were evaluated. The results showed that Mentawai taro corm fiber significantly reduced blood glucose levels (59.5% reduction), accompanied by a positive trend toward improved sensory responses (25% increase) and a marked enhancement of motor function (41.6% increase) in diabetic mice. Additionally, fiber supplementation reduced MDA levels in brain tissue (19.3% reduction) and attenuated Purkinje cell degeneration in the cerebellum (27.3% reduction). In contrast, Mentawai taro corm whole flour and starch exerted minimal protective effects, with starch supplementation improving motor function only. Overall, among the various Mentawai taro corm preparations tested, the fiber extract was the most effective in ameliorating symptoms of diabetic neuropathy.*

Keywords: *Colocasia esculenta; diabetes mellitus; motoric balance; oxidative stress; sensory function.*

Introduction

Diabetes mellitus is one of the most prevalent health issues worldwide today [1]. As the disease progresses, it is often accompanied by various comorbidities that exacerbate health problems, one of which is diabetic neuropathy [2]. This condition is defined as a complication of diabetes characterized by progressive structural and functional impairment of peripheral and/or autonomic nerves due to sustained hyperglycemia and associated metabolic and microvascular abnormalities, resulting in sensory, motor, and autonomic deficits [2]. Chronic exposure to hyperglycemia induces oxidative stress, which is recognized as a major contributing factor to the development of diabetic neuropathy [3]. The accumulation of free radicals subsequently leads to peripheral nerve dysfunction [4], thereby reducing sensory function and causing severe pain, most

commonly in the feet [5]. In addition, diabetic neuropathy disrupts motor balance and impairs overall agility in daily activities [6].

Various commercially available synthetic drugs are widely used to treat diabetic neuropathy; however, they are associated with a range of adverse side effects [7], [8]. For instance, naltrexone has been reported to cause gastrointestinal disturbances [9]. Other drugs, such as duloxetine and gabapentin, are also associated with similar adverse effects [10]. Previous reports further indicate that gabapentin and pregabalin increase the risk of cardiovascular diseases [11]. Therefore, there is an urgent need to identify alternative therapeutic approaches that are effective in managing diabetic neuropathy while minimizing side effects. One promising strategy is to explore the potential of functional foods for the management of this condition.

Among various plant-based natural products used as functional foods for the treatment of diabetic neuropathy, Mentawai taro (*Colocasia esculenta* var. Mentawai) from the family Araceae appears to be a promising candidate. Native to the Mentawai Islands (West Sumatra, Indonesia), the corm of this species has long been consumed as both a staple and supplemental food by the local Mentawai people [12]. Nutritional analyses have shown that the corm of common *C. esculenta* contains 34.6% carbohydrates, 0.52% protein, 0.11% fat, 0.97% ash, and 5.1% dietary fiber [13]. In contrast, the Mentawai taro corm exhibits higher contents of carbohydrates (65%), protein (8.15%), fat (0.18%), and dietary fiber (22.2%), but a lower ash content (0.23%) [14]. Plants within the Colocasia genus are also known to contain various bioactive compounds, including digalactosyldiacylglycerols, polyphenols, and tarin [13] suggesting their potential role in counteracting neurological disorders, including neuropathy. These compounds are proposed to exert complementary antioxidant, anti-inflammatory, and neuroprotective effects that may contribute to neuropathy mitigation [13]. previous study has demonstrated the neuroprotective effects of Mentawai taro corm flour against high-fat diet–induced neurodegeneration in mice [14]. However, to date, it remains unclear whether Mentawai taro corm, when formulated as a functional food, can effectively manage diabetes mellitus–associated comorbidities, particularly diabetic neuropathy.

Mentawai taro corm is primarily composed of starch and dietary fiber, which can be separated through specific extraction processes [14]. In the absence of extraction, simple milling of the corm yields whole flour. The present study aims to evaluate the efficacy of Mentawai taro corm administered in the form of whole flour, fiber extract, and starch extract in ameliorating diabetic neuropathy. The findings of this study are expected to provide insight into the most effective form of Mentawai taro corm preparation as a functional food candidate for diabetic patients. We hypothesize that dietary administration of Mentawai taro corm as whole flour, fiber extract, or starch extract can alleviate diabetic neuropathy, with differential efficacy among these preparations.

Materials and Methods

Sample Collection and Preparation

Mentawai taro corms were obtained from Boshua Village, Sipora Island, Mentawai Islands, West Sumatra, Indonesia. Taxonomic validation of the species was performed by Dr. Nurainas at the Herbarium, Department of Biology, Faculty of Mathematics and Natural Sciences, Universitas Andalas. The corms were washed, peeled, and ground under laboratory conditions. The ground material was then divided into two portions. One portion was immediately steamed at 100 °C for 30 min and subsequently oven-dried at 70 °C for 14 h until a constant weight was achieved, resulting in whole flour (WFL).

The second portion was soaked in distilled water at a ratio of 1:10 (w/v) at room temperature overnight to allow separation of starch (sediment) and fiber (supernatant). Both the starch (STR) and fiber (FIB) fractions were collected, steamed at 100 °C for 30 min, and oven-dried at 70 °C for 14 h to obtain powdered forms. All three preparations (WFL, STR, and FIB) were stored in sterile, airtight containers until further use. The preparation procedure was conducted according to previously described protocols [14], [15].

