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### **ORIGINAL ARTICLE**

# CONSUMPTION OF COCONUT MILK DID NOT INCREASE CARDIOVASCULAR DISEASE RISK IN MICE

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#### ARTICLE INFO

## ABSTRACT

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#### Key words:

Cardiovascular disease, Coconut milk, Serum lipid profile Freshly prepared coconut milk (25%, 50% and 100% preparations) was administered to mice in three test groups for 14 days. The control group was given an equal volume of tap water for the same period. Consumption of the different concentrations of coconut milk was found not to alter significantly (p>0.05) the serum lipid profile and proatherogenic indices of mice in the test groups, relative to the control group. Coconut milk may not increase the cardiovascular disease risk of those who consume it for food or for its medicinal values

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## **INTRODUCTION**

The coconut palm (cocos nucifera) belongs to the family Aracacea, and grows widely in the humid tropics. In countries where it is grown, coconut is usually a part of the traditional food (Foale, 2003). Coconut water contains sugars, proteins, antioxidants, vitamins and minerals. It is used as a refreshing drink throughout the tropics and in the preparation of isotonic sports drinks (Campbell-Falck et al., 2000). Coconut milk, the best known product of coconut meat (endosperm), is a sweet, milky-white, oilprotein-water emulsion, obtained when grated coconut meat is squeezed through a muslin cloth (Akpan et al., 2006). The fat content of the milk is about 17% and 90-92% of these are saturated fats. Though coconut milk is more saturated than most other oils and fats, about twothirds of the saturated fatty acids are medium chain fatty acids (Dayrit, 2003). The milk is used in the production of virgin coconut oil by controlled low temperature heating and removal of the oil fraction (Akpan et al., 2006). Coconut oil is useful as cooking oil, and finds application as biofuel (Jones, 1991).

Coconut milk has been shown to be useful in Ayuverdic medicine and in healing mouth ulcers (Nneli and Woyike, 2008) and in the folk-loric management of lower urinary tract symptoms (LUTS) and gastrointestinal cramps. However, little is known above the effect of its consumption on the cardiovascular risk state of those who consume it.

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## **MATERIALS AND METHODS**

Twenty male albino mice (average weight  $30 \pm 3g$ ) were acclimatized to the animal house for one week and thereafter assigned to four groups of 5 mice each. The mice were given feed and water ad libitum, exposed to 12hr dark/light cycles under humid tropical conditions. Mice in Group A served as the control, and were administered 0.2ml of tap water per os daily for 14 days. Those in Groups B, C and D were administered 0.2ml of 25%, 50% and 100% freshly prepared coconut milk respectively, per os daily for the same period. After 14 days, the mice were bled exhaustively by cardiac puncture, under light chloroform anesthesia. The blood was allowed to stand at ambient temperature for clotting to take place, before centrifugation at 2000g for 5 minutes. Serum was used for the analysis of total cholesterol, HDLcholesterol and triacylglycerol using standard colorimetric procedures (Allain et al., 1974; Lopes-Virella et al., 1977; Tietz, 1990). LDL- cholesterol, Non-HDL cholesterol and serum lipid pro-atherogenic indices were derived using standard formulae (Friedwald et al., 1972; Packard and Saito, 2003; Budzynski et al., 2003). Descriptive statistics was carried out on the data generated and are reported as means ± standard deviations. Differences between injections" and "olive oil means were separated by one way ANOVA and multiple comparisons test. A significant threshold of p < 0.05 was employed for the analysis. Data analysis was done using SPSS for windows version 11.0 (SPSS Inc., Chicago, IL).

## **RESULTS AND DISCUSSION**

Table 1 shows that all the lipid profile parameters were (each) highest in the control group, though the differences

between each test group and the control group were all insignificant (p>0.05). From Table 2, it is seen that the serum lipid pro-atherogenic indices were also similar (p>0.05) between the control group and each of the test groups. Though diets containing high quantities of

Budzynski J, Klopocka M, Swiakowski M, Pulkowski G and Ziółkowski1 M. 2003. Lipoprotein(a) in alcoholdependent male patients during a six-month abstinence period. *Alcohol & Alcoholism*, 38:157–162
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Group С D В A TAG (mmol/l)  $4.66 \pm 1.54$  $4.42 \pm 1.03$  $3.86 \pm 0.85$  $4.16 \pm 0.23$ 0.483 0.636 0.838 TCHOL (mmol/l)  $4.59 \pm 0.77$  $3.86 \pm 0.21$  $3.63 \pm 0.42$  $3.50 \pm 0.38$ р 0.194 0.114 0.098 HDL (mmol/l)  $2.03 \pm 0.25$  $1.58 \pm 0.32$  $1.62 \pm 0.32$  $1.38 \pm 0.30$ 0.188 0.199 0.084 р LDL (mmol/l)  $0.30 \pm 0.07$  $0.27 \pm 0.19$  $0.25 \pm 0.04$  $0.28 \pm 0.35$ 0.842 0.364 0.939 Р Non-HDL (mmol/l)  $2.28 \pm 0.53$  $2.01 \pm 0.36$  $2.17 \pm 0.39$  $2.41 \pm 0.70$ 0.804 0.629 Р 0.429

	Table 1. Serum lipid	profile of mice given	different concentrations of	oral coconut milk for 14 days
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TAG, TCHOL, HDL, LDL represent Triacyglycerol, Total Cholesterol, High Density Lipoprotein Cholesterol and Low Density Lipoprotein Cholesterol, respectively.

. Table 2: Serum lipid pro-atherogenic indices of mice given different c oncentrations of oral coconut milk for 14 days

Group					
	А	В	С	D	
TCHOL/HDL	$2.13\pm0.37$	$2.52\pm0.58$	$2.28\pm0.40$	$2.64 \pm 0.53$	
Р		0.393	0.625	0.193	
Non-HDL/HDL	$1.13 \pm 0.37$	$1.52 \pm 0.58$	$1.28 \pm 0.40$	$1.64 \pm 0.53$	
Р		0.393	0.625	0.193	
LDL/HDL	$0.14 \pm 0.05$	$0.18 \pm 0.15$	$0.16 \pm 0.03$	$0.23 \pm 0.31$	
Р		0.699	0.640	0.599	

TCHOL, HDL, LDL represent Total Cholesterol, High Density Lipoprotein Cholesterol and Low Density Lipoprotein Cholesterol, respectively.

saturated fats are known to result in dyslipidemia, medium chain fatty acids differ from the other saturated animal and dairy fats in their metabolism in the body. Medium chain fatty acids are rapidly absorbed in the intestines, even without pancreatic lipase. They are then carried in the portal vein to the liver and are rapidly oxidized to produce energy (Dayrit, 2003). It has also been shown that medium chain fatty acids, unlike long chain fatty acids, do not enter the cholesterol cycle and are not deposited in fat depots (Pamplona-Roger, 2005). These may explain the absence of any significant change in the serum lipid profile and pro-atherogenic indices of the mice. The data are instructive, especially given that each mouse consumed an equivalent of 50cl for a 70kg man daily for 14 days. Consumption of coconut milk may therefore not increase the risk of cardiovascular diseases in those who consume it either for its food value or its medicinal properties.

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