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Sweet Potato (*Ipomoea batatas* (l.) Lam.) Tuber Extract Preserves Mitochondrial Ca^{2+} Homeostasis, Limits Oxidative Stress and Induces Spasmolytic Effects: Molecular Evidence for Functional Nutrition

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ABSTRACT

Sweet potato (*Ipomoea batatas* (L.) Lam.) is increasingly recognized as a functional food rich in bioactive compounds; however, its molecular effects on mitochondrial function and smooth muscle physiology remain insufficiently characterized. In this study, we investigated the impact of sweet potato tuber extract on mitochondrial Ca^{2+} homeostasis, oxidative stress parameters, and smooth muscle contractility using in vitro experimental models. Flavonoid profiling by HPLC confirmed the presence of quercetin, rutin, and isorhamnetin as major constituents. Treatment with the extract significantly enhanced mitochondrial membrane potential ($\Delta\Psi m$), reaching up to 126% of control values, while lipid peroxidation, assessed by malondialdehyde (MDA) levels, was reduced by approximately 40% compared with untreated mitochondria. The extract markedly attenuated Ca^{2+}-induced mitochondrial swelling, indicating inhibition of mitochondrial permeability transition pore (mPTP) opening. In isolated intestinal smooth muscle preparations, the extract produced a clear dose-dependent spasmolytic effect, achieving 55–65% relaxation at the highest concentration, with kinetics slower than verapamil but comparable to papaverine, consistent with a multi-target mechanism. Collectively, these findings demonstrate that sweet potato-derived bioactives modulate mitochondrial stability, Ca^{2+} signaling, and smooth muscle excitability, providing strong mechanistic evidence for Ipomoea batatas as a functional food with clinically relevant antioxidant and spasmolytic potential in molecular nutrition and preventive health contexts.

Keywords: *Ipomoea batatas,* Functional food, Mitochondria, Ca^{2+} signaling, Flavonoids, Oxidative stress, Smooth muscle.

INTRODUCTION

Diet-derived bioactive compounds are increasingly recognized as critical regulators of cellular homeostasis, exerting their effects through modulation of mitochondrial metabolism, redox balance, and intracellular signaling networks. Mitochondria serve as the central hub of cellular energy production, integrating nutrient-derived substrates with oxidative phosphorylation and calcium (Ca^{2+})-dependent regulatory mechanisms. In this context, mitochondrial Ca^{2+} homeostasis represents a key molecular link between energy metabolism, reactive oxygen species (ROS) generation, and functional responses of excitable and non-excitable tissues, including smooth muscle (Bernardi et al. 2015a; Giorgi et al. 2018).

Physiological Ca^{2+} uptake by mitochondria stimulates ATP synthesis by activating dehydrogenases of the tricarboxylic acid cycle. However, excessive Ca^{2+} influx leads to mitochondrial dysfunction through depolarization of the inner mitochondrial membrane, activation of the mitochondrial permeability transition pore (mPTP), and subsequent release of pro-oxidant and pro-apoptotic factors

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(Halestrap and Richardson 2015). Persistent mPTP opening results in impaired oxidative phosphorylation, enhanced lipid peroxidation, and disruption of cellular bioenergetics, processes that are particularly detrimental to tissues with high metabolic and contractile demands such as smooth muscle (Duchen et al. 2008; Baines 2010; Ergasheva et al. 2021).

Smooth muscle contractility is tightly regulated by intracellular Ca^{2+} signaling, membrane potential dynamics, and the phosphorylation state of myosin light chains. Dysregulation of Ca^{2+} homeostasis contributes to hypercontractility, spasm formation, and functional disorders of the gastrointestinal and vascular systems (Sanders et al. 2012). Importantly, mitochondrial Ca^{2+} buffering capacity and redox status directly influence cytosolic Ca^{2+} oscillations and Ca^{2+}-dependent signaling cascades, highlighting mitochondria as an essential modulator of smooth muscle physiology (Rizzuto et al. 2012).

In recent years, growing attention has been directed toward dietary bioactive compounds capable of modulating mitochondrial function and Ca^{2+} signaling in a multi-target manner. Among these, plant-derived flavonoids have emerged as potent modulators of redox balance, ion channel activity, and mitochondrial integrity. Flavonoids such as quercetin, rutin, and isorhamnetin have been shown to scavenge ROS, stabilize mitochondrial membranes, and partially inhibit voltage-dependent Ca^{2+} channels, thereby exerting cytoprotective and spasmolytic effects (Panche et al. 2016; Salehi et al. 2019).

