Plant Foods Hum Nutr (2017) 72:418–424
https://doi.org/10.1007/s11130-017-0643-0

ORIGINAL PAPER

Coconut Products Improve Signs of Diet-Induced Metabolic Syndrome in Rats

Sunil K. Panchal[1] · **Sharyn Carnahan**[1,2] · **Lindsay Brown**[1,2]

Published online: 27 October 2017

Abstract Increasing prevalence of obesity and metabolic syndrome warrants identification of potential therapeutic options for intervention. This study tested commercially available Virgin Coconut Oil and Coconut Nourish, as coconuts are rich sources of lauric and myristic acids. Male Wistar rats were fed either corn starch diet (C); high-carbohydrate, high-fat diet (H); high-carbohydrate, high-virgin coconut oil diet (HV); or high-carbohydrate, high-coconut Nourish diet (HN) for 16 weeks. Metabolic, liver, and cardiovascular health parameters were measured during and at the end of the study. Virgin coconut oil lowered body weight (C 386±8g, H 516±13g, HV 459±10g), blood glucose concentrations (C 4.2±0.1 mmol/L, H 5.4±0.2 mmol/L, HV 4.6±0.2 mmol/L), systolic blood pressure (C 127±5mmHg, H 149±4mmHg, HV 133±3mmHg,) and diastolic stiffness (C 25.0±1.7, H 31.4±1.2, HV 25.2 ±2.3,) with improved structure and function of the heart and liver. Coconut Nourish increased total body lean mass (C 255 ±10g, H 270±16g, HN 303±15g) and lowered plasma total cholesterol concentrations (C 1.6±0.2 mmol/L, H 1.7 ±0.1 mmol/L, HN 1.0±0.0 mmol/L), systolic blood pressure (C 127±5mmHg, H 149±4mmHg, HN 130±3mmHg) and diastolic stiffness (C 25.0±1.7, H 31.4±1.2, HN 26.5±1.0), improved structure and function of the heart and liver but increased plasma concentrations of triglycerides (C 0.3 ±0.1 mmol/L, H 1.1±0.4 mmol/L, HN 1.8±0.2 mmol/L) and non-esterified fatty acids (C 1.2±0.3 mmol/L, H 3.3 ±0.8 mmol/L, HN 5.6±0.4 mmol/L). Thus, the fiber and protein in coconut Nourish and the medium-chain saturated fatty acids in virgin coconut oil may improve cardiovascular and liver complications in obesity.

Keywords Metabolic syndrome · Obesity · Coconut · Saturated fatty acids · Lauric acid

Introduction

Metabolic syndrome is a cluster of risk factors for cardiovascular disease and type 2 diabetes including obesity, insulin resistance, hypertension, dyslipidemia, and impaired glucose tolerance [1]. The role of dietary saturated fatty acids as a risk factor for cardiovascular and metabolic disease has been widely debated [2, 3]. Current results suggest that the biological effects of saturated fatty acids depend on the source of the fat and the chain length of individual fatty acids, supporting the classification of these fatty acids based on biological effects rather than chemical structure [3]. Lauric and myristic acids are medium-chain saturated fatty acids found in large amounts in coconuts [4]. The 12-carbon lauric acid is rapidly oxidized in the cell, the 14-carbon myristic acid has an intermediate rate of oxidation while the longer-chain saturated fatty acids such as the 18-carbon stearic acid are oxidized at a slower rate [5]. Cold-pressed coconut oil, also referred to as virgin coconut oil (VCO), is mainly composed of saturated fatty acids (~91%), predominantly lauric and myristic acids [6].

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s11130-017-0643-0) contains supplementary material, which is available to authorized users.

✉ Sunil K. Panchal
Sunil.Panchal@usq.edu.au

[1] Functional Foods Research Group, Institute for Agriculture and the Environment, University of Southern Queensland, QLD, Toowoomba 4350, Australia

[2] School of Health and Wellbeing, Faculty of Health, Engineering and Sciences, University of Southern Queensland, Toowoomba 4350, Australia

Traditionally, components of coconuts such as shell fiber, pulp, and water have been used to treat diseases including diabetes, asthma, stomach pain, dermatitis, and diarrhea, as well as for their antipyretic and diuretic responses [7]. The husk from coconut fruit as a source of fiber has been associated with health effects including antimalarial, antibacterial, and antidepressant responses [8, 9]. Further, our recent studies showed that lauric acid prevented induction of obesity and osteoarthritis in high-carbohydrate, high-fat diet-fed rats [10]. A review of studies with coconut oil suggested that the consumption of coconut oil did not improve lipid profile and hence cardiovascular risk when compared to *cis* unsaturated fatty acids [11].

