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RESEARCH ARTICLE

Coconut Milk Phenolics Enhance General Oxidative-stress Resistance and Specifically Protect Against Lipid Peroxidation in Selected Bacteria

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ABSTRACT

Coconut milk, the crude aqueous extract of the scraped endosperm of the nutty fruit of Cocos nucifera, is an edible plant product used as a food or food additive. Previous studies have reported that it contains, among other biomolecules, phenolic compounds that can act as antioxidants against oxidative stress caused by Reactive Oxygen Species. Pretreatment with phenolic compounds from coconut milk has been previously reported to augment antioxidant protection in the eukaryote Saccharomyces cerevisiae and other organisms. This study aimed to determine whether the phenolic extract of coconut milk offers any antioxidant protection against selected bacteria subjected to oxidative stress. It was observed that the phenolic extract of coconut milk, once absorbed into cells, conferred significant protection against induced oxidative stress in both Escherichia coli and Lactobacillus spp. This protective effect was demonstrated by a reduction in lipid peroxidation ($p<0.05$; $n=3$) and by a significant increase in cell survival following oxidative challenge ($p<0.05$; $n=3$). These findings demonstrated that the phenolic extract of coconut milk provides both general and specific antioxidant protection to beneficial bacteria, offering insights into potential interactions between dietary coconut products and gut microbial resilience.

***Keywords**: Coconut milk, Lipid peroxidation, Oxidative stress in bacteria, Phenolic extract of coconut milk, Reactive Oxygen Species*

INTRODUCTION

Molecular oxygen has the potential to acquire a single electron from various cellular biomolecules, thereby generating the free radical superoxide anion, which can subsequently produce hydroxyl radicals and other oxidising species. These reactive oxygen species (ROS) impose a serious threat to cellular integrity by oxidising biomolecules, a condition referred to as oxidative stress (Halliwell and Gutteridge, 2015). Effective antioxidant mechanisms enable aerobic and facultative microorganisms to survive in oxygen-rich environments. By contrast, organisms lacking these systems, such as obligate anaerobes, are confined to anaerobic niches (Imlay, 2013).

In the facultative anaerobe *Escherichia coli*, antioxidant defense mechanisms are dominated by enzymes including superoxide dismutase and catalase (Imlay, 2013). Meanwhile, microaerophilic genera such as *Lactobacillus spp.* survive under lower-oxygen conditions and rely on comparatively weaker systems, such

as Mn-catalase, NADH oxidase, and NADH peroxidase, to manage ROS (Kang *et al.*, 2013). Beyond endogenous defences, microorganisms (and their hosts) may benefit from exogenous antioxidant substances derived from the diet or environment.

The edible extract of the coconut kernel, commonly known as coconut milk (from *Cocos nucifera*), is obtained by homogenising and squeezing the grated endosperm with water. Coconut milk contains a variety of phenolic compounds, including gallic acid, caffeic acid, chlorogenic acid, vanillic acid, syringic acid, ferulic acid, and p-hydroxybenzoic acid (Karunasiri *et al.*, 2020). These compounds are capable of donating hydrogen atoms or electrons to neutralise ROS and are therefore associated with antioxidant activity (Di Meo *et al.*, 2013; Rudrapal *et al.*, 2022).

Recent studies highlight the importance of phenolic compounds in the human diet and their relevance to microbiological systems. Wadanambi *et al.* (2023) reported that a phenolic extract of coconut milk provided protection to *Lactobacillus spp.* against oxidative stress, indicating possible interactions between dietary phenolics and gut-microbiota resilience. A broader review by Arslan *et al.* (2025) described the antioxidant potential of bacterial metabolites, including phenolics, across multiple bacterial phyla, suggesting under-explored mechanisms of microbial oxidative-stress resistance. Chandimali *et al.* (2025) further summarised how dietary phenolics, flavonoids, and carotenoids reduce oxidative damage and lipid peroxidation in eukaryotic systems. Despite these insights, the functional role of a phenolic extract from coconut milk in protecting prokaryotic cells from oxidative damage remains poorly understood. Existing evidence provides only preliminary indications of its potential effects on bacterial oxidative-stress responses, leaving a clear gap in understanding how dietary phenolics interact with microbial cells at a mechanistic level.