Sweet potato (*Ipomoea batatas* L.) is a globally important root crop and a rich source of complex carbohydrates, dietary fiber, vitamins (A, C, and B group), essential minerals, and diverse phenolic compounds. Numerous studies have demonstrated the antioxidant, anti-inflammatory, and metabolic regulatory properties of sweet potato extracts and isolated constituents (Alam et al. 2016; Wang et al. 2016; Ruziev et al. 2024). In particular, the high flavonoid content of sweet potato tubers has been associated with enhanced antioxidant capacity and protection against oxidative stress-induced cellular damage.

Despite extensive nutritional characterization, the molecular mechanisms underlying the physiological effects of sweet potato bioactives remain insufficiently explored. Specifically, limited information is available regarding their influence on mitochondrial Ca^{2+} handling, mPTP regulation, and smooth muscle contractility at the cellular and subcellular levels. Understanding these mechanisms is essential for substantiating the classification of sweet potato as a functional food with defined molecular targets rather than merely a source of dietary antioxidants.

This study aimed to determine the molecular and physiological effects of *Ipomoea batatas* root extract and its flavonoid-rich fraction on mitochondrial function and smooth muscle activity, and to evaluate the potential of bioactive substances to modulate Ca^{2+} homeostasis, mitochondrial stability, and contractile responses by combining mitochondrial bioenergetics assays with oxidative stress markers and in vitro smooth muscle contraction models.

MATERIALS AND METHODS

Preparation and characterization of sweet potato extract

Fresh tubers of sweet potato (*Ipomoea batatas* (L.) Lam.) were thoroughly washed, peeled, sliced, and air-dried at 40 °C to constant weight. The dried material was milled into a fine powder using a laboratory grinder. Extraction was performed using a hydroethanolic solvent system (70% ethanol, v/v) at a solid-to-solvent ratio of 1:10 (w/v). The mixture was subjected to continuous stirring at room temperature for 24 h, followed by filtration and solvent removal under reduced pressure using a rotary evaporator at 40 °C. The resulting extract was lyophilized and stored at −20 °C until further analysis.

The flavonoid profile of the extract was characterized by high-performance liquid chromatography (HPLC). Quantification of quercetin, rutin, and isorhamnetin was carried out using a reverse-phase C18 column under gradient elution conditions, with detection at 360 nm. Identification and quantification were performed by comparison with authentic standards, confirming a flavonoid-rich composition consistent with functional food quality criteria (Panche et al. 2016; Salehi et al. 2019).

Experimental animals and ethical considerations

Adult male Wistar rats (180–200 g) were obtained from a certified laboratory animal facility and housed under standard conditions (22 ± 2°C, 12 h light/dark cycle) with free access to food and water. All experimental procedures were conducted in accordance with internationally accepted guidelines for the care and use of laboratory animals and approved by the institutional ethics committee.

Isolation of rat liver mitochondria

Rat liver mitochondria were isolated by differential centrifugation as described previously (Giorgi et al. 2018). Briefly, liver tissue was excised, rinsed in ice-cold isolation buffer, and homogenized in a medium containing 250 mM sucrose, 10 mM Tris-HCl, and 1 mM EDTA (pH 7.4). The homogenate was centrifuged at 800 × g for 10 min to remove nuclei and cell debris, followed by centrifugation of the supernatant at 10 000 × g for 15 min to obtain the mitochondrial pellet. The pellet was washed and resuspended in isolation buffer, and protein concentration was determined using the modified Lowry method.

Assessment of mitochondrial functional parameters

Mitochondrial Membrane Potential (ΔΨm).

Mitochondrial membrane potential was assessed spectrophotometrically using a ΔΨm-sensitive dye under energized conditions. Mitochondria (0.3–0.4 mg protein/mL) were incubated in respiration buffer containing sucrose, Tris-HCl, KH_2PO_4, succinate, and rotenone. Changes in membrane potential were expressed as percentage of control values.