Intakes of simple sugars (fructose and sucrose), long-chain saturated fatty acids and *trans* fatty acids in the modern diet are major predictors of the increase in metabolic syndrome [2, 3, 12]. Our previous studies have reported that the feeding of high-carbohydrate, high-fat diet using fructose and sucrose as simple sugars and beef tallow as the source of long-chain saturated fatty acids and *trans* fatty acids induced metabolic, cardiovascular, and hepatic complications in rats that are similar to the metabolic syndrome in humans [10, 13, 14]. Further, foods can also prevent or reverse disease states, and current research has focused on these functional foods for metabolic diseases [15].

This study investigated two commercial coconut-derived products, Banaban virgin coconut oil and Banaban coconut Nourish, as sources of medium-chain saturated fatty acids and fiber, respectively, replacing beef tallow in diet-induced metabolic syndrome in rats. Structural changes in the rat heart were characterized by histology and echocardiography, while heart function was assessed *in vivo* using echocardiography and *ex vivo* in isolated perfused hearts. Systolic blood pressure and responses of isolated thoracic aortic rings were measured to identify vascular reactivity. Metabolic parameters (oral glucose tolerance, body composition, and plasma lipid concentrations) were assessed. Changes in liver structure and function were defined by histology and plasma activities of liver enzymes.

Materials and Methods

Rats and Diets 40 male Wistar rats aged 9–10 weeks old (337 ± 1 g) were purchased from Animal Resource Centre (Canning Vale, WA, Australia) and housed individually in a temperature-controlled (21 ± 2 °C), 12-h light/dark cycle environment with free access to group-specific food and water at the University of Southern Queensland Animal House. Rats were randomly divided into four separate groups ($n = 10$ each) and were fed with one of the following diets for 16 weeks: corn starch diet (C); high-carbohydrate, high-fat diet (H); high-carbohydrate, high-VCO diet (HV) and high-carbohydrate, high-coconut Nourish diet (HN). The composition of C and H diets have been reported previously [13, 14]. In HV and HN diets, beef tallow in the H diet was replaced by Banaban virgin coconut oil (200 g/kg) and Banaban coconut Nourish powder (200 g/kg), respectively. The composition of VCO and coconut Nourish powder are described in Supplementary Table 1.

Physiological Measurements Body weight and intakes of food and water were recorded daily for all rats. Energy intakes and feed conversion efficiency were calculated [13]. Abdominal circumference was measured every eighth week using a standard measuring tape under light anesthesia with Zoletil (tiletamine 10 mg/kg, zolazepam 10 mg/kg, intraperitoneal; Virbac, Peakhurst, NSW, Australia) [13]. Oral glucose tolerance tests were performed on rats at 0, 8, and 16 weeks [13]. Dual-energy X-ray absorptiometric measurements were performed on rats after 16 weeks of feeding using a Norland XR36 DXA instrument (Norland, Fort Atkinson, WI, USA) [13]. Systolic blood pressure and echocardiographic examination were measured [13]. After terminal euthanasia, left ventricular function was assessed using isolated Langendorff heart preparation [13]. Responses of thoracic aortic rings to noradrenaline, acetylcholine, and sodium nitroprusside were measured [13].

Euthanasia Euthanasia was induced by intraperitoneal injection of Lethabarb (pentobarbitone sodium; 100 mg/kg; Virbac, Peakhurst, NSW, Australia). Heparin (200 IU; Sigma-Aldrich Australia, Sydney, NSW, Australia) was administered through the right femoral vein followed by withdrawal of ~5 mL blood from the abdominal aorta. Blood was collected into heparinized tubes and centrifuged at 5000 × *g* for 15 min within 30 min of collection. Plasma was collected and stored at −20 °C.

Organ Weights and Histology Following perfusion experiments, right ventricle and left ventricle (with septum) were separated and weighed. Liver, kidney, and retroperitoneal, epididymal, and omental fat contents were removed and weighed. Organ weights were normalized relative to tibial length (mg/mm). Two rats *per* group were used exclusively for histopathological analysis. Thin sections (5 μm) of tissue were cut and stained with haematoxylin and eosin stain for determination of inflammatory cell infiltration (heart and liver, ×20) and fat vacuole deposition (liver, ×20). Picrosirius red staining was used to define collagen distribution in the left ventricle (×20) [13].

Plasma Biochemistry Plasma activities of alanine transaminase and aspartate transaminase and plasma concentrations of triglycerides, total cholesterol, and non-esterified fatty acids were determined [13].

Statistical Analysis Data are presented as mean ± SEM. GraphPad Prism version 6.00 for Windows (San Diego, CA, USA) was used for statistical analyses of differences between the groups by one-way analysis of variance. Statistically significant variables were treated with Neumann–Keuls *post hoc* test to compare all groups of animals. $P < 0.05$ was considered statistically significant.

