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Research Article

EFFECT OF THE HIGH INTAKE OF PALM OIL ON THE PLASMA LIPID PROFILE AND ARTERIAL BLOOD PRESSURE IN RATS

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ABSTRACT

The aim of this study was to investigate the effect of the excessive intake of fresh palm oil and refined palm oil (PROMOR) on some physiological parameters such as growth rate, lipid profile and arterial blood pressure in rats. Three groups of Wistar albino rats were submitted to different types of diets rich in fresh palm oil (FPO) or in refined palm oil (RPO) at the doses of 10%, 15% and 20% for each against a control group receiving only the basal diet, within a period of 12 weeks. The effects of these different diets were evaluated on the weight of some target organs, the variation of body weight, the lipid profile and the arterial blood pressure. The intake of these oils led chronologically to growth retardation (31%); atherogenic dyslipidemia characterized by a highly significant increase of the atherogenic index (117%) and plasma level of total cholesterol (59%), triglycerides (200%) and LDL cholesterol (108%). However, a marked decrease in HDL cholesterol (79.9%) was also noted, with hypertension as a consequence for which an increase of 76.5% of the blood pressure was recorded in the group of animals receiving 20% RPO. A high intake of fresh or refined palm oil could probably cause growth retardation, atherogenic dyslipidemia and arterial hypertension.

Keywords: Palm oil, weight gain, dyslipidemia, arterial hypertension, atherosclerosis.

INTRODUCTION

Hyperlipidemia is defined as an abnormal increase in the total plasma concentrations of cholesterol (hypercholesterolemia), triglycerides (hypertriglyceridemia), low density lipoproteins (LDL cholesterol) with a reduction in the level of high density lipoproteins (HDL cholesterol). Just like hypertension and diabetes, these abnormal functioning are the primary cause of cardiovascular diseases, which by themselves, constitute a major problem in public health[1,2].

In Cameroon, about 43% of people are obese especially in the West region. In Dschang (Menoua Division of the West region), out of 87 women tested, 33.3% were obese[3]. Obesity is a major risk factor for the development of arterial hypertension,[4] which represents the most important cardiovascular affection in the world[5]. At least 5% of deaths in adults are in relation with arterial hypertension[6]. Cameroon meals are very rich in lipids of vegetable origin (palm oil) which would be susceptible to increase the risks of development of dyslipidemia and more precisely atherogenic dyslipidemia which is characterized by a high plasma level of total cholesterol, triglycerides and LDL cholesterol and a low level of HDL cholesterol[7]. Palm oil has been used in food preparation for over 5,000 years. Palm oil, obtained from the fruits of the palm trees, is the most widely produced edible vegetable oil in the world and it's nutritional and health attributes have been well documented[8]. This oil is consumed in its fresh state and/or at various levels of oxidation. Feeding experiments in various animal species and humans have highlighted controversial evidences on the beneficial and harmful effects of fresh palm oil to health. On one hand, these benefits include reduction in the risk of arterial thrombosis and atherosclerosis, inhibition of cholesterol

biosynthesis and platelet aggregation, and reduction in blood pressure[9]. However, on the other hand, when used in the oxidized state, it possesses potential dangers to the physiological and biochemical functions of the body. The reduction of the dietetic level of oxidized oil and/ or the level of oxidation may reduce the health risk[10]. For many years now, it has been established that the primary cholesterol-elevating fatty acids are the saturated fatty acids with 12 (lauric acid), 14 (myristic acid) and 16 (palmitic acid) carbon atoms with a concomitant increase in the risk of coronary heart disease. The World Health Organization in its report (2005)[11] states that there is convincing evidence that palmitic oil consumption contributes to an increased risk of developing cardiovascular diseases. Past research confined to epidemiological observations, intervention trials and studies on experimental animals and humans have provided vital information on the obvious facts of the dietetic fats on blood pressure (BP). So, the excessive or chronic intake of dietary oils rich in saturated fatty acids could increase the risk of metabolic and cardiovascular diseases[8]. Therefore, the aim of this study was to investigate the effect of the chronic intake of fresh palm oil (obtained from Dschang, local market) and refined palm oil (PROMOR) on some physiological and metabolic parameters such as growth rate, plasma lipid profile and arterial blood pressure in rats.

MATERIALS AND METHODS

Experimental design

A total number of 168 Wistar albino rats of both sex aged within 3 to 4 weeks, were used. These rats were divided into 3 comparable groups of 56 rats each. The later was then divided into 7 groups of 8 rats each. The first group was fed with basal diet (control) and the subsequent groups were fed with basal diet fortified with 10%, 15% or 20% fresh palm oil (FPO) or refined palm oil (RPO), respectively for 4, 8 and 12 consecutive weeks. The composition of the various diets, as described by Mbogning[12], is as shown in Table 1. All rats had free access to food and water throughout the experiment. The males were separated from the females in other to avoid mating. Blood pressure was measured at the 8th and 12th week of treatment. Blood was obtained at the end of each treatment phase through the abdominal artery. The blood obtained was immediately centrifuged at 3000 rounds per minute for 15 minutes. The plasma obtained was labelled and conserved at -20^{o}C. This helped to evaluate the lipid profile. After this, the heart, aorta and liver were delicately isolated, from conjunctive tissues, cleaned in NaCl 0.9%, dried and weighed. The left ventricle was then separated from the heart and weighed.