Animal models

All animal handling procedures and experimental protocols were approved by the Research Ethics Committee of Andalas University (Approval No. Bio-08-24-UA/2024). Thirty adult male mice (8 weeks old; body weight 20–24 g) were obtained from Balai Veteriner Baso, West Sumatra, Indonesia. The animals were acclimatized for 10 days in a controlled animal facility under regulated temperature (26.0–26.5 °C), relative humidity (67.0–67.8%), and a 12 h light/12 h dark illumination cycle. Following acclimatization, the mice were randomly assigned to experimental treatments. Twenty-four mice received a single intraperitoneal injection of alloxan monohydrate (150 mg/kg body weight) to induce diabetes mellitus [16], whereas six mice were injected with physiological saline and served as the healthy (non-diabetic) control group. Three days after injection, blood glucose levels were measured using an automatic glucometer (AGM-4000) via tail-tip blood sampling. Mice with fasting blood glucose levels ≥ 250 mg/dL were classified as diabetic. In this study, all alloxan-treated mice (n=24) successfully developed diabetes, whereas saline-injected mice (n=6) maintained normal blood glucose levels (100–150 mg/dL).

Experimental Diet Treatments

For experimental treatments, the mice were randomly allocated into five groups as follows: Group 1 (Control): non-diabetic mice; Group 2 (Diabetes): diabetic mice fed a standard chow diet (Ratio®, a commercial rodent diet; Citra Inna Feed); Group 3 (Diabetes + WFL): diabetic mice fed a standard diet supplemented with 15% Mentawai taro corm whole flour; Group 4 (Diabetes + FIB): diabetic mice fed a standard diet supplemented with 15% Mentawai taro corm fiber; Group 5 (Diabetes + STR): diabetic mice fed a standard diet supplemented with 15% Mentawai taro corm starch. The dietary supplementation level (15%) was selected based on a two-week preliminary study using Mentawai taro corm fiber, which demonstrated a significant reduction in blood glucose levels in alloxan-induced diabetic mice.

Blood Glucose Measurements

Blood glucose levels were measured at two time points: at the beginning of the dietary intervention and at the end of the experimental period. Blood samples were collected from the tail tips, and glucose concentrations were determined using a glucometer (AGM-400).

Assessment of Sensory Function by Hot Plate Test

At the end of the treatment period, sensory function was evaluated using the hot plate test in accordance with a standard protocol [17]. Each mouse was placed on a hot plate maintained at a constant temperature of 50 °C. The animals' initial responses to thermal stimulation of the paws were recorded using a video camera. Latency time was defined as the duration from placement on the hot plate to the first nociceptive response, including paw licking, jumping, or rapid movement. Latency times were determined through analysis of the recorded videos. All assessments were conducted in a blinded manner by masking the cage labels, ensuring that the observer was unaware of the animals' original group assignments during evaluation.

Assessment of Motor Function by Balance Beam Test

Motor coordination was assessed at the end of the treatment period using the balance beam test, following procedures described previously [18]. Prior to testing, the mice were trained to traverse the beam for three consecutive days. During the test, each mouse was placed at the starting point of the beam and allowed to cross to the end while being recorded using a video camera. The time required to reach the end of the beam was determined through analysis of the recorded videos. All assessments were conducted in a blinded manner by masking the cage labels, ensuring that the observer was unaware of the animals' original group assignments during evaluation.

Measurement of Malondialdehyde (MDA) in Brain Tissue

Upon completion of all behavioral assessments, on day 29, the animals were euthanized by lethal injection of ketamine. A portion of brain tissue (0.3 g) was dissected, immediately transferred into a microcentrifuge tube, and stored at −80 °C. The tissue samples were subsequently homogenized in phosphate-buffered saline (PBS) using a tissue homogenizer and centrifuged at 3,000 rpm for 10 min. The resulting supernatant was collected for the determination of malondialdehyde (MDA) levels. MDA concentrations were quantified using a lipid peroxidation assay kit (Sigma-Aldrich), and absorbance was measured using a UV–Vis spectrophotometer (Thermo Fisher Scientific). MDA levels were expressed as nmol/g tissue. All procedures were performed in accordance with the manufacturer's instructions provided with the assay kit.

Histopathological Examination of the Cerebellum

Histological examination was performed following a previously described procedure [14]. Briefly, cerebellar samples were collected and fixed in neutral buffered formalin overnight. The tissues were then dehydrated through a graded ethanol series, cleared in xylene, and infiltrated with paraffin. Following paraffin embedding, the tissues were trimmed and sectioned at a thickness of 5 µm using a rotary microtome (Leica). The sections were stained with hematoxylin and eosin using a commercial staining kit (Sigma-Aldrich). Representative tissue sections exhibiting clear staining and minimal artifacts were examined under a light microscope (Olympus). For each mouse, Purkinje cell counts were performed on four representative slides, with five randomly selected fields of view analyzed per slide.

Statistical Analysis

All quantitative data were tested for normality and homogeneity of variance. Data were then analyzed using one-way analysis of variance (ANOVA), followed by Bonferroni's post hoc test. Statistical significance was set at $P < 0.05$.

Results and Discussion

As shown in Figure 1A, all alloxan-treated mice exhibited significantly elevated blood glucose levels compared with the control group ($P < 0.01$), confirming the successful induction of diabetes mellitus. Subsequent blood glucose measurements at the end of the treatment period (Figure 1B) demonstrated that diabetic mice fed with Mentawai taro corm fiber showed a significant reduction in blood glucose levels compared with diabetic mice receiving the standard diet alone ($P < 0.01$). Notably, blood glucose levels in the fiber-treated group were not significantly different from those of the control group ($P > 0.05$). In contrast, mice fed with Mentawai taro corm whole flour or starch maintained elevated blood glucose levels comparable to those observed in the untreated diabetic group ($P > 0.05$).

