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Protective Effect of Coconut Milk Phenolic Antioxidants on Oxidative Stress Induced Macromolecular Damage in *Saccharomyces cerevisiae*

M. G. A. N. Karunasiri,[1] M. M. Gunawardena,[2] D. Damunupola,[1] K. N. Seneviratne,[1] and N. Jayathilaka[1*]

[1]*Department of Chemistry, University of Kelaniya, Kelaniya*
[2]*Department of Microbiology, University of Kelaniya, Kelaniya*

Coconut milk is the aqueous extract of coconut endosperm used as a major source of fat in culinary applications. Nowadays, blended (machine-pressed) coconut milk (BCM) or commercial coconut milk products, such as liquid coconut milk (LCM) and powdered coconut milk (PCM), are used in place of hand-pressed coconut milk. Coconut milk is rich in polyphenolic antioxidants (PA). In this study, inhibition of H_2O_2 induced oxidative damage on lipids, proteins and mitochondrial DNA byPA extracted from various domestic and commercially available coconut milk preparationswas evaluated using *Saccharomyces cerevisiae* (yeast). Yeast can be used as a eukaryotic model organism to study oxidative stress since these cells share many basic biological properties with human cells including the genes involved in the oxidative stress response. PA were extracted from coconut milk with methanol/ water (50%). The total polyphenol content was measured by the Folin Ciocalteu method (BCM; PCM; LCM, 821 ± 14, 823 ± 14, 824 ± 12µg/100mL respectively). Pretreatment of the yeast cells with 0.5 mg/mL PA from BCM, PCM and LCM prior to H_2O_2 induced oxidative damage, significantly ($P<0.05$) increased the cell viability (87 ± 2, 90 ± 0, 86 ± 2%) compared to the control without PA (80 ± 2%). Pretreatment with PA from BCM, PCM and LCM completely inhibited the formation of thiobarbituric acid reactive species due to oxidative stress-induced lipid peroxidation in Yeast cells (0.016 ± 0.001, 0.011 ± 0.006, 0.019 ± 0.001 µg/mL, respectively) compared to the control without PA (0.045 ± 0.002 µg/mL) ($P < 0.05$).While BCM and PCM decreased the formation of protein carbonyls due to oxidative stress-induced protein oxidation (14.2 ± 0.1; 14.0 ± 0.1 nmol/mL) significantly ($P < 0.05$) compared to the control without PA (15.2 ± 0.3 nmol/mL), LCM (14.8 ± 0.3 nmol/mL) did not significantly decrease protein oxidation to levels present in un-stressed cells (9.1 ± 0.3 nmol/mL). The protective effect of PA on oxidative stress-induced mitochondrial DNA damage in yeast cells was evaluated based on the effect on the percentage of respiratory deficient cells as determined by CFU in YPG supplemented plates. Compared to the control without PA (10 ± 3%) PCM and LCM (3 ± 1, 7 ± 1%) showed a significantly lower percentage ($P < 0.05$) of respiratory deficient cells while BCM did not show a significant difference (5 ± 4%). PA from BCM, PCM and LCM imparted protection against oxidative stress induced damage on lipid, proteins and DNA in yeast cells.

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E-mail: njayathi@gmail.com