Lipid peroxidation is a notable form of oxidative damage. ROS may extract hydrogen atoms from lipids, forming lipid radicals that react with molecular oxygen to generate lipid peroxyl radicals, thereby propagating chain reactions that impair membrane integrity and function (Sydney Rose *et al.*, 2022). For bacteria, such membrane damage compromises nutrient uptake, energy transduction, and overall survival. Suppressing lipid peroxidation, therefore, represents a specific protective mechanism distinct from general oxidative-stress tolerance.

The bacterial species selected for this study, *Escherichia coli* and *Lactobacillus spp.*, represent physiologically distinct yet ecologically relevant members of the gut microbiota. *E. coli* is a facultative anaerobe frequently exposed to oxidative bursts, whereas *Lactobacillus spp.* are microaerophilic commensals with comparatively weaker endogenous antioxidant systems. Together, they provide a meaningful contrast for evaluating the protective effects of a phenolic extract of coconut milk in prokaryotic cells.

In this study, a phenolic extract of coconut milk that contains a natural mixture of phenolic compounds was prepared and evaluated for its potential protective effects against bacteria. Specifically, we investigated whether pretreatment with the phenolic extract of coconut milk enhances general oxidative-stress resistance (survival rate) and simultaneously reduces lipid peroxidation (specific protection) in *E. coli* and *Lactobacillus spp*. subjected to oxidative stress induced by hydrogen peroxide together with ferrous chloride.

MATERIALS AND METHODS

Preparation of coconut milk

Mature coconuts (12–14 months) collected from *Cocos nucifera* L. typica (tall type) coconut trees were used to prepare coconut milk as previously reported (Karunasiri *et al.*, 2020). Scraped coconut kernel (100 g) with distilled water (100 mL) was mixed using a kitchen blender (3 min). The homogenised mixture was squeezed through cheesecloth to separate the liquid portion, which was taken as coconut milk. The density of the preparation was 1.0±0.0 g/mL.

Preparation of the methanolic extract of coconut milk

The methanolic extract of coconut milk was prepared by the method previously reported by Karunasiri *et al*. (2020). Lipids were removed from coconut milk by centrifugation (1080 *g*, 10 min). The resultant aqueous extracts of coconut milk (1 mL) were mixed with methanol (1 mL) and chloroform (1 mL), vortexed (30 Hz, 1 min), centrifuged (3800 *g*, 10 min), and the top methanol/water layer was separated as the methanolic extract of coconut milk.

Extraction of phenolic compounds from the methanolic extract of coconut milk

Methanolic extract of coconut milk was used, as reported by Karunasiri *et al*. (2020), to make the stock solution of phenolic compounds. The methanolic extract was first evaporated, and the residue, which consisted of phenolics, was dissolved in distilled water to a known concentration of 20.0 mg/mL, and then filter-sterilised using a 0.45 μm bacteriological filter to obtain a sterile solution of phenolic compounds.

Bacterial strains

Lyophilised cultures of *E. coli* (ATCC 25922) and five Lactobacilli, namely, *Lactobacillus acidophilus* (ATCC 4356), *L. plantarum* (DMBUK 113080), *L. lactis* (ATCC 12315), *L. casei* (ATCC 393), and *L. fermentum* (ATCC 14931) were obtained from the culture collection, DMBUK (Department of Microbiology, University of Kelaniya Culture Collection), reconstituted in relevant media, and maintained in glycerol (20%) at -80°C. *E. coli* for the experiment was grown in LB broth (tryptone 10 g/L, yeast extract 5 g/L, and NaCl 10 g/L) at 37°C at 250 rpm. The Lactobacilli were grown in MRS broth (peptone 10 g/L, beef extract 10 g/L, yeast extract 5 g/L, glucose 20 g/L, sorbitan monooleate 1 g/L,

di-potassium hydrogen orthophosphate 2 g/L, magnesium sulphate 0.1 g/L, manganese sulphate 0.05 g/L, ammonium citrate 2 g/L, sodium acetate 5 g/L) at 37°C under anaerobic conditions.

Determining the concentration of phenolics that was below bactericidal levels

As phenolic compounds at high concentrations are bactericidal, the concentration of phenolic substances that is below the bactericidal concentration had to be determined for the use of phenolics in experiments that are meant to define the antioxidant support of those compounds. The concentration of the phenolic extract of coconut milk that would need to kill 50% of each bacterial population, i.e., the LD_{50} concentrations, was determined (n=3) by exposing bacterial cultures (3 h) to a concentration series (0-1.5 mg/mL) of phenolic extract of coconut milk, and subsequently counting the number of colonies, that is, colony forming units (CFUs) produced on solid medium. A culture not exposed to phenolics was used as the control, and CFU counts at the tested concentrations were calculated as percentages of the CFUs produced by the control. The concentration of phenolics that would need to reduce the CFUs down to 50% of the CFUs produced by the control was taken as the LD_{50}. The concentration of antioxidants used for the pretreatment of bacteria was kept below the LD_{50} values to ensure no harm would happen to the bacterial populations.