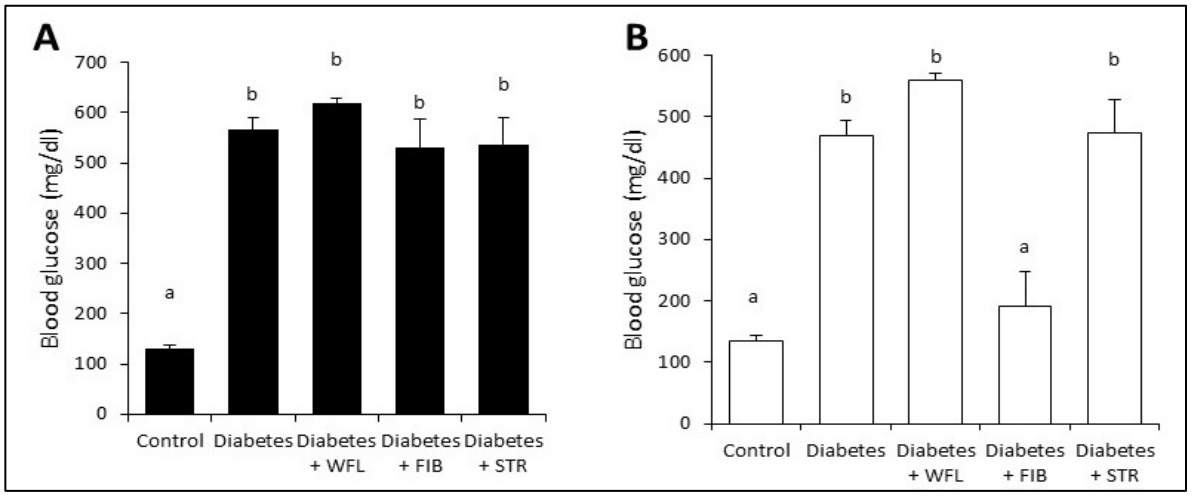

Figure 1. Effect of Mentawai taro corm on blood glucose levels in mice. (A) Blood glucose levels before treatment, (B) Blood glucose levels after 28-day treatment. WFL (whole flour), FIB (Fiber), STR (Starch). Lower case letters above the bars indicate statistical significance ($P < 0.05$).

Assessment of sensory function using the hot plate test at the end of the treatment period (Figure 2) showed that diabetic mice exhibited significantly prolonged response latencies to thermal stimulation compared with the control group ($P < 0.05$), indicating the presence of neuropathy. In contrast, diabetic mice fed with Mentawai taro corm fiber demonstrated a reduction in response latency that was comparable to that of the control group ($P > 0.05$). However, this improvement was not statistically significant when compared with the other diabetic treatment groups.

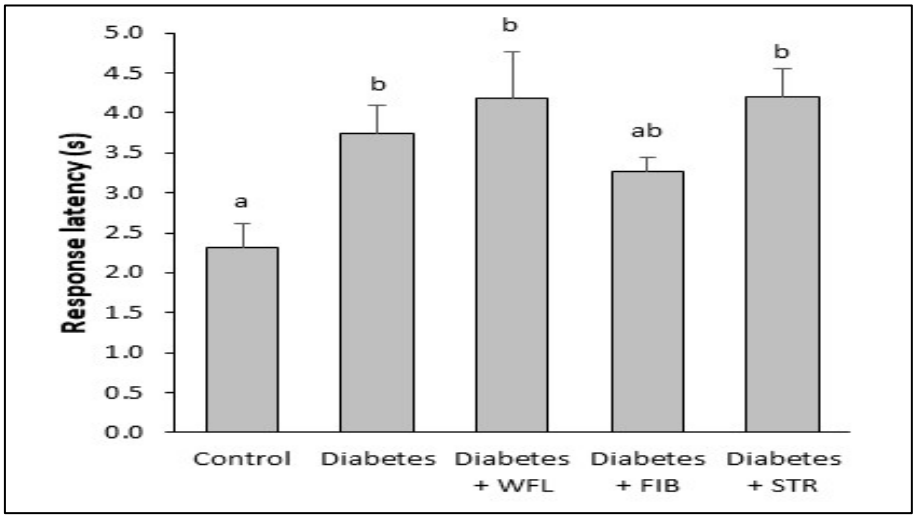

Figure 2. Effect of Mentawai taro corm on paw sensitivity of mice against heat stimulus in hot plate test. Response latency indicates the time needed to the initial response of mice paw upon heat stimulus. WFL (whole flour), FIB (Fiber), STR (Starch). Lower case letters above the bars indicate statistical significance ($P < 0.05$).

In addition to sensory function assessment, motor control was evaluated using the balance beam test. As shown in Figure 3, diabetic mice fed the standard diet exhibited a significantly longer beam traversal time compared with the control group ($P < 0.01$). Similarly, diabetic mice fed with Mentawai taro corm whole flour also showed a marked increase in completion time. In contrast, diabetic mice

fed with Mentawai taro corm fiber or starch demonstrated shorter traversal times that were comparable to those of the control group ($P > 0.05$).

