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Low dietary fiber causes low-risk atherogenicity in male Wistar rats fed a cereal-containing infant formula

Chiedozie O. Ibegbulem¹, Linus A. Nwaogu¹, Ubong S. Udoala¹,
Donatus C. Belonwu², Chiduzie N. Udechukwu¹

ABSTRACT

Background and Objectives: Commercially available infant and non-infant cereal formulas are widely used as weaning meals. We hypothesized that differences in their fiber contents affect the lipid profiles of consumers. The aim of the study was to assess the plasma lipid profiles in rats fed two infant formulas and one non-infant formula. **Materials and Methods:** Weanling male rats were assigned to control group (Group A, pelletized guinea feed containing 10.0% fiber), infant formula B Group containing 7.0% fiber, infant formula C Group containing 1.7% fiber and non-infant formula D Group containing 7.2% fiber. Experimental rats were fed *ad libitum* with cereal formulas B, C, and D for 11 days under the same circadian rhythm and room temperature ($28.0 \pm 2.0^\circ\text{C}$). Body weight gained, total feed consumed, feed conversion ratio, plasma lipid profiles, and atherogenicity markers were evaluated. **Results:** Digestion and assimilation were not perturbed ($P > 0.05$). Formulas B and C reduced ($P < 0.05$) high density lipoprotein-cholesterol (HDL-c) and increased low density lipoprotein-cholesterol (LDL-c)/HDL-c ratio. Feed and formulas B and D were not atherogenic: Triacylglycerol (TG)/HDL-c ratio = 0.44 ± 0.21 , 0.86 ± 0.21 and 0.51 ± 0.00 , respectively; log (TG/HDL-c ratio) = -0.41 ± 0.22 , -0.08 ± 0.28 and -0.28 ± 0.01 , respectively. Formula C was low-risk atherogenic: TG/HDL-c ratio = 1.34 ± 0.44 and log (TG/HDL-c ratio) = 0.10 because of low fiber (1.7%) and/or no gluten contents; unlike formula B with 7.0% fiber + gluten, and formula D with 7.2% fiber + gluten. Effect size, r , of its fiber on LDL-c/HDL-c ratio was 0.3928 ($r^2 = 15.43\%$) unlike $r = 0.9863$ ($r^2 = 97.28$) and 0.9978 ($r^2 = 99.56\%$) for formulas B and D, respectively. **Conclusion:** Atherogenic tendency of formula C could be eliminated by increasing its fiber content.

KEY WORDS: Atherogenicity, cereal formulas, fiber, lipid profile, rats

¹Department of Biochemistry, Federal University of Technology, Owerri, Nigeria,
²Department of Biochemistry, University of Port Harcourt, Port Harcourt, Nigeria

Address for correspondence:
Dr. Chiedozie O. Ibegbulem,
Department of Biochemistry,
Federal University of
Technology, Owerri, Nigeria.
E-mail: ibemog@yahoo.com

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INTRODUCTION

Children, adolescents and adults rely on cereal formulas as sources of tasty nourishing meals. These nutritious supplements are commercially available and can be prepared at home, and have also been used to wean infants. Infancy is characterized by rapid physical growth, physiological, immunological, and mental developments [1]. The human milk satisfies infant nutrient needs for the first 4-6 months of life; the possible exceptions being the relative lack of fluoride, iron and vitamin D [2]. The World Health Organization recommends exclusive breastfeeding for the first 6 months of life, with continued breastfeeding along with appropriate complementary foods up to 2 years of age or beyond [3]. After this period, there is a gradual shift to semi-solid and solid food formulas; most of which are used to wean the infant. Infant formulas generally use heat-treated cow's milk as base; with soya-bean-based formulas accounting for 25% of the infant formulas used in the United States [2]. Wardlaw and Kessel [2] also reported that the concern over the use of soy milk in infant feeding is because of the isoflavone content;

that isoflavones may cause endocrine disruption, developmental delay in boys and precocious puberty in girls.