Results and Discussion

Physiological and Metabolic Parameters with Virgin Coconut Oil (VCO) VCO-fed HV rats had higher body weight, body weight gain, and abdominal circumference compared to C rats while these parameters were lower in HV rats compared to H rats (Table 1). Energy intake and feed conversion efficiency were higher in HV rats compared to C rats while these parameters were lower than H rats (Table 1). During oral glucose tolerance test, basal blood glucose concentration in HV rats was not different from C rats while it was lower than H rats (Table 1). Although VCO lowered basal blood glucose concentrations, it failed to improve the overall glucose tolerance as evident from the area under the curve (Table 1). Retroperitoneal, epididymal, and omental fats were higher in HV rats compared to C rats while they were not different between H and HV rats (Table 1). Total body fat and lean mass were similar in C and HV rats whereas both these parameters were also similar in H and HV rats (Table 1). Plasma total cholesterol concentrations were not different in C and HV rats while HV rats had higher total cholesterol concentrations compared to H rats (Table 1). Plasma triglyceride concentrations were higher in HV rats compared to C rats while HV and H rats had similar triglyceride concentrations (Table 1). Plasma non-esterified fatty acid concentrations were higher in HV rats compared to C rats while H and HV rats had no differences in plasma concentrations of non-esterified fatty acids (Table 1).

The responses of diet supplementation with virgin coconut oil may be due to replacement of longer-chain saturated fatty acids with medium-chain fatty acids [16]. Virgin coconut oil components may induce increased energy expenditure leading to a lower weight gain than beef tallow. The major medium-chain fatty acids in virgin coconut oil are lauric acid and myristic acid [16], which are rapidly absorbed in the intestine even without pancreatic lipase [17]. Lauric acid is more likely to be oxidized by the mitochondria than longer-chain saturated fatty acids such as stearic and palmitic acids [18]. We have also compared lauric, myristic, palmitic, and stearic acids for their effects on obesity and osteoarthritis with lauric acid markedly reducing both obesity and osteoarthritis compared

Table 1 Dietary intakes, body composition, and plasma biochemistry

Variables	*C*	*H*	*HV*	*HN*
Initial body weight (g)	340 ± 1	337 ± 1*	337 ± 1*	337 ± 2
Final body weight (g)	386 ± 8	516 ± 13a*	459 ± 10b*	512 ± 11a*
Body weight gain (%)	13.3 ± 2.3	52.2 ± 3.7a*	36.5 ± 4.1b*	51.7 ± 2.8a*
Food intake (g/day)	38.8 ± 1.4	24.4 ± 0.5a*	21.4 ± 0.8b*	24.5 ± 0.9a*
Water intake (g/day)	30.0 ± 1.5	25.9 ± 1.9b	21.7 ± 1.2b*	36.7 ± 1.5a*
Energy intake (kJ/day)	378 ± 16	524 ± 10a*	441 ± 14b*	458 ± 14b*
Feed conversion efficiency (g/kJ)	0.12 ± 0.02	0.34 ± 0.02a*	0.27 ± 0.03b*	0.38 ± 0.02a*
Abdominal circumference (cm)	18.5 ± 0.2	22.7 ± 0.5a*	20.7 ± 0.3b*	21.8 ± 0.2a*
Basal blood glucose (mmol/L)	4.2 ± 0.1	5.4 ± 0.2a*	4.6 ± 0.2b	5.2 ± 0.2a*
Area under the curve (mmol/L·minutes)	765 ± 26	866 ± 22*	862 ± 32*	893 ± 29*
Retroperitoneal fat (mg/mm)	177 ± 21	420 ± 43*	415 ± 50*	380 ± 47*
Epididymal fat (mg/mm)	120 ± 11	229 ± 15*	198 ± 20*	252 ± 19*
Omental fat (mg/mm)	134 ± 13	242 ± 24*	237 ± 19*	250 ± 22*
Total abdominal fat (mg/mm)	430 ± 41	892 ± 73*	850 ± 87*	881 ± 84*
Total body fat mass (g)	121 ± 12	235 ± 19*	176 ± 26	169 ± 15*
Total body lean mass (g)	255 ± 10	270 ± 16	256 ± 11	303 ± 15*
Fat:lean mass ratio	0.49 ± 0.07	0.81 ± 0.10*	0.71 ± 0.12	0.58 ± 0.08
Plasma total cholesterol (mmol/L)	1.6 ± 0.2	1.7 ± 0.1b	2.0 ± 0.0a	1.0 ± 0.0c*
Plasma triglyceride (mmol/L)	0.3 ± 0.1	1.1 ± 0.4	1.4 ± 0.3*	1.8 ± 0.2*
Plasma NEFA (mmol/L)	1.2 ± 0.3	3.3 ± 0.8*	4.8 ± 0.8*	5.6 ± 0.4*