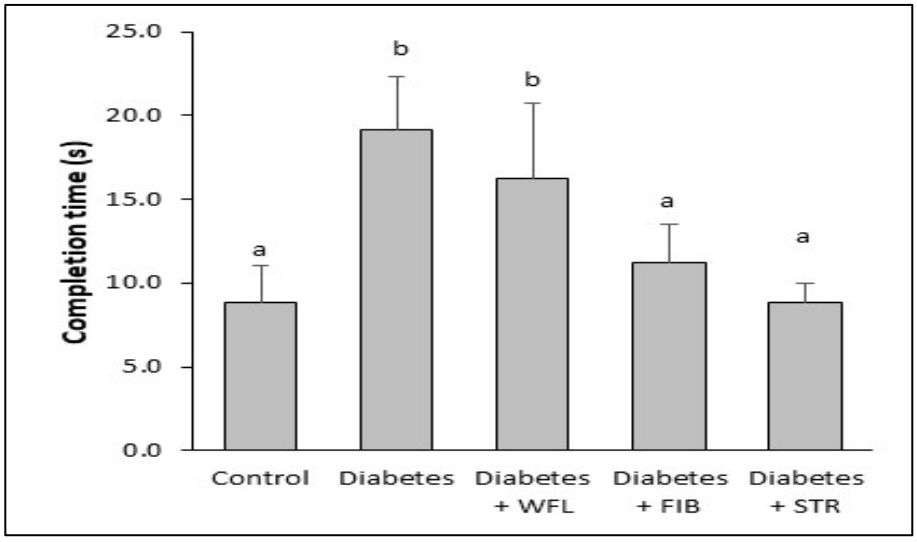

Figure 3. Effect of Mentawai taro corm on motoric control of mice based on balance beam test. Completion time indicates the time needed to cross the beam. WFL (whole flour), FIB (Fiber), STR (Starch). Lower case letters above the bars indicate statistical significance ($P < 0.05$).

To evaluate the ability of Mentawai taro corm to counteract oxidative stress in the brain, malondialdehyde (MDA) levels were measured at the end of the treatment period. As shown in Figure 4, diabetic mice exhibited significantly elevated brain MDA levels compared with the control group ($P < 0.05$). Similarly, increased MDA levels were observed in diabetic mice fed with Mentawai taro corm whole flour or starch. In contrast, diabetic mice fed with Mentawai taro corm fiber showed a significant reduction in MDA levels, which were comparable to those of the control group ($P > 0.05$).

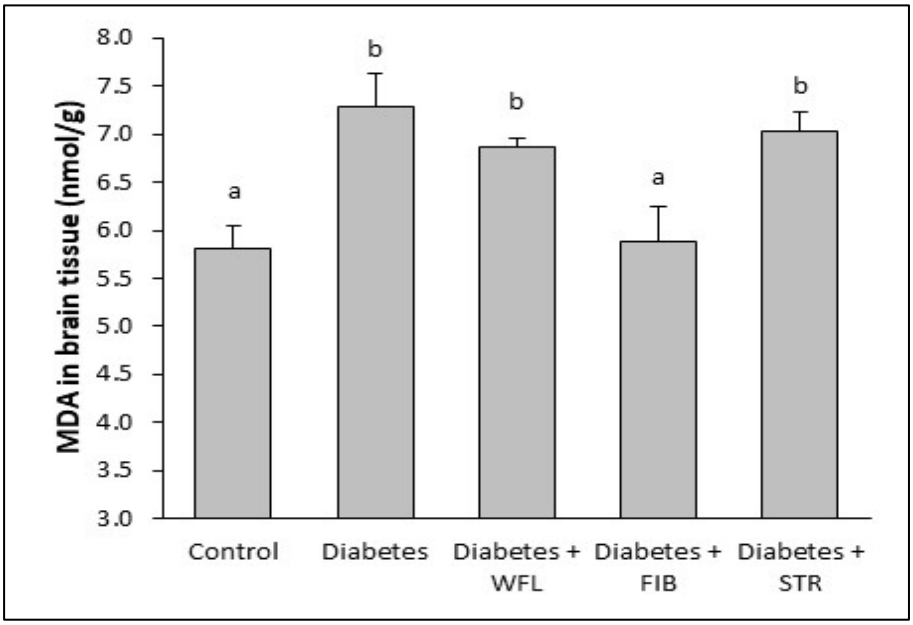

Figure 4. Effect of Mentawai taro corm on malondialdehyde (MDA) level in brain tissue of mice. WFL (whole flour), FIB (Fiber), STR (Starch). Lower case letters above the bars indicate statistical significance ($P < 0.05$).

To evaluate the protective effects of Mentawai taro corm on cerebellar integrity, histopathological analyses were performed at the end of the treatment period. As shown in Figure 5, diabetic mice exhibited extensive degeneration of Purkinje cells in the cerebellum compared with the control group. Similar degenerative changes were also observed in diabetic mice fed with Mentawai taro corm whole flour, fiber, or starch. However, the extent of Purkinje cell degeneration was noticeably reduced in mice fed with the fiber preparation compared with the other diabetic groups.