Commercially available infant and non-infant formulas contain different levels of fiber, even when they are produced by the same manufacturer and lead us to hypothesize that they have different effects on plasma lipid profiles of consumers. Water-soluble fiber reduces serum total cholesterol (TC) and low density lipoprotein cholesterol (LDL-c) without affecting triacylglycerol and high density lipoprotein cholesterol (HDL-c) [4,5]. Most of the infant and non-infant formulas sold in Nigeria are products of a popular conglomerate with manufacturing and distribution concerns in Spain, Nigeria, Côte d'Ivoire, Gambia, Cameroon, Burkina Faso, Mali, Togo, Benin, Niger, Equatorial Guinea, Gabon, Ghana, Guinea, Senegal, Democratic Republic of Congo, Chad, and Sao Tome and Principe. Their trademarks were not revealed, but coded and sufficiently described in Tables 1 and 2 using the manufacturer's information. In previous studies, the protein efficiency ratios of some of them were reported to a range between 1.69 and 2.40 [6,7]; indicating that their proteins were of high

qualities. Whereas oat-based cereal may contribute to a lipid-lowering diet [5], parenteral administration of soybean to children undergoing bone marrow transplantation reduced HDL-c and apolipoprotein A [8]. On the other hand, soybean milk diet improves plasmatic lipid profiles by decreasing serum TC and LDL-c and increasing serum triglycerides, HDL-c in experimental animals; thus, lowering LDL/HDL ratio [9]. There are little or no reports on the effects of the maize- and soybean-based cereal formulas sold in Nigeria on the lipid profiles of consumers; hence, a precognition is desired. The aim of this study was to determine the effects of some randomly selected cereal infant and non-infant formulas on the plasma triacylglycerol (TG), TC, LDL-c, HDL-c and very LDL-c (VLDL-c) levels and their atherogenic tendency in rats.

MATERIALS AND METHODS

Animals and Diets

Twenty weanling male albino rats of the Wistar strain were purchased from the animal house of the Department of Veterinary Medicine, University of Nigeria, Nsukka, Nigeria. The rats were 5-6 weeks old, had mean initial body weights of 49.98 ± 5.45 g and were housed individually in wire-screened cages with provisions for feed and fluid troughs. They were maintained under the same circadian cycle and a

Table 1: Codes, descriptions and ingredients of feed and cereal-containing formulas

Code	Description	Ingredients*
A	Guinea feed	Cereals/grains, vegetable protein, premix (vitamins/mineral), essential amino acids, salt, antioxidant, anti-toxins, prebiotic and enzymes
B	Infant cereal	Degermed maize flour, soya bean flour (dehulled), sucrose, palm olein, calcium carbonate, sodium phosphate, sodium chloride, soya lecithin, vitamins, ferric pyrophosphate, vanillin, zinc sulfate, potassium iodide. May contain traces of milk and gluten
C	Infant cereal	Cereal (enzymatically hydrolyzed wheat flour 36.1%, wheat flour 23.5%), skimmed milk powder (23.3%), vegetable oils, honey (5%), mineral mix (calcium carbonate, iron fumarate, zinc sulfate, potassium iodide), vitamin mix (C, niacin, E, calcium pantothenate, thiamin, A, B ₆ , K, folic acid, biotin, D), culture (<i>Bifidobacterium lactis</i>), and flavoring
D	Non-infant cereal	Whole maize flour, sucrose, soya bean flour (dehulled), sodium chloride, calcium carbonate, acidity regulators: Potassium phosphate, sodium phosphate, ferrous fumarate, vitamin A. May contain traces of milk and gluten

*Manufacturers' information

Table 2: Nutritional information of feed and cereal formulas

Parameter	Feed and cereal formulas*				Mean	SD	CV%
	A [#]	B [#]	C [#]	D [#]			
Energy (kcal/100 g)	255.0±0.00	398.0±0.00	427.0±0.00	373.4±0.00	363.35	65.37	17.99
Protein (g/100 g)	15.0±0.00	15.0±0.00	15.0±0.00	13.0±0.00	14.50	0.87	5.97
Carbohydrates (g/100 g)	NG	64.2±0.00	68.3±0.00	65.5±0.00	66.00 [§]	1.71 [§]	2.59 [§]
Fat (g/100 g)	7.0±0.00	9.0±0.00	10.0±0.00	6.6±0.00	8.15	1.97	24.14
Dietary fiber (g/100 g)	10.0±0.00	7.0±0.00	1.7±0.00	7.2±0.00	6.48	3.00	46.31

Values are mean±SD of 10 data sources. *Manufacturers' descriptions, [#]Based on the codes in Table 1, [§]Computed without carbohydrates from pelletized guinea feed. SD: Standard deviation, CV: Coefficient of variation, NG: Not given

room temperature of $28.0 \pm 2.0^\circ\text{C}$ and treated humanely as encapsulated in National Institutes of Health [10] guidelines. The research was approved by the Departmental Ethical Committee on the use of animals for the research, Department of Biochemistry, Federal University of Technology, Owerri, Nigeria.

