

**EFFECT OF CEREALS AND NATA DE COCO
SUPPLEMENTATION IN HYPERLIPIDEMIA**

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EFFECT OF CEREALS AND NATA DE COCO SUPPLEMENTATION IN
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ABSTRACT

The purpose of this study was to evaluate the effect of the cereals and nata de coco supplementation on lipid status, serum vitamin A, β -carotene, vitamin E levels, lipid peroxidation and its acceptability, tolerance, compliance and safety in twenty two subjects with hyperlipidemia. Subjects consisted of eleven men and eleven women aged 32-75 yr, and had serum TC level of ≥ 5.2 mmol/L, serum TG level of ≥ 1.69 mmol/L and LDL-C level of ≥ 3.4 mmol/dL. The study consisted of four wks of control and twenty wks of supplementation period. They were supplemented twice daily with 15g of the supplement for twenty wks. This 30g of supplement provided 122.6 kcal, 5.5g of protein, 0.5g of fat and 24.1g of carbohydrate and 2.76g of fiber. After twenty wks, the subjects were classified into two groups, according to their compliance, **group A**; compliance ≥ 90 % of assigned supplement intake and **group B**; compliance < 90 % of assigned supplement intake.

In group A significant differences were seen between the mean TG level at wk zero and the mean values at wk four, eight, and twelve ($p < 0.05$) but no significant differences were seen in TC, LDL-C and HDL-C except TC level at wk sixteen was significantly lower than that at wk zero ($p < 0.05$). All subjects had normal serum vitamin A, β -carotene and vitamin E levels during the study and had no significant differences from wk zero. Serum MDA concentration of the twenty two subjects at wk four, eight, twelve, sixteen and twenty were significantly decreased from those at wk zero. ($p < 0.05$)

KEY WORDS: SUPPLEMENT / DIETARY FIBER / HYPERLIPIDEMIA /
VITAMIN A / VITAMIN E / LIPID PEROXIDATION

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ผลของการเสริมผลิตภัณฑ์อาหารจากธัญพืชและวุ้นน้ำมะพร้าวในผู้ป่วยไขมันในเลือดสูง
(EFFECT OF CEREALS AND NATA DE COCO SUPPLEMENTATION IN
HYPERLIPIDEMIA)

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บทคัดย่อ

การศึกษาครั้งนี้มีวัตถุประสงค์ เพื่อประเมินผลของการเสริมผลิตภัณฑ์อาหารจากธัญพืช และวุ้นน้ำมะพร้าวต่อภาวะโภชนาการด้านไขมัน, ระดับวิตามินเอ, เบต้า - แคโรทีน, วิตามินอี ในเลือด และลิปิดเพอร์ออกซิเดชั่น ตลอดจนการยอมรับผลิตภัณฑ์ของผู้ป่วย และความปลอดภัยของผลิตภัณฑ์ของผู้ป่วยภาวะไขมันในเลือดสูง จำนวน 22 คน ซึ่งประกอบด้วย ผู้ชาย 11 คน และผู้หญิง 11 คน อายุระหว่าง 32 – 75 ปี ผู้ป่วยทุกคนมีระดับโคเลสเตอรอลในซีรัม ≥ 5.2 มิลลิโมล/ลิตร, ระดับไตรกลีเซอไรด์ ≥ 1.69 มิลลิโมล/ลิตร และแอล ดีแอล โคเลสเตอรอล ≥ 3.4 มิลลิโมล/ลิตร ระยะเวลาศึกษาทั้งสิ้น 24 สัปดาห์ ประกอบด้วย 4 สัปดาห์แรกเป็นระยะควบคุม และ 20 สัปดาห์ต่อมาเป็นระยะเสริมผลิตภัณฑ์อาหาร ระหว่างสัปดาห์ที่ 0 ถึงสัปดาห์ที่ 20 ให้ผู้ป่วยดื่มผลิตภัณฑ์อาหารจากธัญพืชและวุ้นน้ำมะพร้าว วันละ 2 ครั้ง ก่อนมื้ออาหารเช้าและเย็น โดยแต่ละครั้งละลายผงผลิตภัณฑ์อาหาร 15 กรัม ในน้ำอุ่นให้ได้ปริมาตรครบ 150 มล. ผลิตภัณฑ์อาหารจากธัญพืชและวุ้นน้ำมะพร้าว 30 กรัมให้พลังงาน 122.6 กิโลแคลอรี, โปรตีน 5.5 กรัม, ไขมัน 0.5 กรัม, คาร์โบไฮเดรต 24.1 กรัม และใยอาหาร 2.76 กรัม

หลังจากได้รับผลิตภัณฑ์อาหารเป็นเวลา 20 สัปดาห์ ได้แบ่งอาสาสมัครออกเป็น 2 กลุ่มคือ กลุ่มที่รับประทานผลิตภัณฑ์อาหารมากกว่า 90 % ของปริมาณที่กำหนด มี 15 คน (กลุ่ม A) และกลุ่มที่รับประทานผลิตภัณฑ์อาหารน้อยกว่า 90 % ของปริมาณที่กำหนด มี 7 คน (กลุ่ม B)

อาสาสมัครในกลุ่ม A มีค่าดัชนีของระดับไตรกลีเซอไรด์ในซีรัมลดลงจากระดับก่อนได้รับการรักษาอย่างมีนัยสำคัญทางสถิติยกเว้นสัปดาห์ที่ 16 และ 20 แต่ไม่พบการเปลี่ยนแปลงอย่างมีนัยสำคัญของค่าดัชนีของระดับโคเลสเตอรอล, แอล ดีแอล โคเลสเตอรอล และแอล ดีแอล โคเลสเตอรอลในอาสาสมัครทุกคน ยกเว้นในกลุ่ม A ระดับโคเลสเตอรอล สัปดาห์ที่ 16 ลดลงอย่างมีนัยสำคัญเมื่อเทียบกับก่อนได้รับการรักษา ทั้งกลุ่ม A และกลุ่ม B ไม่พบการเปลี่ยนแปลงของระดับวิตามินเอ, เบต้า - แคโรทีน, วิตามินอี ในเลือด และระดับวิตามินดังกล่าวอยู่ในเกณฑ์ปกติ การศึกษาพบว่าผู้ป่วยทั้ง 2 กลุ่ม ซึ่งได้รับผลิตภัณฑ์อาหารจากธัญพืชและวุ้นน้ำมะพร้าว วันละ 30 กรัม เป็นเวลา 20 สัปดาห์ มีระดับมาดอนดีแอลดีไฮด์ในซีรัมลดลงจากระดับก่อนได้รับการรักษาอย่างมีนัยสำคัญในสัปดาห์ที่ 4, 8, 12, 16 และ 20

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LIST OF ABBREVIATIONS

ALT	Alanine aminotransferase
AP	Alkaline phosphatase
AST	Aspartate aminotransferase
BFM	Body fat mass
BMI	Body mass index
BP	Blood pressure
BST	Biceps skinfold thickness
CHD	Coronary heart disease
CHO	Carbohydrate
CRBP	Cellular retinol – binding protein
d	day
DF	Dietary fiber
dL	Deciliter
FBG	Fasting blood glucose
FFM	Fat free mass
g	gram
GGT	Gamma glutamyl transferase
HDL-C	High density lipoprotein-cholesterol
Ht	Height
IBW	Ideal body weight
IDF	Insoluble dietary fiber
kg	kilogram
LDL-C	Low density lipoprotein-cholesterol
m	meter
MDA	Malondialdehyde
mg	milligram
mm	millimeter
MUAC	Mid upper arm circumference
RBP	Retinol – binding protein

LIST OF ABBREVIATIONS (CONT.)

RDA	Recommended dietary allowance
SCFAs	Short – chain fatty acids
SDF	Soluble dietary fiber
SEM	Standard error of mean
SIT	Supra iliac skinfold thickness
SST	Sub scapula skinfold thickness
TBA	Thiobarbituric acid
TBW	Total body water
TC	Total cholesterol
TDF	Total dietary fiber
TG	Triglyceride
TST	Triceps skinfold thickness
UAMC	Upper arm muscle circumference
WHC	Water holding capacity
WHR	Waist over hip circumference ratio
wk	Week
Wt	Weight
yr	Year

CHAPTER I

INTRODUCTION

Dietary fiber (DF) is the portion of plant cells that cannot be digested by human alimentary enzymes and cannot be absorbed from small bowel but are partly hydrolyzed by bacteria in the colon. In general, dietary fiber can be divided into 2 types according to its solubility: water soluble dietary fiber and water insoluble dietary fiber. Each type has different physiological effect. Water insoluble dietary fiber is associated with the reduction of blood cholesterol and the diminution in the intestinal absorption of glucose, whereas the water insoluble dietary fiber is related to both water absorption and intestinal regulation. Several studies suggest that a high intake of dietary fiber from cereals is associated with a low risk of coronary heart disease (CHD) (7-13).

Recently, the eating pattern of Thai people has changed towards the consumption style of ready-to-eat and convenient western diets. That diets including fast food and prepared foods such as hamburger, pizza, fried chicken and bakery products (14). The reason for their popularity is they are easier to prepare and consume when compared with the long hours of preparation required for traditional Thai food (15). The changing consumption pattern can result in the growth of nutrition problems since these foods often provide high fat, sugar, refined carbohydrate and energy but low dietary fiber (16-18). According to one study on the dietary fiber consumption of Thai adolescents in Bangkok, the average intake of dietary fiber in males was only 7.32 g/day (mostly obtained from grains) and the average intake of dietary fiber in females was only 8.88 g/day (mostly obtained from fruits). This study showed that the dietary fiber intake of adolescents was lower than the recommended amount of dietary fiber (19).

Numerous health organizations suggest increasing the consumption of dietary fiber, with specific recommendations of 25-30 g/day (20). It is difficult to meet daily requirement because fruits and vegetable, which are the main sources of dietary fiber for Thai people, contains only 1-3 % of total weight and large consumption of them are needed to be ingested. The formulation of high fiber products, which include a number of high fiber ingredients such as wheat bran, oat bran, corn bran, cellulose and so forth, may be another alternative. However, most of these ingredients are imported into Thailand and some of them are expensive and they may not be appropriate for Thai tastes (21). In Thailand, several agricultural products have a potential for use as dietary fiber source and should be investigated.

Source of dietary fiber in our study are plenty plant raw material agricultural products in Thailand such as cereal and legume; sweet corn, unpolished rice, mung bean and nata de coco for processing in form of supplement food. Cereals and legume intake is reportedly associated with the reduction of blood cholesterol and triglyceride.

Study of David JA Jenkins et al (7) The authors assessed the effect of leguminous seeds in the dietary management of 7 male with hyperlipidemia. All subjects were substituted approximately 140 grams of dried beans daily for other sources of starch in their diet over a 4-month period. After this the energy intake and body weight in these subjects were held constant. However, the results revealed that high-legume diet significantly reduced serum triglyceride and serum cholesterol levels. However, LDL-C and HDL-C levels remained unaltered.

Study of Janet R Mahalko et al (160), the effects of consuming fiber from corn bran, soy hulls, or apple powder on glucose tolerance and plasma lipids were investigated in 2 studies of patients with type II diabetes. Patients with diabetes were given control low fiber bread or bread containing one of three fiber sources for incorporation into their usual self-selected diets. The fiber sources used were soy hulls, corn bran, or dehydrated powdered apple, substituted for 25 % of the white wheat flour in bread. The 10 subjects in study A consumed an average of 26 grams of fiber source/d. incorporated into 7 slices of bread. The eight subjects in study B consumed an average of 52 grams of fiber source/d, half incorporated into 7 slices of bread, and half into other foods in their diets. Three subjects were in both studies. This study reported that soy hull consumption slightly improved some measures of glucose

tolerance with results varying between the studies whereas consumption of 52 grams of corn bran decreased VLDL-C, TG and glycosylated Hb, but subject tolerance was poor. In addition, consumption of 52 grams of apple powder increased LDL-C and TC levels.

Rice bran is a unique cereal bran because of the amount and type of fiber and the amount of fat. Rice bran contains predominantly insoluble fiber but is much lower in fiber (181 g/kg) than either corn bran (890 g/kg) or wheat bran (453 g/kg), the other two cereals bran rich in insoluble fiber. Rice bran has been shown to have cholesterol-lowering activity in hypercholesterolemic animals and human subjects (165). Proposed mechanisms of cholesterol-lowering by rice bran include fecal excretion of fat, cholesterol, and bile acids. Soluble fiber and viscosity do not appear to play a significant role in the hypocholesterolemic activity of rice bran.

Study of Raghuram and coworkers (161) reported a significant reduction in serum TC in 15 and 30 days when 12 hypercholesterolemic subjects replaced their customary cooking oil with rice bran oil. Kestin and coworkers (162) observed a significant increase in HDL-C / TC ratio after 4 weeks of feeding 11.8 grams of dietary fiber from rice bran or oat bran (total nonstarch-polysaccharides, 21 g/d) to mildly hypercholesterolemic free-living men. Hegsted and coworkers (163) reported that consumption of stabilized rice bran (100 g/d) for 2 and 3 weeks periods resulted in a 4-10 % reduction in TC in moderately hypercholesterolemic subjects fed diets containing 37 % of calories as fat. In normocholesterolemic men, feeding rice bran (15 or 30 g/d) for 3 weeks resulted in non-significant plasma cholesterol reductions (164).

Nata de coco is an organic high dietary fiber food product, cultivated by bacterial fermentation action on coconut water. It is high in cellulose, low in fat and calories and contains no cholesterol. Nata de coco is beneficial effects to control weight, protect diverticular disease and cancer of colon and rectum.

From the study by Mesomya W. et al (24), they have developed food product from plant raw material agricultural in Thailand such as cereals and legume; sweet corn, unpolished rice, mung bean and nata de coco for processing in form of health food, source of dietary fiber then evaluates the serum lipid lowering effect compare with health food from Bangkok market in the experimental rat. The result from study by Mesomya W. et al, reported that Formula 2 of health food product from cereals

(6 % unpolished rice, 18 % mung bean, 30 % sweet corn) and 40 % nata de coco was significant lower the serum triglyceride level in the experimental rats.

In addition, some studies on the effect of dietary fiber on vitamin absorption have been conducted for most vitamins. Due to the close relationship between the absorption of triglyceride and fat-soluble vitamins, disorders of fat absorption are invariably always associated with a disorder of the absorption of fat-soluble vitamins. Especially, vitamin A and vitamin E are interesting because of their antioxidant activity. Although differences in the type, duration of intake, amount of fiber fed and the methods for determining uptake make comparisons across studies difficult, it appears that generally fiber has little effect on vitamin absorption (22). Some evidences suggested that high dietary fiber diet reduced the antioxidative activity of a carotenoid and alpha-tocopherol mixture on LDL oxidation *ex vivo* in human. (23)

So, the purpose of this research is to study the effect of the cereals and nata de coco supplementation in patients with hyperlipidemia on serum lipid level, vitamin A, E concentrations and determine of lipid peroxidation as an indication of antioxidant activity of vitamin A and vitamin E. The result from this study can be used as a guideline advising hyperlipidemic patients and general people who have problem on serum lipid levels.

OBJECTIVES

General objective

The purpose of this study is to study the effect of the cereals and nata de coco supplementation in patients with hyperlipidemia.

Specific objectives

This study aims to evaluate

1. The effects of the cereals and nata de coco supplementation on serum lipid, serum vitamin A, E and lipid peroxidation.
2. The acceptability, tolerance and compliance of the cereals and nata de coco supplement.

CHAPTER II

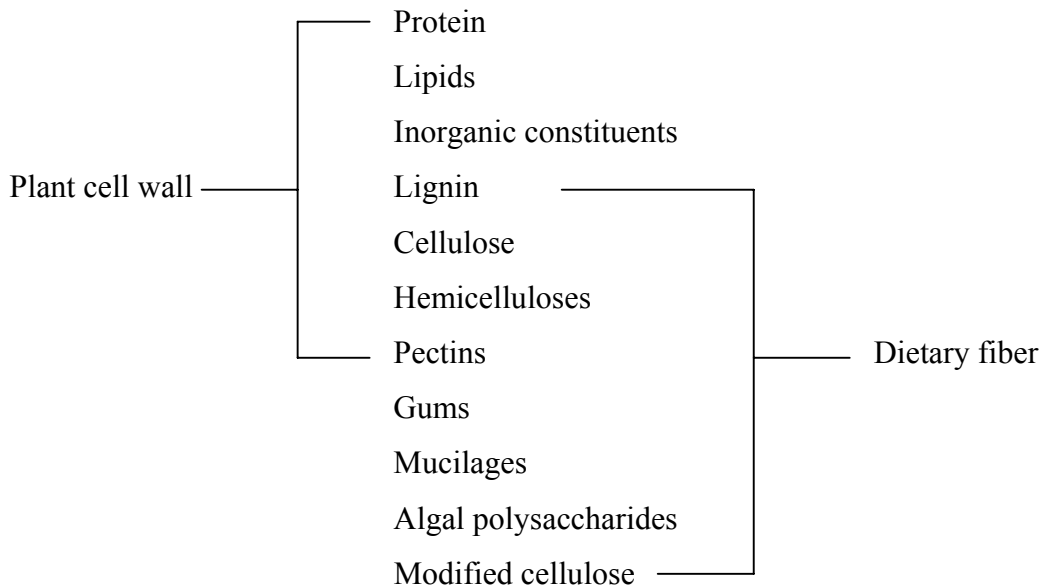
LITERATURE REVIEW

2.1 Dietary fiber

2.1.1 Definition

Hipsley first used the term “Dietary Fiber (DF)” in 1953 to describe plant cell walls in the diet (25), and later other investigators expanded this definition. Previously, the term crude fiber was popularly used. It is the portion of plant foods resistant to hydrolysis by acid and subsequently by alkali (26). It includes only celluloses, some hemicelluloses and lignin. Most of the fiber values in food composition tables are in the amount of crude fiber that are less than the amount of total dietary fiber (TDF) because of the loss of some fiber components during the analysis with digesting by acid or alkaline (27). Trowell, in 1972, wishing to show the inadequacy of crude fiber figures in foods nutrition tables and the importance of analyzing all the nondigestible plant cell wall materials in the diets, defined DF as “the remnants of the plant cell wall that are not digested by human alimentary enzymes” (4). This definition did not include polysaccharides present in some food additives such as plant gum, algal polysaccharides, pectin, modified cellulose, and modified starches. This was subsequently modified to include all plant polysaccharides and lignin that are not digested by endogenous secretions of the human digestive tract (28). The physiological definition is most widely accepted, whereas some researchers also considered a purely chemical definition, i.e. non-starch polysaccharide and lignin (29). For practical purposes, this simplified chemical definition may also be acceptable (30).

DF is not a single entity but a complex mixture of substances and different in chemistry (31-33). It is composed of a variety of polysaccharides including cellulose, hemicelluloses, pectin, gum, mucilage, algal polysaccharide (e.g. agar and carrageenan) and lignin which is a nonpolysaccharide (31, 34-36). The relationship between the plant cell wall and the current dietary fiber definition is shown in Fig 1.

Fig 1 Relationship between the plant cell wall and dietary fiber

From: Asp N-G. Definition and analysis of dietary fiber. Scand J Gastroenterol 1987; 22 (suppl 129): 16-20 (31)

Superficially, the concept of DF involves a material found in or associated with food that is not digested and does not provide energy (calories) or building blocks for the structural growth and maintenance of consuming organism (37). Furthermore, accepting a physiological definition rather than a chemical definition, the most popular definition for DF is that it is the portion of plant cells that cannot be digested by human alimentary enzymes and cannot be absorbed from small bowel but are partly hydrolyzed by bacteria in the colon (31, 40).

Currently, a variety of definitions of DF are worldwide. Based on the Food and Nutrition Board, 2001, the following definitions have been developed: *Dietary Fiber consists of nondigestible carbohydrates and lignin that are intrinsic and intact in plants. Functional Fiber consists of isolated, nondigestible carbohydrates that have beneficial physiological effects in humans. Total Fiber is the sum of Dietary Fiber and Functional Fiber* (39).

2.1.2 Classification of DF and its component

As described, DF does not have a defined composition because the quantities of component vary in different parts of plant cell and vary from source to source. Therefore, the components of DF can be categorized into several types depending on the basis of classification.

Based on water solubility, DF can be divided into 2 groups: insoluble dietary fiber (IDF) and soluble dietary fiber (SDF). Unlike the IDF, SDF is soluble in warm or hot water, but is reprecipitated when that water is mixed with four parts of ethanol. Most foods of plant origin contain both SDF and IDF but they may be rich in one type of fiber or another, and each have individual chemical characteristics and physiological effects (33, 37, 40-41). **Table 1** shows the soluble and insoluble components of DF.

Table 1 Summarizes the soluble and insoluble components of DF (31, 32)

Insoluble components of DF	Soluble components of DF
Cellulose	Pectin
Hemicellulose	β – glucan
Lignin	Gums
Cutin	Mucilages

2.1.2.1 Insoluble dietary fiber

Cellulose: Cellulose is the main structural component of plant cell walls. It is insoluble not only in cold or hot water, but also in hot dilute acids and alkalis as well. Its primary structure is that of an unbranched 1-4 -D-glucose polymer containing about 3,000 glucose units, although values up to 100,000 have been suggested (42). However, it may be of much higher molecular weight since extraction procedure used to purify it is likely to lead to breakdown of the larger polymers. The structure of cellulose is stabilized by extensive hydrogen bonding both inter- and unbranched nature of the cellulose polymer, it is able to pack together quite closely in a three dimensional latticework, forming microfibrils of cellulose. However, some

portions of cellulose are amorphous cellulose that can adsorb water. Because of its chemical makeup, cellulose can be purified for use as food ingredient (37, 41, 43, 44).

Hemicellulose: The hemicellulose component of plant cell wall consists of a wide variety of polysaccharide polymers that contain a mixture of pentose and hexose sugars and many of which are branched. Most contain between two and four different sugars, the ones commonly present being xylose, arabinose, mannose, galactose, glucose, rhamnose and galacturonic and glucuronic acids. Hemicellulose molecules are usually much smaller than cellulose, with between 150 and 200 sugar units. They are also more amorphous than cellulose molecules. In contrast with cellulose, hemicellulose can be dissolved in dilute alkali or hot water (1, 41, 42, 45).

Lignin: Lignin is a highly complex nonpolysaccharide polymer that contains phenylpropane units derived from phenolics such as sinapyl, coniferyl, cinnamyl and p-coumaryl alcohols (46). It is a small polymer having a molecular weight of between 1,000 and 4,500. The basis unit of the polymer is joined by carbon-to-carbon bonds. Lignin is highly insoluble, being a major part of the residue left after treatment of cell wall with 72 % sulfuric acid (47).

Cutin and plant waxes: These hydrophobic liquid materials are typically found in the plant structure, closely associated with the structural polysaccharides or on the outer surface of the plants. They usually present in small quantity (37).

2.1.2.2 Soluble dietary fiber

Pectins: The term pectins, pectic substances, pectic acid, and pectinic acid are all used to describe various groups of pectins. Most are heteropolysaccharides containing mainly galacturonic acid, galactose, arabinose, xylose, rhamnose and fucose (42). Pectins are primarily water-soluble, their solubility being somewhat dependent on the degree of esterification of the galacturonic acid as well as the makeup of the constituent side chain (37).

Beta-glucans: are glucose polymers containing both β -1, 3 links as well as β -1, 4 links in various proportion depending on source which make molecule less linear than cellulose and more soluble in water (37, 48). Glucans with higher

molecular weight and greater proportion of β -1, 4 linkages β -1, 4 link tend to be insoluble in water, but the lower molecular weight species with more β -1, 3 linkages are soluble (48).

Gum: Most of gums come from plant materials that may be either endogenous or exogenous gums (exudates) and the others are products of microbial biosynthesis and chemical modification of natural polysaccharides (49). The molecular structure of gums comprises a long polysaccharide chain with numerous side branches of sugars or oligosaccharides. Many different saccharides are found in gums including hexose, glucose, mannose, and galactose as well as pentose, arabinose, xylose, and rhamnose. Food gums have very unique functionalities that can improve processing and eating characteristics when formulating high-fiber foods (37, 50, 51).

Mucilages and algal polysaccharides: Mucilages are a mixed group of complex polysaccharides that are often associated with the endosperm or storage polysaccharides of plant seeds. They are usually neutral polysaccharide (42). Algal polysaccharides are found in algae and seaweeds such as agar, carrageenan, and alginate (52).

2.1.3 Physio-chemical properties of dietary fiber (1, 53)

For understanding of the role of DF in human body, it is necessary to know the properties of each DF component. Some DF sources possess unique properties that appear to have a relationship to physiological response. Some of these properties and the physiological response elicited by specific fiber fractions are described below and summarized in **Table 2**.

Table 2 Physiological responses affected by the physical properties of DF

Physical property	DF fraction	Physiological response
Bacterial degradation	Polysaccharides	Production of SCFAs, flatulence, and acidity
Water-holding capacity	Polysaccharides with polar groups	Effect on nutrient absorption, fecal weight, and rate of transit in stomach and small intestine
Cation exchange	Acidic Polysaccharides	Increase in mineral excretion
Absorption of organic materials	Lignin Pectin	Binding and excretion of bile acid

2.1.3.1 Bacterial degradation (1, 54-58)

DF cannot be enzymatically degraded in the human small intestine. However, it is fermented to varying degrees by the microflora which naturally occur in the large intestine. The degree of degradation varies considerably among the polysaccharides and depends on many factors such as types, components and polysaccharide structure of DF, water-holding capacity, physical structure of plant and kinds of bacterial flora in large intestine. For example, pectins, mucilages, and gums appear to be completely degraded, whereas, cellulose is only partially broken down. Furthermore, DF from fruits and vegetables appear to be more fermentable than that from cereals and nuts. The extent of bacterial degradation has several potential consequences;

1. Short-chain fatty acids (SCFAs) produced during bacterial metabolism may influence physiological responses to fiber, for example, SCFAs can be used by cells in the colon for energy, and absorption of SCFAs may influence hepatic metabolism of lipid and glucose.
2. The fermentation process may lower the pH of the large bowel and affect the activity of bacterial enzymes.
3. Bacterial cell mass can account for a significant portion of the fecal weight and thus contribute to fecal bulk (1, 59, 60).

2.1.3.2. Water-holding capacity (WHC) (41, 1, 54, 57-62)

WHC measures the ability of DF to hold water and is related to solubility of the polysaccharides. It is significantly enhanced on polysaccharides by the presence of sugar residue with free polar groups. Cellulose and lignin are insoluble and have a relatively low WHC. In contrast, pectins, gums, β -glucans, mucilages and some hemicelluloses have a high WHC. Hydration of the DF results in the formation of the gel matrix. This can raise the viscosity of gastrointestinal contents and partition some water soluble nutrients into the gel matrix and, as a consequence, slow gastric emptying and the diffusion and absorption of the nutrients. Although WHC has also been related to increase fecal bulk, the relationship is not straightforward because of the bacterial degradation of DF within the colon. Typically a higher WHC is

associated with greater fermentability of the fiber sources by allowing greater penetration of microbes into polysaccharide structure.