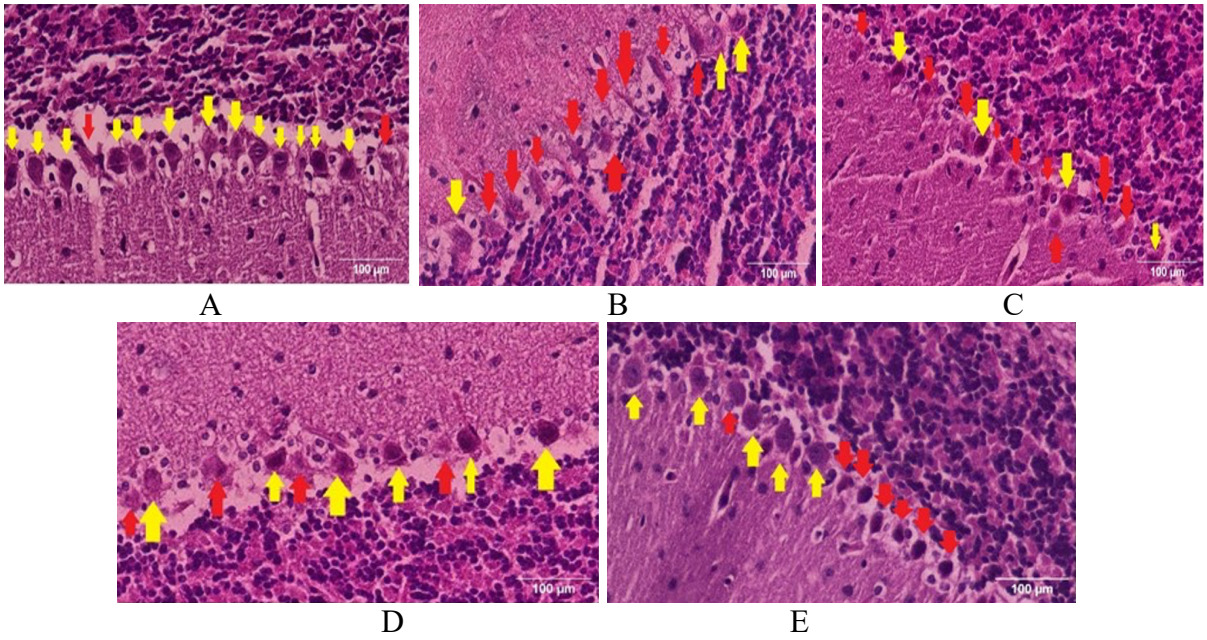

Figure 5. Effect of Mentawai taro corm on Purkinje cells of mice cerebellum. (A) Control, (B) diabetes, (C) Diabetes + whole flour (WFL), (D) Diabetes + fiber (FIB), (E) Diabetes + starch (STR). Yellow arrows indicate normal Purkinje cells, red arrows indicate degenerated Purkinje cells.

Quantitative analysis of Purkinje cells (Figure 6) revealed no significant differences in the total number of Purkinje cells among the experimental groups. Nevertheless, diabetic mice fed the standard diet, as well as those fed with Mentawai taro corm whole flour or starch, showed a significantly higher number of degenerated Purkinje cells compared with the control group ($P < 0.01$). In contrast, diabetic mice fed with Mentawai taro corm fiber exhibited a significant reduction in the number of degenerated Purkinje cells compared with all other diabetic groups ($P < 0.05$). Despite this improvement, the number of degenerated Purkinje cells in the fiber-fed group remained significantly higher than that observed in the control group ($P < 0.05$).

This experimental study demonstrated the beneficial effects of Mentawai taro corm fiber extract against diabetic neuropathy in an alloxan-induced mouse model. After 28 days of dietary fiber supplementation, diabetic mice exhibited improved blood glucose regulation, a positive trend toward enhanced sensory responses, and improved motor control. Moreover, Mentawai taro corm fiber markedly reduced oxidative stress in brain tissue and decreased the number of degenerated Purkinje cells in the cerebellum. In contrast, dietary supplementation with Mentawai taro corm whole flour or starch exerted minimal protective effects compared with the fiber preparation.

Previous studies have demonstrated that chronic exposure to excessive blood

glucose levels promotes oxidative stress in various tissues, including the central and peripheral nervous systems [19], [20]. Increased oxidative stress leads to the accumulation of free radicals, which can damage neural structures, including those involved in sensory function [20]. Other reports have indicated that neurons responsible for motor control, including cerebellar Purkinje cells, are also susceptible to damage under conditions of elevated oxidative stress [21]. Collectively, these pathological alterations result in impairments of sensory and motor functions, which are key hallmarks of diabetic neuropathy [22]. Purkinje cell degeneration has been shown to be closely associated with deficits in sensory integration and motor control, as these neurons provide the sole inhibitory output of the cerebellar cortex [17]. Loss of Purkinje cells can disrupt cerebellar signaling, leading to motor impairments such as ataxia, poor coordination, and abnormal gait, as well as altered processing of proprioceptive and somatosensory inputs essential for sensorimotor integration [6] Consistently, previous studies have reported that diabetic mice with elevated oxidative stress levels exhibit delayed responses to thermal stimulation, indicative of neuropathy [23]. In the present study, diabetic mice fed with Mentawai taro corm fiber exhibited a positive trend toward improved sensory responses and enhanced motor control, accompanied by a substantial reduction in Purkinje cell degeneration and decreased malondialdehyde (MDA) levels in brain tissue. MDA is a well-established biomarker of oxidative stress, as it represents a stable product of lipid peroxidation induced by reactive oxygen species [19]. Taken together, these findings suggest that Mentawai taro corm fiber may ameliorate diabetic neuropathy by mitigating oxidative stress in the brain.