The pelletized guinea feed (product of a subsidiary of UAC Nigeria Plc., Jos, Nigeria) and three commercially available cereal formulas used in the study were purchased at the Relief Market, Owerri, Nigeria.

Rat Feeding Study

The rats were allotted to four groups of five rats each on a weight basis. The rats in each group were acclimatized for 4 days. The control rats were fed pelletized guinea feed (code named A), while the experimental rats were fed with two cereal infant formulas (code named B and C, respectively) and one non-infant formula (code named D) for 11 days. All the rats drank tap water.

Anthropometric Measurements

At the end of the feeding study, each rat was re-weighed and the body weight gained was calculated as the difference between the final and initial body weights. Feed consumed was calculated as the sum of the daily feed intakes throughout the experimental period while feed conversion ratio was calculated as the ratio of the feed consumed to the body weight gained.

Collection of Blood and Preparation of Plasma

The rats were anesthetized by exposure to dichloromethane vapor in a covered transparent plastic container. Incisions were then made into their thoracic regions and were terminally bled by cardiac puncture using 5 mL hypodermic syringes and needles. The blood was immediately transferred into lithium-heparin sequestering bottles. Lithium-heparin causes the release of the tissue-based extracellular enzyme, lipoprotein lipase, *in vivo* [11] but may not interfere with lipid metabolism *in vitro* because of the absence of tissue and lipoprotein lipase. The uncoagulated blood was centrifuged at 3000 g for 10 min and the respective plasma collected by aspiration using Pasteur pipette.

Assay for Plasma Lipid Profile

Plasma was analyzed for its contents of TG, TC, LDL-c and HDL-c using a standard diagnostic kit (Biosystems[®],

S.A. Costa Brava 30, Barcelona, Spain) with the aid of a digital spectrophotometer model 590 (Turner®, USA). Briefly, TG concentration was determined when it was converted to glycerol 3-phosphate by lipase and glycerol kinase. The hydrogen peroxide generated when the glycerol 3-phosphate was converted to dihydroxyacetone phosphate by glycerol 3-phosphate dehydrogenase at pH 7.0. TC concentration was determined when free and esterified cholesterol were used to generate hydrogen peroxide using cholesterol esterase and cholesterol oxidase and the H₂O₂ later used to generate a chromophore, through the action of peroxidase. HDL-c concentration was determined when VLDL-c and LDL-c were precipitated with phosphotungstate and magnesium ions and the HDL-c content of the supernatant monitored quantitatively by the actions of cholesterol esterase, cholesterol oxidase, and a chromophore (quinoneimine) formed from dichlorophenol sulfonate by peroxidase at pH 7.0. LDL-c concentration was determined when it was precipitated with polyvinyl sulfate and its concentration calculated from the difference between the serum TC and cholesterol in the supernatant after centrifugation. VLDL-c was calculated by dividing the TG content (mmol/L) by 2.2 [12].

Atherogenic Tendency

Atherogenic tendency was determined using the TG/HDL-c ratio, which is a strong predictor of myocardial infarction and coronary disease [13,14]; log (TG/HDL-c ratio), which is the atherogenic index of plasma (AIP) [15]; TC/HDL-c and LDL-c/HDL-c ratios which are measures of atherogenicity [16].

Statistical Analysis

Results were expressed as mean \pm standard deviation. Data between groups on the same column were analyzed relative to the control using one-way analysis of variance. Effect size (as coefficient of correlation, *r*) between two variables was calculated by converting the *t*-statistics into *r* value at 95% as described by Field [17]; briefly, *r* was calculated as the square root of the ratio of the square of the *t* value to the sum of the square of the *t* value and the degree of freedom was calculated. Data on the same row were analyzed using percentage coefficient of variation (CV%).

