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Effect of coconut milk on intestinal barrier function and management of oxidative stress

Ambanpola N.[1]*, Anjali N. V. P.[1], Manilgama T.[1], Gunawardane M.[1], Seneviratne K. N.[1], Jayathilaka N.[1]

Coconut milk (CM) is a major source of dietary fat in a Sri Lankan meal. It is rich in saturated, medium-chain fatty acids (MCFA) and various polyphenols. Some of the ingested fats and polyphenols are not absorbed in the small intestine and reach the colon. This study assessed the formation of metabolic products from CM and the influence of CM on intestinal barrier function. Twelve-week-old female Wistar rats were housed at 25 ± 1°C with a 12 h light and dark cycle. Rats were randomly assigned to two experimental groups (12 rats/ group). Ad libitum access to water and a diet containing 4.2 % total fat; from that 3% fat by means of soybean oil (SOD) control or CM (CMD) was provided for four weeks. Six rats from each group fasted for 10–12 h and were treated with ethanol (20%, 6 g/kg body weight) by oral gavage (SODM and CMDE groups were obtained). Blood (1 mL) was then drawn from the tail vein. Plasma antioxidant capacity, lipid peroxidation, and protein carbonyl content were determined by 2,2-diphenyl-1-picryl-hydrazy (DPPH) assay, ferric reducing antioxidant power (FRAP) assay, protein carbonyl assay, and thiobarbituric acid reactive substances (TBARS) assay according to previously reported methods. At the end of the feeding experiments, animals were subjected to barbiturate euthanasia and a transverse abdominal incision was made. The cecal wash samples with phosphate-buffered saline (pH 7.4) were stored at -80 °C. Liver and brain samples were also harvested. All experimental procedures were approved by the Ethics Review Committee, University of Kelaniya. Short-chain fatty acids (SCFAs) in the cecal wash, plasma, liver, and brain samples were quantified by Gas chromatography. SCFA levels were determined by the standard curves of each SCFA. Shapiro-Wilk normality test ($P<0.05$) and t-test was used for the statistical comparison. Acetate, propionate, and butyrate concentrations were 802.9±0.4 µg/mL, 156.3±2.1 µg/mL, 20.5±0.4 µg/mL and 802.8±0.4 µg/mL, 153.5±1.7 µg/mL, 19.9±0. µg/mL in CMD and SOD cecal wash samples respectively. Acetate, propionate, and butyrate concentrations were 236.2±0.1 µg/mL, 16.2±0.2 µg/mL, 1.3±0.0 µg/mL and 226.3±1.4 µg/mL, 14.4±0.2 µg/mL, 1.2±0.0 µg/mL in CMD and SOD plasma samples respectively. There was a significant ($P<0.05$) difference between plasma acetate and propionate levels in CMD compared to SOD. SCFAs were not detected in liver and brain samples. Saccharolytic microbes ferment oligo- and polysaccharides and produce SCFAs. Following their production SCFAs are rapidly absorbed by colonic cells and those not metabolized by colonic cells pass into the liver. Thus, only a small amount of the SCFAs reach systemic circulation and other tissues. Alcohol causes oxidative stress by releasing reactive oxygen species (ROS) during alcohol metabolism. Polyphenols serve as exogenous antioxidants, and they scavenge free radicals to control ROS. According to the four assays, there were no significant differences in the antioxidant capacity between the four groups suggesting no antioxidant effect of coconut milk over soy oil control. Thus, CM has a significant ($P<0.05$) impact on SCFAs passing through the intestinal barrier but no effect on the management of oxidative stress than soy oil.

Keywords: Coconut milk, Wistar rats, Medium-chain fatty acids, Intestinal barrier, Oxidative stress

[1] University of Kelaniya, Sri Lanka
∗ nimrothambanpola@gmail.com