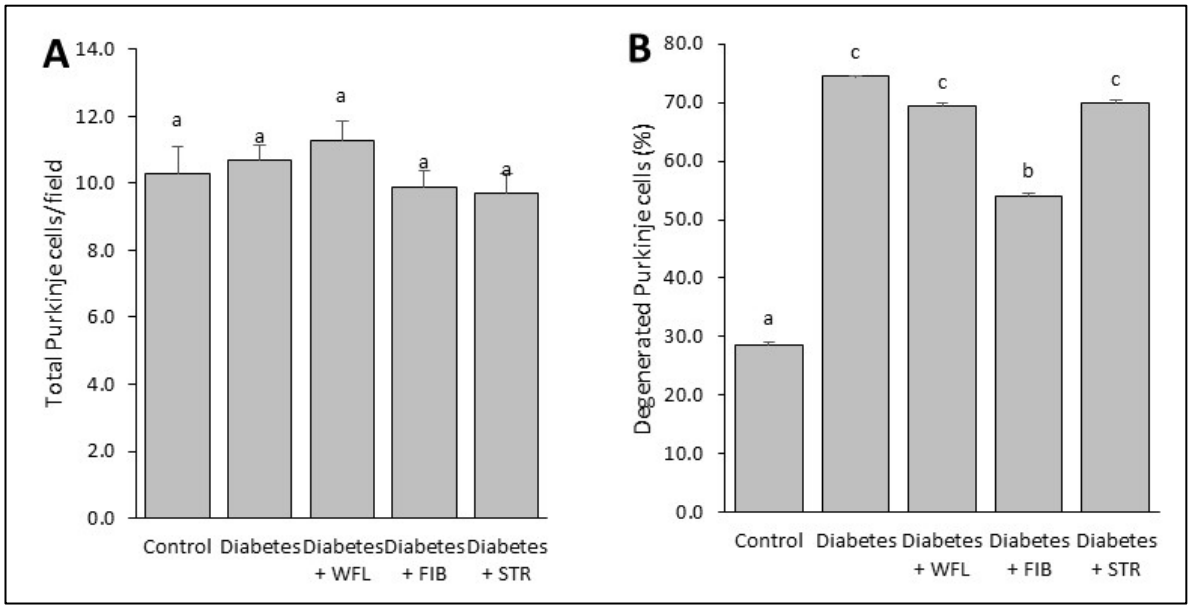

Figure 6. Effect of Mentawai taro corm on number of Purkinje cells of mice cerebellum. (A) Total number of Purkinje cells per each field view, (B) Percentage of degenerated Purkinje cells. WFL (whole flour), FIB (Fiber), STR (Starch). Lower case letters above the bars indicate statistical significance ($P < 0.05$).

Dietary fiber can exert health benefits by enhancing the production of short-chain fatty acids (SCFAs) in the intestine [24]. SCFAs are fermentation products of the gut microbiota, with soluble fiber serving as a primary substrate for microbial fermentation [25]. Our previous study demonstrated that dietary supplementation

with jicama fiber promotes the growth of SCFA-producing bacteria in mice [26]. Other studies have shown that increased levels of SCFAs, including butyrate, propionate, and acetate, are closely associated with improved blood glucose homeostasis [27] and enhanced neurological health [28]. The beneficial effects of SCFAs are thought to be mediated through multiple mechanisms, including attenuation of oxidative stress [29], enhancement of insulin sensitivity [30], and promotion of cellular proliferation and tissue homeostasis [31]. Although SCFA levels were not measured in the present study, it is plausible that Mentawai taro corm fiber may alleviate diabetic neuropathy, at least in part, by increasing intestinal SCFA production. Further studies are warranted to determine whether Mentawai taro corm fiber supplementation directly enhances SCFA production and to elucidate its contribution to the observed neuroprotective effects.

Among all tested forms of Mentawai taro preparations, the fiber extract was the most effective in managing diabetic neuropathy. The lower fiber content in the whole flour and starch preparations, compared with the fiber extract, may account for their reduced efficacy. The higher starch content in the whole flour and starch extracts may contribute to elevated blood glucose levels and may be less effective in promoting the growth of SCFA-producing gut microbiota. Consequently, SCFA levels in mice fed with whole flour or starch preparations may be lower than those in mice receiving the fiber extract. Therefore, in terms of neuropathy management, the fiber extract appears to be the most favorable preparation, whereas the whole flour and starch preparations confer comparatively limited benefits.

The translational relevance of this study lies in the identification of Mentawai taro corm fiber as a promising plant-based functional food ingredient with potential applications beyond preclinical settings for the management of diabetic neuropathy. The observed improvements in glycemic control, sensory responses, motor coordination, and neurohistological outcomes in diabetic mice suggest that fiber-rich dietary interventions may target key pathogenic mechanisms of neuropathy, particularly hyperglycemia-induced oxidative stress and neuronal damage. Given that Mentawai taro is traditionally consumed and locally accessible, its fiber extract holds potential for development as a dietary component or functional food formulation for future applications. Nevertheless, well-designed follow-up studies, including clinical trials, are required to confirm its safety, efficacy, and optimal dosage in translational contexts.

Several limitations of this study should be acknowledged. First, only a single dietary dose (15%) of Mentawai taro corm preparations (fiber, whole flour, and starch) was evaluated; therefore, further studies are required to determine whether different doses could elicit comparable or enhanced protective effects against diabetic neuropathy. Second, the treatment duration was relatively short (28 days), and a longer intervention period may yield more pronounced neuroprotective effects. Additionally, analyses of fiber composition (soluble and insoluble fractions) and phytochemical profiles were not conducted.

This study is also limited by the absence of detailed macronutrient composition data for the whole flour, fiber extract, and starch extract, which differ substantially and may have influenced total energy and nutrient intake. Moreover, the lack of food intake and body weight measurements restricts a comprehensive evaluation of whether the observed effects were driven by dietary composition or differences in consumption. With respect to disease modeling, neuropathy was assessed at a single endpoint without baseline measurements, limiting the ability to evaluate disease progression. Finally, the molecular mechanisms underlying the protective effects of Mentawai taro corm fiber were not investigated. Future studies

addressing these limitations are warranted to strengthen the translational relevance of these findings.

Conclusion

Our findings demonstrate that dietary supplementation with Mentawai taro corm fiber at a dose of 15% significantly improves diabetic neuropathy in alloxan-induced mice. These improvements were reflected by a positive trend in sensory response (25% increase), enhanced motor balance (41.6% increase), reduced malondialdehyde (MDA) levels in brain tissue (19.34% reduction), and a significant decrease in the number of degenerated Purkinje cells in the cerebellum (27.3% reduction). In contrast, supplementation with Mentawai taro corm whole flour or starch did not confer comparable beneficial effects. Collectively, these findings suggest that Mentawai taro corm fiber holds promise as a potential functional food candidate for the management of diabetic neuropathy.

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Conflict of interest

The authors declare that no conflict of interest in this study.