All values are mean ± SEM. * indicates significantly different values compared to C. Means between H, HV and HN without a common letter are significantly different, $P < 0.05$. C, corn starch diet-fed rats; H, high-carbohydrate, high-fat diet-fed rats; HV, high-carbohydrate, high-VCO diet-fed rats; HN, high-carbohydrate, high-coconut Nourish diet-fed rats; NEFA, non-esterified fatty acids

to H rats, in contrast to palmitic and stearic acids [10]. Other studies with coconut oil have shown similar health benefits [19–21]. Previous studies with palmitic and stearic acids confirmed their role in inducing insulin resistance [22, 23]. Although lauric and myristic acids are also saturated fatty acids with similar chemical properties to palmitic and stearic acids, all saturated fatty acids cannot be considered to produce the same physiological responses [3]. Fat to lean mass ratio can predict metabolic dysfunction [24]. In our study, fat to lean mass ratio was higher in obese rats compared to healthy lean rats reflecting the metabolic disturbances in obese rats. Further, the improved fat to lean mass ratio in VCO and coconut Nourish supplemented rats suggests that these interventions improve the metabolic status of rats.

Physiological and Metabolic Parameters with Coconut Nourish HN rats had higher body weight, body weight gain, and abdominal circumference compared to C rats while these parameters were similar in HN and H rats (Table 1). Energy intake was higher in HN rats compared to C rats while it was lower than H rats (Table 1). Feed conversion efficiency was higher in HN rats compared to C rats while it was similar in H and HN rats (Table 1). During oral glucose tolerance test, basal blood glucose concentrations in HN rats were higher than C rats while it was similar to H rats (Table 1). Area under the curve was unchanged between the groups (Table 1). Retroperitoneal, epididymal, and omental fats were higher in HN rats compared to C rats while they were not different between H and HN rats (Table 1). Total body fat mass in HN rats was higher than C rats while it was similar to H rats; total body lean mass in HN rats was higher than C rats while it was similar to H rats (Table 1). Plasma total cholesterol concentrations were lower in HN rats compared to C and H rats (Table 1). Plasma triglyceride concentrations were higher in HN rats compared to C rats while HN and H rats had similar triglyceride concentrations (Table 1). Plasma non-esterified fatty acid concentrations were higher in HN rats compared to C rats while H and HN rats had no differences in plasma concentrations of non-esterified fatty acids (Table 1). In summary, replacement of beef tallow with coconut Nourish caused no change in body weight while increasing total body lean mass. Coconut Nourish contains nutrients including protein and fiber in addition to saturated fatty acids. This high content of protein may be responsible for the decreases in fat mass to lean mass ratio [25]. Moreover, the increase in lean mass occurred in the presence of lower energy intake compared to the obese rats, thus increasing the feed efficiency. Coconut Nourish is also the source of fiber that may also help in the improvements seen in these rats.

Cardiovascular and Hepatic Structure and Function with VCO Virgin coconut oil, a rich source of medium-chain fatty acids, reduced blood pressure in Spontaneously Hypertensive Rats and heated palm oil-induced hypertensive rats [26, 27]. Increased nitric oxide production may be a possible mechanism for this decrease in blood pressure [26, 27]. Fructose-induced hypertension in rodents is well-studied as this model has been used for intervention trials throughout the world [28, 29].

HV rats showed similar systolic blood pressure to C rats while it was lower in HV rats compared to H rats (Table 2). LVIDs was higher while fractional shortening and ejection fraction were lower in HV rats compared to C rats (Table 2). HV rats showed similar left ventricular diastolic stiffness to C rats while stiffness was lower in HV rats compared to H rats (Table 2). Left ventricular wet weight was similar in C and HV rats while it was lower in HV rats than H rats (Table 2). HV rats showed inhibition of infiltration of inflammatory cells (Fig. 1c) and collagen deposition (Fig. 1g) compared to H rats (Fig. 1b and f).

Livers from HV rats showed inhibition of infiltration of inflammatory cells and deposition of fat vacuoles (Fig. 1k) compared to H rats (Fig. 1j). HV rats had higher liver wet weight compared to C rats while it was not different compared to H rats (Table 2). Plasma ALP activity was higher in HV rats compared to C rats while it was unchanged compared to H rats (Table 2).

In summary, replacement of beef tallow with VCO as a source of saturated fatty acids lowered body weight, blood glucose concentrations, systolic blood pressure, and diastolic stiffness while improving structure and function of the heart and liver but without decreasing central obesity. These changes may be correlated to the lower inflammatory cell infiltration and subsequent collagen deposition in the heart and liver of virgin coconut oil-supplemented rats.

Previous studies reporting the impact of saturated fatty acids on human health concluded that lauric and myristic acids raised plasma total cholesterol concentrations, with lauric acid increasing LDL cholesterol and myristic acid increasing both LDL and HDL cholesterol concentrations [30, 31]. Lauric acid lowered the ratio of total cholesterol to HDL, while myristic acid did not change this ratio [32]. The current study found that total plasma cholesterol concentrations increased with virgin coconut oil supplementation in rats, although it cannot be confirmed if the changes were due to increases in HDL-cholesterol or LDL-cholesterol.