Pretreatment of bacterial strains with the phenolic extract of coconut milk

A loopful of *E. coli* from the glycerol stock was used to inoculate 1 mL of sterile LB broth and was incubated overnight at 37°C and 250 rpm on a shaker. The culture was added to 1 mL of sterile LB broth until the suspension reached an optical density of 0.5. A 10 µL aliquot from a sterile phenolic extract solution was added to the 1 mL culture and was incubated overnight at 37°C and 250 rpm. Lactobacilli were subjected to the same procedure, using the previously described specific medium and growth conditions required for Lactobacilli. In a control (n=3), 10 µL of distilled water was used instead of phenolics.

Induction of oxidative stress in the bacterial strains

The broth cultures were centrifuged (100 *g*, 3 min) to harvest the pretreated cell pellets, washed with 1x Phosphate- Buffered Saline (PBS) (1 mL) to wash residual antioxidants, centrifuged (100 *g*, 3 min) again, and resuspended in 1x PBS (1 mL). As ferrous ions generate hydroxyl free radicals, 15 mM $FeCl_2$ was added to reach a concentration of 150 µM $FeCl_2$, and the mixture was kept for 30 min at room temperature before hydrogen peroxide was added to a final concentration of 2 mM. The samples were incubated under induced oxidative stress for 1 h at room temperature, with E. coli at 250 rpm, and with lactobacilli under anaerobic conditions. Sterile distilled water was used in place of hydrogen peroxide in the control experiments (n=3).

Evaluation of lipid peroxidation in *E. coli* pretreated with the phenolic extract

The thiobarbituric acid reactive substances (TBARS) assay was used to measure lipid peroxidation, a test that quantifies the total amount of certain products of lipid oxidation, namely malondialdehyde (MDA) and other reactive aldehydes, which react with thiobarbituric acid (TBA), producing colored compounds. The preventive effect of coconut milk antioxidants against oxidation induced by H_2O_2 and $FeCl_2$ together on linoleic acid, a lipid taken in from the medium into the bacterial cells pretreated with coconut milk antioxidants, was measured by TBARS assay (n=3), with the help of a standard curve prepared using a concentration series (0-5 µM) of MDA using the method reported by Do *et al.* (1996) and Senanayake *et al*. (2018) with some modifications. Linoleic acid was homogenised in a solution of Tween 20 in H_2O (1:2000 v/v) at room temperature for 5 min at 250 rpm. The homogenate was added to the overnight cultures of pretreated bacterial cells to a final concentration of 0.03 mg/mL, and the cultures were incubated at 250 rpm at room temperature for 4 h before induction of oxidative stress. The control sample contained linoleic acid homogenate, $FeCl_2$, and H_2O instead of H_2O_2, so no peroxidation was expected. The cells were harvested by centrifugation (380 *g*, 10 min), washed with 1xPBS (1 mL), and lysed. 1 mL of TBA (15% TCA, 0.8% TBA in 0.25 N HCl) was added, mixed well, 8 µL of butylated hydroxytoluene (BHT) (0.2%) was added, and the mixture was heated at 95 °C for 15 min. The mixture was allowed to cool and centrifuged at 3800 *g* for 20 min. The absorbance of the supernatant was measured at 532 nm by a Multiskan GO spectrophotometer (Thermo Scientific). The standard curve was prepared using malondialdehyde (n=3).

Evaluation of the survival of pretreated bacteria subjected to oxidative stress

1 mL of the culture was transferred to the appropriate solid medium, incubated under the growth conditions required for each bacterial strain, and the CFUs were counted. The number of CFUs produced by the test that was neither pretreated nor oxidative stressed (CFUs not pretreated, not stressed) was counted. The number of CFUs produced by the test subjected to oxidative stress without pretreatment (CFUs not pretreated, stressed) was also counted and recorded as a percentage of the CFUs produced by the test that was neither pretreated nor subjected to oxidative stress. The number of CFUs of the test that was pretreated before applying oxidative stress (CFUs pretreated, stressed) was counted, and it was also recorded as a percentage of CFUs produced by the test that was neither pretreated nor oxidative stressed. The difference between the percentages of CFUs produced without pretreatment and with pretreatment was statistically analysed (n=3) to determine whether the differences, if any, were significant or not.