References

[1] K. Yang, X. Yang, C. Jin, S. Ding, T. Liu, B. Ma, H. Sun, J. Zhang, Y. Li, "Global burden of type 1 diabetes in adults aged 65 years and older, 1990-2019: population based study," *BMJ*, vol. 385, p. e078432, 2024, doi: 10.1136/bmj-2023-078432.

[2] E. L. Feldman, B.C. Callaghan, R. Pop-Busui, D.W. Zochodne, D.E. Wright, D.L. Bennett, V. Brill, J.W. Russell, V. Viswanathan, "Diabetic neuropathy," *Nat Rev Dis Primers*, vol. 5, no. 1, p. 41, 2019, doi: 10.1038/s41572-019-0092-1.

[3] S. Dewanjee, S. Das, A. K. Das, N. Bhattacharjee, A. Dihingia, T. K. Dua, J. Kalita, P. Manna, "Molecular mechanism of diabetic neuropathy and its pharmacotherapeutic targets," 2018. doi: 10.1016/j.ejphar.2018.06.034.

[4] D. M. Teleanu, A. G. Niculescu, I. I. Lungu, C. I. Radu, O. Vladâcenco, E. Roza, B. Costăchescu, A. M. Grumezescu, R. I. Teleanu, "An Overview of Oxidative Stress, Neuroinflammation and Neurodegenerative Diseases," 2022. doi: 10.3390/ijms23115938.

[5] R. Galiero, A. Caturano, E. Vetrano, D. Beccia, C. Brin, M. Alfano, J. Di Salvo, R. Epifani, A. Piacevole, G. Tagliaferri, M. Rocco, I. Iadicicco, G. Docimo, L. Rinaldi, C. Sardu, T. Salvatore, R. Marfella, F. C. Sasso, "Peripheral Neuropathy in Diabetes Mellitus: Pathogenetic Mechanisms and Diagnostic Options," 2023. doi: 10.3390/ijms24043554.

[6] N. D. Reeves, G. Orlando, and S. J. Brown, "Sensory-motor mechanisms increasing falls risk in diabetic peripheral neuropathy," 2021. doi: 10.3390/medicina57050457.

[7] M. M. Asrar, S. Kumari, B. C. Sekhar, A. Bhansali, and D. Bansal, "Relative efficacy and safety of pharmacotherapeutic interventions for diabetic peripheral neuropathy: A systematic review and bayesian network meta-analysis," *Pain Physician*, vol. 24, no. 1, 2021, doi: 10.36076/ppj.2021.24.e1-e14.

[8] S. Derry, R. F. Bell, S. Straube, P. J. Wiffen, D. Aldington, and R. A. Moore, "Pregabalin for neuropathic pain in adults," *Cochrane Database Syst Rev*, vol. 1, no. 1, p. CD007076, 2019, doi: 10.1002/14651858.CD007076.pub3.

[9] A. Srinivasan, P. Dutta, D. Bansal, A. Chakrabarti, A. K. Bhansali, and D. Hota, "Efficacy and safety of low-dose naltrexone in painful diabetic neuropathy: A randomized, double-blind, active-control, crossover clinical trial," *J Diabetes*, vol. 13, no. 10, 2021, doi: 10.1111/1753-0407.13202.

[10] S. Khasbage, R. Shukla, P. Sharma, and S. Singh, "A randomized control trial of duloxetine and gabapentin in painful diabetic neuropathy," *J Diabetes*, vol. 13, no. 7, 2021, doi: 10.1111/1753-0407.13148.

[11] Y. Pan, P. B. Davis, D. C. Kaebler, R. P. Blankfield, and R. Xu, "Cardiovascular risk of gabapentin and pregabalin in patients with diabetic neuropathy," *Cardiovasc Diabetol*, vol. 21, no. 1, 2022, doi: 10.1186/s12933-022-01610-9.

[12] Z. Syarif, N. Akhir, and B. Satria, "Identification of plant morphology of taro as a potential source of carbohydrates," *Int J Adv Sci Eng Inf Technol*, vol. 7, no. 2, 2017, doi: 10.18517/ijaseit.7.2.1323.

[13] P. R. Pereira, É. B. de A. Mattos, A. C. N. T. F. Corrêa, M. A. Vericimo, and V. M. F. Paschoalin, "Anticancer and immunomodulatory benefits of taro (Colocasia esculenta) corms, an underexploited tuber crop," 2021. doi: 10.3390/ijms22010265.

[14] P. Santoso, F. Ramadhan Marviano, and R. Rahayu, "Neuroprotective Effect of *Colocasia esculenta* Var. Mentawai Corm Flour High-Fat Diet Fed Mice," *Journal of Cellular Neuroscience and Oxidative Stress*, vol. 16, no. 1, pp. 1173–1182, 2024, doi: 10.37212/jcnos.1470198.

[15] A. M. P. Dewi and E. F. Tethool, "Modification of Starch with Amylosucrase: Methods, Physicochemical Properties and Health Implications," *Biota : Jurnal Ilmiah Ilmu-Ilmu Hayati*, vol. 9, no. 2, pp. 210–218, 2024, doi: 10.24002/biota.v9i2.9095.

[16] A. Murdiati, H. Wuryastuty, Y. Marsono, and E. Harmayani, "Penurunan Glukosa dan Perubahan Profil Lipida Serum Tikus Sprague-Dawley Hiperglikemia-Hiperkolesterolemia Akibat Asupan Sorbitol-Oleat Poliester (SOPE)," *Biota : Jurnal Ilmiah Ilmu-Ilmu Hayati*, 2019, doi: 10.24002/biota.v14i3.2576.