RESULTS

The ingredients pelletized guinea feed, two infant and non-infant formulas, coded A, B, C, and D, respectively, as described by their manufacturer are presented in Table 1. The CV% for their proximate compositions ranged from 2.59% to 46.31%, but the carbohydrate content of the pelletized guinea feed was not provided by the manufacturer [Table 2]. The control rats gained the most ($P < 0.05$) body weight followed by the rats that were placed on infant formula B, infant formula C, then non-infant formula D in that order. It also shows that the control rats and the rats that were placed on formula B ate more ($P < 0.05$) feed than those on formulas C and D. However, the feed conversion ratios of all the rat groupings did

not significantly ($P > 0.05$) vary [Table 3]. The plasma TC, TG, LDL-c, and VLDL-c contents of the rats did not significantly ($P > 0.05$) vary, whereas the plasma HDL-c contents of the rats that were placed on formulas A and D were significantly ($P < 0.05$) higher than those of the rats that were placed on formulas B and C [Table 4]. The atherogenicity indices of the rats show that the plasma TG/HDL-c ratios and the logarithms of these ratios for the rats that were placed on formula C is significantly ($P < 0.05$) higher than those of the rats in Groups A, B, and D. The plasma TC/HDL-c ratio of the rats in Groups A, B, and C were significantly ($P < 0.05$) higher than that of rats in Group D. It also shows that their plasma LDL-c/HDL-c ratios were in the order: Groups B and C > Group A > Group D [Table 5]. The effect size (*r*) between dietary fat and LDL-c, dietary fat and LDL-c/HDL-c ratio, dietary fiber and HDL-c, dietary fiber and LDL-c and dietary fiber and LDL-c/HDL-c ratio for rat placed on pelletized guinea feed A and formulas B and D were very large. However, for rats placed on formula C, the effect size (*r*) between dietary fat and LDL-c, and dietary fat and LDL-c/HDL-c ratio were very large, and those between dietary fiber and HDL-c, and dietary fiber and LDL-c were fairly large. Table 6 also shows that the effect size of the dietary fiber content of formula C on the plasma LDL-c/HDL-c ratio of the rats that ate it was weak.

DISCUSSION

Detailed descriptions of the ingredients used in preparing the feed and cereal formulas [Table 1] may help the probing mind to easily identify them. The feed and formulas were nutritionally blended by the manufacturers to contain different types of cereals, vitamins and minerals that are supposed to

Table 3: Anthropometric measurements at the end of the experimental period (11 days)*

Group [#]	Number of rats	Weight gained (g)	Feed consumed (g)	Feed conversion ratio
A	5	33.97 \pm 1.69 ^a	102.54 \pm 6.12 ^a	3.02 \pm 0.03 ^m
B	5	29.97 \pm 1.49 ^b	77.62 \pm 22.12 ^a	2.56 \pm 0.61 ^m
C	5	21.04 \pm 1.20 ^c	60.72 \pm 14.49 ^b	2.86 \pm 0.53 ^m
D	5	21.00 \pm 1.05 ^c	68.80 \pm 14.31 ^b	3.25 \pm 0.52 ^m

*Values are mean \pm SD, [#]Based on codes in Table 1. Values on the same column bearing the same superscript letter a, b, c, or m are not significantly different ($P > 0.05$). SD: Standard deviation

Table 4: Effects of feed and cereal formulae on lipid profiles (mmol/L) of rats at the end of the experimental period (11 days)*

Group [#]	TC	TG	HDL-c	LDL-c	VLDL-c
A	2.45 \pm 0.41 ^a	0.64 \pm 0.37 ^b	1.36 \pm 0.21 ^c	0.80 \pm 0.63 ^a	0.29 \pm 0.16 ^m
B	2.46 \pm 0.33 ^a	0.65 \pm 0.18 ^b	0.86 \pm 0.42 ^d	1.31 \pm 0.29 ^a	0.29 \pm 0.08 ^m
C	2.67 \pm 0.36 ^a	0.72 \pm 0.42 ^b	0.72 \pm 0.55 ^d	1.12 \pm 0.29 ^a	0.33 \pm 0.19 ^m
D	2.26 \pm 0.40 ^a	0.75 \pm 0.19 ^b	1.38 \pm 0.28 ^c	0.54 \pm 0.48 ^a	0.34 \pm 0.08 ^m