Statistical analysis

All experiments were conducted in triplicate. A two-sample t-test was conducted to assess significant differences ($p \leq 0.05$) between the means. Bars

sharing the same superscript letter are not significantly different ($p \leq 0.05$), whereas bars with different letters differ significantly. Data were analysed using Minitab statistical software (Version 17 for Windows).

RESULTS AND DISCUSSION

Bactericidal concentrations of the phenolic extract against the selected bacteria

Although the research was intended to determine and quantify antioxidant support, if any, given to the tested bacteria by the phenolic extract of coconut milk, it was found that these phenolic compounds showed antibacterial activity in high concentrations, as is typically expected with phenolics. Hence, it was necessary to determine concentrations that would not be high enough to reduce bacterial growth significantly but would be sufficient to provide antioxidant support.

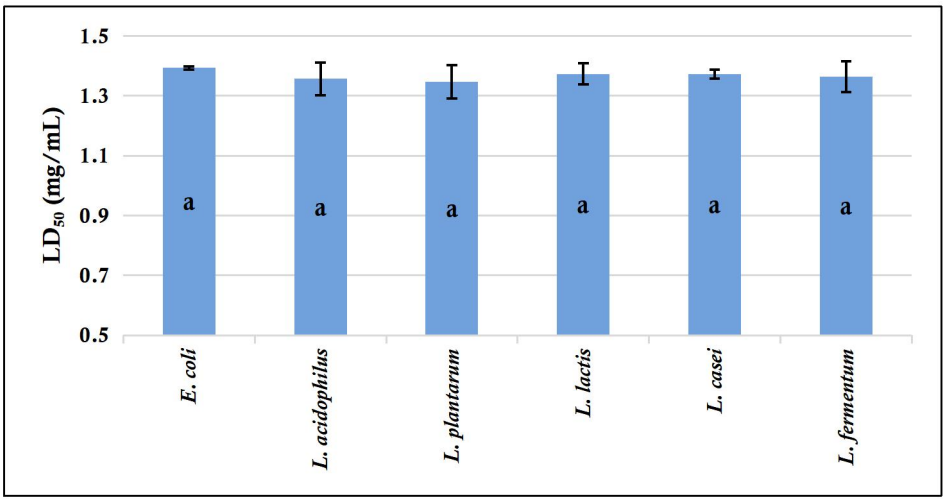

Figure 1: Antibacterial concentrations (LD_{50} mg/mL) of phenolic extract of coconut milk against the tested bacteria (mean±SD; n=3) measured in terms of the reduction of CFUs by 50%. Bars sharing the same letter (a) are not significantly different ($p \leq 0.05$)

Figure 1 presents the bactericidal activity of the phenolic extract of coconut milk against the bacterial strains used in this study, expressed as LD_{50} values (mean±SD; n=3). The phenolic extract exhibited mild antibacterial effects, with *E. coli* showing an LD_{50} of 1.39±0.006 mg/mL. The *Lactobacillus spp.* demonstrated LD_{50} values within a comparable range, varying slightly among species (Figure 1). These LD_{50} determinations were essential for selecting appropriate phenolic concentrations for subsequent assays. Accordingly, all pretreatment concentrations used in the oxidative-stress and lipid-peroxidation experiments were maintained at 0-0.9 mg/mL, which was well below their respective LD_{50} values (all were in the range of between 1.3-1.5 mg/mL (Figure 1), which ensured that the phenolics did not exert bactericidal effects but would instead allow assessment of their antioxidant protective capacity.