2.1.3.3. Adsorption of organic materials (1, 41, 54, 57, 61)

Adsorption of organic materials including bile acids, cholesterol, and toxic compounds is the third interesting physical property of DF. *In vitro* studies have demonstrated that lignin is an effective bile acid adsorbent. Pectin and other acidic polysaccharides also seem to sequester bile acids. Cellulose, in contrast, has little bile acid binding ability. Bile acid adsorption is measured *in vivo* as the ability to increase fecal bile acid and steroid excretion. The ability to increase fecal bile acid excretion has been correlated to the plasma cholesterol-lowering effect of certain soluble, noncellulose polysaccharides, such as pectin, oat bran, guar gum, and psyllium (1, 33, 59, 63-69).

Other organic materials are also bound by plant DF. The binding of toxic or carcinogenic substances or alterations in microbial metabolism associated with high DF diets may protect against the development of colonic cancer in man (1, 59)

2.1.3.4. Cation exchange capacity

This property represents an adverse effect of high DF food. The reduced mineral availability and electrolyte absorption associated with certain high-fiber diets are undoubtedly due to the binding of minerals and electrolytes on fiber sources, resulting in increased fecal excretion of minerals and electrolytes. The number of free carboxyl groups on the sugar residues and uronic acid content of polysaccharides appear to be related to the cation exchange properties of DF (1, 59, 61).

2.1.3.5. Particle size

The degree to which the cell wall matrix, which is rich in DF components, is disrupted by grinding to a finer particle size will influence the physiological response to fiber source (61). The fermentability of DF is influenced by particle size. For instance, coarsely ground wheat bran was found to be more effective in increasing stool weight and reducing intracolonic pressure than finely ground wheat

bran. The reason was the finely ground structure has increased surface area that allows greater microbial degradation (70). However, decreasing particle size of oat bran and microcrystalline cellulose can increase WHC. Likewise, if the cell wall is completely intact, digestive enzymes may penetrate and release nutrients from the food slower than if the cell wall has been disrupted by grinding (1, 56, 59).

2.1.4 Physiological effects of dietary fiber

DF has been reported to have several physiological effects, depending upon the physical and chemical properties of the individual DF sources. These effects include increasing fecal bulk and improving large bowel function, decreasing nutrient availability, reducing levels of plasma cholesterol, and reducing glycemic responses to a meal. Some DF sources are more effective than others in eliciting these responses (59, 61). Factors contributing to increase fecal bulk include the presence of undegraded DF residue, an increase in fecal water content, and increase in microbial cell mass arising from the fermentation of DF. IDF is more effective in this physiological effect than SDF. **Table 3** shows results from several studies demonstrating that fecal bulk is increased by various DF supplements.

Table 3 Fecal bulk associated with DF supplements (1, 59)

DF source	% Increase in fecal wet weight
Oat bran	15
Pectin	16-35
Guar gum	20
Apple	40
Carrot	59
Cabbage	67
Cellulose	75
Wheat bran, coarse	80-127
Wheat bran, fine	24

Another effect of DF on large bowel function is to decrease transit time that is the time for a marker to pass in the feces after consumption. Addition of fruits and

vegetables or wheat bran to human diets but not pectins or gums, have been shown to shorten transit time. Transit time and stool weight are inversely related, however, once a transit time of 20-30 hours is reached, further increases in stool weight do not substantially shorten transit time. Further definition of the effects of DF on these two parameters may allow better assessment of the adequacy of DF intake with respect to maintaining normal physiological function (61).

As shown in **table 4**, sources of non-cellulose polysaccharides that are viscous appeared to be most effective in reducing plasma cholesterol levels which resulting from capacity of binding bile acid, an effect readily demonstrated in hyperlipidemic individuals. Foods formulated with gums for treatment of hypercholesterolemia have been tested. These studies suggested that the formulated foods are palatable to the individual and effective in treatment of the disorder (1, 37, 61). High-fiber foods are “chewy” and chewing stimulates the flow of saliva and the secretion of gastric juices. Water-soluble fiber, such as pectin, form gels and increase the viscosity and stickiness of stomach contents (61).

Table 4 Effect of DF sources on plasma cholesterol levels (1)

Source of DF	DF ingestion (g/d)	% Reduuction of TC level
Corn bran	52	4
Guar gum	24	16
Leguminous seed	140	7
Pectin	25	13
Rice bran	100	10
Wheat bran	17	1
Whole oats	15	11

Fiber may slow down the passage of food in the mouth and stomach (71) and help to promote a sensation of satiety and, by delaying entry of food into the small intestine, modify the rate of absorption of nutrients. Pectins and gums slowed down movement in the jejunum by increasing the viscosity of its content. In this way they

may reduce the rate of digestion and delay the absorption of glucose and other nutrient (56). The percentage increase in plasma glucose can be reduced when sources of viscous polysaccharides are mixed with a glucose load in a glucose tolerance test. In a food system, the presence of DF may attenuate the plasma glucose and insulin response to a meal, however, the interactions are clearly more complex than those observed in the glucose tolerance test.

Some concern has been raised that sources of DF can lower the availability of minerals. Although consumption of high DF diets may increase fecal mineral excretion, the nutritional consequences will depend on the overall adequacy of mineral intake and the total amount of DF consumed. Any recommendations for increasing DF intake should be limited if mineral balance is not compromised (61). **Table 5** shows some of the physico-chemical properties of DF and the gastrointestinal events it may modify (72).

2.1.5. Effect of dietary fiber intake on health

2.1.5.1. Effect of DF intake on heart diseases

From previous studies, DFs were found to have some protection against ischemic heart disease. Wolk *et al.* (73) studied the relationship between ischemic heart disease and DF intake in woman. They found that DF, especially the ones from grains and cereals, could reduce the rate of atherosclerosis and ischemic heart disease. This finding corresponded to that found by Liu *et al.* (74) who studied the effect of whole grains could reduce the risk of ischemic heart disease. However, from these studies, it can be concluded that DF do not have a direct effect in protecting ischemic heart disease but can reduce the cholesterol level in blood, especially the soluble dietary fibers such as pectin, gum and mucilages. Soluble dietary fiber will form a gel-like structure when combined with water and this structure can obstruct the diffusion of nutrients (and fat) at the early intestines. Besides, this gel-like structure will block the working of bile system and result in less diffusion of cholesterol to the body, more bile is then evacuated with feces. It is therefore not surprising that people with high DF intake will normally have more bile in their feces.

Table 5 Physico-chemical, physiological, and clinical aspects of DFs (72)

Physicochemical Property	Type of DF	Physiological effects	Clinical Implication
Particle size and WHC	Wheat bran,	↑Gastric emptying	Pectic ulcer
	Pentosan content, Polysaccharides, Lignin mixtures	↓Mouth to cecum transit, ↓Total gastrointestinal tract transit time, ↓Colonic intraluminal pressure ↑Fecal bulk	Constipation Diverticular disease Dilute potential carcinogens
Cation exchange	Acidic polysaccharides (e.g. pectins)	Tend to increase small intestinal losses of minerals, trace elements, heavy metals	Negative mineral balance, possibly compensated for by colonic salvage, antitoxic effect
Antioxidant	Lignin (reducing phenolic groups)	↓Free radicals in digestive tract	Anticarcinogenesis
Degradability (colonic bacteria)	Polysaccharides (free of lignin)	↑Production of gas and SCFAs, ↓Cecal pH	Flatus, energy production
Absorption and nonspecific Effect	Lignin, pectin mixed DF	↑Fecal steroid output ↑Fecal fat and N loses (small)	Hypercholesterolemia Cholelithiasis
Viscosity	Gums,	↓Gastric emptying	Dumping syndrome
	Mucilages, Pectins	↑ Mouth to cecum transit ↓Rate of small intestinal absorption (e.g. of glucose, bile acid)	Diabetes Hypercholesterolemia

↑ = Increase, ↓ = decrease

Key and Truswell (75) studied the effect of pectin intake on the evacuation of bile by asking voluntary healthy subjects to take 15 grams of pectin everyday for three consecutive weeks. They found that bile acid was increased to 40 % of normal amount of evacuated feces. From the study of Anderson *et al.* (76) on the safety and efficiency of phylimum, one of soluble dietary fibers, on cholesterol level reduction in blood. It was found that the intake of 10.2 grams phylimum with low fat diets everyday could reduce the cholesterol level in blood. Serum total cholesterol was reduced by 4 %, LDL cholesterol reduced by 7 % and the ratio between apo A and apo A-I reduced by 6 % when compared with low fat diets without phylimum. This could be concluded that phylimum, one of soluble dietary fibers, could reduced the cholesterol leveling blood. Shane and Waller (77) studied the effect of two DF, which were corn bran and wheat bran, on the reduction of cholesterol level in blood and triglyceride for 29 patients with hypercholesterolemia. They asked the first group of patients to take 20 grams of corn bran, and the second group to take 20 grams of wheat bran, everyday for 98 days. They found that fiber in corn bran had a significant effect in reduction of triglyceride and VLDL cholesterol level. However, wheat bran did not have such effect. The results of this study show that soluble dietary fibers from corn bran can reduce the cholesterol level in blood and triglyceride better than wheat bran which are insoluble dietary fiber. Therefore, most soluble dietary fibers are widely accepted to have a significant effect in preventing and protecting ischemic heart disease. In the United States, US Food and Drug Administration, USDA, also accept such fact and recommend that the intake of soluble dietary fibers can reduce the risk of atherosclerosis and ischemic heart disease.

2.1.5.1. Effect of dietary fiber intake on diabetes

From the properties of DF, particularly their gel-like structure formation when combined with water which has a direct impact on diffusion of nutrients i.e. fat, carbohydrate, sugar, and other minerals, many researchers were interested in investigating the effect of DF intake on diabetes. Wolven and Jenkins (78) studied the effect of stickiness of fiber gel, such as guar gum, pectin, phylimum and wheat bran, on resistance test to glucose in blood. They found that guar gum resulted

in the best reduction of glucose level and reduced the cholesterol to 44%. According to the journal of American Medical Association in 1997, there were some studies on the effect of DFs in 65,000 middle-aged women in the US and it was found that DFs, especially from grains and cereals, could reduce the risk of diabetes. These studies showed that DFs have a good effect on dietary glycemic load which results in diabetes. Another experiment conducted on 915 women who usually consumed grains and found that these could also reduce the risk of diabetes (79). The intake of DFs, specifically soluble dietary fiber, can reduce the rate of glucose diffusion to the body. In addition, the intake of DFs can block the enzyme at pancreas that is used to digest carbohydrate and hence reduce the reaction of insulin level and intestinal hormone (80).

2.1.5.3. Effect of dietary fiber intake on colon cancer, constipation and diverticular disease of colon

From the occurrence of growing number of patients with colon cancer in the west and in many developed countries, eating habit has been identified as one of contributing factors to colon cancer. Some studies found that DFs could play an important role in preventing colon cancer. The metabolism process of DFs would reduce the risk of colon cancer because:

1. DFs can remove the secondary bile acid process which prevent the generation of dehydroxylase, the substance that causes cancer;
2. DFs will result in higher feces mass and hence can relieve peak time that feces (and substance that causes cancer) would stay in the large intestine;
3. Some DFs act as antioxidant.

Bandura (81) found that DFs from wheat bran had some effect on preventing and reducing the substance that causes colon cancer. In addition, patients with tumor at large intestine, when consumed wheat bran for some period, were found to have relieved from the disease. Many studies have supported the importance of higher DF intake especially for insoluble dietary fibers as they increase the feces mass and, as a result, reduce the time that feces would stay in large intestine. Substances that may cause cancer will also be less concentrated (82). As feces mass increases, the transfer of wastes from the beginning of large intestine to the rectum takes place in

shorter time. This helps smoothing the evacuation and reduces the causes of constipation. Suffering from continuing constipation requires higher force in the evacuation process causing high pressure in the anus and rectum frequently and could lead to diverticular disease of colon (83). Daily intake of appropriate DFs could be a way that prevents suffering from constipation, diverticular disease of colon and colon cancer.

2.1.5.4. Effect of dietary fiber intake on obesity

Heaton (84) asked 10 voluntary subjects to consume whole apples, minced apples and apple juice (fiber-filtered) alternately and found that whole apples caused the subjects to feel full longest followed by minced apples and apple juice, respectively. Heaton also found that the difference of this feeling could be as long as 2 hours and the glucose level in blood was increased and reduced most rapidly (also to the lowest) with apple juice (no fiber). This rapid falling of glucose level may stimulate hunger and the feel to take more diets in shorter time. However, because the insulin level in blood was also higher with apple juice, fat is likely to be built up more in the fat tissues. It is clear that intake of diets with no fiber results in shorter feeling full, more rapid hunger and more risk to obesity than diets with high DFs.

2.1.5.5. Effect of dietary fiber intake on hyperlipidemia

A tremendous number of studies in both humans and experimental animals have been conducted examining the ability of different types of DF to lower plasma cholesterol concentrations. From these studies certain generalities can be deduced. Most isolated fibers that are water soluble dietary fiber will lower plasma cholesterol in humans and plasma and liver cholesterol in animals. These include pectins, phylum, and various gums such as guar gum, locust bean gum, and modified celluloses such as carboxymethylcellulose. Consumption of fiber-rich sources containing water soluble dietary fibers, such as oat bran and barley (sources of mixed-linkage β -glucans), legumes, and vegetables, usually results in a lowering of plasma cholesterol. Reductions in total plasma cholesterol up to 2.5 % have been reported, but most studies find reductions in the range of 5-10 %. Almost invariably the reductions occur in the low-density lipoprotein fraction, with little or no change in high-density

lipoprotein cholesterol. In contrast, isolated fibers of fiber source that are not water soluble dietary fiber have rarely been found to alter plasma cholesterol. These fibers include cellulose, lignin, corn bran, and wheat bran.

How cholesterol-lowering DFs mediate their action remains a subject of controversy. One hypothesis is that a fiber-induced increase in bile acid excretion leads to an increased demand for bile acid synthesis, resulting in an increased rate of conversion of cholesterol to bile acids (85). If cholesterol synthesis rates do not increase sufficiently to compensate for the loss of cholesterol to bile acids, then cholesterol concentrations will decrease. However, not all fibers that lower cholesterol increase bile acid excretion (86). A corollary to this hypothesis is that cholesterol-lowering fibers alter the bile acid profile by differential binding to bile acids, which could lead to decreases in absorption or synthesis of cholesterol (87). Changes in the profile of the bile acid pool with feeding of cholesterol-lowering fibers have been noted in several studies (88-89). Future studies with additional fiber types will be necessary to establish the importance of this correlation. Many water-soluble dietary fibers form a viscous matrix within the small intestine (90), which could interfere with cholesterol or bile acid absorption in the small intestine. Guar gum has been found to delay cholesterol disappearance from the small intestine in one study (91), but to have no effect on absorption in another (92). Likewise, pectin has been found to reduce cholesterol absorption in one study (93) but not in another (94). Using a highly viscous but nonfermentable modified cellulose, hydroxypropyl methylcellulose, it has been shown that cholesterol absorption decreases linearly with the logarithm of intestinal contents viscosity (95). Another hypothesis is that sources of fiber will modify cholesterol synthesis. Cholesterol synthesis as measured by ^{14}C -acetate incorporation into cholesterol (96) or hepatic 3-OH-3-methyl glutaryl coenzyme A activity (97) is elevated in rats fed pectin, a hypocholesterolemic fiber source (98). This elevation is undoubtedly due to reduced cholesterol absorption or enhanced bile acid excretion in pectin-fed rats. Studies with isolated hepatocytes have demonstrated that propionate, which can be produced by fermentation of soluble dietary fiber, inhibits fatty acid synthesis and ^{14}C -acetate incorporation into cholesterol but does not inhibit total cholesterol synthesis (99). A preliminary report indicates that in humans *in vivo* lipogenesis may be suppressed in subjects fed a high-carbohydrate diet that is rich in

complex carbohydrate, including starch and DF (100). These results suggest that the effect of fermentable fibers on hepatic fatty acid synthesis and secretion should be investigated further, especially since hepatic-derived triglyceride-rich lipoproteins are the precursors of the low-density lipoprotein fraction. Overall, the evidence suggests that more than one mechanism contributes to the cholesterol-lowering effect of DF. The physical properties of fiber that seem most likely to be responsible are bile acid binding (or entrapment) and viscosity. DF binds components of mixed micelle such as bile salts, fatty acids, monolene, phospholipids, etc and may therefore impair fat absorption in the upper jejunum. Reduced lymphatic absorption of both cholesterol and triglyceride in rats receiving soluble dietary fiber.

2.1.6 Dietary fiber sources

Thailand is an agricultural country and agricultural industry, we have a lot of agricultural products which have potential for use as dietary fiber sources such as cereals, legume, rice bran , etc. They are a high dietary fiber content. They could be considered as a source of dietary fiber for human.

The major sources of dietary fiber come from plants such as fruits, vegetables, whole-grain, cereals, legumes and nuts (39). The amount of dietary fiber content in food varies according to types, age, including the part of plant as well as history of processing treatment (101, 102). As a product of agriculture, the sources of dietary fiber can be categorized into four main sources as follows:

Fruits : Dietary fiber content of fruits is relatively low on a fresh weight basis. Fruit juice is very low but dried fruits are moderately high in dietary fiber content. The concentrated sources of fruit fibers can be obtained through dehydration processes (42). Pectin is an important example of commercial fruit fibers which is extracted from citrus, apple peels and cores (103, 104)

Vegetables : Vegetable is another good source of dietary fiber. Their concentration is relatively high in a wide variety of dehydrated products that can potentially be used as ingredients for high-fiber food. (37)

Cereals and seeds : cereals and seeds are excellent sources of dietary fiber. Wheat, rice, oat, corn, and soy are examples. The whole cereal grains consist of bran,

endosperm, and germ, which contain low to high dietary fiber content. Usually, the highest dietary fiber content is found in cereal bran and legume hulls such as wheat bran, oat bran, corn bran, soy bran, rice bran, soy hull, pea hull, sunflower hull, etc (105, 106)

Other potential fiber sources : Other sources of dietary fiber are not found in plants but derived from animals such as chitin and chitosan. These can be obtained as waste materials from shrimp and crab processing procedures. Chitin and chitosan appear to have great potential in food, but they have not been approved as food ingredients. However, greater interest should focus on new dietary fiber sources which are derived from waste or by-products in food industries such as apple pomace, sugar beet pule, citrus peel, residue of mango, papaya, coffee, and cocoa processing, etc.(107)

2.1.7 Dietary fiber recommendation

At present, from all many studies, we cannot identify the exact amount of daily dietary fiber intake which would balance our body requirement. Too much intake of dietary fibers could have an effect and interfere the diffusion system of some minerals and nutrients to the body, while too little intake of fiber will affect the evacuation process and result in risks for many diseases, as mentioned, i.e. diabetes, colon cancer etc. Therefore, dietary fibers are important and the daily intake of fibers is necessary. While there is still no regulation on suitable daily intake of dietary fibers in many countries, some countries, such as Sweden, the United Kingdom, Austria and Ireland, have recommended that the suitable average daily intake of dietary fiber is 25 – 30 grams, including both insoluble and soluble fibers. The Public Health Department of New Zealand also recommended their people to take 25-30 grams of dietary fiber daily, a quarter of which or about 7-8 grams should be soluble dietary fibers, (108). The South America even specify the recommendations of dietary fiber intake according to sex and age. Older men and women should take 18 grams daily and 12 grams daily, respectively. For younger men and women, daily dietary fiber intake is increased to 20-25 grams (20). According to the recommendation of diet intake for good health in Thailand, the daily intake of dietary fibers is 25 grams. The good intake

must include both soluble and insoluble dietary fibers as each have their own particular properties (109). The intake of many different types of dietary fibers in suitable amounts would balance our digestive system.

2.1.8 Adverse effects of dietary fiber

Even though DF have benefits on health, over-consumption of fiber bring about potential adverse effects. Some adverse effects may occur with the consumption of 50-60 gram fiber diets (110). Gastrointestinal effects resulting from inclusion of high-fiber foods in the diet include gastrointestinal obstruction, which has been reported primarily with gel-forming fiber supplements but also with wheat bran and other fiber-rich source. It is unclear whether a similar relationship holds for other types of fiber but these reports stress the importance of adequate fluid intake in order to derive beneficial effects of fiber and to avoid potential hazards(31, 58, 110). Abdominal fullness and flatulence are among most common adverse effects when fiber intake increase significantly. Intestinal distention and diarrhea may also occur. These side effects are caused by bacterial fermentation of fiber with release of volatile fatty acids, hydrogen, carbon dioxide and methane. These gastrointestinal disturbances should subside within 24 to 48 hours(31). DF intake should be increased gradually along with adequate fluid intake so the gastrointestinal tract can adjust to the change.

The effect of DF on mineral balances continue to be a controversial issue since a number of studies have demonstrated reduced or even negative mineral balance with the intake of certain fiber at moderate to high levels(111). The mechanism by which DF influence mineral absorption is related to ability of DF to act as weak cation exchanger, decrease transit time, dilute mineral concentration by increasing fecal bulk and resisting digestion in the large bowel. Many other factors in addition to DF can affect the absorption of minerals(111) such as the use of mineral supplements, the level of mineral intake, the length of study period, the health status of the subjects, and the presence of other dietary constituents which may impair (phytate and oxalate) or enhance (ascorbate or citrate) absorption. Unfortunately, these factors are not always controlled in metabolic studies so inconsistencies are inevitable.

On vitamin, few studies have been reported that riboflavin and niacin may not be as easily absorbed from whole wheat bread as from enriched white bread. Niacin bound to cellulose in cereal bran may not be readily available and wheat bran is shown to decrease vitamin B6 availability (37, 110).

2.1.9 Effects of dietary fiber on vitamin absorption

Among the fat-soluble vitamins, vitamin A has been the most extensively studied in man. Results of human studies indicated that carotene was less available from vegetables than as pure carotene in oil, and this was later attributed to the presence of fiber (112). Barnard and Heaton (113) found that in human subjects lignin given with a test meal containing 5,000 I.U. of vitamin A per kilogram body weight had no effect on serum rise in vitamin A after the meal. In addition, Kasper et al (114) reported that when wheat bran cellulose, pectin, Guar flour, carob bean flour, or carrageenan was given with a test meal containing 300,000 I.U. of vitamin A palmitate, the area under the curve for serum concentration of vitamin A for the 9 hours afterward was significantly decreased by all fiber sources. These two studies were conducted because there had been reports of binding of bile acids by fiber, and it was postulated that fiber might also affect bioavailability of vitamin A through changes in intestinal absorption. The three well-known long-term studies on man on serum vitamin A concentrations subsequent to increased ingestion of dietary fiber come to different conclusions. During an experiment on 68 patients receiving two tablespoons of bran per day over a period of at least 6 months, it was found that serum vitamin A concentrations were higher compared to the initial figures (115), whereas in another experiment on 4 subjects after the daily intake of 15 g apple pectin or 30 g wheat bran over a period of 50 days, a decrease in vitamin A concentration was found, while carotene concentrations remained unchanged (113). Wahal and co-workers (116) tested the effect of wheat bran on serum vitamin A levels in healthy subjects during a 6-week trial. The addition of wheat bran to a standard diet with 20,000 units of vitamin A significantly lowered serum vitamin A levels within 1 week, and this trend continued over 3 weeks. They suggest that bran in the wheat flour which forms the staple diet in some parts of India may contribute towards the vitamin A deficiency state.

commonly observed in this country. These studies with both human and animal indicate that if there is a decrease in vitamin A bioavailability because of dietary fiber and the effect is small.

There are several dietary fibers and vitamin E studies conducted on rat, vitamin E supplemented diet containing 10 % of pectin that was fed for 56 days resulted in lower body weight as well as decreased plasma and red blood cell vitamin E and increased hemolysis (117) Feeding 20% of the diet as bread or cereals for 30 days (three different breads and two cereals) did not effect plasma vitamin E level (118). In rat fed 5 % or 20 % wheat bran for 56 days, plasma vitamin E level declined at 5 weeks but reverted to pre-study levels at 8 weeks (119) Rats fed 6% or 8 % pectin for 8 weeks had lower body weight, lower liver vitamin E levels, and higher hemolysis rates than those fed 0 or 3 % pectin (120). From these studies on rats it appears that intakes of pectin at a level of 6% or more of the diet decrease vitamin E bioavailability, but fiber - containing breads or cereals as 20 % of the diet have only transient or no effect.

Additionally, dietary fiber has an effect to decrease in bioavailability of vitamin A and vitamin E. They also may effect on antioxidative activities of both vitamins.

Burton and Lngold (121) concluded their series of experiments that vitamin A and β -carotene have potential role in lipid antioxidant in those membranes and organelle that are exposed to the lowest partial pressure of oxygen. Vitamin E will tend to be concentrated in those lipid region that are exposed to the highest partial pressures of oxygen for example cells lining the other surface of the lung and red blood cell membrane.

It is observed that α - tocopherol has been shown in vitro and in vivo to inhibit oxidative damage especially in LDL , thus may lower or even prevent atherosclerotic processes induced by oxidized LDL (122). The antioxidant activity of carotenoids shown in vitro was studied in randomized intervention trials by administration of pure β -carotene. Several studies showed a significant association between a low antioxidant concentration and increased risk of disease or progression of disease.

Hoffmann et al present study (23), the influence of dietary fiber on the antioxidant enrichment and the oxidation resistance of LDL after antioxidant supplementation is Investigated. They reported that result after antioxidant

supplementation the isolated LDL revealed significantly ($p < 0.05$) increased antioxidant concentrations but addition of pectin, guar, or cellulose to the meal depressed this increase. Concomitantly the observed increase in the resistance of LDL against oxidation (measured as lag phase) was lower with dietary fiber supplementation than that found without. These results indicate that dietary fiber supplementation decreases the antioxidant effect of a supplement consisting of carotenoids and α - tocopherol in LDL. Additionally, an effect that is likely to be mediated by a reduced bioavailability of these antioxidants in the gut.

2.2. Vitamin A and β -carotene

Vitamin A is represented primarily by the cyclic polyene alcohol vitamin A with an empirical formula of $C_{20}H_{30}O$ and those conjugated double bonds in the side chain are in the *trans* arrangement. The structure of vitamin A is shown in **Fig. 1**.

There are two forms of vitamin A as such, retinol and dehydroretinol. Retinol, found as an ester (retinyl palmitate) is biologically active as an alcohol, the most common form usually referred to as retinol; an aldehyde, as retinal; and an acid, as retinoic acid.

Another representative of vitamin A occurring in nature is vitamin A₂, which has an additional double bond in the ring at the 3-4 position. It has biological activity only 40 percent of vitamin A₁. A third such representative is neovitamin A-a in which the terminal double bond in the side chain of vitamin A₁ is *cis*. It has low biological activity.

In addition, carotene, a related compound to vitamin A, is also called provitamin A, because it can be converted to vitamin A in the body, and precursor of vitamin, because it precedes vitamin. At least, 10 different carotenoids exhibit provitamin activity. Four of those carotenoids, μ , β , γ and cryptoxanthine are important, of which β -carotene being the most important.