Cardiovascular and Hepatic Structure and Function with Coconut Nourish HN rats showed similar systolic blood pressure to C rats while it was lower in HN rats compared to H rats (Table 2). LVPWd, IVSd, and relative wall thickness were higher while fractional shortening and ejection fraction were lower in HN rats compared to C rats (Table 2). LVPWd, IVSd, and relative wall thickness were higher in HN rats while ejection time was lower compared to H rats (Table 2). HN rats showed similar diastolic stiffness to C rats while it was lower in HN rats compared to H rats (Table 2). Left ventricular wet

Table 2 Cardiovascular and hepatic structure and function

Variables	*C*	*H*	*HV*	*HN*
Cardiovascular variables				
Systolic blood pressure (mmHg)	127 ± 5	149 ± 4^{a*}	133 ± 3^{b}	130 ± 3^{b}
LVIDd (mm)	7.63 ± 0.13	8.31 ± 0.30	7.94 ± 0.21	7.76 ± 0.26
LVPWd (mm)	1.73 ± 0.08	1.79 ± 0.04^{b}	1.89 ± 0.05^{ab}	1.97 ± 0.03^{a*}
LVIDs (mm)	3.41 ± 0.22	4.34 ± 0.34*	4.40 ± 0.22*	4.00 ± 0.23
IVSd (mm)	1.71 ± 0.09	1.77 ± 0.03^{b}	1.86 ± 0.06^{ab}	1.94 ± 0.03^{a*}
Relative wall thickness	0.45 ± 0.02	0.43 ± 0.02^{b}	0.47 ± 0.02^{ab}	0.51 ± 0.02^{a*}
Fractional shortening (%)	55.6 ± 2.4	45.5 ± 3.4*	44.5 ± 1.7*	48.7 ± 1.6*
Ejection fraction (%)	90.5 ± 1.4	85.7 ± 3.0	82.8 ± 1.5*	86.3 ± 1.2*
Ejection time (ms)	83.8 ± 3.6	88.0 ± 2.3^{a}	89.0 ± 1.5^{a}	81.7 ± 2.3^{b}
Left ventricle wet weight (mg/mm)	19.7 ± 0.4	22.3 ± 0.9^{a*}	20.6 ± 0.3^{b}	23.8 ± 1.0^{a*}
Right ventricle wet weight (mg/mm)	4.28 ± 0.18	4.68 ± 0.35^{ab}	3.84 ± 0.23^{b}	5.66 ± 0.73^{a}
Diastolic stiffness constant, κ	25.0 ± 1.7	31.4 ± 1.2^{a*}	25.2 ± 2.3^{b}	26.5 ± 1.0^{b}
Hepatic variables				
Liver wet weight (mg/mm tibial length)	205 ± 18	293 ± 13^{b*}	301 ± 8^{b*}	372 ± 21^{a*}
Plasma ALP activity (U/L)	124 ± 15	196 ± 19^{a*}	222 ± 9^{a*}	152 ± 11^{b}

All values are mean ± SEM. * indicates significantly different values compared to C. Means between H, HV and HN without a common letter are significantly different, $P < 0.05$. C, corn starch diet-fed rats; H, high-carbohydrate, high-fat diet-fed rats; HV, high-carbohydrate, high-VCO diet-fed rats; HN, high-carbohydrate, high-coconut Nourish diet-fed rats; LVIDd, left ventricular internal diameter during diastole; LVPWd, left ventricular posterior wall thickness during diastole; LVIDs, left ventricular internal diameter during diastole; IVSd, interventricular septal thickness during diastole; ALP, alkaline phosphatase