The extent of lipid peroxidation in the selected bacteria pretreated with the phenolics

Formation of MDA resulting from linoleic acid peroxidation was quantified to assess the extent of oxidative damage in *E. coli*. Pretreatment with the phenolic extract of coconut milk produced a clear, concentration-dependent protective effect against lipid peroxidation (Figure 2). In the absence of phenolics, *E. coli* showed the highest MDA level (0.098±0.003 µmol/mL). Progressive reductions in MDA were observed with increasing phenolic concentrations, with values decreasing to 0.096±0.003 µmol/mL at 0.4 mg/mL, 0.092±0.003 µmol/mL at 0.5 mg/mL, and further to 0.079±0.002 µmol/mL at 0.6 mg/mL. The most substantial protective effect occurred at the highest phenolic concentration tested, 0.9 mg/mL, which reduced MDA levels to 0.064±0.002 µmol/mL. This reduction was statistically significant compared with the untreated control (p=5.27×10^{-5}; n=3), confirming that the phenolic extract of coconut milk effectively suppressed lipid peroxidation. The reduction in MDA formation was possibly due to the specific antioxidant action at the main lipid-containing structure, the membrane. Therefore, this protection may be interpreted as protecting the integrity and function of bacterial membranes under oxidative stress, complementing the observed enhancement in overall survival.

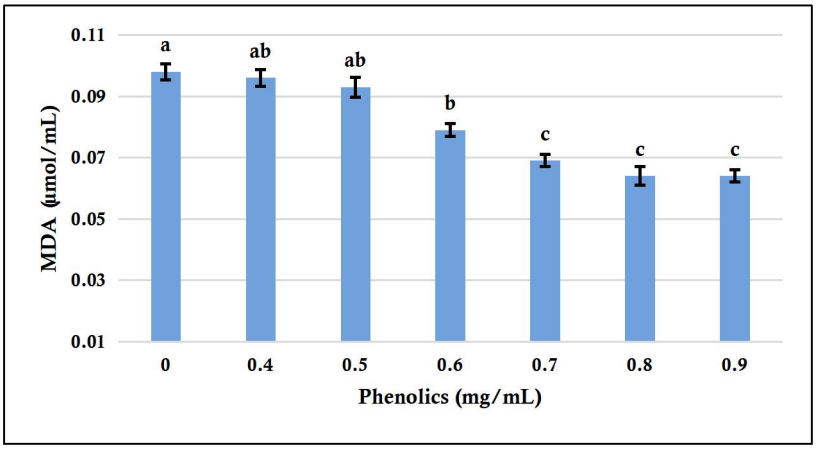

Figure 2: Protection effect of coconut milk phenolics against lipid peroxidation in *E. coli*. Bars with different letters (a,ab,c) differ significantly (p≤0.05)

Pretreatment with the phenolic extract of coconut milk also conferred a protective effect against lipid peroxidation in all *Lactobacillus spp.* tested (Figure 3). For each species, MDA levels were highest at 0 mg/mL phenolics and progressively declined with increasing phenolic concentration. Although the magnitude of MDA reduction varied slightly among species, all five Lactobacilli, *L. acidophilus*, *L. plantarum*, *L. lactis*, *L. casei*, and *L. fermentum*, showed a consistent downward trend across the phenolic range from 0 to 0.9 mg/mL. To evaluate the overall protective effect, the average MDA formed by

all Lactobacilli at 0 mg/mL phenolics was compared with that at the highest phenolic concentration tested (0.9 mg/mL). This comparison showed a significant decrease in MDA formation (p=5.86×10^{-6}; n=3), demonstrating that coconut milk phenolics substantially suppressed lipid peroxidation in all the *Lactobacillus spp.* exposed to oxidative stress.

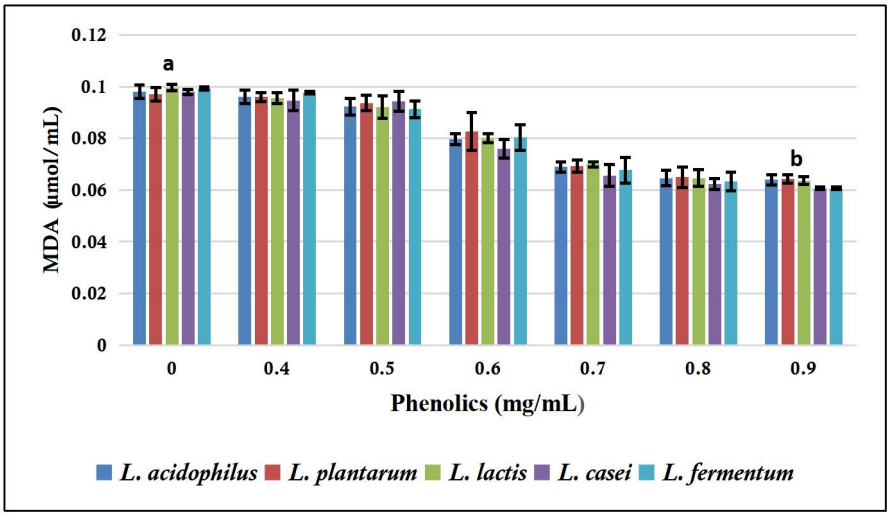

Figure 3: Protection effect of coconut milk phenolics against lipid peroxidation in Lactobacilli. Bars with different letters (a,b) indicate significant differences (p≤0.05)