Ca^{2+}-Induced mPTP opening (Mitochondrial Swelling Assay)

Opening of the mitochondrial permeability transition pore (mPTP) was evaluated by measuring Ca^{2+}-induced mitochondrial swelling. Swelling kinetics were monitored as a decrease in absorbance at 540 nm at 26°C in a medium containing sucrose, EGTA, succinate, rotenone, oligomycin, and HEPES buffer (pH 7.4). Where indicated, cyclosporin A was used as a specific mPTP inhibitor to confirm pore involvement (Halestrap and Richardson 2015).

Lipid peroxidation assay

Lipid peroxidation was induced using an Fe^{2+}/ascorbate system and quantified by measuring malondialdehyde (MDA) formation via the thiobarbituric acid-reactive substances (TBARS) assay. Absorbance was recorded at 540 nm, and MDA concentration was expressed as nmol/mg protein, providing an index of oxidative damage (Baines 2010).

Smooth muscle contractility experiments

Isolated segments of rat intestinal smooth muscle were prepared and mounted in an organ bath containing Tyrode's solution (NaCl 118 mM, KCl 8 mM, $CaCl_2$ 2.5 mM, KH_2PO_4 4 mM, glucose 9 mM, $NaHCO_3$ 11 mM; pH 7.4) maintained at 36.5 °C and continuously aerated with carbogen (95% O_2, 5% CO_2). Spontaneous contractile activity was recorded using a mechanotransducer system coupled to a digital acquisition interface.

Graded concentrations of sweet potato extract (50–200 mg/kg equivalent) were applied cumulatively. Spasmolytic responses were quantified as percentage reduction in contraction amplitude relative to baseline. Classical spasmolytic agents, verapamil and papaverine, were used as reference controls to compare efficacy and kinetic profiles (Sanders et al. 2012).

Interpretation of extract concentrations

The concentrations of sweet potato tuber extract used in the present study represent direct in vitro experimental concentrations applied to isolated biological systems, including mitochondrial suspensions and organ bath preparations of smooth muscle tissue. These concentrations were selected to evaluate the mechanistic effects of the flavonoid-rich extract on mitochondrial bioenergetics, Ca^{2+} homeostasis, oxidative stress parameters, and smooth muscle contractility under controlled experimental conditions.

It should be noted that the reported concentrations do not represent calculated physiological plasma equivalents following dietary consumption of sweet potato. Instead, they reflect the pharmacologically relevant concentration range commonly employed in in vitro mitochondrial and tissue-level functional assays, allowing characterization of dose-dependent biological responses and molecular mechanisms of action.

Data presentation and statistical analysis

All experiments were performed in triplicate or quadruplicate, and data are presented as mean ± standard error of the mean (SEM) (Rayimova et al. 2024; Akramov et al. 2025). Statistical comparisons between groups were conducted using one-way analysis of variance (ANOVA) followed by appropriate post hoc tests. A value of $P < 0.01$ and $P < 0.05$, was considered statistically significant.

RESULTS

Sweet potato extract enhances mitochondrial stability

Sweet potato tuber extract exerted a pronounced stabilizing effect on isolated rat liver mitochondria. Assessment of mitochondrial membrane potential (ΔΨm) revealed a statistically significant, dose-dependent increase following extract treatment (Fig. 1).

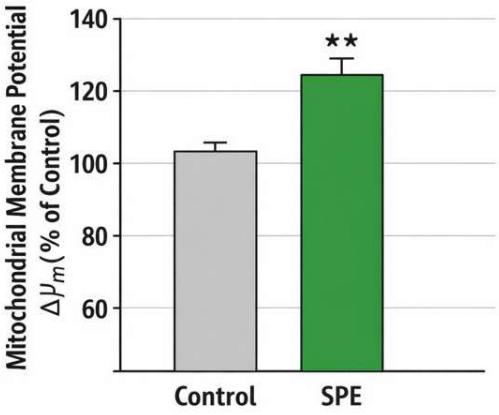

Fig. 1: Effect of sweet potato tuber extract on mitochondrial membrane potential (ΔΨm). Data are expressed as percentage of control (mean ± SEM). **P < 0.01 versus control.

At 50 mg/mL extract concentration, ΔΨm increased to 108 ± 3% of control values, while higher concentrations of 100 mg/mL and 200 mg/mL elevated ΔΨm to 118 ± 4% and 126 ± 5%, respectively (Table 1). These data indicate enhanced proton gradient preservation across the inner mitochondrial membrane, reflecting improved bioenergetic efficiency.