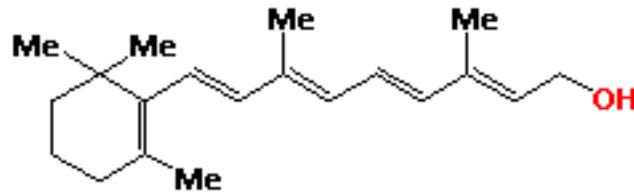


Fig. 2 Structure of vitamin A (retinol)

Foods supply vitamin A in the form of vitamin A, vitamin A ester, and carotene, of which carotene are converted to vitamin A at the wall of the intestine. Vitamin A, incorporated into chylomicrons, is absorbed apparently at intestinal cell by a carrier mediated process, facilitated by protein, so-called cellular retinol-binding protein (CRBP).

It reaches a peak in plasma about four hours after die ingestion. The absorption is reduced by abnormalities of fat absorption and in patients with pancreatic or hepatic disease, intestinal infection and cystic fibrosis. In blood, vitamin A are transported in association with a retinol-binding protein (RBP). Storage of vitamin A is largely in the liver, and a lesser in the lung, body fat and kidney.

The principal source of vitamin A in the diet is likely to be from the carotenes, which are widespread in those plant foods that have high green or yellow colorings, such as beet greens, collards, dandelion green, kale, mustard greens, spinach, Swiss chard, turnip greens, carrots, apricot, cantaloupe, peaches, pumpkins, squash (winter), sweet potatoes and yellow corn. Also, the vitamin A from animal food are liver, butter, egg yolk, cheese, whole milk and fish.

The role of vitamin A in the visual process is established best. It is necessary for growth and differentiation of epithelial tissue and is required for growth of bone, reproduction and embryonic development. It appears to enhance the function of the immune system, to reduce the consequences of some infectious diseases and to protect against the development of certain malignancies (123).

A deficiency of vitamin A may be due to a daily lack of vitamin A and/or provitamin A, or poor absorption. The existence of vitamin A deficiency among preschool children in developing countries is still public health. WHO reported that 31 million preschool children suffered from xerophthalmia and 227 million preschool

children were subclinical deficiencies (124).

In Thailand Changbumrung *et al.* (125) report smaller amounts of retinol and other vitamins intake than those recommended by RDA in all age groups studied of villager in Khon Kaen. In 1990 survey in the north and the northeast Thailand marginal serum retinol levels between 10-20 $\mu\text{g}/\text{dl}$ were found in 14 and 7 percent of the preschool children in the dry and rainy seasons, respectively (126).

Excessive intake may cause serious injury to health. The symptoms are loss of appetite, headache, blurred vision, excessive irritability, loss of hair, drying and flaking of the skin, swelling over the long bones, drowsiness, diarrhea, nausea and enlargement of liver and spleen (127).

Assessment of vitamin A status

The only direct assessment of vitamin A status is by the liver biopsy and measurement of retinyl ester reserves, an invasive procedure that cannot be considered for routine investigations and population surveys. Status can also be assessed by clinical and functional tests, the plasma concentrations of retinol (**Table 6**) and retinol binding protein, and the response to a test dose of vitamin A (128).

Table 6 Vitamin A status indicated by plasma retinol concentrations

Vitamin A status	Blood retinal concentration	
	$\mu\text{mol}/\text{l}$	$\mu\text{g}/\text{l}$
Deficient	< 0.35	< 100
Unsatisfactory	0.35 – 0.70	100 – 200
Normal	0.70 – 1.75	200 – 500
Elevated	> 1.75	> 500

* Bender and Bender, 1997 (128)

2.3. Vitamin E

Eight naturally occurring tocopherols with vitamin E activities are now known. α -tocopherol (5,7,8-trimethyl tocol) is considered to be the most important tocopherol since it comprises about 90 percent of the tocopherol in animal tissues and displays the greatest biological activity in most bioassay. The structure of vitamin E is shown in **Fig. 2**.

Absorption of vitamin E, with the presence of both bile and fat, is taken place in the small intestine, and pass through the intestinal wall into the lymph. They can most stores at adipose tissue, liver and muscle.

Vitamin E occurs mainly in a variety of plants, especially in oil seed crops, some grains, nuts, and green leafy vegetables. Animal tissues and animal products are usually low to poor sources of vitamin E.

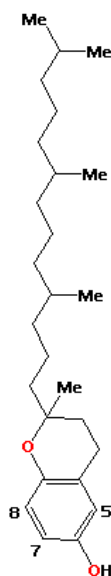


Fig. 3 Structure of vitamin E

The primary function of vitamin E is to help to protect the integrity of cellular and intracellular structures and to prevent destruction of certain enzymes and intracellular components. Vitamin E is more important in human nutrition, since they play role as an oxidant (129). Vitamin E converts the free radical into vitamin E radical, a less reactive and non-harmful form. Consequently, vitamin E has been

attended as a possible role in the prevention of cancer and cardiovascular disease (130) and cataract (131).

Vitamin E deficiency symptoms as such rarely occur in human. However, clinical evidence of deficiency has been observed in infants, in individuals suffering from kwashiorkor and in children and adults who have impaired fat absorption. The symptoms include increased red blood cell fragility and shortened red blood cell life-span, and increased urinary excretion of creatine.

Vitamin E is relatively nontoxic. Excess intake of vitamin E is excreted in feces.

Assessment of vitamin E status

The most commonly used index of vitamin E nutritional status is the plasma concentration of α -tocopherol; because tocopherol is transported in plasma lipoproteins, thus this is best expressed as per mole of cholesterol or per milligram of total plasma lipids (128).

Table 7 Vitamin E status was indexed by plasma tocopherol concentrations

Vitamin E status	Blood tocopherol concentrations ($\mu\text{mol/l}$)
Deficient	< 12
Low	12 –16
Acceptable	> 16

* Bender and Bender, 1997 (128)

Action of vitamin A and E as antioxidants

Vitamin A is a peroxy radical scavenger. Its lipid nature makes this antioxidant effective in reducing membrane lipid peroxidation by acting as a chain breaking antioxidant (132).

α -Tocopherol acts as an antioxidant primarily by scavenging active oxygen radicals, which attack substrates such as lipid, protein, sugars and DNA to initiate chain reactions and/or carry chain propagation. It inhibits lipid peroxidation in cells by

slowing the rate of lipid peroxidation (133). When α -tocopherol react with a lipid peroxide radical, the tocopheroxyl radical are formed. α -Tocopherol radical can be reduced to α -tocopherol by reacting with ascorbate or glutathione. Also an electron transport chain linked enzymic system in which the oxidation of NADH, succinate or reduced cytochrome C is linked.

Burton and Ingold (134) concluded their series of experiments that β -carotene has potential role in lipid antioxidant in those membranes and organelle that are exposed to the lowest partial pressures of oxygen. Vitamin E will tend to be concentrated in those lipid region that are exposed to the highest partial pressures of oxygen for example cells lining the outer surface of the lung and red blood cell membranes.

It is observed that in a condition associated with increased oxidation metabolism, the levels of antioxidant in plasma and leukocytes are decreased. That is because of increased consumption of those antioxidant vitamins during neutralization of reactive oxidants (135). Several studies showed a significant association between a low antioxidant concentration and increased risk of disease or progression of disease. Although the direct effects of antioxidant intake on progression of disease are yet to be determined in human; however, their supplementary effects have been evaluated (136). Conclusively, two steps may help to extend or enhance survival. One is to decrease exposure to high-risk oxidant situation. A second is to optimize antioxidant defenses (137). The intake may be achieved by consumption of vitamin-enriched food or vitamin supplement.

2.4. Malondialdehyde (MDA)

One of the most frequently used biomarkers providing an indication of the overall lipid peroxidation level is the plasma concentration of malondialdehyde (p-MDA). It is one of several byproducts of lipid peroxidation processes. Free radical attacks on polyunsaturated fatty acids in biological systems is thought to produce the sequence of reaction shown in Figure 3, resulting in the formation of both conjugated dienes and lipid hydroperoxides. Lipid hydroperoxides breakdown in the presence of iron or other metal complexes to form aldehydes, e.g. malondialdehyde (MDA), via

the formation of cyclic peroxides and endoperoxides. The measurement of free radical activity is to measure the end products of lipid peroxidation. The most commonly applied test is the thiobarbituric acid reaction (TBA) for the measurement of MDA.

Kohn and Liversedge (138) observed that the first biological application of the TBA reaction that gains tissue incubated aerobically produced a color with 2-thiobarbituric acid. Berheim et al. (139) found that this color to result from the formation of a complex between oxidation products of unsaturated fatty acids and TBA, whilst Sinnhuber et al. (140) showed that the pigment which was produced resulted from the condensation of 2 moles of TBA with 1 mole of MDA (figure 4). Some MDA is formed by breakdown of lipid peroxides *in vivo* in the presence of iron and copper complexes. In the TBA reaction, heating of the specimen at acid pH results in the breakdown of lipid peroxides to form MDA and release of MDA from protein adducts (141). Yagi et al (142) proposed that lipid peroxides be isolated from other TBA-reacting substances by precipitation of lipids and serum proteins using phosphotungstic / sulphuric acid mixture and detection of lipid peroxide by the TBA reaction in acetic acid, with absorbance measurement at 532 nm.

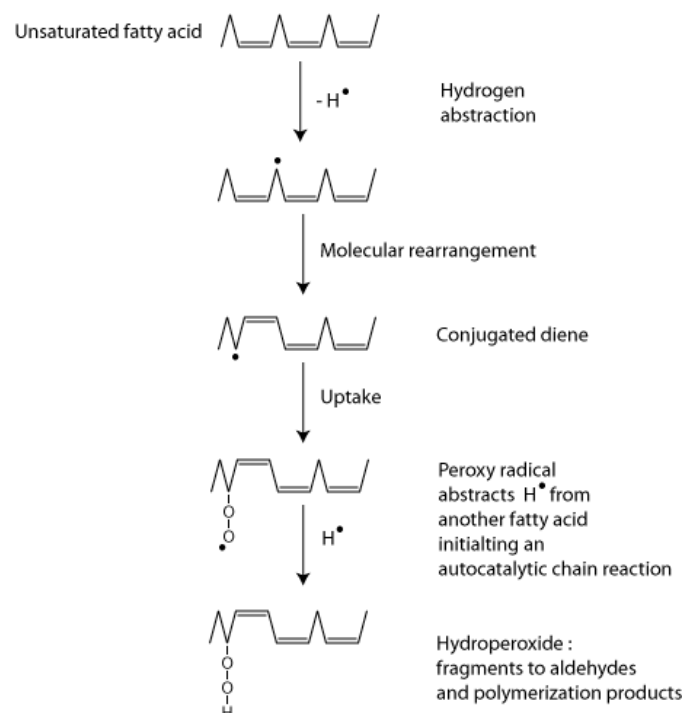


Fig 4 The formation of conjugated dienes and lipid hydroperoxides following hydrogen abstraction from a polyunsaturated fatty acid

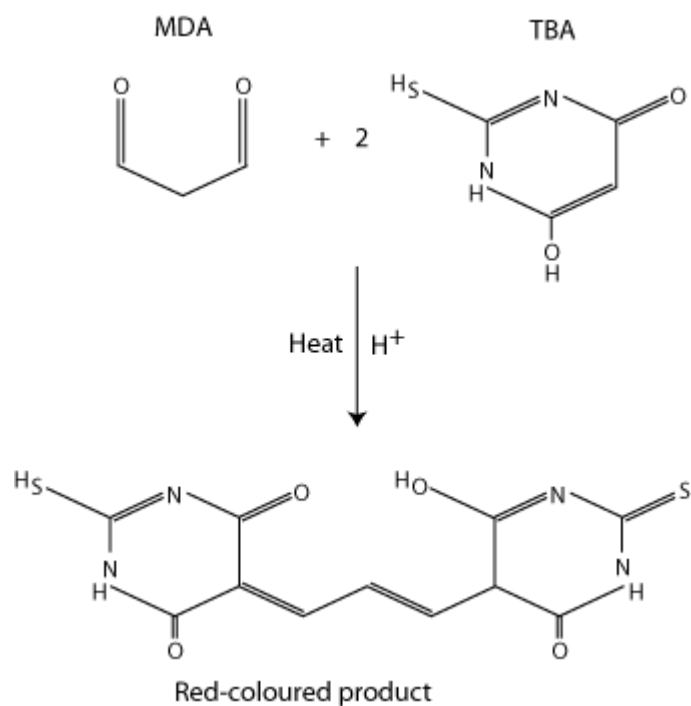


Fig 5 The reaction of malondialdehyde and thiobarbituric acid forming a pink-colored product

CHAPTER III

MATERIALS AND METHODES

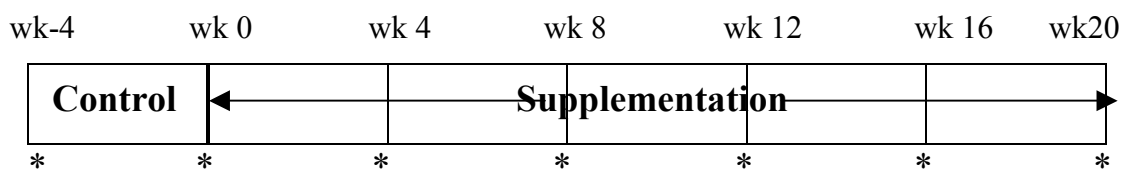
3.1. Subjects

The study included 22 subjects with hyperlipidemia who attended the Nutritional Clinic, Department of Medicine, Ramathibodi Hospital, Mahidol university, Bangkok, Thailand and Medical and Health office, Electricity Generating Authority of Thailand (EGAT), Nonthaburi, Thailand.. They consisted of 11 men and 11 woman with the mean (\pm SEM) age of 55.2 ± 2.80 yrs, height of 160.4 ± 2.03 cm., body weight of 68.2 ± 2.86 kg. and body mass index (BMI) 26.6 ± 1.06 kg./ m² (table 10)

The criteria of hyperlipidemia in these subjects was base on serum TC level of ≥ 200 mg./dL (≥ 5.2 mmol/L), serum TG level of ≥ 150 mg./dL (≥ 1.7 mmol/L) and LDL-C level of ≥ 130 mg./dL (≥ 3.4 mmol/dL). This study excludes subject with secondary causes of hyperlipidemia such as hypothyroidism, nephrotic syndrome, diabetes, and glucocorticoid therapy. No subjects received hypocholesterolemic medications within the 3 months before the study. All of subjects are requested to participate this study for 24 wks.

3.2. Experimental design

Each subject was anticipated to participate in a 24-wks. study consisting of 4 wks. (wk - 4 - wk 0) of contro1 and 20 wks. (wk 0 - wk 20) of supplementation periods (**Fig 6**).



* Dietary assessment, anthropometry, physical examination and biochemical assessment

Fig 6 Experimental design

Before starting of the trial in control period, all of subjects were given dietary counseling and educated for appropriate diet. Throughout the study the subjects were instructed to consume diets with energy distribution of 15 % protein, 30 % fat, and 55 % carbohydrate. Regarding to restrict their cholesterol intake to less than 300 mg./day. After control period, we will exclude subjects if his/her serum lipid was reduced in normal range. The remained subjects must have high level of serum lipid (TC > 200 mg./dL, TG > 150 mg./dL and LDL-C >130 mg/dL).

During the supplementation period, the subjects were supplemented with the cereals and nata de coco as their dietary supplement twice daily before breakfast and dinner. However, they were allowed to follow their own menus. The cereals and nata de coco supplement was formulated by Dr.Wanpen Mesomya and co-worker. **Table 8** shows the ingredients of the cereals and nata de coco supplement. This product was manufactured in powder form. For the subject's consumption, they were instructed to add hot water to 15 g. of the cereals and nata de coco supplement (1 package) to get a final volume of 150 ml.

Table 8 The ingredients of the cereals and nata de coco supplement

Ingredients	% (w/w)
Unpolished rice	6
Mung bean	18
Sweet corn	36
Nata de coco	40

Table 9 Proximate analysis of the cereals and nata de coco supplement (per100 g)

Composition	
Moisture (% wet wt)	5.73
Protein (% wet wt)	18.23
Fat (% wet wt)	1.55
Carbohydrate (% wet wt)	80.22
Ash (% wet wt)	2.68
Soluble dietary fiber (g)	0.7
Insoluble dietary fiber (g)	8.5
Vitamin A (μg)	Not detected
Vitamin C (mg)	Not detected
Vitamin E (mg)	0.5
Calcium (mg)	40
Phosphorus (mg)	305
Sodium (mg)	68
Potassium (mg)	757
Iron (mg)	3.0
Copper (mg)	0.40
Zinc (mg)	2.3
Chloride (mg)	44
Vitamin B ₁ (mg)	0.16
Vitamin B ₂ (mg)	0.02
Vitamin B ₆ (mg)	0.29
Vitamin B ₁₂ (mg)	0.30
Biotin (μg)	12
Folic acid (μg)	176
Niacin (mg)	1.1
Pantothenic acid (mg)	0.6

3.3. Dietary assessment

3.3.1. Dietary record

The subjects were asked to keep an estimated record of all foods and beverages consumed over a three-days food record (2 weekdays – 1 weekend) before their visit to the Nutrition Clinic. Detailed descriptions of all foods and beverages consumed and their method of preparation and cooking were recorded. The example of dietary record was given to all subjects. Food portion size was estimated by the subjects using standard household measuring cups and spoons supplemented by measurements with a ruler (for meat and cake) and counts (for eggs and breads slices). They were asked to provide additional information about any unclear food items. Measuring cups, spoon and face-to-face dietary interview was used to increase the accuracy of estimation of size and amount of each food item.

3.3.2. Nutrient data analysis

Portion size measures are usually converted into grams by investigator before calculating nutrient intakes. The food records were analyzed for energy intake and its distribution derived from protein, fat and carbohydrate. Vitamin, mineral and dietary fiber intakes in each subject were calculated. Data were analyzed by using the computerized food composition analysis package “INMUCAL program version ND2 fiber” modified for Thai food by the Institute of Nutrition, Mahidol University. Mean \pm SEM of each nutrient intake for the whole groups for each visit were then calculated.

At wk 4, wk 12 and wk 20, the taste, smell, acceptability and tolerance to the cereals and nata de coco supplement and its safety as well as compliances in consuming the cereals and nata de coco supplementation in 22 subjects are evaluated.

Their feelings of taste and smell to the cereals and nata de coco supplement were scored as follows: 1 = very good, 2 = good, 3 = acceptable and 4 = unacceptable. The acceptance to the cereals and nata de coco supplement was scored as follows: 1 = whole volume being taken without any problem, 2 = effort being needed to take the whole volume, 3 = the whole volume being partially taken and 4 = the whole volume being rejected. The tolerance to the cereals and nata de coco supplement was rated as

follows: 1 = no diarrhea, 2 = diarrhea without clinical significance, 3 = diarrhea and required for the adjustment of the cereals and nata the coco supplement dosage, 4 = diarrhea and required for the discontinuation of the cereals and nata de coco supplement. The safety to the cereals and nata de coco supplement was scored as follows: 1 = no adverse reaction, 2 = adverse reaction but not requiring treatment, 3 = adverse reaction and requiring treatment and 4 = adverse reaction and requiring discontinuation of the cereals and nata de coco supplement. Their compliances to the cereals and nata de coco supplement were based on their actual intake compare to the assigned intake and rated as percentage of the assigned intake.

3.4. Anthropometric measurement

After a 10-12 hr. fasting, body weight, height mid upper arm circumference (MUAC), triceps skinfold thickness (TST), waist and hip circumferences in each subject were measured by using the standard techniques (143-145). The upper arm muscle circumference (UAMC) was calculated from the following formula (143) : $UAMC = MUAC - 3.1416 TST$. Standards of TST, MUAC, UAMC were taken from the standard source (143). BMI (146) was calculated from body weight in kg. divided by height in m^2 . Standard of weight for height was base on Metropolitan Life Insurance Company. Waist and hip circumferences were measured and waist – over - hip circumference ratio (WHR) was computed (145). The body fat of each subject was measured, using infrared light absorption analysis with the Body Composition Analyzer Futrex-5000A (Futrex Inc, Gaithersburg, MD, USA) (147). Height was measured at wk-4 only whereas the remaining anthropometric parameters were measured at 4-wk intervals.

3.4.1. Weight (Wt)

Weight was measured using a beam balance. Clothing should be minimal when measuring weight. Shoes and socks should not be worn.

3.4.2. Height (Ht)

The subjects were generally measured in a standing position using a stadiometer. When measuring height, the subject stands straight with the head positioned such that the Frankfurt plane is horizontal, feet together, knees straight, and heels buttocks and shoulder blades in contact with the vertical surface of the stadiometer. Arm should be hanging loosely at the sides with palms facing the thighs; the head is not necessarily in contact with the vertical surface. Height was recorded to the nearest millimeter.

3.4.3. Mid-upper-arm-circumference (MUAC)

MUAC has been used as an indicator of the inadequacy of both caloric and protein store. The arm contains subcutaneous fat and muscle. Therefore, decreases in MUAC many reflect either reduction in muscle mass, a reduction in subcutaneous tissue, or both. Changes in MUAC measure can be used to monitor progress during nutritional therapy (148), correlating positively with changes in weight. The MUAC is measured in centimeters at the same level as the triceps skinfold thickness. The measurement is taken at the midpoint of the upper left arm between the acromion process of the scapula and tip of olecranon. After locating the midpoint, the left arm is extended so that it is hanging loosely by the side, with the palm facing inwards. The taps is wrapped gently but firmly around the arm at the midpoint.

3.4.4. Skinfold thickness measurement

Triceps skinfold thickness

Skinfold thickness are estimate of total body fat or caloric stores. The thickness of subcutaneous tissue at different sites of measurement change proportionately with weight or loss, and total body fat (149). The measurement of the triceps skinfold is performed at the midpoint of the upper left arm, between the acromion process and the tip of the olecranon, with the arm hanging relaxed. If marked when doing circumference, measure this and use the mean of three consecutive reading. The instrument used was a Harpenden skinfold caliper (British Indicator Ltd., England).

Biceps skinfold thickness

The measurement of the biceps skinfold is performed at above the center of the cubital fossa at the same level as the triceps skinfold and midarm circumference. The arm hangs relaxed at the patient's side, and the crest of the fold should run parallel to the long axis of the arm.

Subscapular area

The measurement of the subscapular is performed by lift the skin 1 cm. Under the inferior angle of the scapula with the shoulder and arm relaxed. The fold should run parallel to the natural cleavage lines of the skin; this is usually a line about 45 degrees from the horizontal extending medially upward.

Supraliac area

The measurement of the suprailiac is performed by pick up this skinfold 2 cm. above the iliac crest in the midaxillary line. The crest of this fold should run horizontally.

3.4.5. Ideal body weight (IBW)

These weights are derived from the actuarial table of the Metropolitan Life Insurance Company (150). They are the weights for a given height and sex. The IBW can be expressed as follows:

$$\text{IBW (\% std)} = \frac{\text{observed body weight}}{\text{desired weight}} \times 100$$

3.4.6. Body mass index (BMI)

Calculated from the following formula :

$$\text{BMI, kg./m}^2 = \text{weight, kg. / (height, m.)}^2 \quad (151)$$

3.5. Biochemical assessment

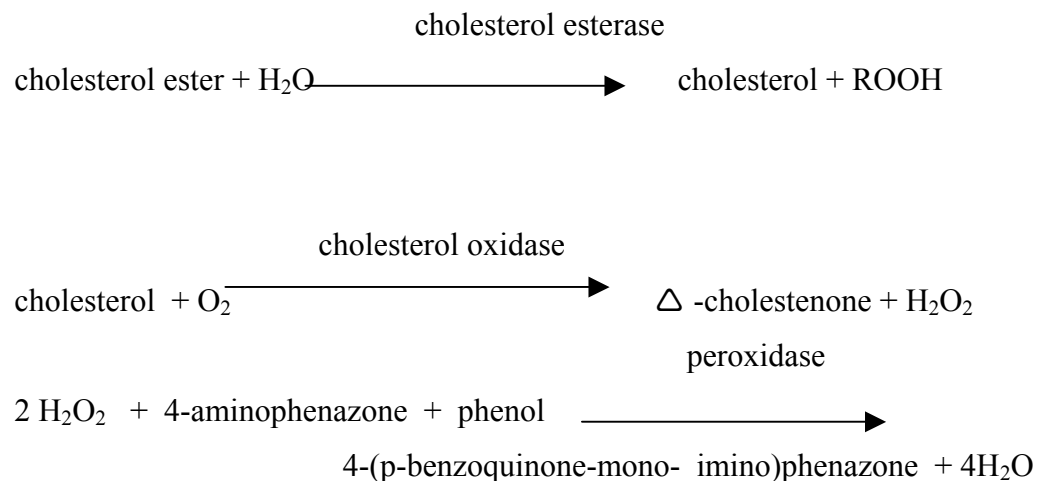
Blood collection

Venous blood was obtained from each subject after a 10-12 hr fast at 4-wk interval. Whole blood serum or plasma was appropriately prepared for biochemical and hematological determination: 1.) serum lipid, 2.) serum vitamin A and E and 3.) lipid peroxidation.

Determination of serum lipoproteins

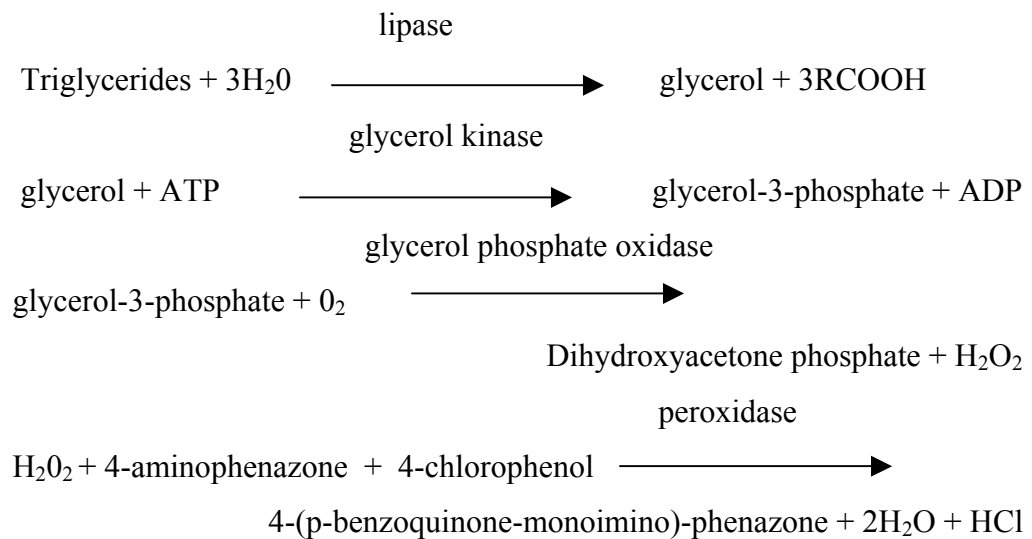
3.5.1. Determination of serum total cholesterol

An Enzymatic-colorimetric method was employed to measure serum total cholesterol by using Human Cholesterol liquicolor test kit. The test principles are as follow:



3.5.2. Determination of serum triglycerides

Enzymatic hydrolysis of triglycerides with subsequent determination of the liberated glycerol by Human Triglycerides GPO Liquicolor test kit was used to measure serum triglycerides. The enzymmatic reactions are as follow:



3.5.3. Determination of high density lipoprotein cholesterol (HDL-C)

The method of Bustein (152) and Lopes-Virella (153) were used for the determination of HDL-C. The Chylomicron, very low density lipoprotein (VLDL) and low density lipoprotein, (LDL) are precipitated by addition of phosphotungstic acid and magnesium chloride. After centrifugation the supernatant fluid contains the HDL-C fraction, which is assayed for HDL-C with the Human Cholesterol liquicolor test kit.