Survival at oxidative stress of the selected bacteria pretreated with the phenolic extract

The results showed that overnight growth in the presence of coconut milk phenolic compounds helped the tested bacteria to survive under the combined oxidative attack that was induced on the following day by means of hydroxyl radicals formed by hydrogen peroxide and ferrous chloride. Induction of oxidative stress reduced the percentage of CFUs from 100% under normal conditions to 72.67±1.20%. However, after pretreatment with the phenolic extract of coconut milk, the *E. coli* cultures produced 90.33±1.53% CFUs (Figure 4). A statistical comparison of the stressed cultures with and without pretreatment confirmed that the increase in survival due to pretreatment was significant (p=<0.05; n=3), demonstrating that coconut milk phenolics significantly enhanced the resistance of *E. coli* to induced oxidative stress.

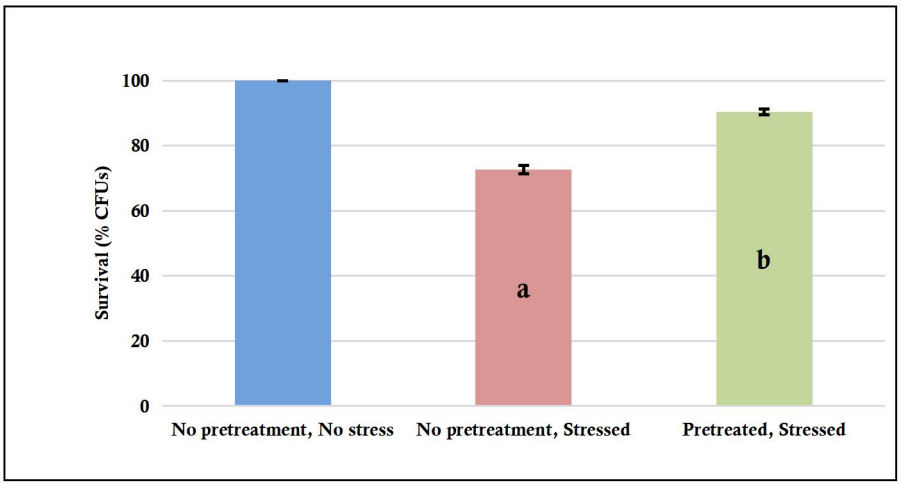

Figure 4: Under oxidative stress, the survival of *E. coli* pretreated with the phenolic extract. Bars with different letters (a,b) differ significantly ($p \leq 0.05$). (The increase in the survival rate in pretreated cultures was significant ($p=0.0011<0.05$; n=3)

Figure 5 shows that pretreatment with coconut milk phenolics increased the survival of all *Lactobacillus spp.* under oxidative stress. In every species tested, the percentage of surviving cells (% CFUs) in the "pretreated and stressed" group was higher than in the "stressed but not pretreated" group, indicating that the phenolic fraction provided measurable antioxidant protection. For each *Lactobacillus spp.*, survival following oxidative challenge was compared between cultures with no pretreatment and those pretreated with phenolics. In all cases, pretreatment with 0.09 mg/mL phenolics, the highest concentration tested, resulted in a clear improvement in survival, confirming that coconut milk phenolics confer broad-spectrum antioxidant support to Lactobacilli.