*Values are mean \pm SD, [#]Based on the codes in Table 1. Values on the same column bearing the same superscript letter a, b, c, d or m are not significantly different ($P > 0.05$). HDL-c: High density lipoprotein-cholesterol, LDL-c: Low density lipoprotein-cholesterol, TG: Triacylglycerol, TC: Total cholesterol, VLDL-c: Very low density lipoprotein-cholesterol, SD: Standard deviation

Table 5: Effects of feed and cereal formulae on atherogenicity indices of rats at the end of the experimental period (11 days)*

Group [#]	Number of rats	TG:HDL-c	Log (TG:HDL-c)	TC:HDL-c	LDL-c:HDL-c
A	5	0.44±0.21 ^c	-0.41±0.22 ^c	1.80±0.03 ^b	0.53±0.38 ^b
B	5	0.86±0.21 ^a	-0.08±0.11 ^a	3.37±1.48 ^a	1.79±0.54 ^a
C	5	1.32±0.44 ^a	0.10±0.15 ^a	1.88±0.52 ^b	3.00±1.89 ^a
D	5	0.51±0.00 ^c	-0.29±0.00 ^c	1.65±0.05 ^c	0.33±0.28 ^c

Values are mean±SD. *Computed from Table 4, [#]Based on the codes in Table 1. Values on the same column bearing the same superscript letter a, b or c are not significantly different ($P>0.05$). HDL-c: High density lipoprotein-cholesterol, LDL-c: Low density lipoprotein cholesterol, TG: Triacylglycerol, TC: Total cholesterol, SD: Standard deviation

Table 6: Effect size (*r*) of dietary fat and LDL-c, dietary fat and LDL-c/HDL-c ratio, dietary fiber and HDL-c, dietary fiber and LDL-c and dietary fiber and LDL-c/HDL-c ratio*

Group [#]	Dietary fat and LDL-c	Dietary fat and HDL-c	Dietary fiber and HDL-c	Dietary fiber and LDL-c	Dietary fiber and LDL-c: HDL-c
A	0.9896	0.9955	0.9992	0.9941	0.9979
B	0.9981	0.9928	0.9940	0.9966	0.9863
C	0.9986	0.9169	0.7360	0.7801	0.3928
D	0.8273	0.9974	0.9973	0.9933	0.9978

*Computed using mean values in Table 1 and mean±SD values in Tables 4 and 5, [#]Based on the codes in Table 1. LDL-c: Low density lipoprotein-cholesterol; HDL-c: High density lipoprotein-cholesterol, SD: Standard deviation

meet basic nutritional requirements. While formulas B and D contained gluten, formula C did not. The variations between their proximate compositions were high in their fat and dietary fiber contents [Table 2]. Their energy contents are normally derived from their fat, protein, and carbohydrate contents. The variations in their protein and carbohydrate contents were low; with their protein contents maintaining the recommended level (>10%) that allows for reasonable growth in young rats [18]. Consuming formula D (with the lowest energy value) at 215 g/day would certainly meet the recommended daily minimum of 800 kcal [19].

The formulas did not affect the abilities of the experimental rats to convert feed mass to body mass [Table 3]. This was confirmed by their body weight gains, which suggested that they grew; with the rats that were placed on formula B growing more than those placed on formulas C and D probably because it was more palatable. Change in body mass is a frequently used indicator of growth [20]. Taste, aroma and texture affect food consumption [2].

The ability to synthesize plasma HDL-c was lower in the rats that were placed on the cereal infant formulas B and C than in the rats that were placed on guinea feed and the non-infant formula D [Table 4]. Even though the rats that were placed on formulas B and C were adjudged to have been exposed to the same cardiovascular risk, their effect size ($r = 0.4376$, $P > 0.05$) indicated that there was a medium effect on the difference between their plasma LDL-c levels, thus suggesting that formula C was more atherogenic. The plasma pro- to anti-atherogenic lipoprotein particles ratios for the rats (for instance

their TG/HDL-c ratios) also suggested that only the rats that were placed on formula C were atherogenic and prone to coronary disease [Table 5]. The AIP index for these vulnerable rats; however, suggested that they were at low-risk of developing cardiovascular disease (CVD); since the index was lower than 0.11. The range for an intermediate risk of developing atherogenicity is 0.11-0.21 while indices above 0.21 indicate increased risk of developing CVD [14]. Abnormal TG/HDL-c ratio indicates an atherogenic lipid profile and a risk for the development of coronary disease [12,13]. Lifshitz *et al.* [21] also reported that the ratio is also predictive of risk to CVD and incidence of ischemic heart disease.