[17] M. Ostovar, A. Akbari, M. H. Anbardar, A. Iraji, M. Salmanpour, S. Hafez Ghoran, M. Heydari, M. Shams, "Effects of Citrullus colocynthis L. in a rat model of diabetic neuropathy," *J Integr Med*, vol. 18, no. 1, 2020, doi: 10.1016/j.joim.2019.12.002.

[18] D. Bartikofsky, M. J. Hertz, D. S. Bauer, R. Altschuler, W. M. King, and C. E. Stewart, "Balance beam crossing times are slower after noise exposure in rats," *Front Integr Neurosci*, vol. 17, 2023, doi: 10.3389/fnint.2023.1196477.

[19] M. A. Babizhayev, I. A. Strokov, V. V. Nosikov, E. L. Savel'yeva, V. F. Sitnikov, Y. E. Yegorov, V. Z. Lankin, "The Role of Oxidative Stress in Diabetic Neuropathy: Generation of Free Radical Species in the Glycation Reaction and Gene Polymorphisms Encoding Antioxidant Enzymes to Genetic Susceptibility to Diabetic Neuropathy in Population of Type I Diabetic Patients," *Cell Biochem Biophys*, vol. 71, no. 3, 2015, doi: 10.1007/s12013-014-0365-y.

[20] K. Luc, A. Schramm-Luc, T. J. Guzik, and T. P. Mikolajczyk, "Oxidative stress and inflammatory markers in prediabetes and diabetes," 2019. doi: 10.26402/jpp.2019.6.01.

[21] Q. Lin, K. Li, Y. Chen, J. Xie, C. Wu, C. Cui, B. Deng, "Oxidative Stress in Diabetic Peripheral Neuropathy: Pathway and Mechanism-Based Treatment," 2023. doi: 10.1007/s12035-023-03342-7.

[22] G. Sloan, D. Selvarajah, and S. Tesfaye, "Pathogenesis, diagnosis and clinical management of diabetic sensorimotor peripheral neuropathy," 2021. doi: 10.1038/s41574-021-00496-z.

[23] P. Santoso, A. S. Simatupang, A. Fajria, R. Rahayu, and R. Jannatan, "Andaliman (Zanthoxylum acanthopodium DC.) fruit ethanolic extract exerts attenuative effect on hyperglycemia, sensory and motoric function's disorders in alloxan-induced diabetic mice," *J Adv Vet Anim Res*, vol. 10, no. 4, pp. 608–619, 2023, doi: 10.5455/javar.2023.j716.

[24] C. Martin-Gallausiaux, L. Marinelli, H. M. Blottière, P. Larraufie, and N. Lapaque, "SCFA: mechanisms and functional importance in the gut," *Proceedings of the Nutrition Society*, vol. 80, no. 1, 2021, doi: 10.1017/s0029665120006916.

[25] P. Yu, M. Fang, Y. Li, L. Cai, R. Han, W. Sun, X. Jiang, L. Chen, J. Du, Z. Zhu, "Interactions between Gut Microbiota and Natural Bioactive Polysaccharides in Metabolic Diseases: Review," *Nutrients*, vol. 16, no. 17, pp. 1–45, 2024, doi: 10.3390/nu16172838.

[26] P. Santoso, R. Maliza, R. Rahayu, Y. Astrina, F. Syukri, and S. Maharani, "Extracted yam bean (*Pachyrhizus erosus* (L.) Urb.) fiber counteracts adiposity, insulin resistance, and inflammation while modulating gut microbiota composition in mice fed with a high-fat diet," *Res Pharm Sci*, vol. 17, no. 5, 2022, doi: 10.4103/1735-5362.355213.

[27] L. Zhao, F. Zhang, X. Ding, G. Wu, Y. Y. Lam, X. Wang, H. Fu, X. Xue, C. Lu, J. Ma, L. Yu, C. Xu, Z. Ren, Y. Xu, S. Xu, H. Shen, X. Zhu, Y. Shi, Q. Shen, W. Dong, R. Liu, Y. Ling, Y. Zeng, X. Wang, Q. Zhang, J. Wang, L. Wang, Y. Wu, B. Zeng, H. Wei, M. Zhang, Y. Peng, C. Zhang, "Gut bacteria selectively promoted by dietary fibers alleviate type 2 diabetes," *Science (1979)*, vol. 359, no. 6380, 2018, doi: 10.1126/science.aao5774.

[28] M. Van de Wouw, M. Boehme, J. M. Lyte, N. Wiley, C. Strain, O. O'Sullivan, G. Clarke, C. Stanton, T. G. Dinan, J. F. Cryan, "Short-chain fatty acids: microbial metabolites that alleviate stress-induced brain–gut axis alterations," *Journal of Physiology*, vol. 596, no. 20, 2018, doi: 10.1113/JP276431.

[29] W. Huang, H. L. Guo, X. Deng, T. T. Zhu, J. F. Xiong, Y. H. Xu, Y. Xu., "Short-Chain Fatty Acids Inhibit Oxidative Stress and Inflammation in Mesangial Cells Induced by High Glucose and Lipopolysaccharide," *Experimental and Clinical Endocrinology and Diabetes*, vol. 125, no. 2, 2017, doi: 10.1055/s-0042-121493.

[30] N. H. T. Pham, M. V. Joglekar, W. K. M. Wong, N. T. Nassif, A. M. Simpson, and A. A. Hardikar, "Short-chain fatty acids and insulin sensitivity: A systematic review and meta-Analysis," 2024. doi: 10.1093/nutrit/nuad042.

[31] L. L. Yang, V. Millischer, S. Rodin, D. F. MacFabe, J. C. Villaescusa, and C. Lavebratt, "Enteric short-chain fatty acids promote proliferation of human neural progenitor cells," *J Neurochem*, vol. 154, no. 6, 2020, doi: 10.1111/jnc.14928.