Estimation of low density lipoprotein cholesterol (LDL-C)

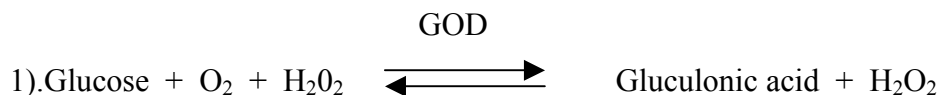
LDL-C level were calculated from Friedewald's formula (154) as follow:

$$\text{LDL-C} = \text{total cholesterol} - (\text{triglycerides} / 5) - \text{HDL-C}$$

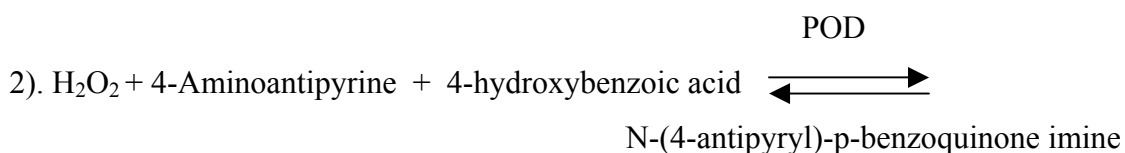
3.5.4. Determination of glucose

The levels of glucose in plasma were determined using the enzymatic method described by Barham and Trinder (155). Plasma was obtained from blood which was preserved by the glycolysis inhibitor, sodium fluoride, mixed with the anticoagulant, calcium oxalate. Glucose oxidase (GOD) catalyzes the oxidation of glucose in the

accordance with the following equation:



Hydrogen peroxide formed was reacted with 4-axninoantipyrene and 4-Hydroxybenzoic acid which was catalized by enzyme peroxidase (POD) as the following equation:



Since the amount of quinoneimine dye formed is in molar equivalent to the amount of glucose, the concentration of the dye in the test tube would represent the amount of glucose in the specimen. The dye concentration was quantitated by spectrophotometer at wavelength 510 nm. Glucose concentration was calculated according to the following equation:

$$\text{Glucose concentration} = (A \times 100) / A_s$$

Where A is the absorbance of the specimen reaction tube and there was 100 mg. per deciliter standard glucose in the standard reaction tube. The amount of glucose concentration was expressed as mg./dL. Appropriate dilution was made if the specimen contained high level of glucose.

3.5.5. Determination of serum vitamin A, β -carotene and α -Tocopherol

Reverse-phase high-performance liquid chromatography method by Miller et al (156) was used for the determination of serum retinol, β -carotene and α -tocopherol in serum after extraction with hexane and retinyl acetate was used as an internal standard. Twenty micro liter of sample were injected into a 3.9x150 mm Nova Pak[®] C18 column and eluted with a 100% methanol for 8 min at 1 ml/min and then switched to a mixture of methanol : acetonitrile : chloroform (47 : 42 : 11) and a flow rate of 2

ml/min by using Waters HPLC system. Retinol and α -tocopherol were detected at 280 nm and β -carotene was detected at 436 nm. With retinyl acetate as an internal standard, standard curves were developed for each compound on the basis of peak area ratios. The coefficient of variation was less than 10% in all cases within a run. Between-run reproducibility was within ± 2 standard deviations.

3.5.6. Determination of lipid peroxide (157)

Oxidation of polyunsaturated fatty acids leads to numerous peroxidic and aldehydic compound, in particular the volatile low molecular weight aldehyde, malondialdehyde (MDA). The reaction of lipid peroxides in serum and tissues with thiobarbituric acid (TBA) was dependent on pH of the reaction mixture as was the case for linoleic acid hydroperoxide. The optimum pH was found to be 3.5. Serum was mixture with sodium dodecyl sulfate, acetate buffer (pH 3.5), and aqueous solution of thiobarbituric acid. After heating at 95 °C for 60 min, the pink pigment produced was extracted with n-butanol-pyridine mixture and estimated by the absorbance at 532 nm. As an external standard, tetramethoxypropane was used, and lipid peroxide level was expressed in terms of nmol malondialdehyde. The pink pigment product was formed by reaction of one molecule of malondialdehyde (MDA) with two molecules of TBA.

3.6. Statistics

Conventional statistical methods were used for the calculation of mean \pm standard error of mean (SEM), The Independent samples t-test was used to compare means for significant change between group with a significance level of $p < 0.05$. The repeated measurement was used to compare for significant change within group above baseline with a significance level of $p < 0.05$.

CHAPTER IV

RESULTS

The selected volunteers were hyperlipidemic patients from the Nutritional Clinic, Department of Medicine, Ramathibodi Hospital, Mahidol university, Bangkok, Thailand and Medical and Health office, Electricity Generating Authority of Thailand (EGAT), Nonthaburi, Thailand. Twenty-two subjects were participated in the study, 11 of them were men and 11 were women, age ranged from 32 to 75 years (mean \pm SEM = 55.2 ± 2.80 yr). At initial characteristics of the volunteers, most of them (68.2 %) were obese. (BMI > 24.9 kg/m²) In group A, their waist circumferences and waist – hip ratio (WHR) were over than the normal 60 % and 60 %, respectively. For group B, their waist circumferences and WHR were over than the normal 43 and 0 %, respectively. (**Table 10**) Most of them had normal blood pressure (BP) was normal, all of them were non-smokers. Three subjects dropped after wk 12 of the study because their personal problem and car accident.

After receiving the supplementation with the cereals and nata de coco supplement for 20 wks, the subjects were classified into 2 groups according to their compliance, **group A**; compliance ≥ 90 % of assigned cereals and nata de coco intake and **group B**; compliance < 90 % of assigned cereals and nata de coco intake.

Table 10 shows initial characteristics including: age, height, weight, BMI, waist, hip circumference, WHR and BP of subjects to the cereals and nata de coco supplementation.

Table 11 shows that the appearance of the product, taste, smell, acceptability, tolerance and safety to the cereals and nata de coco supplement by subjects during the study were satisfactory.

The cereals and nata de coco supplement was well received. In the first wk 0 to wk 4, twelve and eleven of 22 subjects rated the taste and smell of the cereals and nata de coco supplement being acceptable (scored as 3). A few subject commented on taste

and smell but after the initial period this no longer appeared to be a problem. At the end of wk 20, most of subjects rated the taste and smell of the supplement being good (scored as 2). (**Table 11**) All of 22 hyperlipidemic subjects were able to take whole volume of the supplement prescribed without any problem, whereas, they didn't have diarrhea and have adverse reaction (abdominal pain). In addition, a half of the 22 volunteers wished to continue taking the cereals and nata de coco supplement as part of their normal diet.

Table 12, 13 show that compliances in consuming the cereals and nata de coco supplement by subjects during the study were slightly decreased. The remaining data are presented as individual values and mean \pm SEM for each parameter and the statistical significance between the periods of the study for each parameter within group and the corresponding periods between the 2 groups are shown in the footnotes of each table.

Table 14, 15 show their mean \pm SEM of protein, carbohydrates, fat and cholesterol intake; their energy intake and its distribution.

Table 16 shows their mean \pm SEM of total carbohydrate, sugar and total dietary fiber intakes.

Table 17, 18 show their mean \pm SEM of vitamins intake and minerals and phytate intakes.

Table 19, 20 and 21, 22, 23, 24 show their body weight; BMI and body composition parameters.

Table 25, 26, 27, 28 and 29 show their TST and % standard of TST, BST, SST and SIT.

Table 30, 31, 32 and 33 show their MUAC and % standard of MUAC, UAMC and % standard of UAMC.

No significant change in body compositions including: body fat mass, fat-free mass, and total body water were seen between wk 0 and anytime throughout the study. This phenomenon is also in 4 positions of skinfold thickness including: triceps, biceps, sub scapula and supra iliac of 22 hyperlipidemic subjects.

Tanphaichitr et al (unpublished data) have shown that in 453 female Ramathibodi Hospital staff aged 19-64 yrs, there was a significantly positive relationship between body fat mass (BFM) and triceps skinfold thickness (TST) ($r =$

0.77, $p < 0.005$). This is also observed in our 22 hyperlipidemic subjects. ($r = 0.57$, $p < 0.01$) These findings indicate that TST is also a useful anthropometric parameter to assess energy store.

Table 34, 35 and 36 show their waist, hip circumferences and WHR

In our study, acceptable weight and obesity are defined by BMI of 20.00 – 24.99 and more than 25.00 kg/m² whereas abdominal obesity is defined by WHR > 0.80 for female and WHR > 1.00 for male (159).

The incidences of acceptable weight and obesity in 22 hyperlipidemia at wk 0 were 40.9 and 59.1 %, respectively, whereas the corresponding figures at wk 20 were 47.4 and 52.6 %, respectively. (**Table 19**) The incidences of their abdominal obesity at wk 0 and wk 20 were 40.9 and 26.3 %, respectively. (**Table 36**) These findings indicate that they were facing more overall obesity than abdominal obesity. They had the highest incidence of combined overall and abdominal obesity 31.8 % at wk 0 which declined to 15.8 % at wk 20. (**Table 19, 36**)

Table 37, 38, 39, 40, 41, 42 and 43 show individual values and means \pm SEM of serum lipid levels, serum lipid ratios and fasting blood glucose levels in 22 hyperlipidemia on the cereals and nata de coco supplement.

Group A, significant differences were seen between the mean of TG level at wk 0 and the mean values at wk 4, 8, and 12 ($p < 0.05$) but no significant differences in TC, LDL-C and HDL-C in all subjects except in group A, TC level at wk 16 was significantly lower than that at wk 0 ($p < 0.05$).

The homeostasis of blood glucose before and during receiving the cereals and nata de coco supplementation was based on the determination of fasting blood glucose (FBG). The WHO criteria (166) are employed for the diagnosis of diabetes mellitus when FBG is above 6.7 mmol/L (120 mg/dL). In our study, at wk -4 to wk 12, diabetes mellitus was detected in only 1 subject (P.Ke) but after these periods her FBG level turned to the normal range. (**Table 43**) As opposed to the result of K.Ch because his FBG level was slightly increased at wk 20. (**Table 43**) However, there were no significant changes in FBG level in these 22 subjects with hyperlipidemia during receiving the cereals and nata de coco supplementation.

Table 44, 45 show their serum sodium, potassium, chloride, calcium, phosphorus levels and carbon dioxide content.

Table 46, 47 show their serum total and direct bilirubin levels and serum enzyme levels including AST, ALT, AP, and GGT.

Table 48, 49 show their serum total protein, albumin, urea nitrogen, creatinine and uric acid levels.

Evaluation of safety of ingesting the cereals and nata de coco supplement was based on biochemical parameters assessing serum mineral levels and renal and liver function of the subjects.

The serum sodium, potassium, chloride, CO₂ content, calcium and phosphorus, total and direct bilirubin, AST, ALT, alkaline phosphatase and GGT, total protein, albumin, urea nitrogen, creatinine and uric acid levels in 22 hyperlipidemic subjects before and during receiving the cereals and nata de coco supplementation were within the normal limits (167).

It is evident that the consumption of the cereals and nata de coco supplement is safe. It does not cause any hazard to hematopoietic, renal and liver functions.

Table 50, 51 show individual values and means \pm SEM of serum vitamin A, β -carotene and vitamin E levels in 22 hyperlipidemia on the cereals and nata de coco supplement.

All subjects had normal serum vitamin A, β -carotene and vitamin E levels during the study and no significant differences from wk 0.

Table 52 show individual values and means \pm SEM of serum malondialdehyde (MDA) levels in 22 hyperlipidemia on the cereals and nata de coco supplement.

Both group A and group B, significant differences were seen between the mean serum MDA concentration at wk 0 and the means serum MDA values at wk 4, 8, 12, 16 and 20. ($p < 0.001$)

Table 10 Initial characteristics in 22 subjects with hyperlipidemia

Subject	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m ²)	Circumference		WHR	BP (mmHg)
					Waist (cm)	Hip (cm)		
Group A								
A.So	58	160	64.0	25.00	91.0	102.0	0.89	129/73
B.Ja	56	150	77.9	34.62	110.0	108.0	1.02	120/80
B.Ju	59	164	66.5	24.72	94.0	96.0	0.98	130/75
B.Ke	51	154	63.1	26.60	82.0	104.0	0.79	130/70
J.Bu	59	181	77.2	23.56	90.0	97.0	0.93	130/70
P.Ai	32	173	100.2	33.48	111.0	112.0	0.99	120/80
P.Ke	45	148	58.7	26.80	79.0	97.0	0.81	110/70
P.Ra	65	137	55.5	29.57	94.0	99.0	0.95	140/100
P.Sm	68	163	61.0	22.96	84.5	92.0	0.92	120/70
P.So	74	162	63.6	24.23	99.0	103.0	0.96	120/80
P.Wa	55	155	61.3	25.52	82.5	96.8	0.85	120/60
S.Bu	42	160	55.7	21.75	76.0	95.0	0.8	120/80
S.Po	49	153	72.5	30.97	102.0	108.0	0.94	130/70
S.Si	58	149	50.8	22.88	82.0	90.0	0.91	125/67
Y.Ng	60	150	70.2	31.20	102.5	113.5	0.90	130/70
Mean	55.4	157.2	66.6	26.9	92.0	100.8	0.9	
±	±	±	±	±	±	±	±	125/74
SEM	2.8	2.0	3.1	1.0	2.8	1.8	0.1	
Group B								
A.Mo	59	165	65.4	24.02	85.4	98.0	0.87	130/70
I.So	53	159	63.2	25.00	84.0	92.0	0.91	130/70
K.Ch	56	163	75.0	28.23	99.0	104.0	0.95	130/70
P.Ch	58	170	73.0	25.23	91.0	96.0	0.95	140/70
P.Sr	43	175	72.5	23.67	86.0	96.0	0.90	130/70
T.Li	57	167	70.8	25.39	90.0	96.0	0.94	130/70
T.Ni	56	170	82.5	28.55	98.7	107.0	0.92	130/68
Mean	54.5	166.9	71.8	25.7	90.5	98.4	0.9	
±	±	±	±	±	±	±	±	131/70
SEM	2.5	2.1	2.7	0.7	2.6	2.1	0.1	

no significant difference between group A and group B

Table 11 The appearance of the product, taste, smell, acceptability, tolerance and safety to the cereals and nata de coco supplement in 22 subjects with hyperlipidemia

Subject	Appearance*			Taste*			Smell*			Acceptance [†]			Tolerance [‡]			Safety [§]		
	4	12	20	4	12	20	4	12	20	4	12	20	4	12	20	4	12	20
Group A																		
A.So	3	3	-	3	3	-	3	3	-	1	1	-	1	1	-	1	1	-
B.Ja	2	1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1
B.Ju	1	1	1	1	1	1	1	1	3	1	1	1	1	1	1	1	1	1
B.Ke	1	2	1	2	2	1	2	2	1	1	1	1	1	1	1	1	1	1
J.Bu	3	3	3	3	3	3	3	3	2	1	1	1	1	1	1	1	1	1
P.Ai	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
P.Ke	3	3	2	3	3	2	3	2	2	1	1	1	1	1	1	1	1	1
P.Ra	3	2	2	3	3	3	3	2	2	1	1	1	1	1	1	1	1	1
P.Sm	3	2	2	3	2	2	2	2	2	1	1	1	1	1	1	1	1	1
P.So	2	2	2	2	2	2	2	2	2	1	1	1	1	1	1	1	1	1
P.Wa	3	3	3	3	3	3	2	3	3	1	1	1	1	1	1	1	1	1
S.Bu	2	2	2	3	3	2	2	2	2	1	1	1	1	1	1	1	1	1
S.Po	3	3	-	3	3	-	3	3	-	1	1	-	1	1	-	1	1	-
S.Si	1	1	2	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1
Y.Ng	3	2	2	3	3	2	3	2	2	1	1	1	1	1	1	1	1	1
Mean	2.3	2.1	1.8	2.3	2.3	1.9	2.2	2.2	2.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

* 1=very good, 2=good, 3=acceptable, 4=unacceptable

[†] 1 = whole volume being taken without any problem, 2 = effort being needed to take the whole volume,

3 = the whole volume being partially taken and 4 = the whole volume being rejected.

[‡] 1 = no diarrhea, 2 = diarrhea without clinical significance, 3 = diarrhea and required for the adjustment of the cereals and nata the coco supplement dosage, 4 = diarrhea and required for the discontinuation of the cereals and nata de coco supplement

[§] 1 = no adverse reaction, 2 = adverse reaction but not requiring treatment, 3 = adverse reaction and requiring treatment and 4 = adverse reaction and requiring discontinuation of the cereals and nata de coco supplement

Table 11 The appearance of the product, taste, smell, acceptability, tolerance and safety to the cereals and nata de coco supplement in 22 subjects with hyperlipidemia (continued)

Subject	Appearance*			Taste*			Smell*			Acceptance [†]			Tolerance [‡]			Safety [§]		
	4	12	20	4	12	20	4	12	20	4	12	20	4	12	20	4	12	20
Group B																		
A.Mo	2	2	2	2	2	2	3	2	2	1	1	1	1	1	1	1	1	1
I.So	3	2	2	3	2	2	3	2	2	1	1	1	1	1	1	1	1	1
K.Ch	1	1	1	2	2	1	2	2	1	1	1	1	1	1	1	1	1	1
P.Ch	3	2	2	3	3	2	3	2	2	1	1	1	1	1	1	1	1	1
P.Sr	3	3	-	3	3	-	3	3	-	1	1	-	1	1	-	1	1	-
T.Li	2	2	3	3	3	3	2	2	2	1	1	1	1	1	1	1	1	1
T.Ni	2	2	1	3	3	3	3	3	2	1	1	1	1	1	1	1	1	1
Mean	2.3	2.0	1.8	2.7	2.6	2.2	2.7	2.3	1.8	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

* 1=very good, 2=good, 3=acceptable, 4=unacceptable

[†] 1 = whole volume being taken without any problem, 2 = effort being needed to take the whole volume,

3 = the whole volume being partially taken and 4 = the whole volume being rejected.

[‡] 1 = no diarrhea, 2 = diarrhea without clinical significance, 3 = diarrhea and required for the adjustment of the cereals and nata de coco supplement dosage, 4 = diarrhea and required for the discontinuation of the cereals and nata de coco supplement

[§] 1 = no adverse reaction, 2 = adverse reaction but not requiring treatment, 3 = adverse reaction and requiring treatment and 4 = adverse reaction and requiring discontinuation of the cereals and nata de coco supplement

Table 12 Compliances of consuming the cereals and nata de coco supplement in group A

Subject	wk0-wk4			wk4-wk8			wk8-wk12			wk12-wk16			wk16-wk20		
	Actual intake*		Compliance % of assigned intake [†]	Actual intake*		Compliance % of assigned intake [†]	Actual intake*		Compliance % of assigned intake [†]	Actual intake*		Compliance % of assigned intake [†]	Actual intake*		Compliance % of assigned intake [†]
	g/4 wk	g/d		g/4 wk	g/d		g/4 wk	g/d		g/4 wk	g/d		g/4 wk	g/d	
A.So	810	28.9	96.4	840	30.0	100	765	27.3	91.9	-	-	-	-	-	-
B.Ja	840	30.0	100	840	30.0	100	840	30.0	100	810	28.9	96.4	840	30.0	100
B.Ju	840	30.0	100	840	30.0	100	840	30.0	100	810	28.9	96.4	765	27.3	91.9
B.Ke	840	30.0	100	840	30.0	100	840	30.0	100	810	28.9	96.4	840	30.0	100
J.Bu	840	30.0	100	840	30.0	100	840	30.0	100	810	28.9	96.4	780	27.9	92.86
P.Ai	840	30.0	100	795	28.4	94.6	780	27.8	92.8	765	27.3	91.9	765	27.3	91.9
P.Ke	840	30.0	100	840	30.0	100	795	28.4	94.6	780	27.8	92.7	810	28.9	96.4
P.Ra	840	30.0	100	840	30.0	100	840	30.0	100	810	28.9	96.4	765	27.3	91.9
P.Sm	840	30.0	100	780	27.9	92.9	840	30.0	100	840	30.0	100	840	30.0	100
P.So	840	30.0	100	840	30.0	100	840	30.0	100	810	28.9	96.4	810	28.9	96.4
P.Wa	840	30.0	100	840	30.0	100	825	29.5	98.2	780	27.9	96.4	810	28.9	96.4
S.Bu	810	28.9	96.4	780	27.8	92.8	780	27.9	92.9	765	27.3	91.9	765	27.3	91.9
S.Po	840	30.0	100	840	30.0	100	810	28.9	96.4	-	-	-	-	-	-
S.Si	840	30.0	100	840	30.0	100	810	28.9	96.4	780	27.8	92.8	765	27.3	91.9
Y.Ng	840	30.0	100	840	30.0	100	810	28.9	96.4	840	30.0	100	780	27.9	92.9
Mean	836	29.8	99.5	829	29.6	98.7	817	29.2	97.3	676 ^{b3}	28.6 ^{b3}	80.5 ^{b3}	795 ^{b3}	28.4 ^{b3,c4}	94.6 ^{b3}
±	+	+	±	+	+	±	+	+	±	+	+	±	+	+	±
SEM	6.3	0.2	0.8	8.7	0.3	1.0	10.8	0.4	1.3	12.6	0.4	1.5	13.2	0.5	1.6

* the cereals and nata de coco supplement.

significant difference from wk0 – wk4 : ^{b3} p < 0.02

significant difference from wk4 – wk8 : ^{c4} p < 0.05

Table 13 Compliances of consuming the cereals and nata de coco supplement in group B

Subject	wk0-wk4			wk4-wk8			wk8-wk12			wk12-wk16			wk16-wk20		
	Actual intake*	Compliance	Actual intake*	Compliance	Actual intake*	Compliance	Actual intake*	Compliance	Actual intake*	Compliance	Actual intake*	Compliance	Actual intake*	Compliance	
	g/4wk	% of assigned intake [†]	g/4wk	% of assigned intake [†]	g/4wk	% of assigned intake [†]	g/4wk	% of assigned intake [†]	g/4wk	% of assigned intake [†]	g/4wk	% of assigned intake [†]	g/4wk	% of assigned intake [†]	
A.Mo	840	100	780	92.9	750	89.3	720	85.7	720	85.7	720	85.7	720	85.7	
I.So	750	89.3	720	85.7	690	82.1	600	71.4	630	75.0	630	75.0	630	75.0	
K.Ch	840	100	780	92.9	720	85.7	690	82.1	720	85.7	720	85.7	720	85.7	
P.Ch	840	100	840	100	780	92.9	750	89.3	750	89.3	750	89.3	750	89.3	
P.Sr	750	89.3	720	85.7	780	92.9	-	-	-	-	-	-	-	-	
T.Li	750	89.3	840	100	720	85.7	690	82.1	690	82.1	690	82.1	690	82.1	
T.Ni	840	100	750	89.3	720	85.7	720	85.7	720	85.7	720	85.7	720	85.7	
Mean	801.4	95.4	775.7 ^{C4}	92.3 ^{C4}	737.1	87.7	695.0 ^{b, b3, c4}	82.7 ^{b3}	700	83.3	700	83.3	700	83.3	
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	
SEM	16.6	2.0	19.0	2.3	19.0	2.3	28.4	3.4	28.4	3.4	28.4	3.4	28.4	3.4	

* The cereals and nata de coco supplement.

significant difference from wk0 – wk4 : ^{b3} p < 0.02

significant difference from wk0 – wk4 : ^{c4} p < 0.05

significant difference from group A at wk4 – wk8 : ^{c4} p < 0.05

Table 15 Mean \pm SEM of energy and its energy distribution in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

wk	Energy kcal/d	Protein			CHO	Fat
		Total	Animal	Vegetable		
← % of total calories →						
Group A						
-4	1754.9 \pm 23.6	12.2 \pm 0.9	8.7 \pm 0.6	3.5 \pm 0.7	58.3 \pm 2.8	31.4 \pm 2.3
0	1558.1 \pm 19.3	13.0 \pm 0.9	9.1 \pm 1.0	3.9 \pm 0.8	53.0 \pm 2.8	33.9 \pm 2.6
4	1530.3 \pm 20.9	14.3 \pm 1.0	10.0 \pm 0.8	4.3 \pm 0.6	54.4 \pm 1.3	32.4 \pm 1.6
8	1566.3 \pm 19.4	14.3 \pm 1.2	10.0 \pm 0.6	4.3 \pm 0.6	53.3 \pm 2.6	32.4 \pm 2.2
12	1675.5 \pm 33.4	13.6 \pm 0.7	9.5 \pm 1.0	4.1 \pm 1.1	52.4 \pm 2.2	35.3 \pm 1.9
16	1529.0 \pm 21.5	15.3 \pm 1.1	10.7 \pm 1.1	4.6 \pm 1.0	53.4 \pm 2.7	30.4 \pm 2.6
20	1632.3 \pm 15.3	13.6 \pm 1.1	9.5 \pm 1.1	4.1 \pm 1.0	53.4 \pm 3.9	34.3 \pm 3.6
Group B						
-4	2029.4 \pm 20.5	12.1 \pm 1.2	8.9 \pm 0.7	3.2 \pm 0.8	53.9 \pm 1.9	36.0 \pm 1.7 ^{A4}
0	1837.8 \pm 27.8	12.1 \pm 1.2	8.5 \pm 0.9	3.6 \pm 0.8	56.2 \pm 2.0	32.2 \pm 1.8
4	1926.7 \pm 39.5	12.4 \pm 0.7	8.6 \pm 0.9	3.7 \pm 0.7	58.2 \pm 3.1	30.3 \pm 2.8 ^{a4}
8	1717.8 \pm 26.4	14.5 \pm 0.8 ^{b4}	10.1 \pm 0.6 ^{b4}	4.3 \pm 1.2	56.4 \pm 2.3	30.5 \pm 2.3 ^{a4}
12	2252.7 \pm 36.6 ^{b3,E4}	13.5 \pm 0.7	9.4 \pm 1.0	4.0 \pm 1.0	52.4 \pm 2.8	35.6 \pm 2.8 ^{b4}
16	1957.5 \pm 40.6	12.5 \pm 1.4	8.7 \pm 1.0	3.7 \pm 0.9	57.4 \pm 2.5	31.6 \pm 1.8 ^{a4}
20	1936.8 \pm 48.8	12.5 \pm 0.9	8.8 \pm 0.7	3.8 \pm 1.1	56.3 \pm 1.4	32.5 \pm 1.8 ^{a4}

significant difference from -4: ^{a4} $p < 0.05$

significant difference from 0: ^{b3} $p < 0.02$, ^{b4} $p < 0.05$

significant difference from 12: ^{e4} $p < 0.05$

significant difference from group A at wk -4: ^{A4} $p < 0.05$; at wk 12: ^{E4} $p < 0.05$