Table 1: Effect of sweet potato extract on mitochondrial membrane potential and lipid peroxidation (n=5)

Treatment	ΔΨm (% of control)	MDA (nmol/mg protein)
Control	100	5.2 ± 0.3
Extract 50 mg/mL	108 ± 3*	4.6 ± 0.3*
Extract 100 mg/mL	118 ± 4*	3.8 ± 0.2**
Extract 200 mg/mL	126 ± 5*	3.1 ± 0.2*

Note. * P < 0.05; ** P < 0.01.

Simultaneously, lipid peroxidation was markedly suppressed. Malondialdehyde (MDA) levels, a key indicator of oxidative membrane damage, decreased significantly in extract-treated mitochondria (Fig. 2). Compared with control mitochondria (5.2 ± 0.3 nmol/mg protein), MDA content was reduced to 3.8 ± 0.2 nmol/mg protein at 100 mg/mL and further to 3.1 ± 0.2 nmol/mg protein at 200 mg/mL extract concentration ($P < 0.01$). Treatment with sweet potato extract (SPE) significantly increased mitochondrial membrane potential compared with the control group.

Collectively, these results demonstrate that sweet potato extract enhances mitochondrial stability by simultaneously reinforcing membrane potential and attenuating oxidative stress. The observed effects are consistent with the antioxidant and membrane-protective properties of flavonoid-rich plant extracts.

Inhibition of Ca^{2+}-Induced mPTP opening

To further elucidate mitochondrial protection mechanisms, the effect of sweet potato extract on Ca^{2+}-induced mitochondrial permeability transition pore (mPTP) opening was examined using a swelling assay.

In control mitochondria, Ca^{2+} addition triggered rapid and pronounced swelling, reflected by a steep decrease in absorbance at 540 nm (Fig. 3). In contrast, pre-incubation

with sweet potato extract significantly attenuated Ca^{2+}-induced swelling in a concentration-dependent manner.

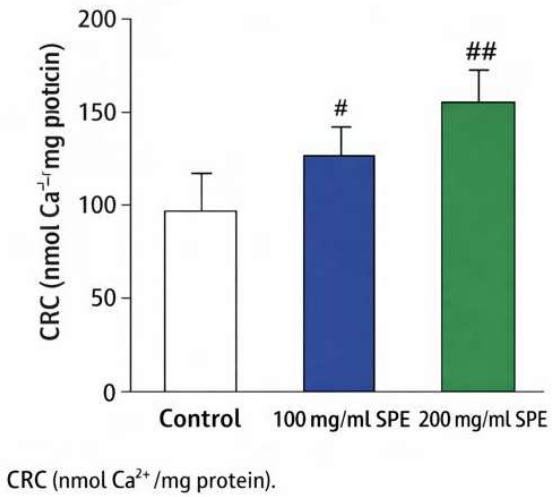

Fig. 2: Effect of sweet potato extract on mitochondrial Ca^{2+} retention capacity (CRC) in isolated rat liver mitochondria (# $P < 0.05$, ## $P < 0.01$ vs. control).

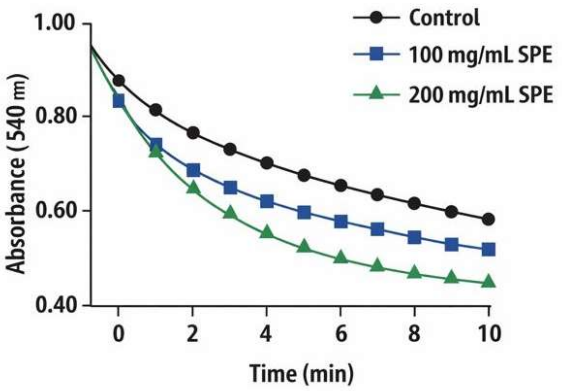

Fig. 3: Sweet potato tuber extract attenuates Ca^{2+}-induced mitochondrial swelling in isolated rat liver mitochondria (Time-dependent changes in mitochondrial swelling were monitored as a decrease in absorbance at 540 nm (A_{540}). Compared with the control, treatment with sweet potato extract (SPE) at 100 and 200 mg/mL significantly delayed Ca^{2+}-induced swelling, indicating dose-dependent stabilization of the inner mitochondrial membrane and inhibition of mPTP opening).