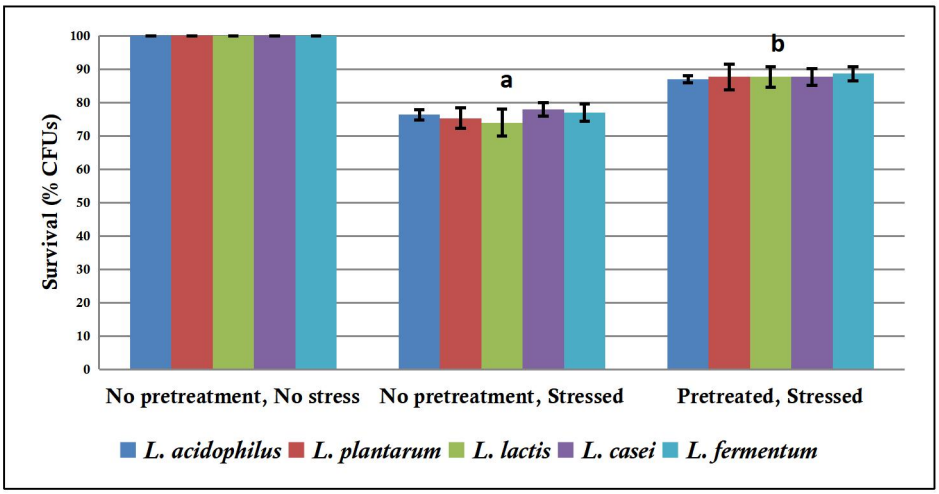

Figure 5: Under oxidative stress, survival of Lactobacilli pretreated with phenolic compounds of coconut milk. Bars with different letters (a,b) indicate significant differences ($p \leq 0.05$)

The statistical analysis further confirmed that the survival advantage conferred by coconut-milk phenolic pretreatment was significant in every *Lactobacillus spp.* tested (Table 1). For each organism, the triplicate survival percentages of the pretreated, stressed cultures were compared with those of the non-pretreated, stressed cultures using an unpaired two-tailed t-test (n=3). All five species, *L. acidophilus*, *L. plantarum*, *L. lactis*, *L. casei*, and *L. fermentum*, showed highly significant increases in survival following pretreatment, with *p*-values ranging from 0.0129 to 0.0011, all of which were well below the threshold of 0.05. These results demonstrated that, across phylogenetically diverse Lactobacillus strains, coconut-milk phenolics consistently enhance cellular tolerance to oxidative stress, reinforcing the conclusion that these phenolic compounds provide a broad-spectrum protective effect against ROS-mediated cell death.

Table 1: The increase in the survival rate of each species pretreated with phenolics (The phenolics significantly protected Lactobacilli against oxidative stress)

Organism	Stressed without pretreatment (n=3) (mean % CFUs±SD)	Stressed after pretreatment (n=3) (mean % CFUs±SD)	Increase in the survival after pretreatment
L. acidophilus	76.34±1.52	87.00±1.00	p=<0.05
L. plantarum	75.34±3.05	87.67±3.78	p=<0.05
L. lactis	74.00±4.00	87.67±3.05	p=<0.05
L. casei	78.00±2.00	87.67±2.51	p=<0.05
L. fermentum	77.00±2.64	88.67±2.08	p=<0.05

The results showed that coconut milk, due to its phenolic content, offers specific antioxidant action against lipid peroxidation and an overall antioxidant protection, possibly due to antioxidant action against detrimental oxidation of other biomolecules, in the tested bacteria, which may occur due to the action of ROSs in those organisms, which happened to be members of the human intestinal microbiome. Given the fact that coconut milk is an ingredient or an additive to human foods, any antioxidant support of coconut milk antioxidants on intestinal organisms would help to draw inferences about interactions between dietary coconut milk and the intestinal microbiome.

CONCLUSION

The study was conducted to determine whether phenolic compounds in coconut milk, once absorbed by cells grown in their presence, would protect selected bacteria against oxidative stress. The phenolic compounds extracted from coconut milk conferred significant protection against sub-lethal oxidative stress, induced by hydrogen peroxide and ferrous chloride, on both *E. coli* and *Lactobacillus spp*. after overnight exposure to the extract.

The phenolic extract of coconut milk exerted protection against induced oxidative stress separately in *E. coli* and *Lactobacillus spp*. caused a statistically significant ($p<0.05$; n=3) increase in the rate of survival, measured in terms of CFUs, compared with the bacteria that were not pretreated with those compounds. The bacteria, once pretreated with those phenolic compounds, showed a statistically significant ($p<0.05$; n=3) degree of reduction in the extent of lipid peroxidation as well.

The results confirmed that coconut milk contains phenolic antioxidants capable of mitigating ROS-induced cellular injury in bacteria. Since many *Lactobacillus spp.* and *E. coli* are key members of the human intestinal microbiome, these findings suggest that dietary coconut milk could enhance microbial resilience to oxidative stress, potentially benefiting gut health and diet-microbiome interactions. However, in vivo studies are required to confirm these effects.

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