Table 16 Mean \pm SEM of total carbohydrate, sugar and total dietary fiber intakes in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

wk	Carbohydrate (g/d)		
	Total	Sugar	Total dietary fiber*
Group A			
-4	255.8 \pm 17.0	14.3 \pm 0.4	16.7 \pm 1.7
0	206.4 \pm 15.6	13.8 \pm 0.5	15.1 \pm 2.0
4	208.1 \pm 6.7 ^{a4}	13.6 \pm 0.4	18.5 \pm 1.0
8	208.7 \pm 17.0 ^{b1}	13.6 \pm 0.4	18.9 \pm 1.3
12	232.0 \pm 8.1 ^{b1}	15.6 \pm 0.5 ^{b1,c1,d1}	18.2 \pm 1.0
16	204.1 \pm 12.8	14.1 \pm 0.4	17.9 \pm 0.7
20	217.9 \pm 14.9 ^{b2}	15.1 \pm 0.4	20.2 \pm 1.5
Group B			
-4	273.5 \pm 15.0	26.3 \pm 0.7 ^{A1}	15.6 \pm 0.8
0	258.2 \pm 14.8	26.4 \pm 0.5 ^{B1}	15.0 \pm 1.2
4	280.3 \pm 7.2	25.3 \pm 0.5 ^{C1}	19.6 \pm 1.0
8	242.2 \pm 12.0	25.2 \pm 0.9 ^{D1}	17.8 \pm 0.7
12	295.1 \pm 16.3 ^{b4,E4}	29.4 \pm 0.6 ^{b1,c1,d1, E1}	19.8 \pm 1.2
16	280.9 \pm 15.5	25.5 \pm 0.2 ^{F1}	20.2 \pm 1.6
20	272.6 \pm 19.1	25.7 \pm 0.3 ^{G1}	19.3 \pm 0.8

* total dietary fiber at week 4 - 20 including dietary fiber from cereals and nata de coco supplement

significant difference from 0: ^{b1} p < 0.005, ^{b2} p < 0.01, ^{b4} p < 0.05

significant difference from group A at wk -4 : ^{A1} p < 0.005; at wk 0 : ^{B1} p < 0.005; at wk 4 : ^{C1} p < 0.005; at wk 8 : ^{D1} p < 0.005; at wk 12 : ^{E1} p < 0.005; at wk 16 : ^{F1} p < 0.005; at wk 20 : ^{G1} p < 0.005

Table 17 Mean \pm SEM of vitamin intake in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

wk	A RE/d	Retinol μ g/d	β -carotene μ g/d	B-1 mg/d	B-2 mg/d	Niacin mg/d	C mg/d
Group A							
-4	310.4 \pm 50.7	160.5 \pm 42.2	899.4 \pm 31.7	0.6 \pm 0.2	1.2 \pm 0.2	8.3 \pm 1.1	142.7 \pm 7.2
0	335.2 \pm 51.8	187.3 \pm 52.0	887.5 \pm 35.9	0.7 \pm 0.2	1.4 \pm 0.3	9.4 \pm 0.9	146.8 \pm 6.2
4	338.2 \pm 47.0	208.4 \pm 25.5	778.9 \pm 17.1	0.6 \pm 0.4	0.9 \pm 0.2	10.4 \pm 1.0	133.2 \pm 21.7
8	412.3 \pm 46.5 ^{a4,b4}	261.5 \pm 43.6 ^{a4,b4}	904.8 \pm 37.4	1.0 \pm 0.2	1.4 \pm 0.2	14.1 \pm 1.4 ^{a4}	165.4 \pm 5.4
12	440.5 \pm 19.6 ^{a4,b4}	288.8 \pm 19.5 ^{a4,b4}	910.4 \pm 25.1	1.4 \pm 0.8 ^{a4,b4}	1.4 \pm 0.2	13.0 \pm 1.3	111.4 \pm 16.1
16	489.4 \pm 27.1 ^{a4,b4}	331.9 \pm 24.3 ^{a4,b4}	945.1 \pm 18.6	2.1 \pm 1.5 ^{a4,b4}	0.9 \pm 0.1	10.2 \pm 1.1	105.3 \pm 27.6
20	455.8 \pm 41.5 ^{a4,b4}	295.4 \pm 55.4 ^{a4,b4}	962.2 \pm 28.7	0.7 \pm 0.2	1.0 \pm 0.1	9.7 \pm 1.2	184.0 \pm 8.2
Group B							
-4	411.6 \pm 47.4	292.8 \pm 45.9	712.6 \pm 27.5	2.1 \pm 1.2	1.5 \pm 0.2	13.8 \pm 1.6	55.9 \pm 16.5 ^{A4}
0	398.2 \pm 46.5	307.7 \pm 46.2	543.2 \pm 27.7	2.1 \pm 1.2	1.4 \pm 0.2	14.2 \pm 1.5	56.0 \pm 16.4 ^{B4}
4	429.0 \pm 14.4	334.4 \pm 19.3	567.8 \pm 30.8	1.4 \pm 0.8	1.2 \pm 0.2	10.8 \pm 1.0	96.0 \pm 20.5
8	465.8 \pm 48.7 ^{D4}	378.7 \pm 49.2	522.6 \pm 32.7	1.8 \pm 1.2	1.4 \pm 0.3	9.4 \pm 1.1 ^{b4}	137.5 \pm 6.1
12	463.5 \pm 31.0 ^{E4}	375.4 \pm 25.0 ^{a4,E4}	528.6 \pm 25.2	0.9 \pm 0.2 ^{a4,b4}	1.2 \pm 0.2	13.0 \pm 0.9	99.3 \pm 19.7
16	409.6 \pm 32.9	294.0 \pm 50.9	689.7 \pm 15.9	2.2 \pm 1.3	1.2 \pm 0.1	10.6 \pm 1.2	116.9 \pm 12.5
20	421.7 \pm 29.7	296.6 \pm 51.7	750.4 \pm 45.7 ^{b4}	0.8 \pm 0.1 ^{a4,b4}	1.3 \pm 0.2	12.8 \pm 0.7	75.8 \pm 23.6 ^{G4}

significant difference from -4: ^{a4} $p < 0.05$

significant difference from 0: ^{b4} $p < 0.05$

significant difference from group A at wk -4: ^{A4} $p < 0.05$; at wk 0: ^{B4} $p < 0.05$; at wk 12: ^{E4} $p < 0.05$; at wk 20: ^{G4} $p < 0.05$

Table 18 Mean \pm SEM of mineral intakes in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

Mineral	Unit	Wk -4 (N=22)	Wk 0 (N=22)	Wk 4 (N=22)	Wk 8 (N=22)	Wk 12 (N=22)	Wk 16 (N=19)	Wk 20 (N=19)
Group A								
Ca	mg/d	647.3 \pm 59.0	561.0 \pm 65.1	570.8 \pm 18.1	557.8 \pm 34.8	573.1 \pm 63.3	673.1 \pm 16.9	549.9 \pm 64.9
Cu	mg/d	0.3 \pm 0.1	0.4 \pm 0.1	0.3 \pm 0.1	0.6 \pm 0.1	0.5 \pm 0.1	0.3 \pm 0.1	0.6 \pm 0.2
Fe								
Fe-Ani	mg/d	4.0 \pm 0.7	5.1 \pm 0.8	4.0 \pm 0.8	5.5 \pm 1.4	4.5 \pm 0.6	4.8 \pm 1.0	3.5 \pm 1.0
Fe-Veg	mg/d	4.3 \pm 0.9	5.4 \pm 0.9	3.0 \pm 0.4	5.9 \pm 1.0	3.9 \pm 0.7	3.0 \pm 0.6	5.4 \pm 0.8
Mg	mg/d	12.0 \pm 1.5	13.6 \pm 1.7	6.2 \pm 1.5	12.6 \pm 2.0	8.6 \pm 2.6	9.6 \pm 2.8	9.6 \pm 2.4
P	mg/d	623.1 \pm 59.2	639.2 \pm 61.3	574.6 \pm 75.2	772.7 \pm 78.0	656.9 \pm 66.3	645.1 \pm 78.1	569.9 \pm 63.7
K	mg/d	1136 \pm 29	1148 \pm 23	1226 \pm 13	1389 \pm 33	1609 \pm 17	1261 \pm 33	1426 \pm 17
Se	mg/d	0.2 \pm 0.1	0.2 \pm 0.1	0.6 \pm 0.2	0.4 \pm 0.2	0.4 \pm 0.2	0.2 \pm 0.1	0.2 \pm 0.1
Na	mg/d	1963 \pm 26	2027 \pm 25	2645 \pm 19	2169 \pm 29	2806 \pm 27	2508 \pm 21	2129 \pm 29
Zn	mg/d	1.5 \pm 0.3	1.6 \pm 0.3	1.6 \pm 0.2	2.5 \pm 0.4	1.9 \pm 0.3	1.8 \pm 0.2	2.0 \pm 0.4
Phytate	mg/d	6.6 \pm 2.0	7.8 \pm 1.7	4.4 \pm 1.8	24.6 \pm 15.6	16.4 \pm 11.6	2.6 \pm 0.9	40.3 \pm 22.2
Group B								
Ca	mg/d	664.5 \pm 15.4	665.8 \pm 15.1	595.3 \pm 61.2	569.0 \pm 11.4	667.5 \pm 10.2	660.5 \pm 11.4	649.3 \pm 64.2
Cu	mg/d	0.4 \pm 0.1	0.5 \pm 0.1	0.4 \pm 0.1	0.2 \pm 0.1	0.5 \pm 0.1	0.5 \pm 0.1	0.4 \pm 0.1
Fe								
Fe-Ani	mg/d	9.6 \pm 1.3	9.5 \pm 1.3	3.1 \pm 0.6	4.2 \pm 1.0	4.1 \pm 0.8	5.3 \pm 1.3	4.2 \pm 1.2
Fe-Veg	mg/d	4.4 \pm 0.9	4.9 \pm 1.0	3.5 \pm 0.6	4.4 \pm 0.8	4.2 \pm 0.6	3.4 \pm 0.8	4.2 \pm 0.9
Mg	mg/d	12.1 \pm 1.9	12.4 \pm 1.9	12.9 \pm 4.4	13.4 \pm 1.6	10.8 \pm 1.7	11.5 \pm 1.4	10.9 \pm 2.4
P	mg/d	806.6 \pm 75.1	813.8 \pm 73.4	538.7 \pm 38.3	680.3 \pm 64.4	733.4 \pm 79.6	747.9 \pm 51.1	662.0 \pm 57.9
K	mg/d	1245 \pm 13	1247 \pm 13	1289 \pm 19	1006 \pm 59	1495 \pm 15	1194 \pm 19	1278 \pm 78
Se	mg/d	0.4 \pm 0.2	0.4 \pm 0.2	0.2 \pm 0.1	0.2 \pm 0.1	0.4 \pm 0.2	0.2 \pm 0.1	0.2 \pm 0.1
Na	mg/d	2324 \pm 30	2311 \pm 30	2055 \pm 24	2168 \pm 27	2517 \pm 21	2089 \pm 21	2551 \pm 26
Zn	mg/d	2.3 \pm 0.4	2.2 \pm 0.4	1.9 \pm 0.3	1.2 \pm 0.2	2.2 \pm 0.4	2.2 \pm 0.4	1.4 \pm 0.2
Phytate	mg/d	6.7 \pm 1.8	7.5 \pm 2.3	12.8 \pm 6.0	4.7 \pm 0.8	21.1 \pm 12.3	20.9 \pm 17.7	8.8 \pm 2.3

Table 19 Body weight in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

subject	Body weight (kg)						
	Wk -4	Wk 0	Wk 4	Wk 8	Wk 12	Wk 16	Wk 20
Group A							
A.So	64.0	64.1	63.4	64.3	64.5	-	-
B.Ja	77.9	79.3	79.9	80.4	80.6	78.2	75.4
B.Ju	66.5	66.1	65.2	64.3	65.8	64.8	63.8
B.Ke	63.1	63.4	63.4	64.2	64.2	63.0	64.1
J.Bu	77.2	81.2	80.9	80.4	82.2	81.2	79.6
P.Ai	100.2	98.6	97.7	97.1	97.6	97.6	99.5
P.Ke	58.7	57.5	57.1	56.6	56.3	55.4	54.6
P.Ra	55.5	54.3	54.2	54.5	54.9	55.3	55.1
P.Sm	61.0	60.5	60.0	59.3	59.0	59.0	58.4
P.So	63.6	64.1	64.3	64.0	63.7	64.6	64.8
P.Wa	61.3	61.0	59.7	60.1	61.2	61.4	60.2
S.Bu	55.7	55.8	55.6	55.8	55.3	56.4	55.8
S.Po	72.5	72.5	70.2	71.1	73.3	-	-
S.Si	50.8	51.4	52.6	51.4	51.8	52.2	52.4
Y.Ng	70.2	69.5	72.8	73.6	74.7	75.3	74.8
Mean	66.3	66.4	66.4	66.3	66.7	66.5	66.0
±	±	±	±	±	±	±	±
SEM	3.6	3.6	3.6	3.6	3.7	3.6	3.7
Group B							
A.Mo	65.4	64.9	64.3	65.4	65.6	66.4	66.6
I.So	63.2	64.3	63.4	64.0	63.8	64.2	65.4
K.Ch	75.0	75.6	77.4	77.6	77.4	77.0	78.4
P.Ch	73.0	72.1	71.6	70.0	71.8	70.6	71.8
P.Sr	72.5	71.2	69.8	69.7	69.5	-	-
T.Li	70.8	70.5	72.6	72.4	73.0	73.0	73.4
T.Ni	82.5	83.0	84.4	82.4	83.4	82.8	83.8
Mean	71.6	71.7	72.3	72.0	72.5	72.3	73.2
±	±	±	±	±	±	±	±
SEM	2.8	2.9	3.2	2.9	3.0	2.8	2.9

Table 20 BMI in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

subject	BMI (kg / m ²)						
	Wk -4	Wk 0	Wk 4	Wk 8	Wk 12	Wk 16	Wk 20
Group A							
A.So	25.00	25.04	24.77	25.12	25.20	-	-
B.Ja	34.62	35.24	35.51	35.73	35.82	34.76	33.51
B.Ju	24.72	24.58	24.24	23.91	24.46	24.09	23.72
B.Ke	26.60	26.73	26.73	27.07	27.07	26.56	27.03
J.Bu	23.56	24.79	24.69	24.54	25.09	24.79	24.30
P.Ai	33.48	32.94	32.64	32.44	32.61	32.61	33.25
P.Ke	26.80	26.25	26.07	25.84	25.70	25.29	24.93
P.Ra	29.57	28.93	28.87	29.04	29.25	29.46	29.36
P.Sm	22.96	22.77	22.58	22.32	22.21	22.21	21.98
P.So	24.23	24.42	24.50	24.39	24.27	24.62	24.09
P.Wa	25.52	25.39	24.85	25.02	25.47	25.56	25.06
S.Bu	21.75	21.80	21.71	21.79	21.60	22.03	21.80
S.Po	30.97	30.97	29.99	30.37	31.31	-	-
S.Si	22.88	23.15	23.69	23.15	23.33	23.51	23.60
Y.Ng	31.20	30.89	32.36	32.71	33.20	33.47	33.24
Mean	26.8	26.8	26.8	26.8	26.9	26.8	26.6
± SEM	± 1.2	± 1.1	± 1.2	± 1.2	± 1.2	± 1.2	± 1.2
Group B							
A.Mo	24.02	23.84	23.62	24.02	24.10	24.39	24.46
I.So	25.00	25.43	25.08	25.32	25.24	25.39	25.87
K.Ch	28.23	28.45	29.13	29.21	29.13	28.98	29.51
P.Ch	25.23	24.95	24.78	24.22	24.84	24.43	24.84
P.Sr	23.67	23.24	22.80	22.76	22.69	-	-
T.Li	25.38	25.28	26.03	25.96	26.18	26.18	26.32
T.Ni	28.55	28.72	29.20	28.51	28.86	28.65	29.00
Mean	25.9	25.9	25.9	25.75	25.9	26.2	26.30
± SEM	± 0.93	± 0.90	± 0.95	± 0.93	± 0.93	± 0.97	± 1.06

Table 21 Body fat and fat-free mass in group A on the cereals and nata de coco supplement

Subject	Body fat (% of body weight)										Body fat (kg)										Fat – free mass									
	-4	0	4	8	12	16	20	-4	0	4	8	12	16	20	-4	0	4	8	12	16	20									
A.So	41.2	40.7	37.4	37.8	39.6	-	-	27.4	26.1	23.7	24.3	25.5	-	-	37.6	38.0	39.7	40.0	39.0	-	-									
B.Ja	44.7	43.2	45.4	43.6	43.0	43.1	42.4	35.2	34.3	36.3	35.1	34.7	33.7	32.0	44.8	45.0	43.6	45.3	45.9	44.5	43.4									
B.Ju	33.2	32.2	33.8	32.6	32.4	32.8	32.3	23.8	21.3	22.0	21.0	21.3	21.3	20.6	45.6	44.8	43.2	43.3	44.5	43.5	43.2									
B.Ke	39.4	42.2	42.9	38.7	38.5	38.7	38.9	24.9	26.8	27.2	24.8	24.7	24.4	24.9	38.2	36.6	36.2	39.4	39.5	38.6	39.2									
J.Bu	27.8	26.2	28.3	24.8	26.7	25.7	24.4	22.4	21.3	22.9	19.9	21.9	20.9	19.4	58.6	59.9	58.0	60.5	60.3	60.3	60.2									
P.Ai	36.7	36.5	34.9	35.3	34.4	34.3	35.7	36.8	36.0	34.1	34.3	33.6	33.5	35.5	63.4	62.6	63.5	62.8	64.0	64.2	64.0									
P.Ke	40.9	40.8	40.5	40.2	41.7	39.7	40.4	24.0	23.9	23.1	22.6	23.5	22.0	22.1	34.7	35.2	34.0	33.4	32.8	33.4	32.5									
P.Ra	42.0	41.9	43.8	42.4	41.8	42.9	42.4	22.8	23.0	23.7	23.2	22.9	23.7	23.4	31.5	32.0	30.5	31.4	32.0	31.6	31.7									
P.Sm	26.8	27.6	29.7	31.0	28.1	28.6	28.6	17.2	16.7	17.8	18.4	16.6	16.9	16.7	42.2	43.8	42.2	40.9	42.4	42.1	41.7									
P.So	33.2	32.5	33.6	32.4	34.0	32.5	33.2	23.8	20.8	21.6	20.7	21.7	21.0	21.5	47.8	43.3	42.7	43.3	42.0	43.6	43.3									
P.Wa	40.2	39.6	39.5	37.1	38.4	38.9	38.6	25.2	24.3	23.6	22.3	23.5	23.9	23.1	37.4	37.0	36.1	37.8	37.7	37.5	37.0									
S.Bu	40.3	35.8	32.6	29.1	33.3	33.9	34.7	22.4	20.0	17.7	16.2	18.4	19.1	19.4	33.3	35.8	38.6	39.4	36.9	37.3	36.4									
S.Po	43.7	42.7	41.0	42.1	40.6	-	-	31.7	31.3	29.2	30.9	28.8	-	-	40.8	42.0	41.9	42.4	50.8	-	-									
S.Si	37.2	39.0	38.8	33.0	33.3	34.6	36.3	18.5	21.4	20.4	17.0	17.2	18.1	19.0	33.4	31.3	32.2	34.4	34.6	34.1	33.4									
Y.Ng	47.6	45.4	42.7	42.8	43.8	43.4	44.5	33.5	30.2	31.1	31.5	32.7	32.7	33.3	34.8	41.6	41.7	42.1	42.0	42.6	41.5									
Mean	37.5	37.1	37.4	35.6	36.1	36.1	36.3	25.4	24.6	24.7	23.6	24.0	23.9	23.9	42.0	42.2	41.7	42.6	42.7	42.6	42.1									
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±									
SEM	1.7	1.7	1.6	1.6	1.5	1.6	1.6	1.7	1.6	1.6	1.7	1.7	1.6	1.6	2.8	2.7	2.6	2.6	2.7	2.7	2.7									

Table 22 Body fat and fat-free mass in group B on the cereals and nata de coco supplement

Subject	Body fat (% of body weight)								Body fat (kg)								Fat – free mass							
	-4	0	4	8	12	16	20	-4	0	4	8	12	16	20	-4	0	4	8	12	16	20			
A.Mo	30.2	29.3	29.3	28.6	27.5	29.1	29.9	19.8	19.0	18.8	18.7	18.0	19.3	19.9	44.1	45.9	45.5	46.7	47.6	47.1	46.7			
I.So	26.8	25.6	31.5	25.2	32.4	26.2	27.5	17.8	16.5	20.0	16.1	20.7	16.8	18.0	46.3	47.8	43.4	47.9	43.1	47.4	47.4			
K.Ch	36.2	35.5	35.0	34.0	33.3	31.9	35.0	29.6	28.4	27.1	26.4	25.8	24.6	27.4	48.2	49.6	50.3	51.2	51.6	52.4	51.0			
P.Ch	33.8	33.9	33.2	31.4	30.9	31.8	31.1	24.2	24.4	23.8	22.0	22.2	22.5	22.3	48.3	47.7	47.8	48.0	49.3	48.1	49.5			
P.Sr	33.0	28.8	29.7	30.0	27.9	-	-	23.9	20.2	21.3	20.9	19.4	-	-	48.6	51.0	52.4	48.8	50.1	-	-			
T.Li	31.8	31.1	30.4	30.1	29.5	28.4	28.1	22.5	22.3	22.1	21.8	20.4	20.7	20.6	48.1	49.5	50.5	50.6	51.3	52.3	52.8			
T.Ni	33.8	33.2	33.3	30.7	31.5	32.7	31.7	28.9	28.3	28.1	25.3	26.3	27.1	26.6	56.2	56.1	56.3	57.1	57.1	55.7	57.2			
Mean	32.1	31.4	32.1	30.0	30.8	30.0	30.5	23.8	23.1	23.3	21.7	22.2	21.8	22.5	48.5	49.4	49.0	50.2	50.0	50.5	50.8			
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±			
SEM	1.3	1.5	0.8	1.2	0.8	1.0	1.1	1.9	2.0	1.5	1.6	1.3	1.5	1.5	1.7	1.4	1.8	1.5	1.9	1.4	1.6			

Table 25 Triceps skinfold thickness in group A on the cereals and nata de coco supplement

Subject	TST (mm)										TST (% std)										
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
A.So	34.0	34.0	33.0	32.8	30.6	-	-	206.1	206.1	200.0	198.8	185.5	-	-	206.1	206.1	200.0	198.8	185.5	-	-
B.Ja	29.0	31.4	29.2	30.6	30.3	30.2	31.0	175.8	190.3	177.0	185.5	183.6	183.0	187.9	175.8	190.3	177.0	185.5	183.6	183.0	183.0
B.Ju	25.4	25.2	25.4	25.2	25.8	25.0	24.6	203.2	201.6	203.2	201.6	206.4	200	196.8	203.2	201.6	203.2	201.6	206.4	200	200
B.Ke	26.8	26.0	26.0	25.4	25.6	25.4	26.0	162.4	157.6	157.6	153.9	155.2	153.9	157.6	162.4	157.6	157.6	153.9	155.2	153.9	153.9
J.Bu	12.2	13.2	14.0	14.0	13.6	14.0	14.0	97.6	105.6	112	112	108.8	112	112	97.6	105.6	112	112	108.8	112	112
P.Ai	21.4	21.4	21.0	20.8	21.0	21.4	22.0	171.2	171.2	168	166.4	168	171.2	176	171.2	171.2	168	166.4	168	171.2	171.2
P.Ke	30.2	30.4	30.2	30.2	29.8	29.4	29.2	183.0	184.2	183.0	183.0	180.6	178.2	177.0	183.0	184.2	183.0	183.0	180.6	178.2	178.2
P.Ra	27.0	26.2	26.0	26.2	26.4	26.6	26.4	163.6	158.8	157.6	158.8	160.0	161.2	160.0	163.6	158.8	157.6	158.8	160.0	161.2	161.2
P.Sm	13.8	14.0	14.0	12.4	12.2	12.4	12.0	110.4	112.0	112.0	99.2	97.6	96	96	110.4	112.0	112.0	99.2	97.6	99.2	99.2
P.So	25.2	24.2	24.0	24.0	23.8	24.0	25.4	152.7	146.7	145.5	145.5	144.2	145.5	153.9	152.7	146.7	145.5	145.5	144.2	145.5	145.5
P.Wa	19.6	19.4	19.8	19.7	19.2	19.6	19.0	118.8	117.6	120.0	119.4	116.4	115.2	115.2	118.8	117.6	120.0	119.4	116.4	118.8	118.8
S.Bu	20.4	20.3	20.4	21.0	20.8	20.0	20.2	123.6	123.0	123.6	127.3	126.1	122.4	122.4	123.6	123.0	123.6	127.3	126.1	121.2	121.2
S.Po	44.6	44.0	43.8	44.0	44.4	-	-	270.3	266.7	265.5	266.7	269.1	-	-	270.3	266.7	265.5	266.7	269.1	-	-
S.Si	11.0	10.6	9.1	10.8	10.5	11.3	11.5	66.7	64.2	55.2	65.5	63.6	68.5	69.7	66.7	64.2	55.2	65.5	63.6	68.5	68.5
Y.Ng	31.4	31.0	30.0	30.5	29.6	30.2	30.0	190.3	187.9	181.8	184.8	179.4	183.0	181.8	190.3	187.9	181.8	184.8	179.4	183.0	183.0
Mean	22.9	22.6	22.2	22.4	22.2	22.3	22.4	147.6	147.7	145.9	146.4	145.4	145.8	146.6	147.6	147.7	145.9	146.4	145.4	145.8	145.8
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	1.9	1.9	1.8	1.9	1.9	1.8	1.8	11.3	11.3	11.1	11.0	11.3	10.8	11.0	11.3	11.3	11.1	11.0	11.3	10.8	10.8

Table 26 Triceps skinfold thickness in group B on the cereals and nata de coco supplement