At 100 mg/mL extract concentration, the rate and magnitude of mitochondrial swelling were reduced by approximately 35–40%, while at 200 mg/mL swelling inhibition reached 55–60% compared to control ($P < 0.01$). The inhibitory pattern closely resembled that observed with cyclosporin A, confirming involvement of the mPTP pathway (Fig. 4).

These findings indicate that sweet potato extract effectively limits pathological Ca^{2+} influx into mitochondria and prevents catastrophic mPTP opening. At the molecular level, this protective effect can be attributed to flavonoid-mediated modulation of membrane lipids, suppression of reactive oxygen species (ROS), and stabilization of protein components regulating pore opening.

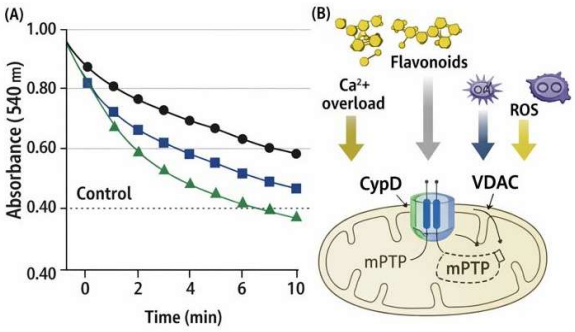

Fig. 4: Modulation of Ca^{2+}-induced mitochondrial swelling and mPTP activity by sweet potato (Ipomoea batatas L.) flavonoids ((A) Kinetics of Ca^{2+}-induced mitochondrial swelling in control and extract-treated mitochondria. (B) Schematic representation of mPTP modulation by sweet potato flavonoids, illustrating reduced Ca^{2+} overload, ROS suppression, and stabilization of cyclophilin D–VDAC interactions).

Dose-dependent spasmolytic effect in smooth muscle preparations

Functional consequences of mitochondrial and Ca^{2+} homeostasis modulation were further evaluated in isolated rat intestinal smooth muscle preparations. Sweet potato extract induced a clear, dose-dependent spasmolytic effect on spontaneous contractile activity (Fig. 5).

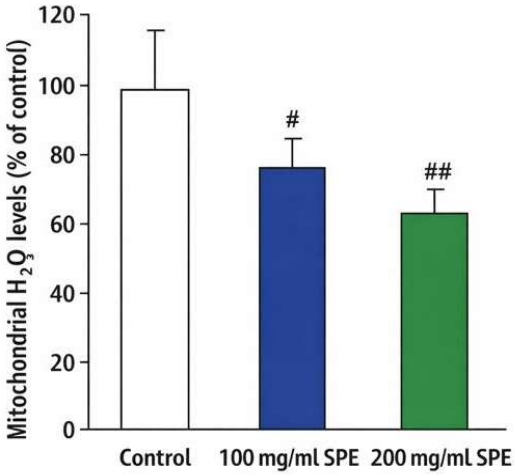

Fig. 5: Effect of sweet potato tuber extract (SPE) on mitochondrial H_2O_2 levels in isolated rat liver mitochondria (# $P < 0.05$, ## $P < 0.01$ vs. control).

At the lowest tested dose (50 mg/kg), contractile amplitude was reduced by 15–18%, indicating mild relaxation. Increasing the dose to 100 mg/kg resulted in 30–40% inhibition of contraction amplitude, while the highest dose (200 mg/kg) produced a robust relaxation effect of 55–65% (Table 2).

Table 2: Spasmolytic effect of sweet potato extract on smooth muscle contractility (n=5)

Dose	Inhibition of contraction (%)
50 mg/kg	15–18
100 mg/kg	30–40
200 mg/kg	55–65

Comparative analysis revealed that the onset of relaxation induced by sweet potato extract was slower than that of verapamil, a classical L-type Ca^{2+} channel blocker, but closely resembled the kinetic profile of papaverine (Fig. 6). However, unlike verapamil, which exerts a highly specific channel-blocking effect, the extract displayed a sustained and stable relaxation pattern.