Formulas B and D contained dehulled soybean flour [Table 1]. Results presented in Tables 4 and 5 seemed to suggest that formula B was the more atherogenic of the two dehulled soybean flour-containing formulas. This may have been as a result of its 2.86% lower fiber content relative to that of formula D [Table 2]. These formulas were less atherogenic than the wheat flour-containing formula C [Table 5]. Formula D improved plasma lipid profile by increasing plasma HDL-c [Table 4], thus lowering LDL-c/HDL-c ratio [Table 5] as reported by Fontenla *et al.* [9], relative to formula B.

Dietary fat and fiber influenced the plasma lipid profiles and atherogenic tendencies of the experimental rats [Table 6]. The relationships were very strong in formulas B and D. While plasma levels of LDL-c and LDL-c/HDL-c ratio were very strongly influenced by the level of fat in formula C, the formula's dietary fiber content exerted strong influences on the plasma HDL-c and LDL-c levels of the rats that ate it. However, the low fiber content of formula C exerted a weak control over the group's plasma LDL-c/HDL-c ratio. The dietary fiber content of formula C had a 15.43% influence over the group's plasma LDL-c/HDL-c ratio unlike 97.28% and 99.56% exerted by the fiber contents of formulas B and D, respectively.

The low-risk atherogenic tendency of formula C can be eliminated by increasing its fiber content and/or incorporating gluten. If the fiber content of formula C is increased to 5.0%, it can increase r to 0.5428, at the present plasma LDL-c/HDL-c ratio. However, this level of fiber can also proportionately decrease the LDL-c/HDL-c ratio to 1.02, other things being equal. That can mean either reducing plasma LDL-c or increasing its HDL-c levels. Recall that formula C had the lowest fiber content that gave rise to the high CV% in Table 2. Dietary fiber has been reported to help lower blood TC, LDL-c, LDL-c/HDL-c ratio, and decrease the risk of coronary heart disease [22-25], while increasing plasma HDL-c concentration [24,26]. Dietary fiber exerts its plasma LDL-c lowering effect by interfering with bile absorption in the intestine and its enterohepatic cycling [27]. Formula C did not also contain gluten [Table 1]. Oat-derived beta-gluten has also been reported to lower TC/HDL-c ratio, LDL-c/HDL-c ratio and increase HDL-c concentration [24]. Thence, formula C's atherogenic nature may be as a result of low fiber content and/or lack of gluten.

The study had a limitation. The trademarks of the formulas were not disclosed for easy identification because of legal

reasons. This means that the reader will need to labor through the identification process using the manufacturer's information provided in Tables 1 and 2.

CONCLUSION

Only formula C was atherogenic because it had low fiber content and/or lacked gluten.