Subject	TST (mm)								TST (%std)							
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20		
A.Mo	14.8	14.0	12.4	11.2	10.8	11.3	10.6	118.4	112	99.2	89.6	86.4	90.4	84.8		
I.So	11.0	10.6	9.4	9.6	11.0	11.2	11.8	88	84.8	75.2	76.8	88	89.6	94.4		
K.Ch	13.2	14.0	15.2	14.6	15.0	15.6	15.2	105.6	112	121.6	116.8	120	124.8	121.6		
P.Ch	18.4	18.2	18.4	18.6	19.3	18.6	18.8	147.2	145.6	147.2	148.8	154.4	148.8	150.4		
P.Sr	8.4	8.8	9.0	8.8	9.0	-	-	67.2	70.4	72	70.4	72	-	-		
T.Li	12.0	12.8	13.4	13.8	13.8	14.2	14.0	96.0	102.4	107.2	110.4	110.4	113.6	112		
T.Ni	15.2	14.8	14.5	14.2	15.0	15.3	15.2	121.6	118.4	116	113.6	120	122.4	121.6		
Mean	14.1	14.1	13.9 ^{C4}	13.7 ^{D4}	14.2 ^{E4}	14.4	14.3	112.8	112.5	111.1	109.3 ^{D4}	113.2	114.9	114.1		
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±		
SEM	1.1	1.0	1.2	1.3	1.3	1.2	1.2	8.6	8.2	9.8	10.1	10.2	9.2	9.4		

significant difference from compliance > 90 % of assigned intake group at wk4: ^{C4} p < 0.05; at wk8: ^{D4} p < 0.05; at wk12: ^{E4} p < 0.05

Table 27 Biceps skinfold thickness in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

Subject	BST (mm)						
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
Group A							
A.So	14.6	14.8	14.8	16.4	14.2	-	-
B.Ja	14.4	13.8	13.6	14.0	13.6	12.6	12.5
B.Ju	15.0	14.5	14.2	13.6	14.6	13.2	13.8
B.Ke	15.4	15.2	15.0	15.0	15.2	15.0	15.6
J.Bu	5.4	4.8	5.4	5.6	5.6	5.5	5.5
P.Ai	13.6	13.2	12.8	12.0	12.6	12.4	13.0
P.Ke	17.0	16.7	16.0	15.8	15.4	15.2	15.0
P.Ra	15.2	15.4	14.2	14.2	13.6	14.0	13.8
P.Sm	6.6	6.8	6.4	6.0	6.2	6.0	6.0
P.So	8.6	8.4	8.0	8.2	8.0	8.2	8.0
P.Wa	13.8	14.0	14.6	13.3	14.0	14.5	13.0
S.Bu	9.2	9.2	8.8	9.0	8.6	8.6	8.4
S.Po	11.0	12.0	12.4	11.6	11.8	-	-
S.Si	5.2	5.0	4.0	4.6	4.5	4.7	5.0
Y.Ng	15.2	15.0	15.0	14.8	14.6	15.0	15.2
Mean	11.9	11.7	11.4	11.2	11.3	11.1	11.1
±	±	±	±	±	±	±	±
SEM	1.2	1.2	1.2	1.1	1.1	1.1	1.1
Group B							
A.Mo	5.0	5.1	6.4	6.4	6.5	7.4	6.8
I.So	5.8	5.4	5.5	5.0	4.8	4.8	5.0
K.Ch	8.5	8.2	8.8	8.4	8.4	8.6	8.8
P.Ch	12.0	12.0	12.4	12.0	12.2	12.2	12.0
P.Sr	8.4	8.6	8.6	8.5	8.2	-	-
T.Li	6.0	6.0	6.8	6.5	6.6	7.2	7.2
T.Ni	8.8	8.0	7.8	7.1	7.3	7.1	7.4
Mean	7.7	7.4	8.0	7.6	7.6	7.9	7.9
±	±	±	±	±	±	±	±
SEM	1.1	1.0	1.0	1.0	1.0	1.0	1.0

Table 28 Sub scapula skinfold thickness in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

Subject	SST (mm)						
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
Group A							
A.So	30.0	29.0	28.0	31.0	31.0	-	-
B.Ja	49.0	51.0	52.0	54.0	55.4	55.4	56.0
B.Ju	47.0	47.2	47.0	46.8	46.8	46.0	46.4
B.Ke	37.6	37.2	37.0	36.8	36.6	36.2	36.8
J.Bu	22.2	23.8	22.6	22.6	23.0	22.4	21.6
P.Ai	52.0	50.8	50.4	50.0	50.2	49.6	50.0
P.Ke	46.6	47.0	47.2	47.0	46.4	46.6	46.0
P.Ra	38.6	38.6	38.0	38.0	37.8	38.2	38.6
P.Sm	29.0	29.4	28.8	28.6	28.4	28.6	28.4
P.So	37.0	36.8	36.8	36.0	36.2	37.0	36.2
P.Wa	40.0	39.8	39.4	40.0	39.8	39.6	39.6
S.Bu	41.2	40.0	38.4	39.2	39.0	38.6	38.4
S.Po	49.6	50.0	50.2	49.8	49.8	-	-
S.Si	22.8	21.6	20.4	20.8	20.3	20.3	20.0
Y.Ng	47.0	47.0	47.0	46.5	46.4	46.8	46.6
Mean	39.2	39.2	38.8	38.9	38.9	38.9	38.8
±	±	±	±	±	±	±	±
SEM	2.7	2.7	2.8	2.8	2.9	2.9	2.9
Group B							
A.Mo	24.4	24.4	24.8	24.0	24.5	25.0	25.1
I.So	19.4	23.0	17.2	17.6	18.4	18.6	18.4
K.Ch	33.2	34.2	35.4	35.3	35.0	35.2	35.7
P.Ch	35.0	34.8	35.2	34.0	35.1	35.0	36.5
P.Sr	27.2	27.4	27.0	26.8	26.4	-	-
T.Li	34.6	34.8	35.5	35.2	35.4	34.8	35.0
T.Ni	23.0	22.8	22.6	22.7	22.8	22.8	22.6
Mean	28.3	29.0	28.4 ^{C4}	28.1 ^{D4}	28.5 ^{E4}	28.6	28.9
±	±	±	±	±	±	±	±
SEM	2.8	2.5	3.2	3.1	3.1	3.0	3.2

significant difference from group A at wk 4 : ^{C4} p < 0.05; at wk 8 : ^{D4} p < 0.05; at wk 12 : ^{E4} p < 0.05

Table 29 Supra iliac skinfold thickness in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

Subject	SIT (mm)						
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
Group A							
A.So	35.0	35.4	32.0	32.4	31.8	-	-
B.Ja	47.0	48.2	51.4	53.0	53.5	49.8	50.0
B.Ju	44.0	44.0	45.5	45.2	45.8	46.0	46.4
B.Ke	39.0	38.8	38.4	38.2	38.4	38.6	39.0
J.Bu	31.2	31.4	28.0	28.4	29.0	28.0	28.0
P.Ai	53.0	51.4	51.0	49.4	50.0	49.4	50.0
P.Ke	50.4	49.2	49.0	49.0	48.0	48.2	48.0
P.Ra	32.2	32.0	32.0	32.0	32.8	33.0	32.8
P.Sm	26.2	26.4	26.0	24.2	24.0	24.0	23.8
P.So	33.2	32.2	32.0	32.0	32.0	32.4	33.0
P.Wa	32.0	31.0	28.8	30.9	31.4	32.2	32.2
S.Bu	35.4	34.8	34.6	34.0	33.0	33.4	33.0
S.Po	39.4	39.0	38.0	37.8	37.6	-	-
S.Si	24.4	24.8	25.6	25.0	25.2	25.1	25.2
Y.Ng	38.0	38.2	36.0	36.5	36.0	36.4	36.6
Mean	37.4	37.1	36.8	36.8	36.8	36.6	36.4
± SEM	± 2.5	± 2.4	± 2.6	± 2.6	± 2.7	± 2.5	± 2.6
Group B							
A.Mo	17.2	17.0	16.8	16.3	16.8	17.0	17.5
I.So	32.0	32.6	32.4	31.0	29.8	30.0	30.6
K.Ch	22.8	22.6	23.0	23.4	23.5	23.3	22.8
P.Ch	30.2	30.0	29.2	29.2	29.6	29.8	29.6
P.Sr	29.6	29.5	29.4	28.6	28.8	-	-
T.Li	20.0	19.8	19.4	20.8	20.8	21.0	21.8
T.Ni	22.0	21.4	18.3	21.6	20.6	19.8	18.8
Mean	24.0	23.9	23.2	23.7	23.5	23.5	23.5
± SEM	± 2.4	± 2.5	± 2.6	± 2.2	± 2.1	± 2.2	± 2.2

Table 30 Mid upper arm circumference in group A on the cereals and nata de coco supplement

Subject	MUAC (cm)										MUAC (% std)										
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
A.So	30.2	30.0	30.5	30.5	31.0	-	-	106.0	105.3	107.0	107.0	108.8	-	-	106.0	105.3	107.0	107.0	108.8	-	-
B.Ja	36.0	34.0	35.5	36.0	37.0	36.0	37.0	126.3	119.3	124.6	126.3	129.8	126.3	129.8	126.3	119.3	124.6	126.3	129.8	126.3	129.8
B.Ju	29.0	29.2	30.0	31.0	30.0	30.5	30.2	99.0	99.7	102.4	105.8	102.4	104.1	103.1	99.0	99.7	102.4	105.8	102.4	104.1	103.1
B.Ke	29.0	29.5	30.0	29.0	29.2	29.0	30.0	101.8	103.5	105.3	101.8	102.5	101.8	105.3	101.8	103.5	105.3	101.8	102.5	101.8	105.3
J.Bu	31.0	31.5	32.5	32.3	33.5	32.5	32.5	105.8	107.5	110.9	110.2	114.3	110.9	110.9	105.8	107.5	110.9	110.2	114.3	110.9	110.9
P.Ai	33.5	34.0	34.0	34.5	34.5	34.5	35.0	114.3	116.0	116.0	117.7	117.7	117.7	119.5	114.3	116.0	116.0	117.7	117.7	117.7	119.5
P.Ke	28.3	28.5	29.0	28.5	28.5	28.0	27.6	99.3	100.0	101.8	100.0	100.0	98.2	96.8	99.3	100.0	101.8	100.0	100.0	98.2	96.8
P.Ra	29.0	29.0	29.0	29.0	28.5	29.5	29.6	101.8	101.8	101.8	101.8	100.0	103.5	103.9	101.8	101.8	101.8	101.8	100.0	103.5	103.9
P.Sm	28.0	27.5	27.5	26.8	26.8	26.6	26.5	95.6	93.9	93.9	91.5	91.5	90.8	90.4	95.6	93.9	93.9	91.5	91.5	90.8	90.4
P.So	29.0	28.5	28.5	28.2	28.2	28.5	28.7	101.8	100.0	100.0	98.9	98.9	100.0	100.7	101.8	100.0	100.0	98.9	98.9	100.0	100.7
P.Wa	35.5	34.2	33.6	32.7	33.0	32.6	31.8	124.6	120.0	117.9	114.7	115.8	114.4	111.6	124.6	120.0	117.9	114.7	115.8	114.4	111.6
S.Bu	27.5	27.5	27.5	27.5	27.5	27.0	27.0	96.5	96.5	96.5	96.5	96.5	94.7	94.7	96.5	96.5	96.5	96.5	96.5	94.7	94.7
S.Po	35.5	36.0	35.0	34.8	34.8	-	-	124.6	126.3	122.8	122.1	122.1	-	-	124.6	126.3	122.8	122.1	122.1	-	-
S.Si	25.0	25.0	26.0	26.5	26.0	25.5	25.0	87.7	87.7	91.2	93.0	91.2	89.5	87.7	87.7	87.7	91.2	93.0	91.2	89.5	87.7
Y.Ng	34.2	33.5	35.0	34.5	34.4	34.5	34.0	120.0	117.5	122.8	121.1	120.7	121.1	119.3	120.0	117.5	122.8	121.1	120.7	121.1	119.3
Mean	30.4	30.1	30.6	30.5	30.5	30.4	30.4	105.7	104.9	106.5	106.1	106.2	105.6	105.7	105.7	104.9	106.5	106.1	106.2	105.6	105.7
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	0.9	0.8	0.9	0.9	1.0	0.9	1.0	3.3	2.9	3.0	3.1	3.3	3.2	3.4	3.3	2.9	3.0	3.1	3.3	3.2	3.4

Table 32 Mid upper arm muscle circumference in group A on the cereals and nata de coco supplement

Subject	UAMC (cm)										UAMC (% std)										
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
A.So	19.5	19.3	20.1	20.2	21.4	-	-	84.1	83.3	86.8	87.0	92.2	-	-	84.1	83.3	86.8	87.0	92.2	-	-
B.Ja	26.9	24.1	26.3	26.4	27.5	26.5	27.3	115.9	104.0	113.5	113.7	118.4	114.3	117.5	115.9	104.0	113.5	113.7	118.4	114.3	117.5
B.Ju	21.0	21.3	22.0	23.1	21.9	22.6	22.5	83.1	84.1	87.0	91.2	86.5	89.5	88.8	83.1	84.1	87.0	91.2	86.5	89.5	88.8
B.Ke	20.6	21.3	21.8	21.0	21.2	21.0	21.8	88.7	91.9	94.1	90.6	91.2	90.6	94.1	88.7	91.9	94.1	90.6	91.2	90.6	94.1
J.Bu	27.2	27.4	28.1	27.9	29.2	28.1	28.1	107.4	108.1	111.1	110.3	115.5	111.1	111.1	107.4	108.1	111.1	110.3	115.5	111.1	111.1
P.Ai	26.8	27.3	27.4	28.0	27.9	27.8	28.1	105.8	107.8	108.3	110.5	110.3	109.8	111.0	105.8	107.8	108.3	110.5	110.3	109.8	111.0
P.Ke	18.8	18.9	19.5	19.0	19.1	18.8	18.4	81.1	81.7	84.1	81.9	82.5	80.9	79.4	81.1	81.7	84.1	81.9	82.5	80.9	79.4
P.Ra	20.5	20.8	20.8	20.8	20.2	21.1	21.3	88.4	89.5	89.8	89.5	87.1	91.1	91.8	88.4	89.5	89.8	89.5	87.1	91.1	91.8
P.Sm	23.7	23.1	23.1	22.9	23.0	22.7	22.7	93.5	91.3	91.3	90.5	90.8	89.7	89.8	93.5	91.3	91.3	90.5	90.8	89.7	89.8
P.So	21.1	20.9	21.0	20.7	20.7	21.0	20.7	90.9	90.1	90.3	89.0	89.3	90.3	89.3	90.9	90.1	90.3	89.0	89.3	90.3	89.3
P.Wa	29.3	28.1	27.4	26.5	27.0	26.4	25.8	126.5	121.1	118.0	114.3	116.2	114.0	111.3	126.5	121.1	118.0	114.3	116.2	114.0	111.3
S.Bu	21.1	21.1	21.1	20.9	21.0	20.7	20.7	90.9	91.0	90.9	90.1	90.4	89.3	89.0	90.9	91.0	90.9	90.1	90.4	89.3	89.0
S.Po	21.5	22.2	21.2	21.0	20.8	-	-	92.6	95.6	91.5	90.4	89.9	-	-	92.6	95.6	91.5	90.4	89.9	-	-
S.Si	21.5	21.7	23.1	23.1	22.7	21.9	21.4	92.9	93.4	99.7	99.6	97.8	94.6	92.2	92.9	93.4	99.7	99.6	97.8	94.6	92.2
Y.Ng	24.3	23.8	25.6	24.9	25.1	25.0	24.6	104.9	102.4	110.2	107.4	108.2	107.8	105.9	104.9	102.4	110.2	107.4	108.2	107.8	105.9
Mean	23.3	23.1	23.6	23.5	23.6	23.4	23.3	97.7	96.6	99.1	98.4	98.8	97.9	97.8	97.7	96.6	99.1	98.4	98.8	97.9	97.8
SEM	0.9	0.8	0.8	0.8	0.9	0.8	0.9	3.7	3.1	3.2	3.1	3.6	3.2	3.3	3.1	3.1	3.2	3.1	3.6	3.2	3.3

Table 34 Waist and hip circumferences in group A on the cereals and nata de coco supplement

Subject	Waist circumference (cm)										hip circumference (cm)				
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	
A.So	91.0	92.0	88.0	87.0	89.0	-	-	102.0	102.0	100.0	102.0	100.0	-	-	
B.Ja	110.0	102.0	101.0	104.0	104.2	103.0	98.0	108.0	109.0	108.0	108.0	107.8	107.0	104.0	
B.Ju	94.0	94.0	94.0	92.0	92.4	93.0	92.7	96.0	96.5	94.5	94.0	94.7	95.0	94.5	
B.Ke	82.0	82.0	81.5	81.0	81.0	81.5	81.0	104.0	103.0	102.0	102.6	101.5	100.5	101.0	
J.Bu	90.0	91.5	90.5	90.0	91.0	91.5	92.7	97.0	100.0	100.0	99.0	100.0	100.0	100.0	
P.Ai	111.0	110.0	109.5	109.0	109.5	109.5	109.0	112.0	112.0	111.0	111.0	111.0	110.5	112.0	
P.Ke	79.0	78.0	78.0	78.0	77.5	77.0	76.0	97.0	96.0	95.5	94.5	93.0	93.0	93.9	
P.Ra	94.0	100.0	99.5	99.0	98.5	99.0	98.5	99.0	102.0	102.0	102.0	102.0	102.6	102.0	
P.Sm	84.5	84.0	84.0	84.0	84.0	84.0	84.0	92.0	92.0	91.0	91.0	91.0	90.6	90.5	
P.So	99.0	99.0	98.5	98.0	98.0	98.2	98.8	103.0	102.0	102.0	100.0	100.5	101.0	101.5	
P.Wa	82.5	78.5	78.0	78.1	77.8	78.0	76.4	96.8	95.0	93.1	95.5	94.7	94.5	94.5	
S.Bu	76.0	75.5	73.5	73.5	73.0	73.3	73.6	95.0	94.0	93.0	92.5	91.0	91.2	91.8	
S.Po	102.0	93.0	94.0	93.5	94.0	-	-	108.0	106.0	107.5	107.0	107.5	-	-	
S.Si	82.0	82.5	83.0	84.1	84.0	84.0	84.5	90.0	90.0	90.0	87.8	89.3	90.0	90.7	
Y.Ng	102.5	102.0	100.0	99.5	99.0	99.0	99.2	113.5	113.0	113.0	113.0	114.0	114.0	114.0	
Mean	91.3	90.7	90.1	90.0 ^{b4}	90.0	90.1	89.9	100.2	100.3	99.6	99.3	99.3	99.2	99.1	
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	
SEM	3.2	3.1	3.1	3.1	3.2	3.1	3.0	2.0	2.1	2.1	2.2	2.2	2.2	2.2	

significant difference from wk0: ^{b4} p < 0.05

Table 35 Waist and hip circumferences in group B on the cereals and nata de coco supplement

Subject	Waist circumference (cm)										hip circumference (cm)											
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	
A.Mo	85.4	85.0	84.0	86.0	86.0	86.0	87.2	98.0	98.0	96.5	95.0	95.5	96.0	97.0	98.0	98.0	96.5	95.0	95.5	96.0	96.0	97.0
I.So	84.0	84.0	84.5	84.0	86.0	85.0	85.5	92.0	92.5	92.0	92.5	91.0	91.0	91.5	92.5	92.5	92.0	92.5	91.0	91.0	91.0	91.5
K.Ch	99.0	97.5	95.0	95.0	95.0	95.0	96.0	104.0	104.5	104.0	104.0	104.5	104.0	105.0	104.5	104.5	104.0	104.0	104.5	104.0	104.0	105.0
P.Ch	91.0	90.5	90.0	90.0	90.0	89.5	90.8	96.0	96.0	95.5	96.0	96.0	96.5	96.0	96.0	96.0	95.5	96.0	96.0	96.5	96.5	96.0
P.Sr	86.0	87.0	85.0	86.0	86.5	-	-	96.0	96.0	95.5	95.5	95.5	-	-	95.5	96.0	95.5	95.5	95.5	-	-	-
T.Li	90.0	91.5	92.5	92.0	92.5	91.0	92.7	96.0	97.0	97.6	96.8	96.8	97.0	97.5	96.8	96.8	97.6	96.8	96.8	97.0	97.0	97.5
T.Ni	98.7	97.5	97.0	97.0	98.0	96.0	97.5	107.0	106.0	105.6	105.0	105.6	106.0	105.9	105.0	106.0	105.6	105.0	105.6	106.0	106.0	105.9
Mean	91.4	91.0	90.2	90.7	91.2	90.4	91.6	98.8	99.0	98.5	98.2	98.2	98.2	99.0	98.2	98.2	98.5	98.2	98.2	98.4	98.4	99.0
\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm
SEM	2.6	2.4	2.4	2.1	2.0	1.8	1.9	2.3	2.1	2.1	2.1	2.1	2.3	2.1	2.1	2.1	2.1	2.1	2.3	2.3	2.3	2.4

Table 37 Serum TC and TG in group A on the cereals and nata de coco supplement

Subject	TC (mmol/L)										TG (mmol/L)											
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	
A.So	6.5	7.2	7.2	6.6	5.8	-	-	2.3	2.3	2.1	1.3	1.2	-	-	2.3	2.3	2.1	1.3	1.2	-	-	
B.Ja	5.6	6.1	5.0	5.7	6.1	4.9	6.0	2.7	2.7	1.9	2.6	1.7	1.3	1.3	2.7	2.7	1.9	2.6	1.7	1.3	1.3	
B.Ju	5.8	6.0	5.4	5.8	5.7	5.5	5.4	2.9	2.9	3.0	2.4	3.3	3.6	3.2	3.5	3.5	3.0	2.4	3.3	3.6	3.2	
B.Ke	6.7	6.4	7.1	7.2	7.0	7.3	7.4	3.2	3.2	1.9	1.7	2.5	2.1	2.1	2.0	2.0	1.9	1.7	2.5	2.1	2.1	
J.Bu	7.1	6.5	5.1	5.6	6.2	5.4	5.2	2.4	2.4	0.9	1.5	1.6	1.3	1.0	1.8	1.8	0.9	1.5	1.6	1.3	1.0	
P.Ai	7.3	7.6	8.0	5.8	7.3	6.8	7.3	3.1	3.1	2.0	3.1	3.2	3.0	3.3	3.4	3.4	2.0	3.1	3.2	3.0	3.3	
P.Ke	6.2	6.3	5.7	6.1	5.5	5.1	5.6	3.1	3.1	1.4	2.1	1.8	0.7	0.7	2.4	2.4	1.4	2.1	1.8	0.7	0.7	
P.Ra	7.3	7.7	7.2	7.3	6.7	6.7	6.6	3.5	3.5	2.2	1.7	2.4	2.2	2.2	2.5	2.5	2.2	1.7	2.4	2.2	2.2	
P.Sm	10.1	6.2	4.7	4.7	4.7	4.7	5.3	2.2	2.2	1.4	1.5	1.9	1.0	1.2	2.8	2.8	1.4	1.5	1.9	1.0	1.2	
P.So	7.4	7.2	6.9	8.1	6.6	7.1	6.6	1.8	1.8	2.0	1.5	1.6	1.4	1.4	2.2	2.2	2.0	1.5	1.6	1.4	1.4	
P.Wa	8.7	6.9	8.4	7.7	8.4	7.3	8.8	2.3	2.3	1.9	2.0	2.1	2.9	2.9	2.5	2.5	1.9	2.0	2.1	2.9	2.9	
S.Bu	6.1	6.4	6.3	6.5	6.8	6.7	7.1	2.8	2.8	2.8	2.6	2.6	5.0	5.0	2.9	2.9	2.8	2.6	2.6	5.0	5.0	
S.Po	8.6	6.7	5.4	6.3	7.4	-	-	2.0	2.0	1.5	1.4	1.3	-	-	2.1	2.1	1.5	1.4	1.3	-	-	
S.Si	9.4	8.7	8.1	7.8	7.5	7.5	7.3	2.3	2.3	2.7	1.4	2.3	1.3	1.3	2.4	2.4	2.7	1.4	2.3	1.3	1.3	
Y.Ng	7.4	6.4	6.2	6.3	6.6	6.3	7.2	2.6	2.6	2.3	1.9	1.9	1.8	1.8	2.5	2.5	2.3	1.9	1.9	1.8	1.8	
Mean	7.3	6.8	6.5	6.5	6.5	6.2 ^{b4}	6.6	2.7	2.6	2.0 ^{b4}	2.0 ^{b4}	2.2	2.1 ^{b4}	2.1	2.6	2.6	2.0 ^{b4}	2.0 ^{b4}	2.2	2.1 ^{b4}	2.1	
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	0.4	0.2	0.3	0.3	0.3	0.3	0.3	0.1	0.1	0.2	0.1	0.2	0.3	0.3	0.1	0.1	0.2	0.1	0.2	0.3	0.3	

significant difference from wk0: ^{b4} p < 0.05

Table 38 Serum TC and TG in group B on the cereals and nata de coco supplement

Subject	TC (mmol/L)										TG (mmol/L)											
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	
A.Mo	5.9	5.9	6.7	6.9	5.9	6.3	5.5	2.5	2.3	4.4	2.3	3.8	3.3	2.0								
I.So	6.0	7.1	7.3	6.7	7.4	6.6	6.7	2.5	2.1	2.4	2.2	1.6	2.2	2.2								
K.Ch	7.0	6.9	6.8	6.3	6.3	5.3	6.0	2.7	2.5	3.4	1.9	3.4	1.9	1.7								
P.Ch	5.6	5.8	5.5	3.9	6.0	5.5	5.6	2.4	2.1	1.8	1.2	2.8	2.1	2.3								
P.Sr	6.7	6.1	6.4	5.3	5.7	-	-	2.9	2.2	2.8	3.3	2.5	-	-								
T.Li	6.3	5.7	5.4	5.8	6.7	3.4	4.9	2.8	2.1	1.4	1.6	2.0	1.2	2.4								
T.Ni	7.6	6.6	5.9	6.8	7.5	6.0	6.6	2.5	2.7	2.7	2.2	2.5	2.9	2.8								
Mean	±	6.3	±	6.1	±	5.5	±	2.6	±	2.3	±	2.7 ^{E4}	±	2.2								
SEM	0.3	0.2	0.3	0.5	0.3	0.5	0.3	0.1	0.1	0.4	0.2	0.3	0.3	0.2								

significant difference from group A at wk4: ^{C4} p < 0.05; at wk12: ^{E4} p < 0.05

Table 41 Serum TC/HDL-C and LDL-C/HDL-C ratios in group A on the cereals and nata de coco supplement