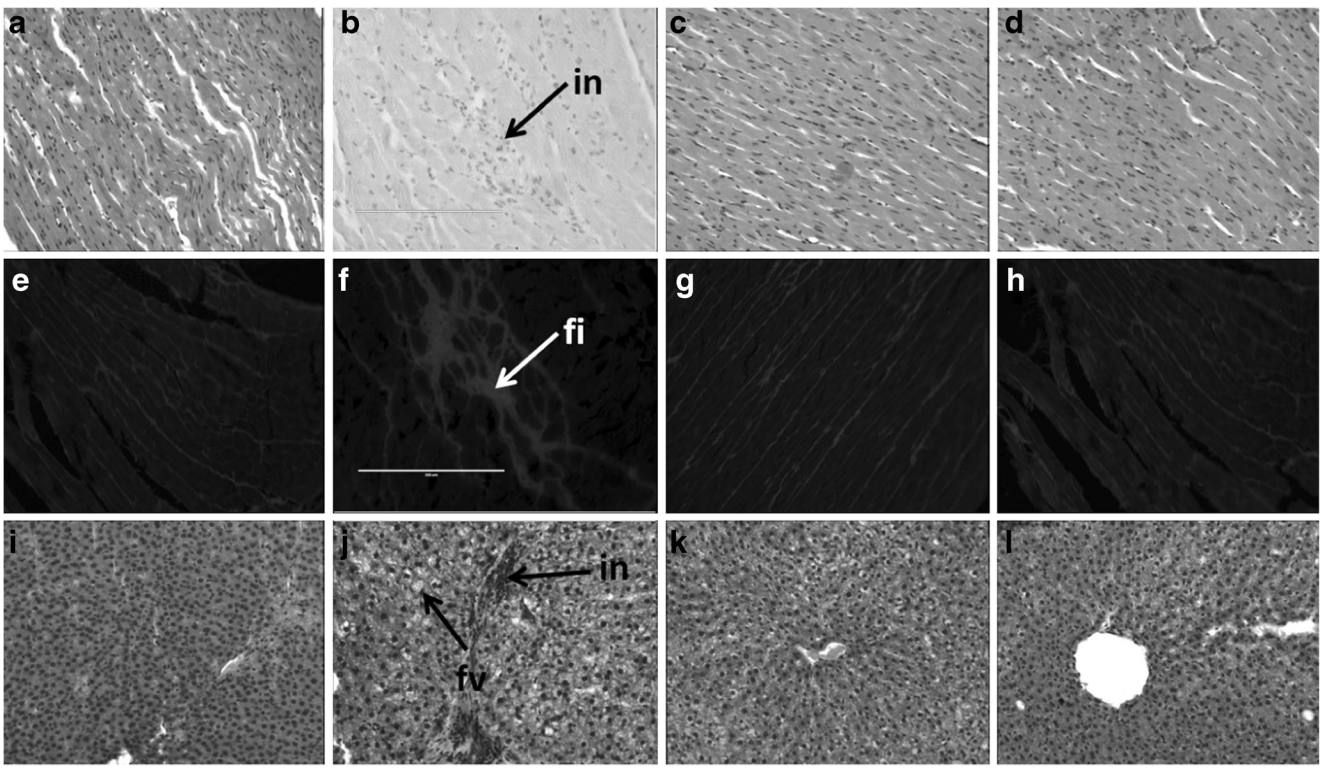

Fig. 1 Effects of coconut products on inflammation and fibrosis in the heart and inflammation and fat deposition in the liver. Haematoxylin and eosin staining of left ventricle showing inflammatory cells ('in'; ×20) from C (**a**), H (**b**), HV (**c**) and HN (**d**) rats. Picrosirius red staining of left ventricle showing fibrosis ('fi'; ×40) from C (**e**), H (**f**), HV (**g**) and HN (**h**) rats. Haematoxylin and eosin staining of liver showing inflammatory cells ('in') and fat deposition ('fv', ×20) from C (**i**), H (**j**), HV (**k**) and HN (**l**) rats. C, corn starch diet-fed rats; H, high-carbohydrate, high-fat diet-fed rats; HV, high-carbohydrate, high-VCO diet-fed rats; HN, high-carbohydrate, high-coconut Nourish diet-fed rats

weights were similar in H and HN rats but higher in HN rats compared to C rats (Table 2). HN rats showed inhibition of infiltration of inflammatory cells (Fig. 1d) with inhibition of collagen deposition (Fig. 1h). HN rats showed improved aortic responses to noradrenaline (Supplementary Figure 1A, $-\log EC_{50}$ values – C = 6.89 ± 0.18; H = 6.42 ± 0.33; HV = 6.31 ± 0.08; HN = 6.98 ± 0.17) with no change in thoracic aortic responses to sodium nitroprusside and acetylcholine (Supplementary Figure 1B and 1C).

Livers from HN rats showed inhibition of infiltration of inflammatory cells and decreased deposition of fat vacuoles (Fig. 1l) compared to H rats (Fig. 1j). HN rats had higher liver wet weight compared to C and H rats (Table 2). Plasma ALP activity was similar in C and HN rats while it was lower in HN rats compared to H rats (Table 2).

Thus, the physiological and metabolic changes with coconut Nourish were accompanied by the prevention of increases in systolic blood pressure and diastolic stiffness while improving structure and function of the heart and liver, but also by increased plasma triglyceride and non-esterified fatty acids concentrations with lowered plasma total cholesterol concentrations. Similar to virgin coconut oil, coconut Nourish was able to normalize the systolic blood pressure and left ventricular diastolic stiffness. While these changes were similar, coconut Nourish was also able to lower total cholesterol concentrations. This reduction may be attributed to the fiber content of the coconut Nourish as shown in previous study with dietary fiber reducing blood pressure and total cholesterol [33].

