Hindawi
Journal of Chemistry
Volume 2020, Article ID 3503165, 8 pages
https://doi.org/10.1155/2020/3503165

*Research Article*

# Protective Effect of Coconut Oil Meal Phenolic Antioxidants against Macromolecular Damage: *In Vitro* and *In Vivo* Study

**A. N. Karunasiri**[1], **C. M. Senanayake**[1,2], **H. Hapugaswatta**[1], **N. Jayathilaka**[1] and **K. N. Seneviratne**[1]

[1]*Department of Chemistry, Faculty of Science, University of Kelaniya, Kelaniya, Sri Lanka*
[2]*Department of Biosystems Technology, Faculty of Technology, University of Sri Jayewardenepura, Nugegoda, Sri Lanka*

Correspondence should be addressed to N. Jayathilaka; njayathi@kln.ac.lk and K. N. Seneviratne; kapilas@kln.ac.lk

Received 27 April 2020; Accepted 8 June 2020; Published 29 June 2020

Academic Editor: Ioannis G. Roussis

Copyright © 2020 A. N. Karunasiri et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Coconut oil meal, a cheap by-product of coconut oil production, is a rich source of phenolic antioxidants. Many age-related diseases are caused by reactive oxygen species- (ROS-) induced damage to macromolecules such as lipids, proteins, and DNA. In the present study, the protective effect of the phenolic extract of coconut oil meal (CMPE) against macromolecular oxidative damage was evaluated using *in vitro* and *in vivo* models. Sunflower oil, bovine serum albumin (BSA), and plasmid DNA were used in the *in vitro* study, and thiobarbituric acid reactive substances (TBARS), protein carbonyl, and nicked DNA were evaluated as oxidation products. The inhibitory effect of CMPE against $H_2O_2$-induced macromolecular damage was evaluated using cultured HEp-2 cells. The results indicate that CMPE inhibits macromolecular damage both *in vitro* and *in vivo*. In addition, CMPE regulates redox status of HEp-2 cells under oxidative stress conditions by maintaining higher reduced glutathione levels. There was no significant difference in the expression of glutathione peroxidase in stressed and unstressed cells suggesting that CMPE regulates the cellular oxidative stress responses without affecting the expression of oxidative stress response genes. Oral feeding of Wistar rats with CMPE improves the serum and plasma antioxidant status without causing any toxic effects.

## 1. Introduction

Oxidative stress-induced modification of macromolecules such as lipids, proteins, and DNA has been identified as a risk factor in various diseases such as cardiovascular disease, neurodegenerative disorders, and cancer [1]. Accumulation of high levels of ROS in cells causes cell toxicity and cell death, while low levels of ROS support cell growth and proliferation [2]. In addition to the health effects caused by breakdown of lipids due to ROS-mediated peroxidation, the resultant products of lipid peroxidation such as malondialdehyde (MDA) and 4-hydroxy-2-nonenal can modify proteins and DNA causing further damage [3]. Oxidation causes irreversible changes to protein structure and activity, while sulfur-containing amino acids are particularly susceptible to ROS-mediated damage [4]. ROS can also cause severe damage to cellular DNA, and the fate of the cell depends on the extent of DNA damage [5]. Therefore, regulating redox status in the living systems by maintaining appropriate levels of ROS is important for proper redox homeostasis [1, 6]. Reduced form of glutathione (GSH) is a natural radical scavenger in the living systems that can protect cells from ROS-mediated oxidative damage. The ratio of GSH and oxidized form of glutathione (GSSG) is an indicator of the redox status of cells [7]. Expression of glutathione peroxidase (GPx) is also known to upregulate at high $H_2O_2$ concentrations resulting from oxidative stress [8].

In addition to cellular endogenous antioxidant systems, natural antioxidants are important in controlling excess ROS