Subject	TC/HDL-C ratio										LDL-C/HDL-C ratio											
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	
A.So	5.62	4.21	4.26	3.85	4.26	-	-	3.71	2.59	2.69	2.48	2.87	-	-	-	-	-	2.48	2.87	-	-	
B.Ja	5.24	5.11	5.33	5.24	4.84	4.15	4.64	3.05	3.09	3.42	3.17	3.20	2.63	3.12	2.63	3.42	3.17	3.20	2.63	3.12	3.12	
B.Ju	4.81	4.36	4.38	4.33	4.68	4.63	4.45	2.70	2.21	2.29	2.54	2.43	2.26	2.23	2.26	2.29	2.54	2.43	2.26	2.23	2.23	
B.Ke	5.91	5.23	5.83	5.94	6.75	5.32	6.38	3.64	3.40	4.11	4.32	4.65	3.62	4.67	3.62	4.11	4.32	4.65	3.62	4.67	4.67	
J.Bu	3.83	3.36	3.03	3.25	3.95	4.00	3.94	2.25	1.96	1.78	1.85	2.49	2.56	2.57	2.56	1.78	1.85	2.49	2.56	2.57	2.57	
P.Ai	6.02	6.21	5.81	4.61	6.39	6.44	6.02	3.85	3.91	4.13	3.80	4.09	4.17	3.77	4.17	4.13	3.80	4.09	4.17	3.77	3.77	
P.Ke	5.36	5.28	4.33	4.43	4.51	3.54	3.54	3.13	3.35	2.84	2.74	2.85	2.30	2.28	2.30	2.84	2.74	2.85	2.30	2.28	2.28	
P.Ra	7.81	7.59	6.80	6.13	6.81	5.76	6.56	5.08	5.46	4.83	4.48	4.63	3.91	4.79	3.91	4.83	4.48	4.63	3.91	4.79	4.79	
P.Sm	10.32	5.17	5.03	3.27	3.62	3.12	4.02	8.32	3.07	2.81	1.78	1.94	1.81	2.59	1.81	2.81	1.78	1.94	1.81	2.59	2.59	
P.So	3.85	4.38	3.72	3.62	3.26	3.29	3.71	2.42	2.78	2.24	2.31	1.90	1.99	2.14	1.99	2.24	2.31	1.90	1.99	2.14	2.14	
P.Wa	6.72	5.70	6.35	6.34	6.23	6.17	6.31	4.88	3.77	4.71	4.57	4.56	4.07	4.28	4.07	4.71	4.57	4.56	4.07	4.28	4.28	
S.Bu	5.49	4.98	4.58	4.74	5.23	5.91	5.73	3.33	2.96	2.64	2.89	3.54	3.41	3.58	3.41	2.64	2.89	3.54	3.41	3.58	3.58	
S.Po	4.83	4.37	3.17	3.94	4.14	-	-	3.33	2.73	1.77	2.55	2.80	-	-	-	1.77	2.55	2.80	-	-	-	
S.Si	7.41	7.98	6.93	6.82	6.59	5.29	6.34	5.57	5.98	4.87	5.25	4.66	3.87	4.52	4.66	4.87	5.25	4.66	3.87	4.52	4.52	
Y.Ng	4.22	4.45	3.73	3.87	3.84	3.92	4.63	2.56	2.64	2.11	2.35	2.34	2.42	2.95	2.64	2.11	2.35	2.34	2.42	2.95	2.95	
Mean	5.9	5.4	5.1	4.8	5.1	4.7	5.1	3.9	3.4	3.3	3.2	3.3	3.0	3.3	3.4	3.3	3.2	3.3	3.0	3.3	3.3	
±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	0.5	0.4	0.3	0.3	0.4	0.3	0.3	0.5	0.3	0.3	0.3	0.3	0.2	0.3	0.3	0.3	0.3	0.3	0.2	0.3	0.3	

Table 42 Serum TC/HDL-C and LDL-C/HDL-C ratios in group B on the cereals and nata de coco supplement

Subject	TC/HDL-C ratio										LDL-C/HDL-C ratio																	
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20							
A.Mo	6.97	5.11	7.19	6.21	6.74	7.59	5.92	5.27	3.22	4.03	4.28	3.74	4.78	3.92	6.97	5.11	7.19	6.21	6.74	7.59	5.92	5.27	3.22	4.03	4.28	3.74	4.78	3.92
I.So	5.68	5.96	6.11	5.53	5.33	5.54	5.73	3.61	4.17	4.20	3.70	3.81	3.72	3.87	5.68	5.96	6.11	5.53	5.33	5.54	5.73	3.61	4.17	4.20	3.70	3.81	3.72	3.87
K.Ch	6.43	6.63	7.08	5.76	7.36	5.57	5.50	4.29	4.53	4.43	4.00	4.55	3.62	3.74	6.43	6.63	7.08	5.76	7.36	5.57	5.50	4.29	4.53	4.43	4.00	4.55	3.62	3.74
P.Ch	5.07	5.07	4.71	4.19	5.92	4.95	5.12	3.02	3.23	2.98	2.64	3.67	3.09	3.14	5.07	5.07	4.71	4.19	5.92	4.95	5.12	3.02	3.23	2.98	2.64	3.67	3.09	3.14
P.Sr	5.20	3.34	5.47	3.53	4.31	-	-	3.18	1.79	3.36	1.55	2.43	-	-	5.20	3.34	5.47	3.53	4.31	-	-	3.18	1.79	3.36	1.55	2.43	-	-
T.Li	6.59	5.55	4.88	5.14	5.79	3.20	4.24	4.24	3.60	3.30	3.48	4.74	1.68	2.31	6.59	5.55	4.88	5.14	5.79	3.20	4.24	4.24	3.60	3.30	3.48	4.74	1.68	2.31
T.Ni	7.51	6.35	5.97	7.54	7.41	6.82	6.56	5.36	4.15	3.74	5.46	5.26	4.32	4.28	7.51	6.35	5.97	7.54	7.41	6.82	6.56	5.36	4.15	3.74	5.46	5.26	4.32	4.28
Mean	6.4	5.8	6.0	5.7	6.4	5.6	5.5	4.3	3.8	3.8	3.9	4.3	3.5	3.5	6.4	5.8	6.0	5.7	6.4	5.6	5.5	4.3	3.8	3.8	3.9	4.3	3.5	3.5
SEM	0.4	0.3	0.4	0.4	0.4	0.6	0.3	0.4	0.2	0.2	0.4	0.3	0.4	0.3	0.4	0.3	0.4	0.4	0.4	0.6	0.3	0.4	0.2	0.2	0.4	0.3	0.4	0.3

Table 43 Fasting blood glucose in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

Subject	FBG (mmol/L)						
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
Group A							
A.So	5.0	5.1	5.2	4.9	4.7	-	-
B.Ja	5.5	4.5	4.7	4.5	4.3	4.8	5.0
B.Ju	5.7	5.2	5.0	4.7	4.6	5.1	4.9
B.Ke	4.4	5.0	4.6	4.3	4.4	4.4	5.0
J.Bu	4.8	4.9	4.9	4.4	4.8	4.6	4.5
P.Ai	4.4	5.5	5.3	4.4	4.6	4.7	5.1
P.Ke	6.8	7.0	7.7	7.0	7.3	6.5	6.2
P.Ra	4.9	5.1	4.8	4.6	4.9	4.6	4.9
P.Sm	4.3	4.3	4.8	4.6	4.4	4.7	4.8
P.So	4.2	4.3	3.9	4.4	4.4	4.8	4.2
P.Wa	5.9	5.9	5.8	5.1	5.0	5.7	5.6
S.Bu	4.7	4.9	4.6	5.5	4.1	4.3	4.3
S.Po	5.0	4.8	4.4	4.8	5.3	-	-
S.Si	5.6	5.0	4.9	5.0	4.6	5.3	4.9
Y.Ng	4.8	5.0	4.9	4.9	5.2	4.8	5.3
Mean	5.1	5.1	5.1	4.9	4.8	4.9	5.0
±	±	±	±	±	±	±	±
SEM	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Group B							
A.Mo	6.4	5.6	6.1	4.6	5.0	5.5	5.3
I.So	4.2	4.2	4.1	3.9	3.3	4.8	4.6
K.Ch	6.2	6.1	6.5	6.5	6.0	6.7	7.1
P.Ch	6.2	5.8	5.6	4.9	5.2	5.9	5.2
P.Sr	5.3	5.4	5.3	5.3	5.2	-	-
T.Li	5.4	5.0	4.5	4.4	4.6	4.7	4.3
T.Ni	5.5	4.9	4.8	4.5	4.5	4.7	4.7
Mean	5.6	5.3	5.3	4.8	4.8	5.4	5.2
±	±	±	±	±	±	±	±
SEM	0.3	0.3	0.4	0.4	0.4	0.3	0.4

Table 44 Serum mineral levels and carbon dioxide content in group A on the cereals and nata de coco supplement

Subject	Sodium (mmol/L)		Potassium (mmol/L)		Chloride (mmol/L)		CO ₂ content (mmol/L)		Calcium (mmol/L)		Phosphorus (mmol/L)	
	wk	wk	wk	wk	wk	wk	wk	wk	wk	wk	wk	wk
0	12	20	0	12	20	0	12	20	0	12	20	0
A.So	143	146	4.6	5.0	100	108	24.0	23.3	-	2.4	2.4	-
B.Ja	146	144	4.1	4.0	105	107	21.2	20.9	21.4	2.3	2.4	2.4
B.Ju	145	146	3.4	3.9	102	104	22.5	21.9	22.7	2.3	2.4	2.4
B.Ke	141	141	4.3	4.5	104	103	26.9	23.2	27.0	2.4	2.4	1.6
J.Bu	143	146	3.8	3.6	106	108	22.4	22.1	23.4	2.4	2.4	1.0
P.Ai	144	139	4.6	4.7	100	102	22.0	23.0	23.4	2.3	2.5	1.4
P.Ke	138	140	4.3	4.7	101	104	24.4	23.5	22.4	2.2	2.4	1.0
P.Ra	144	145	4.5	5.3	103	107	27.9	24.2	26.4	2.4	2.4	1.1
P.Sm	144	144	4.8	4.6	99	107	27.2	25.6	29.0	2.4	2.4	1.1
P.So	144	144	4.4	4.4	106	107	25.8	20.7	22.0	2.6	2.4	1.8
P.Wa	146	146	5.0	4.6	106	102	23.9	22.4	23.8	2.4	2.6	1.1
S.Bu	140	141	4.6	4.6	102	102	27.7	25.2	24.8	2.4	2.5	0.9
S.Po	143	142	3.9	4.1	104	103	25.6	26.6	-	2.4	2.4	1.1
S.Si	146	145	4.6	4.5	99	102	24.5	23.5	22.4	2.3	2.3	1.0
Y.Ng	144	143	3.8	4.3	100	105	32.6	26.5	27.6	2.4	2.6	1.3
Mean	143	143	4.3	4.4	102	105	25.0	23.0	24.0	3.1	2.4	1.2
±	±	±	±	±	±	±	±	±	±	±	±	±
SEM	0.7	0.7	0.1	0.1	0.7	0.6	0.9	0.5	0.7	0.8	0.1	0.1

Table 46 Serum bilirubin and enzyme levels in group A on the cereals and nata de coco supplement

Subject	Total bilirubin (μ mol/L)			Direct bilirubin (μ mol/L)			AST (μ /L)			ALT (μ /L)			Alkaline phosphatase (μ /L)			GGT (μ /L)		
	wk 0	wk 12	wk 20	wk 0	wk 12	wk 20	wk 0	wk 12	wk 20	wk 0	wk 12	wk 20	wk 0	wk 12	wk 20	wk 0	wk 12	wk 20
A.So	12.6	7.5	-	3.8	2.6	-	31.6	29.0	-	60.0	53.0	-	72.0	66.0	-	45.0	42.0	-
B.Ja	11.6	11.4	8.7	3.5	3.5	3.2	22.6	21.0	19.0	43.0	38.0	31.0	92.0	89.0	85.0	36.8	29.0	18.0
B.Ju	21.0	20.6	15.7	4.5	4.6	3.8	22.6	19.0	19.0	60.2	56.0	43.0	70.0	67.0	82.0	65.0	68.0	45.0
B.Ke	11.2	5.3	7.5	5.0	2.6	3.1	16.0	21.0	18.0	48.0	38.0	60.0	66.0	64.0	60.0	27.0	35.0	35.0
J.Bu	10.5	10.7	11.6	3.4	3.7	3.8	28.0	27.0	19.0	46.0	52.0	38.0	100.0	99.0	99.0	140.0	149.0	69.0
P.Ai	11.1	9.3	15.6	3.2	3.0	4.9	32.0	30.0	38.0	60.0	58.0	64.0	89.0	99.0	90.0	78.0	76.0	73.0
P.Ke	10.0	9.8	12.5	4.0	3.4	3.0	15.0	12.0	16.0	39.0	24.0	33.0	96.0	108.0	106.0	28.0	37.0	37.0
P.Ra	7.8	6.3	6.2	3.1	2.6	2.1	16.0	19.0	18.0	33.0	27.0	30.0	91.0	82.0	93.0	25.0	56.0	50.0
P.Sm	13.5	12.2	16.7	6.0	6.4	6.2	26.0	22.0	29.0	48.0	44.0	57.0	110.0	116.0	95.0	111.0	101.0	144.0
P.So	9.6	8.8	8.7	3.3	3.4	3.3	16.0	20.0	23.0	34.0	28.0	29.0	65.0	82.0	73.0	21.0	27.0	24.0
P.Wa	7.5	12.6	8.8	2.6	3.8	2.9	22.0	19.0	22.0	30.0	26.0	28.0	86.0	80.0	63.0	60.0	58.0	64.0
S.Bu	10.4	9.0	7.9	4.8	2.3	3.4	17.0	19.0	19.0	50.0	32.0	32.0	77.0	89.0	60.0	22.0	45.0	24.0
S.Po	13.4	6.9	-	3.7	2.4	-	28.0	20.0	-	74.0	44.0	-	78.0	88.0	-	75.0	25.0	-
S.Si	4.4	4.6	7.7	2.6	2.9	3.3	18.6	17.0	19.0	43.0	35.0	31.0	62.0	59.0	67.0	30.0	28.0	21.0
Y.Ng	15.0	9.0	10.2	3.2	3.2	3.7	24.0	28.0	27.0	30.0	34.0	31.0	68.0	67.0	79.0	20.0	36.0	37.0
Mean	11.0	10.0	10.6	3.8	3.5	3.6	21.2	21.1	22.0	43.4	37.8	39.0	82.5	84.7	80.9	51.1	57.3	49.3
\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm
SEM	1.1	1.1	1.0	0.3	0.3	0.3	1.5	1.3	1.7	2.8	3.2	3.6	4.3	4.9	4.3	10.6	9.8	9.4

Table 47 Serum bilirubin and enzyme levels in group B on the cereals and nata de coco supplement

Subject	Total bilirubin (μ mol/L)		Direct bilirubin (μ mol/L)		AST (μ /L)		ALT (μ /L)		Alkaline phosphatase (μ /L)		GGT (μ /L)							
	wk 0	wk 12	wk 0	wk 12	wk 0	wk 12	wk 0	wk 12	wk 0	wk 12	wk 0	wk 12						
A.Mo	10.0	9.9	12.0	3.6	3.4	3.7	18.9	18.0	15.0	36.4	34.0	26.0	80.0	76.0	78.0	35.0	31.0	20.0
I.So	12.2	13.5	9.1	4.4	4.3	2.9	25.0	22.0	29.0	42.0	39.0	54.0	80.0	77.0	69.0	79.0	82.0	100.0
K.Ch	11.0	11.0	6.8	3.6	3.8	3.6	23.9	25.0	29.0	62.0	63.0	63.0	79.0	74.0	63.0	43.0	45.0	44.0
P.Ch	14.8	14.5	15.5	4.2	4.0	3.8	16.0	14.0	18.0	45.0	44.0	40.0	83.0	79.0	68.0	38.8	40.0	31.0
P.Sr	9.1	10.0	-	4.5	5.2	-	29.0	34.0	-	38.0	42.0	-	102.0	125.0	-	47.0	48.0	-
T.Li	13.2	14.6	13.4	3.6	4.2	4.0	47.0	33.0	42.0	80.0	76.0	84.0	61.0	49.0	59.0	55.0	61.0	44.0
T.Ni	11.1	11.1	10.2	3.6	3.4	3.2	22.0	20.0	19.0	45.0	49.0	42.0	60.0	64.0	80.0	48.0	50.0	39.0
Mean \pm SEM	12.0 \pm 0.7	12.4 \pm 0.8	11.8 \pm 1.3	3.8 \pm 0.2	3.8 \pm 0.2	3.5 \pm 0.2	25.5 \pm 4.5	22.0 \pm 2.7	25.3 \pm 4.1	51.7 \pm 6.6	50.8 \pm 6.5	51.5 \pm 8.3	73.8 \pm 4.2	69.8 \pm 4.7	69.5 \pm 3.3	49.8 \pm 6.5	51.5 \pm 7.3	46.3 \pm 11.4

Table 49 Serum total protein, albumin, urea nitrogen, creatinine and uric acid in group B on the cereals and nata de coco supplement

Subject	Total protein (g/L)			Albumin (g/L)			Urea nitrogen (mmol/L)			creatinine (μmol/L)			uric acid (μmol/L)		
	wk	12	20	wk	12	20	wk	12	20	wk	12	20	wk	12	20
	0			0			0			0			0		
A.Mo	81.2	80.1	78.7	43.0	42.8	44.0	6.0	6.3	5.8	115.0	132.0	116.0	428.0	436.0	444.0
I.So	80.2	77.3	76.2	43.6	46.1	45.4	5.7	5.2	3.5	90.00	95.0	79.0	395.0	388.0	417.0
K.Ch	80.8	81.8	81.4	43.6	43.4	41.2	5.0	3.5	3.7	102.0	100.0	87.0	373.0	375.0	381.0
P.Ch	89.0	85.0	79.9	49.6	47.1	44.7	5.0	5.1	3.7	110.0	115.0	101.0	478.0	486.0	446.0
P.Sr	85.8	85.1	-	47.9	48.3	-	4.6	3.5	-	78.0	91.0	-	478.0	463.0	-
T.Li	83.2	81.8	82.3	47.1	45.3	46.9	7.2	7.8	7.0	102.0	115.0	106.0	410.0	430.0	409.0
T.Ni	94.0	91.0	86.2	46.1	49.8	46.9	5.2	5.7	5.3	104.0	114.0	87.0	240.0	241.0	235.0
Mean	84.7	82.8	80.8	45.5	45.8	44.8	5.7	5.6	4.8	103.8	111.8	96.0	387.3	392.7	388.7
+															
SEM	2.3	1.9	1.4	1.0	1.0	0.9	0.3	0.6	0.6	3.5	5.3	5.7	32.8	34.3	32.3

Table 51 Serum vitamin A, vitamin E and β -carotene levels in group B on the cereals and nata de coco supplement

Subject	Vitamin A ($\mu\text{g/dL}$)						β -carotene ($\mu\text{g/dL}$)						Vitamin E (mg/dL)								
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
A.Mo	130.4	103.1	109.3	107.6	101.9	76.0	93.7	21.4	27.4	16.2	32.6	19.2	22.9	55.9	1.22	1.09	1.51	1.25	1.34	1.43	1.21
I.So	113.5	115.3	110.6	119.4	123.7	124.1	135.0	18.0	27.1	22.7	17.1	19.5	53.6	32.8	1.15	1.09	1.20	1.09	1.00	1.24	1.76
K.Ch	121.4	110.5	95.6	102.6	90.0	119.6	113.5	26.8	9.6	48.7	5.6	15.1	8.9	18.0	0.95	0.92	1.24	1.17	0.96	1.16	1.15
P.Ch	120.7	135.6	131.0	90.6	156.5	140.4	135.1	51.1	26.0	13.1	22.2	18.2	20.6	44.0	0.70	1.03	0.90	1.17	1.07	1.15	1.14
P.Sr	109.5	79.8	110.2	177.9	144.7	-	-	19.3	0.0	0.0	0.0	0.0	-	-	0.67	3.38	0.93	1.42	1.20	-	-
T.Li	100.0	144.0	144.5	135.8	167.2	116.8	144.1	21.8	14.1	29.2	34.3	9.2	74.9	106.4	1.18	1.34	1.32	1.27	1.50	1.02	1.28
T.Ni	105.1	121.1	103.7	146.6	147.8	95.3	103.5	70.7	10.0	13.4	14.3	21.8	25.7	18.9	1.28	1.04	1.00	1.06	1.14	0.85	1.19
Mean	115.2	121.6	115.8	117.1	131.2	112.0	120.8	35.0	19.0	23.9	21.0	17.2 ^{E4}	34.4	46.0	1.1	1.1	1.2	1.2	1.2	1.1	1.3
\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm	\pm
SEM	4.6	6.3	7.5	8.6	12.7	9.3	8.2	8.7	3.6	5.6	4.5	1.8	10.1	13.5	0.1	0.1	0.1	0.1	0.1	0.1	0.1

significant difference from group A at wk12: ^{E4} p < 0.05

Table 52 Serum malondialdehyde (MDA) levels in 22 subjects with hyperlipidemia on the cereals and nata de coco supplement

Subject	Serum malondialdehyde (MDA) levels						
	wk -4	wk 0	wk 4	wk 8	wk 12	wk 16	wk 20
Group A							
A.So	13.15	13.46	11.05	10.47	10.00	-	-
B.Ja	16.20	16.25	13.95	12.93	11.92	10.80	9.96
B.Ju	16.74	16.20	15.33	13.95	13.04	12.10	10.94
B.Ke	16.56	17.54	14.24	13.08	12.21	11.89	10.29
J.Bu	17.72	16.85	15.83	15.07	14.28	13.44	12.43
P.Ai	21.63	21.76	19.75	19.24	18.77	17.54	16.88
P.Ke	16.74	16.41	15.72	14.49	13.15	12.32	11.23
P.Ra	19.38	19.51	17.90	16.78	15.40	14.82	13.91
P.Sm	17.64	16.99	16.41	15.65	14.82	13.70	13.08
P.So	20.18	21.13	18.22	17.46	16.30	15.65	15.00
P.Wa	15.65	15.14	13.91	12.64	11.63	11.12	10.47
S.Bu	16.63	16.01	15.43	14.75	14.06	13.22	13.01
S.Po	17.46	16.71	14.89	14.13	13.70	-	-
S.Si	13.33	14.54	11.92	10.98	10.07	9.38	8.41
Y.Ng	20.40	19.82	18.59	18.15	17.68	17.07	15.94
Mean	17.6	16.8	15.9 ^{b1}	15.0 ^{b1}	14.1 ^{b1}	13.3 ^{b1}	12.4 ^{b1}
± SEM	± 0.6	± 0.6	± 0.6	± 0.6	± 0.7	± 0.7	± 0.7
Group B							
A.Mo	20.43	21.46	18.26	17.46	16.78	16.52	15.18
I.So	17.32	16.34	15.69	14.93	13.91	13.33	12.54
K.Ch	20.40	20.60	18.77	17.36	16.49	15.97	15.07
P.Ch	16.74	17.01	15.00	13.51	13.01	12.88	11.38
P.Sr	21.34	20.47	19.60	18.80	17.54	-	-
T.Li	16.05	16.51	14.42	13.15	12.36	11.74	10.87
T.Ni	13.30	14.43	11.45	10.29	9.53	9.42	8.08
Mean	17.9	17.1	16.2 ^{b1}	15.1 ^{b1}	14.2 ^{b1}	11.1 ^{b1}	10.4 ^{b1}
± SEM	± 1.1	± 1.1	± 1.1	± 1.1	± 1.1	± 2.1	± 2.0

significant difference from wk0: ^{b1} p < 0.001

CHAPTER V

DISCUSSION

This study was designed to evaluate the effect of the cereals and nata de coco supplementation on serum lipid, serum vitamin A, E and lipid peroxidation. In our study the cereals and nata de coco powder, composed of 40 % nata de coco, 6 % unpolished rice, 30 % sweet corn and 18 % mung bean was supplemented to usual diet in hyperlipidemic subjects.

After receiving the supplementation with the cereals and nata de coco supplement for 20 wks, the subjects were classified into 2 groups according to their compliance, **group A**; compliance ≥ 90 % of assigned cereals and nata de coco intake and **group B**; compliance < 90 % of assigned cereals and nata de coco intake.

The mean of energy distribution before and during the study of group A was 14 % protein -, 55 % carbohydrate -, and 31 % fat – calories whereas that of group B was 13 % protein -, 56 % carbohydrate -, and 31 % fat – calories. A mean value equivalent to 29.1 and 26.5 g/d of the cereals and nata de coco supplement was incorporated into the diet of 15 subjects within group A and 7 subjects within group B. In group A, their total protein intake at wk 16 and carbohydrate intakes at wk 12, 20 and total fat intake at wk 12 were significantly increased from wk 0. (**Table 14, Fig 10**) For group B, their total carbohydrate and total fat intakes at wk 12 were higher than wk 0. (**Fig 12**) The sugar intakes at wk 12 of both groups, which were significantly higher than wk 0, 4, and 8. (**Table 16, Fig 10, 12**) Cholesterol intakes of both group, which were nearly at 300 ± 27 mg/d, were reduced by 9.7 % in group A and 7.4 % in group B. In group A, the energy intakes were slightly higher on the supplementation at wk 12 but the increase was not significant. The other hand, group B, their energy intakes at wk 12 were significantly higher than wk 0. (**Table 15, Fig 10, 12**)

Overall the intakes of the macronutrients and energy intake from 2 groups were significantly higher at wk 12 than the values at baseline (wk 0). The reason was the changing of their eating habit of subject in this period. Because the supplementation at wk 12 was same period of the end of year and New Year celebrations. Also, Most of subjects tended to increase their energy intakes, but after this period all of them can decrease energy intakes down to their normal values. Thus, No significant change in body weight and body mass index (BMI) were seen between wk 0 and anytime during the 20 weeks on the supplementation. Also, they were held constant body weight and BMI (**Table 18, 19**) because there was only a slightly change in energy intake throughout the study

Both subject compliance of supplement intake and dietary intake have a major influence on serum lipid level, especially serum TG level.

Group A, significant differences were seen between the mean of TG level at wk 0 and the mean values at wk 4, 8, and 12 ($p < 0.05$) but no significant differences in TC, LDL-C and HDL-C in all subjects except in group A, TC level at wk 16 was significantly lower than that at wk 0 ($p < 0.05$). (**Fig 7**)

This finding confirms those of Mesomya W. et al (24), they studied in five kinds dietary fiber diet in experimental rats for 4 weeks. The result indicated that experimental rat fed 40 % nata de coco, 6 % unpolished rice, 30 % sweet corn and 18 % mung bean (same supplement in our study) was significant lower the serum TG level in rats than those fed the experimental diet from apple pectin and cellulose even though the percentage of total dietary fiber was lower than those two experimental diet (apple pectin and cellulose) but no serum cholesterol-lowering effect.

In according to Anderson JW. Et al (9), the serum TG levels significantly ($p < 0.05$) decreased by 10 % in hypercholesterolemic men consuming wheat bran 40 g/d. The results of our study showed the serum TG lowering effect of supplement food product from 40 % nata de coco, 6 % unpolished rice, 30 % sweet corn and 18 % mung bean which was highly insoluble fiber, so our results may indicate that the insoluble fiber in supplement food product from those composition significantly reduced serum TG level in hyperlipidemic subjects.