Coconuts are known for their great versatility for communities in the tropical and subtropical areas of the world. Coconut or coconut products as part of the regular diet along with seafood decreased cardiovascular disease risk factors in Samoan Islanders, including higher HDL-cholesterol concentrations and lower abdominal circumferences [34]. Similar observations were seen in the Kitava population when compared with the Swedish population. The regular diet of Kitava people includes tubers, coconuts, and seafood and this Kitava population is free from overweight, hypertension, cardiovascular disease, and malnutrition [35]. It is important that the extent of health benefits with coconut and coconut products is explored further to establish the role of coconut as a functional food. These results can then form the basis for controlled human trials to identify translatable responses of coconuts.

Conclusions

Both virgin coconut oil and coconut Nourish contain high amounts of lauric and myristic acids while coconut Nourish also contains increased fiber and protein. When beef tallow was replaced with virgin coconut oil or coconut Nourish in a high-carbohydrate, high-fat diet, the increases in systolic blood pressure and diastolic stiffness in the heart were inhibited. Moreover, the coconut Nourish increased the lean mass in rats even with decreased energy intake. Virgin coconut oil reduced the fasting blood glucose concentrations while Nourish was able to reduce the total cholesterol concentrations in plasma. Both the virgin coconut oil- and Nourish-supplemented diets were rich in fructose and sucrose content, so these results are quite relevant to overweight and obese individuals who consume diets rich in simple sugars.

Acknowledgements This study was funded by Enterprise Connect and Nature Pacific Pty Ltd. as part of the *Researcher in Business* program. Virgin coconut oil and Banaban Nourish were provided by Nature Pacific Pty Ltd., Gold Coast, QLD, Australia as part of this program. We thank Mr. Brian Bynon, School of Veterinary Sciences, The University of Queensland Gatton, for plasma biochemical analyses.

Author's Contributions S.K.P. and L.B. designed the research protocol and interpreted the results; S.K.P. and S.C. conducted the animal experiments and analyzed the data; S.K.P., S.C. and L.B. wrote the manuscript; and S.K.P. had primary responsibility for final content. All authors read and approved the final manuscript.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Statement on the Welfare of Animals All experimental protocols were approved by the Animal Ethics Committee of the University of Southern Queensland under the guidelines of the National Health and Medical Research Council of Australia. The approval number for this study was 13REA008. This article does not contain any studies with human participants performed by any of the authors.

References

1. O'Neill S, O'Driscoll L (2015) Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies. Obes Rev 16: 1–12
2. Hammad S, Pu S, Jones PJ (2016) Current evidence supporting the link between dietary fatty acids and cardiovascular disease. Lipids 51:507–517
3. Poudyal H, Brown L (2015) Should the pharmacological actions of dietary fatty acids in cardiometabolic disorders be classified based on biological or chemical function? Prog Lipid Res 59:172–200
4. DebMandal M, Mandal S (2011) Coconut (*Cocos nucifera* L.: *Arecaceae*): in health promotion and disease prevention. Asian Pac J Trop Med 4:241–247
5. St-Onge M-P, Jones PJH (2002) Physiological effects of medium-chain triglycerides: potential agents in the prevention of obesity. J Nutr 132:329–332
6. Katragadda HR, Fullana A, Sidhu S, Carbonell-Barrachina ÁA (2010) Emissions of volatile aldehydes from heated cooking oils. Food Chem 120:59–65
7. Lima EB, Sousa CN, Meneses LN, Ximenes NC, Santos Junior MA, Vasconcelos GS et al (2015) *Cocos nucifera* (L.) (*Arecaceae*): a phytochemical and pharmacological review. Braz J Med Biol Res 48:953–964
8. Akinpelu DA, Alayande KA, Aiyegoro OA, Akinpelu OF, Okoh AI (2015) Probable mechanisms of biocidal action of *Cocos*