REFERENCES

- Okoye ZS. Biochemical Aspects of Nutrition. New Delhi: Prentice-Hall of India; 1992.
- Wardlaw GM, Kessel MW. Perspective in Nutrition. 5th ed. Boston: McGraw-Hill; 2002.
- WHO. Noncommunicable Diseases and Mental Health. Geneva: World Health Organization; 2013.
- Brown L, Rosner B, Willett WW, Sacks FM. Cholesterol-lowering effects of dietary fiber: A meta-analysis. *Am J Clin Nutr* 1999;69:30-42.
- Poulter N, Chang CL, Cuff A, Poulter C, Sever P, Thom S. Lipid profiles after the daily consumption of an oat-based cereal: A controlled crossover trial. *Am J Clin Nutr* 1994;59:66-9.
- Onyeike EN, Ayalogu EN, Ibegbulem CO. Evaluation of the nutritional value of some crude oil polluted freshwater fishes. *Glob J Pure Appl Sci* 2000;6:227-33.
- Okorie SU, Nwanekezi EC. Effects of processing methods on the quality of maize-groundnut infant weaning food. *Glob J Pure Appl Sci* 2002;8:209-14.
- Baena-Gómez MA, de la Torre Aguilar MJ, Mesa MD, Llorente-Cantarero FJ, Pérez Navero JL, Gil-Campos M. Effects of parenteral nutrition formulas on plasma lipid profile in children with bone marrow transplantation. *Ann Nutr Metab* 2013;63:103-10.
- Fontenla M, Prchal A, Cena AM, Albarracín AL, Pintos S, Benvenuto S, et al. Effects of soy milk as a dietary complement during the natural aging process. *Nutr Hosp* 2008;23:607-13.
- United State Department of Health, Education, and Welfare. Public Health Service; NIH. Guide for the Care and Use of Laboratory Animals. Washington, DC: National Academics Press; 1985, Revised 1996.
- Roth B, Ekelund M, Fan BG, Ekstrom U, Nilsson-Ehle P. Effects of heparin and low molecular weight heparin on lipid transport during parenteral feeding in the rat. *Acta Anaesthesiol Scand* 1996;40:102-11.
- Burnstein MA, Sammille J. A rapid determination of cholesterol bound to A and B lipoprotein. *Clin Chem Acta* 1960;5:601-9.
- Gaziano JM, Hennekens CH, O'Donnell CJ, Breslow JL, Buring JE. Fasting triglycerides, high-density lipoprotein, and risk of myocardial infarction. *Circulation* 1997;96:2520-5.
- da Luz PL, Favarato D, Faria-Neto JR Jr, Lemos P, Chagas AC. High ratio of triglycerides to HDL-cholesterol predicts extensive coronary disease. *Clinics (Sao Paulo)* 2008;63:427-32.
- Dobiášová M, Frohlich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: Correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FER(HDL)). *Clin Biochem* 2001;34:583-8.
- Ibegbulem CO, Chikezie PC. Serum lipid profile of rat (*Rattus norvegicus*) fed palm oil and palm kernel oil containing diet. *Asian J Biochem* 2012;7:46-53.
- Field A. Discovering Statistics Using SPSS. 2nd ed. London: SAGE Publications Ltd.; 2005.
- Henry KM, Kon SK. Effect of level of protein intake and of age of rat on the biological value of proteins. *Br J Nutr* 1957;11:305-13.
- FAO. Energy and protein requirement. Food and Agricultural Organization. Geneva: World Health Organization; 1973.
- Taylor DJ, Green NP, Stout GW. Growth and development. In: Soper R, editor. *Biological Science*. 3rd ed. Cambridge: University Press; 1998. p. 758-75.
- Lifshitz F, Pintos PM, Lezón CE, Macri EV, Friedman SM, Boyer PM. Dyslipidemia is not associated with cardiovascular disease risk in an animal model of mild chronic suboptimal nutrition. *Nutr Res* 2012;32:52-8.
- Rivellese A, Riccardi G, Giacco A, Pacioni D, Genovese S, Mattioli PL, et al. Effect of dietary fibre on glucose control and serum lipoproteins in diabetic patients. *Lancet* 1980;2:447-50.
- Hunninghake DB, Miller VT, LaRosa JC, Kinoshian B, Brown V, Howard WJ, et al. Hypocholesterolemic effects of a dietary fiber supplement. *Am J Clin Nutr* 1994;59:1050-4.
- Reyna-Villasmil N, Bermúdez-Pirela V, Mengual-Moreno E, Arias N, Cano-Ponce C, Leal-Gonzalez E, et al. Oat-derived beta-glucan significantly improves HDLC and diminishes LDLC and non-HDL cholesterol in overweight individuals with mild hypercholesterolemia. *Am J Ther* 2007;14:203-12.
- Bazzano LA. Effects of soluble dietary fiber on low-density lipoprotein cholesterol and coronary heart disease risk. *Curr Atheroscler Rep* 2008;10:473-7.
- Lindgärde F, Larsson L. Effects of a concentrated bran fibre preparation on HDL-cholesterol in hypercholesterolaemic men. *Hum Nutr Clin Nutr* 1984;38:39-45.
- Fernandez ML. Distinct mechanisms of plasma LDL lowering by dietary fiber in the guinea pig: Specific effects of pectin, guar gum, and psyllium. *J Lipid Res* 1995;36:2394-404.

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