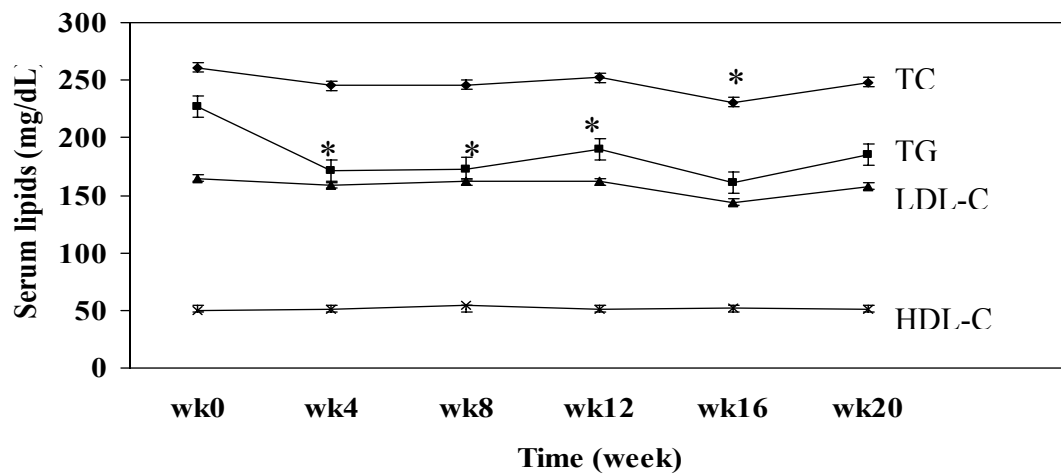


Fig 7 The means of serum cholesterol (TC), triglyceride (TG), LDL-C and HDL-C levels of group A (n = 15) over the 20 weeks the cereals and nata de coco supplemented period. Significance levels refer to differences from the mean of the control. (p < 0.05)

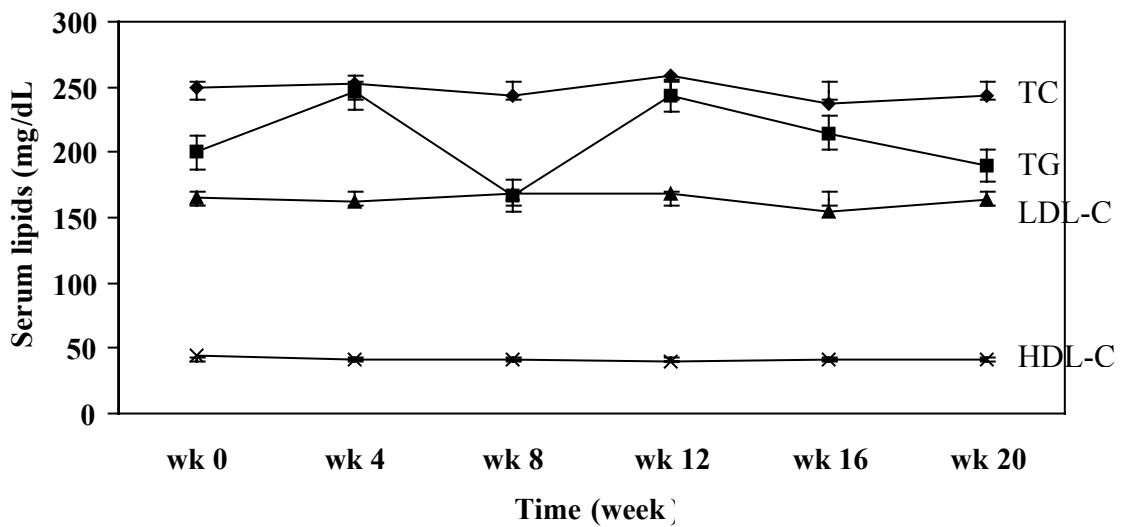


Fig 8 The means of serum cholesterol (TC), triglyceride (TG), LDL-C and HDL-C levels of group B (n = 7) over the 20 weeks the cereals and nata de coco supplemented period.

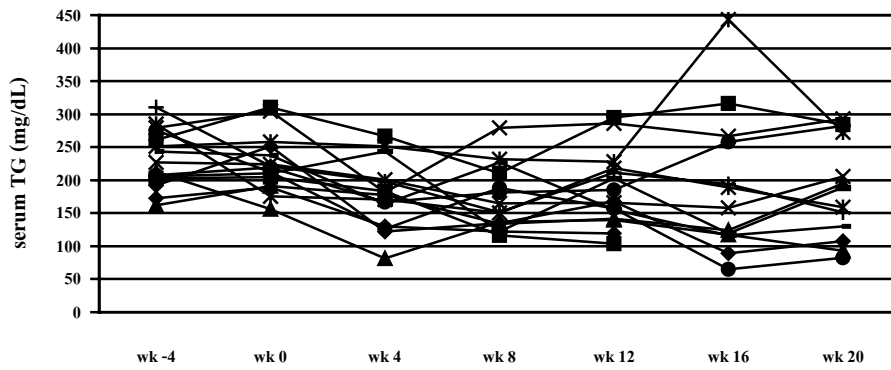
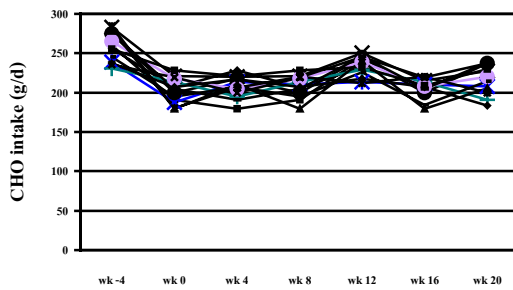
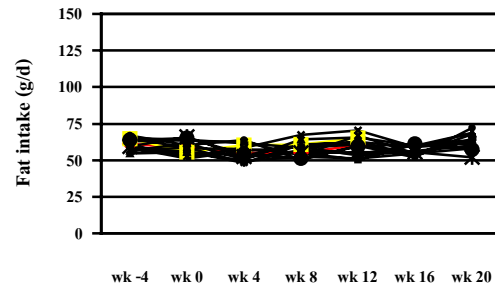


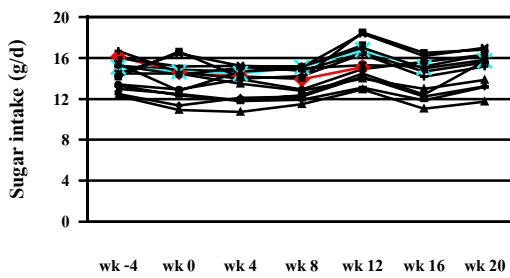
Fig 9 Individual values of serum triglyceride in group A (n = 15)



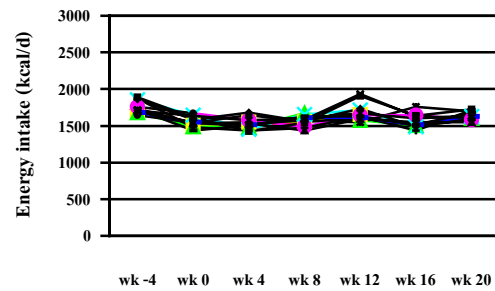
(A)



(B)



(C)



(D)

Fig 10 Individual values of carbohydrate (A), fat (B), sugar (C), and energy intake (D) in group A (n = 15)

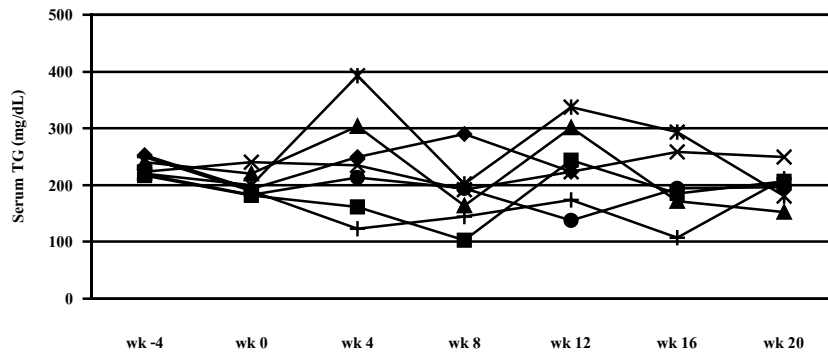


Fig 11 Individual values of serum TG in group B (n = 7)

(A)

(B)

(C)

(D)

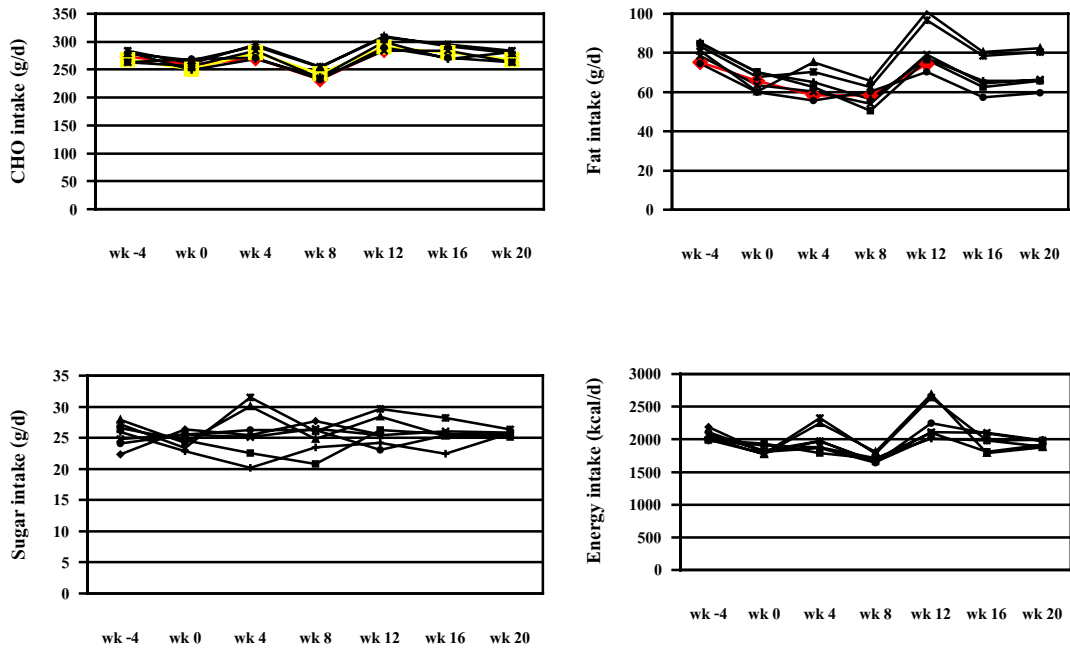


Fig 12 Individual values of carbohydrate (A), fat (B), sugar (C), and energy intake (D) in group B (n = 7)

Study of David JA Jenkins et al (7) The authors assessed the effect of leguminous seeds in the dietary management of 7 male with hyperlipidemia. All subjects were substituted approximately 140 grams of dried beans daily for other sources of starch in their diet over a 4-month period. After this the energy intake and body weight in these subjects were held constant. However, the results revealed that high-legume diet significantly reduced serum triglyceride and serum cholesterol levels. However, LDL-C and HDL-C levels remained unaltered.

Study of Janet R Mahalko et al (160), the effects of consuming fiber from corn bran, soy hulls, or apple powder on glucose tolerance and plasma lipids were investigated in 2 studies of patients with type II diabetes. Patients with diabetes were given control low fiber bread or bread containing one of three fiber sources for incorporation into their usual self-selected diets. The fiber sources used were soy hulls, corn bran, or dehydrated powdered apple, substituted for 25 % of the white wheat flour in bread. The 10 subjects in study A consumed an average of 26 grams of fiber source/d. incorporated into 7 slices of bread. The eight subjects in study B consumed an average of 52 grams of fiber source/d, half incorporated into 7 slices of bread, and half into other foods in their diets. Three subjects were in both studies. This study reported that soy hull consumption slightly improved some measures of glucose tolerance with results varying between the studies whereas consumption of 52 grams of corn bran decreased VLDL-C, TG and glycosylated Hb, but subject tolerance was poor. In addition, consumption of 52 grams of apple powder increased LDL-C and TC levels.

Rice bran is a unique cereal bran because of the amount and type of fiber and the amount of fat. Rice bran contains predominantly insoluble fiber but is much lower in fiber (181 g/kg) than either corn bran (890 g/kg) or wheat bran (453 g/kg), the other two cereals bran rich in insoluble fiber. Rice bran has been shown to have cholesterol-lowering activity in hypercholesterolemic animals and human subjects (165). Proposed mechanisms of cholesterol-lowering by rice bran include fecal excretion of fat, cholesterol, and bile acids. Soluble fiber and viscosity do not appear to play a significant role in the hypocholesterolemic activity of rice bran.

Study of Raghuram and coworkers (161) reported a significant reduction in serum TC in 15 and 30 days when 12 hypercholesterolemic subjects replaced their

customary cooking oil with rice bran oil. Kestin and coworkers (162) observed a significant increase in HDL-C / TC ratio after 4 weeks of feeding 11.8 grams of dietary fiber from rice bran or oat bran (total nonstarch-polysaccharides, 21 g/d) to mildly hypercholesterolemic free-living men. Hegsted and coworkers (163) reported that consumption of stabilized rice bran (100 g/d) for 2 and 3 weeks periods resulted in a 4-10 % reduction in TC in moderately hypercholesterolemic subjects fed diets containing 37 % of calories as fat. In normocholesterolemic men, feeding rice bran (15 or 30 g/d) for 3 weeks resulted in non-significant plasma cholesterol reductions (164).

The homeostasis of blood glucose before and during receiving the cereals and nata de coco supplementation was based on the determination of fasting blood glucose (FBG). The WHO criteria (166) are employed for the diagnosis of diabetes mellitus when FBG is above 6.7 mmol/L (120 mg/dL). In our study, at wk -4 to wk 12, diabetes mellitus was detected in only 1 subject (P.Ke) but after these periods her FBG level turned to the normal range. (**Table 43**) As opposed to the result of K.Ch because his FBG level was slightly increased at wk 20. (**Table 43**) However, there were no significant changes in FBG level in these 22 subjects with hyperlipidemia during receiving the cereals and nata de coco supplementation.

Evaluation of safety of ingesting the cereals and nata de coco supplement was based on biochemical parameters assessing serum mineral levels and renal and liver function of the subjects. The serum sodium, potassium, chloride, CO₂ content, calcium and phosphorus (**Table 44, 45**), total and direct bilirubin, AST, ALT, alkaline phosphatase and GGT (**Table 46, 47**), total protein, albumin, urea nitrogen, creatinine and uric acid levels (**Table 48, 49**) in 22 hyperlipidemic subjects before and during receiving the cereals and nata de coco supplementation were within the normal limits (167). It is evident that the consumption of the cereals and nata de coco supplement is safe. It does not cause any hazard to hematopoietic, renal and liver functions.

The serum vitamin A, β -carotene and vitamin E levels (**Table 50, 51**) in 22 hyperlipidemic subjects before and during receiving the cereals and nata de coco supplementation were within the normal range (128).

In our study, we found that the change of serum vitamin A and β -carotene levels are due to the change of their serum vitamin A, β -carotene intake before and during receiving the cereals and nata de coco supplementation (**Table 16**) evident by

the significantly positive correlation between vitamin A intakes in RE/d and serum vitamin A levels in $\mu\text{g/dL}$ (group A: $r = 0.915$, $p < 0.001$ and group B: $r = 0.940$, $p < 0.001$). This result was similar to the β -carotene intake in $\mu\text{g/d}$ and serum β -carotene concentration in $\mu\text{g/dL}$ (group A: $r = 0.955$, $p < 0.001$ and group B: $r = 0.980$, $p < 0.001$).

Several studies investigated about the effects of dietary fiber on vitamin absorption. Results of experimental animals and human studies are controversy. Baceuse vitamin absorption depended on many factors. Also we may divide the results into 2 ways.

First, Some studies indicated that vitamin absorption was decreased bioavailability by dietary fiber. Barnard and Heaton (113) found that in human subjects lignin give with a test meal containing 5,000 I.U. of vitamin A per kilogram body weight had no effect on serum rise in vitamin A after the meal. In addition, Kasper et al (114) reported that when wheat bran cellulose, pectin. Guar flour, carob bean flour, or carrageenan was given with a test meal containing 300,000 I.U. of vitamin A palmitate, the area under the curve for serum concentration of vitamin A for the 9 hour afterward was significantly decrease by all fiber source. These two studies were conducted because there had been reports of binding of bile acids by fiber, and it was postulated that fiber might also effect bioavailability of vitamin A though changes in intestinal absorption, whereas in another experiment on 4 subjects after the daily intake of 15 g apple pectin or 30 g wheat bran over a period of 50 days, a decrease in vitamin A concentration was found, while carotene concentrations remained unchanged (113). In experimental rats, vitamin E supplemented diet containing 10 % of pectin that was fed for 56 days resulted in lower body weight as well as decreased plasma and red blood cell vitamin E and increased hemolysis (117), whereas rats fed 6% or 8 % pectin for 8 weeks had lower body weight, lower liver vitamin E levels, and higher hemolysis rates than those fed 0 or 3 % pectin (120). From these studies on rats it appears that intakes of pectin at a level of 6% or more of the diet decrease vitamin E bioavailability, but fiber - containing breads or cereals as 20 % of the diet have only transient or no effect.

Secondly, some studies reported that dietary fiber do not effect on vitamin absorption or the effect is very small. Study of Rattan J et al, during an experiment on

68 patients receiving two tablespoons of bran per day over a period of at least 6 months, it was found that serum vitamin A concentration were higher compared to the initial figures (115), whereas Wahal and co-workers (116) tested the effect of wheat bran on serum vitamin A levels in healthy subjects during a 6-weeks trial. The addition of wheat bran to a standard diet with 20,000 unit of vitamin A significantly lower serum vitamin A level within 1 week, and this trend continued over 3 weeks. On rats, feeding 20% of the diet as bread or cereals for 30 days (three different breads and two cereals) did not effect plasma vitamin E level (118). In rat fed 5 % or 20 % wheat bran for 56 days, plasma vitamin E level declined at 5 weeks but reverted to pre-study levels at 8 weeks (119). In our study, 22 hyperlipidemic subjects after the daily intake of 30 grams of the cereals and nata de coco supplement over a period of 20 weeks, we found that serum vitamin A, β -carotene and serum vitamin E concentrations remained unchanged. It is evident that the consumption of the cereals and nata de coco supplement does not effect on the vitamin A, β -carotene and serum vitamin E absorption.

Evaluation of lipid peroxidation was based on serum malondialdehyde (MDA) concentration of 22 hyperlipidemic subjects.

Both group A and group B, significant differences were seen between the mean serum MDA concentration at wk 0 and the means serum MDA values at wk 4, 8, 12, 16 and 20. ($p < 0.001$) (**Table 52, Fig 8**)

This result may be related to supplementation because we did not found the significant decrease in their serum MDA concentration before supplementation.

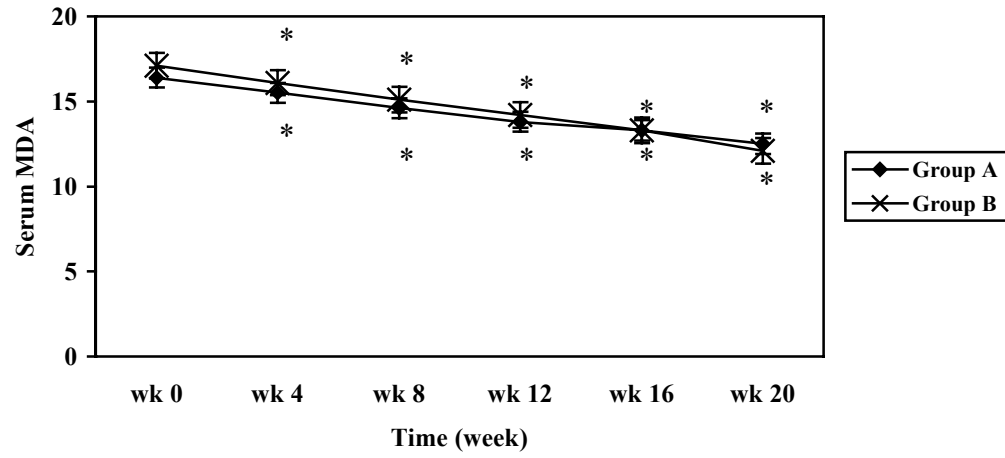


Fig 13 Mean of serum malondialdehyde concentration in group A and group B. Significant levels refer to differences from the mean of the control ($p < 0.001$).

CHAPTER VI

CONCLUSION

The purpose of this study is to investigate the effects of the cereals and nata de coco supplementation on serum lipids, serum vitamin A, E and lipid peroxidation and its acceptability, tolerance and safety in subjects with hyperlipidemia. The study was conducted in 22 subjects with hyperlipidemia. They consisted of 11 men and 11 women with the mean (\pm SEM) age of 55.2 ± 2.80 yrs, height of 160.4 ± 2.03 cm., body weight of 68.2 ± 2.86 Kg. and body mass index (BMI) 26.6 ± 1.06 Kg./ m² (**table10**). The criteria of hyperlipoproteinemia in these subjects was based on serum TC level of ≥ 200 mg./dL (≥ 5.2 mmol/L), serum TG level of ≥ 150 mg./dL (≥ 1.69 mmol/L) and LDL-C level of ≥ 130 mg./dL (≥ 3.4 mmol/dL). Three subjects dropped after wk 12 of the study because their personal problem and car accident.

After receiving the supplementation with the cereals and nata de coco supplement for 20 wks, the subjects were classified into 2 groups according to their compliance, **group A**; compliance ≥ 90 % of assigned cereals and nata de coco intake and **group B**; compliance < 90 % of assigned cereals and nata de coco intake.

Throughout the study, the subjects were instructed to consume diets with 15% protein-, 30% fat-, and 55% carbohydrate-calories. During the cereals and nata de coco supplementation period, the subjects consumed 15 g of the cereals and nata de coco supplement, dissolved in hot water as their dietary supplement twice daily before breakfast and dinner. This 30 g of the cereals and nata de coco supplement provided 122.57 kcal, 5.47 g of protein, 0.46 g of fat, 24.07 g of carbohydrate and 2.76 g of fiber. The results are summarized below.

Lipid status

1. There were no significant changes of serum lipid levels during the control period.
2. Only in group A, significant differences were seen between the mean of serum TG level at wk 0 and the mean values at wk 4, 8, and 12 ($p < 0.05$). For, serum

TC level at wk 16 was significantly lower than that at wk 0 ($p < 0.05$). The results imply that the insoluble fiber in the supplement food from 40 % nata de coco, 6 % unpolished rice, 36 % sweet corn, and 18 % mung bean may reduce serum TG in hyperlipidemic subjects.

3. The changes in serum lipid levels during the cereals and nata de coco supplementation period were mainly due to the consumption of the cereals and nata de coco supplement evidenced by dietary and biochemical data.

3.1 The mean (\pm SEM) cholesterol intakes of group A at wk 0, wk4, wk8, wk 12, wk 16 and wk 20 were 305 ± 14.8 , 283.7 ± 22.7 , 285.1 ± 22.9 , 310.4 ± 14.2 , 282.6 ± 23.1 , and 285.5 ± 12.8 mg/d, whereas group B, their mean (\pm SEM) cholesterol intakes of responder at wk 0, wk4, wk8, wk 12, wk 16 and wk 20 were 315 ± 44.5 , 313.5 ± 16.6 , 287.8 ± 16.6 , 327.0 ± 17.9 , 300.5 ± 27.2 , and 285.2 ± 32.2 mg/d. These values were not significantly different. Thus their cholesterol intake should not be the major factor in altering serum TC and LDL-C levels.

3.2 In group A, their mean (\pm SEM) energy intakes at wk 0, wk4, wk8, wk 12, wk 16 and wk 20 were 1558.1 ± 19.3 , 1530.3 ± 20.9 , 1566.3 ± 19.4 , 1675.5 ± 33.4 , 1529.0 ± 21.5 , and 1632.3 ± 15.3 kcal/d. These values were not significantly different. However, group B, their mean (\pm SEM) energy intakes at wk 0, wk4, wk8, wk 12, wk 16 and wk 20 were 1837.8 ± 27.8 , 1926.7 ± 39.5 , 1717.8 ± 26.4 , 2252.7 ± 36.6 , 1957.5 ± 40.6 , and 1936.8 ± 48.8 kcal/d. Their mean (\pm SEM) energy intakes at wk 12 were significantly higher than those at wk 0. The reason was the changing of their eating habit of subject in this period. Because the supplementation period at wk 12 was the same period of the end of year and New Year celebrations. Also, Most of subjects tended to increase their energy intakes, but after this period all of them can decrease energy intakes down to their normal values.

3.3 There were no significant changes in dietary energy distribution throughout the study: responder, 14 % protein -, 55 % carbohydrate -, and 31 % fat – calories whereas that of non-responder was 13 % protein -, 56 % carbohydrate -, and 31 % fat – calories. Thus their dietary energy distribution was close to that designed in our study.

The acceptability, tolerance and compliance of the subjects to the cereals and nata de coco supplement

4. The cereals and nata de coco supplement was well received. In the first wk 0 to wk 4, twelve and eleven of 22 subjects rated the taste and smell of the cereals and nata de coco supplement being acceptable (scored as 3). A few subject commented on taste and smell but after the initial period this no longer appeared to be a problem. At the end of wk 20, most of subjects rated the taste and smell of the supplement being good (scored as 2). All of 22 hyperlipidemic subjects were able to take whole volume of the supplement prescribed without any problem, whereas, they didn't have diarrhea and have adverse reaction (abdominal pain).

Blood glucose status

5. There were no significant changes in FBG levels during receiving the cereals and nata de coco supplementation.

Safety of ingesting the cereals and nata de coco supplementation

Serum mineral levels

6. Their serum sodium, potassium, chloride, calcium and phosphorous levels during the study were within the normal limits.

Liver and renal function tests

7. Their serum total and direct bilirubin, AST, ALT, alkaline phosphatase, GGT, total protein, albumin, urea nitrogen, creatinine, uric acid and CO₂ levels during the study were within the normal limits. These indicate that the consumption of the cereals and nata de coco supplement are not hazardous to liver and renal functions.

Serum vitamin A, β -carotene, and vitamin E levels

8. The serum vitamin A, β -carotene and vitamin E levels in 22 hyperlipidemic subjects before and during receiving the cereals and nata de coco supplementation were within the normal range.

The change of serum vitamin A and β -carotene levels may be related to the change of their serum vitamin A, β -carotene intake before and during receiving the cereals and nata de coco supplementation evident by the significantly positive correlation between vitamin A intakes in RE/d and serum vitamin A levels in $\mu\text{g/dL}$ (group A: $r = 0.915$, $p < 0.001$ and group B: $r = 0.940$, $p < 0.001$). This result was similar to the β -carotene intake in $\mu\text{g/d}$ and serum β -carotene concentration in $\mu\text{g/dL}$ (group A: $r = 0.955$, $p < 0.001$ and group B: $r = 0.980$, $p < 0.001$).

Lipid peroxidation

9. Evaluation of lipid peroxidation was based on serum malondialdehyde (MDA) concentration of 22 hyperlipidemic subjects.

Both group A and group B, significant differences were seen between the mean serum MDA concentration at wk 0 and the means serum MDA values at wk 4, 8, 12, 16 and 20. ($p < 0.001$).

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