nucifera husk extract and fractions on bacteria isolates. BMC Complement Altern Med 15:116
9. Lima EB, de Sousa CN, Vasconcelos GS, Meneses LN, E Silva Pereira YF, Ximenes NC et al (2016) Antidepressant, antioxidant and neurotrophic properties of the standardized extract of *Cocos nucifera* husk fiber in mice. J Nat Med 70:510–521
10. Sekar S, Shafie SR, Prasadam I, Crawford R, Panchal SK, Brown L et al (2017) Saturated fatty acids induce development of both metabolic syndrome and osteoarthritis in rats. Sci Rep 7:46457
11. Eyres L, Eyres MF, Chisholm A, Brown RC (2016) Coconut oil consumption and cardiovascular risk factors in humans. Nutr Rev 74:267–280
12. Rippe JM, Angelopoulos TJ (2013) Sucrose, high-fructose corn syrup, and fructose, their metabolism and potential health effects: what do we really know? Adv Nutr 4:236–245
13. Panchal SK, Poudyal H, Iyer A, Nazer R, Alam MA, Diwan V et al (2011) High-carbohydrate, high-fat diet-induced metabolic syndrome and cardiovascular remodeling in rats. J Cardiovasc Pharmacol 57:611–624
14. Poudyal H, Panchal SK, Waanders J, Ward L, Brown L (2012) Lipid redistribution by *a*-linolenic acid-rich chia seed inhibits stearoyl-CoA desaturase-1 and induces cardiac and hepatic protection in diet-induced obese rats. J Nutr Biochem 23:153–162
15. Brown L, Poudyal H, Panchal SK (2015) Functional foods as potential therapeutic options for metabolic syndrome. Obes Rev 16: 914–941
16. Marina AM, Che Man YB, Amin I (2009) Virgin coconut oil: emerging functional food oil. Trends Food Sci Technol 20:481–487
17. Liau KM, Lee YY, Chen CK, Rasool AHG (2011) An open-label pilot study to assess the efficacy and safety of virgin coconut oil in reducing visceral adiposity. ISRN Pharmacol 2011:949686
18. Delany JP, Windhauser MM, Champagne CM, Bray GA (2000) Differential oxidation of individual dietary fatty acids in humans. Am J Clin Nutr 72:905–911
19. Lekshmi Sheela D, Nazeem PA, Narayanankutty A, Manalil JJ, Raghavamenon AC (2016) *In silico* and wet lab studies reveal the cholesterol lowering efficacy of lauric acid, a medium chain fat of coconut oil. Plant Foods Hum Nutr 71:410–415
20. Narayanankutty A, Palliyil DM, Kuruvilla K, Raghavamenon AC (2017) Virgin coconut oil reverses hepatic steatosis by restoring redox homeostasis and lipid metabolism in male Wistar rats. J Sci Food Agric. https://doi.org/10.1002/jsfa.8650
21. Famurewa AC, Ekeleme-Egedigwe CA, Nwali SC, Agbo NN, Obi JN, Ezechukwu GC (2017) Dietary supplementation with virgin coconut oil improves lipid profile and hepatic antioxidant status and has potential benefits on cardiovascular risk indices in normal rats. J Diet Suppl. https://doi.org/10.1080/19390211.2017.1346031
22. Shinjo S, Jiang S, Nameta M, Suzuki T, Kanai M, Nomura Y et al (2017) Disruption of the mitochondria-associated ER membrane (MAM) plays a central role in palmitic acid–induced insulin resistance. Exp Cell Res 359:86–93
23. Hirabara SM, Curi R, Maechler P (2010) Saturated fatty acid-induced insulin resistance is associated with mitochondrial dysfunction in skeletal muscle cells. J Cell Physiol 222: 187–194
24. Ezeh U, Pall M, Mathur R, Azziz R (2014) Association of fat to lean mass ratio with metabolic dysfunction in women with polycystic ovary syndrome. Hum Reprod 29:1508–1517
25. Blouet C, Mariotti F, Azzout-Marniche D, Bos C, Mathé V, Tomé D et al (2006) The reduced energy intake of rats fed a high-protein low-carbohydrate diet explains the lower fat deposition, but macronutrient substitution accounts for the improved glycemic control. J Nutr 136:1849–1854
26. Alves NF, Porpino SK, Monteiro MM, Gomes ER, Braga VA (2015) Coconut oil supplementation and physical exercise improves baroreflex sensitivity and oxidative stress in hypertensive rats. Appl Physiol Nutr Metab 40:393–400
27. Kamisah Y, Periyah V, Lee KT, Noor-Izwan N, Nurul-Hamizah A, Nurul-Iman BS et al (2015) Cardioprotective effect of virgin coconut oil in heated palm oil diet-induced hypertensive rats. Pharm Biol 53:1243–1249
28. Tran LT, Yuen VG, Mcneill JH (2009) The fructose-fed rat: a review on the mechanisms of fructose-induced insulin resistance and hypertension. Mol Cell Biochem 332:145–159
29. Klein AV, Kiat H (2015) The mechanisms underlying fructose-induced hypertension: a review. J Hypertens 33:912–920
30. Denke MA, Grundy SM (1992) Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. Am J Clin Nutr 56:895–898
31. Zock PL, De Vries JH, Katan MB (1994) Impact of myristic acid versus palmitic acid on serum lipid and lipoprotein levels in healthy women and men. Arterioscler Thromb 14:567–575
32. Lawrence GD (2013) Dietary fats and health: dietary recommendations in the context of scientific evidence. Adv Nutr 4:294–302
33. Bagger M, Andersen O, Nielsen JB, Ryttig KR (1996) Dietary fibres reduce blood pressure, serum total cholesterol and platelet aggregation in rats. Br J Nutr 75:483–493
34. Dibello JR, Mcgarvey ST, Kraft P, Goldberg R, Campos H, Quested C et al (2009) Dietary patterns are associated with metabolic syndrome in adult samoans. J Nutr 139:1933–1943
35. Lindeberg S, Eliasson M, Lindahl B, Ahrén B (1999) Low serum insulin in traditional pacific islanders-the Kitava study. Metabolism 48:1216–1219