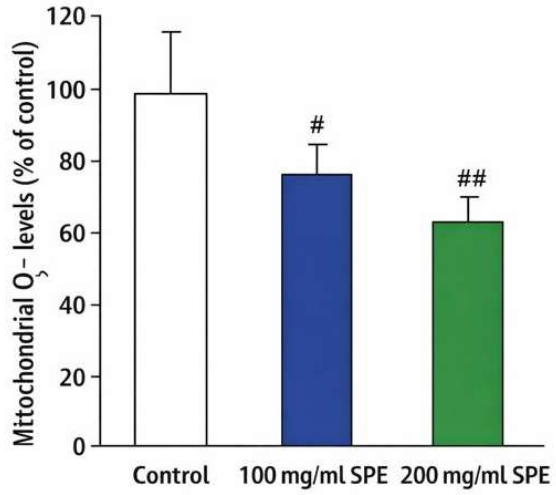

Fig. 6: Effect of sweet potato extract (SPE) on mitochondrial superoxide anion ($O_2^{\bullet-}$) levels in isolated rat liver mitochondria (Data are expressed as percentage of control values. Treatment with SPE at 100 and 200 mg/mL significantly reduced mitochondrial $O_2^{\bullet-}$ production in a dose-dependent manner, indicating attenuation of mitochondrial reactive oxygen species generation and improved redox homeostasis (# $P < 0.05$, ## $P < 0.01$ vs. control).

These observations suggest that the spasmolytic action of sweet potato extract is mediated through a multi-target mechanism, involving partial inhibition of Ca^{2+} influx, attenuation of mitochondrial Ca^{2+} overload, and reduction of oxidative stress. This integrative mode of action likely underlies the sustained relaxation observed in smooth muscle tissues.

Overall integration of results

Taken together, the results demonstrate that sweet potato extract exerts coordinated mitochondrial and smooth muscle protective effects. Enhancement of ΔΨm, suppression of lipid peroxidation, inhibition of Ca^{2+}-induced mPTP opening, and dose-dependent spasmolytic activity collectively support a mechanistic link between mitochondrial stabilization and smooth muscle relaxation. These findings provide strong experimental evidence for the functional relevance of sweet potato-derived bioactives in molecular nutrition and physiological regulation.

DISCUSSION

The present study provides compelling experimental evidence that sweet potato (*Ipomoea batatas* L.) tuber extract exerts coordinated mitochondrial and smooth muscle regulatory effects through a multi-level molecular mechanism. Unlike classical antioxidant paradigms that primarily emphasize free radical scavenging, the findings demonstrate that sweet potato-derived flavonoids function as metabolic and ionic modulators, integrating mitochondrial bioenergetics, Ca^{2+} homeostasis, and contractile signaling pathways.

One of the most significant outcomes of this study is the pronounced stabilization of mitochondrial function following treatment with sweet potato extract. The observed increase in mitochondrial membrane potential (ΔΨm) by up to 126% of control indicates preservation of the proton motive force and improved efficiency of oxidative phosphorylation. Maintenance of ΔΨm is essential for ATP synthesis, Ca^{2+} buffering, and prevention of apoptotic signaling cascades (Nicholls and Ferguson 2013; Ergasheva et al. 2024).

Concomitantly, the marked reduction in malondialdehyde (MDA) levels confirms effective suppression of lipid peroxidation. These findings align with previous reports demonstrating that plant flavonoids, including quercetin and rutin, incorporate into mitochondrial membranes and protect phospholipids from ROS-mediated damage (Sandoval-Acuña et al. 2014; Skulachev et al. 2017; Ergasheva et al. 2021; Ruziev et al. 2024). However, the magnitude of mitochondrial protection observed in the present study suggests not merely antioxidant action, but active modulation of mitochondrial membrane integrity and ion permeability.

Mitochondrial Ca^{2+} overload and subsequent opening of the mitochondrial permeability transition pore (mPTP) represent critical events linking oxidative stress to bioenergetic collapse (Bernardi et al. 2015a). The present results demonstrate that sweet potato extract significantly attenuates Ca^{2+}-induced mitochondrial swelling, indicating inhibition of mPTP opening.

Mechanistically, this effect is likely mediated by several converging pathways: (i) reduction of ROS levels, which are known sensitizers of cyclophilin D-dependent mPTP opening; (ii) stabilization of inner mitochondrial membrane lipids; (iii) modulation of Ca^{2+} flux through mitochondrial Ca^{2+} uniporters. Such a multi-target mechanism is consistent with current models proposing that dietary polyphenols modulate mitochondrial stress thresholds rather than acting as direct pore blockers (Bernardi et al., 2015b; Dorta et al. 2018).

The mitochondrial effects observed in isolated organelles translated into clear functional outcomes at the tissue level. Sweet potato extract induced a robust, dose-dependent relaxation of intestinal smooth muscle, reaching 55–65% inhibition of contractile activity at the highest dose. Importantly, the kinetic profile of this response differed from that of verapamil, a selective L-type Ca^{2+} channel blocker, but closely resembled papaverine, a phosphodiesterase inhibitor with broader intracellular effects.

This distinction is physiologically meaningful. Whereas verapamil exerts rapid and direct inhibition of Ca^{2+} influx, sweet potato extract produced a slower but sustained spasmolytic effect, suggesting indirect regulation of Ca^{2+} signaling. The data support a model in which flavonoids partially inhibit voltage-dependent Ca^{2+} channels, reduce mitochondrial Ca^{2+} overload, and suppress ROS-mediated sensitization of contractile machinery. This integrative mechanism ultimately decreases myosin light chain kinase (MLCK) activity

and favors smooth muscle relaxation (Somlyo and Somlyo 2003).

From a pharmacodynamic perspective, sweet potato extract differs fundamentally from classical spasmolytics. While verapamil and papaverine act on single dominant targets, the extract exhibits pleiotropic activity, simultaneously affecting Ca^{2+} channels, mitochondrial function, and redox balance. Such multi-target behavior is increasingly recognized as a hallmark of food-derived bioactives and underlies their favorable safety and adaptability profiles (Howes and Simmonds 2014).

The present findings therefore position sweet potato extract not as a substitute for pharmacological agents, but as a nutritional modulator capable of enhancing physiological resilience under stress conditions, including oxidative stress and dysregulated smooth muscle excitability.

Within the framework of molecular nutrition, the results strongly support the concept that diet-derived compounds can modulate cellular signaling networks at the organelle level. By preserving mitochondrial bioenergetics and regulating Ca^{2+}-dependent contractility, sweet potato-derived flavonoids may contribute to the prevention of functional gastrointestinal disorders and stress-associated smooth muscle dysfunction.

Importantly, these effects arise from whole-extract activity rather than isolated compounds, emphasizing the relevance of food matrix interactions and synergistic phytochemical action (Liu 2013). This positions sweet potato as a promising candidate for development of functional foods and nutraceuticals targeting mitochondrial health and smooth muscle physiology.

In summary, the present study demonstrates that sweet potato tuber extract exerts coordinated mitochondrial and smooth muscle protective effects through regulation of Ca^{2+} homeostasis, suppression of oxidative stress, and stabilization of mitochondrial membranes. These findings provide a mechanistic bridge between nutritional bioactives and physiological function, reinforcing the role of sweet potato as a scientifically substantiated functional food within the domain of molecular nutrition.

Conclusion

Sweet potato (*Ipomoea batatas* L.) tuber extract exhibits potent and quantifiable bioactivity at the mitochondrial and smooth muscle levels. The extract significantly increased mitochondrial membrane potential relative to control values, while reducing lipid peroxidation, as evidenced by a 40-45% decrease in MDA concentration. Ca^{2+}-induced mitochondrial permeability transition pore (mPTP) opening was significantly attenuated, confirming improved mitochondrial stability and Ca^{2+} buffering capacity. Overall, these findings provide compelling mechanistic evidence that flavonoids from sweet potato act as metabolic and redox modulators rather than simple antioxidants.

DECLARATIONS

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Conflict of Interest: The authors confirm that there are no conflicts of interest related to this study

Data Availability: All data generated and analyzed during this study are included in this article; no additional datasets were produced.

Ethics Statement: This study was approved by the Ethics Commission of the Scientific Research Institute Agrobiotechnologies and Biochemistry of Gulistan State University.

Author's Contribution: KK, RY, DK, SK, US and MM performed the experiments. KK, RY and NR analyzed data. KK, CB and DE statis-tically analyzed results. All authors wrote the draft of the manuscript. KK and RY conducted the critical revision of the manuscript. KK worked out the concept and design, supervised and funded the experiments. All authors read and approved the final manuscript.

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