The relative body weight of each animal was calculated as follows:

$$\text{Relative body weight} = \frac{\text{Absolute body weight of one time interval (g)}}{\text{Body weight of rat on commencement of treating day (g)}} \times 100$$

Measurement of blood pressure

Blood pressure and cardiac frequency were determined by the invasive method. Briefly, Animals were anaesthetized by intraperitoneal administration of sodium thiopental at the dose of 50 mg/kg and a catheter was inserted into the femoral vein for drug administration. Another catheter was inserted in the left carotid artery for direct blood pressure measurement. Both catheters were filled with glucose-saline heparinised solution. The catheter inserted in the carotid artery was connected to a blood pressure transducer model Ugo Basile PRC 21k-10 coupled with an Ugo Basile Unirecord model 7050 for blood pressure recordings. A stabilisation period of 30 minutes was observed before any recording. Heart frequency was determined by the use of pulse intervals.

Measurement of plasma lipids, left ventricular index and atherogenic index

Blood samples were collected from the abdominal artery and centrifuged at 3000 rpm for 15 minutes. The resultant supernatant was assayed for total cholesterol (TC), triglycerides (TG) and high density lipoprotein (HDL). TC, TG, and HDL were estimated using the method described by Richmond[13], Roeschlau[14] and Cole[15] with an available commercial kit IMNESCO. The LDL cholesterol, left ventricular and atherogenic index were determined using the formulas below[16]:

$$\text{LDL cholesterol (mg/dl)} = \text{Total cholesterol} - \frac{\text{Triglycerides}}{5} - \text{HDL cholesterol}$$

$$\text{Left ventricular index} = \frac{\text{Mass of left ventricle}}{\text{Mass of the heart}}$$

$$\text{Atherogenic index} = \frac{\text{LDL cholesterol}}{\text{HDL cholesterol}}$$

Statistical analysis

Statistical analysis was performed using Graph Pad Prism version 5.0. All results are expressed as means ± standard error of the mean. Data were analysed using one-way analysis of variance (*ANOVA*) followed by Tukeys'post test. Differences between means were considered to be significant when $p<0.05$.

RESULTS

Effects of the consumption of fresh palm oil (FPO) and refined palm oil (RPO) at the doses of 10%, 15% and 20% on body weight

Figure 1 illustrates the evolution in body weight of the animals treated after 4, 8 and 12 weeks with fresh palm oil (FPO) and refined palm oil (RPO) at different doses per kilogram per body weight. It comes out of this study that, a significant decrease 31% ($p<0.05$) of the body weight was observed from the second week of treatment in the group of animals treated with ration containing 20% FPO and RPO as compared to the control. However weight loss was more pronounced in FPO treated groups.

Effects of the consumption of fresh and refined palm oil at the dose of 10%, 15% and 20% on the lipid profile

The lipid metabolism in rats treated chronically with fresh and refined palm oil at different doses was significantly disturbed compared to the untreated rats. As shown in tables 2, 3 and 4, there is an increase in the levels of total cholesterol, triglycerides and LDL cholesterol with percentages of 59%, 200%, and of 108% respectively, observed in the group of rats fed with 20% RPO, against a significant ($P<0.01$) decrease of 79.9% in the level of HDL cholesterol, observed in animals fed with 20% RPO as compared to the control.

Effects of palm oils on blood pressure, left ventricular index and on some target organs.

From the results illustrated in Table 5, there was a highly significant increase ($p<0.001$) in blood pressure at the end of the study for all the groups fed with both oils as compared to the control group. The increment was higher in RPO groups (76.5% at 20% diet). In addition, the left ventricular index and the aorta showed significant increase in all the groups treated with both oils, with marked effects or more significant in animals treated with refined palm oil. Concerning the liver weight, an increase in the relative weight of this organ was lower in animals treated with fresh palm oil than in animals treated with refined palm oil, as compared to the control.

DISCUSSION

Palm oil has been used in food preparation for over 5 000 years now. So, the aim of this study was to investigate the effect of the chronic intake of dietetic oils (fresh and refined palm oil) rich in saturated fatty acids on some physiological and metabolic parameters. The results obtained in the present study, showed a significant reduction in body weight observed upon treatment with both oils (fresh and refined palm oil). This could be due to a reduction in food intake observed in the treated groups as compared to the control. It is shown that, this reduction in food intake could be due to the time necessary for the treated groups to adapt to the high fatty diet on one hand, and on the other hand, the mechanisms of stimulation of appetite whose role is to regulate food intake so as to maintain homeostasis between calorie intake and energy loss[17]. The reduction in body weight could also be explained as a deficiency in the hepatic biosynthesis of amino acids necessary for protein synthesis. As such, proteins needed for body building will not be available. In addition an elevation in the blood level of triglycerides; total cholesterol, LDL cholesterol and a decrease in the level HDL cholesterol are associated with the growth hormone deficiency syndrome[18].

Also, an increase in the relative weight of the liver could be due to, firstly, an increase in the metabolic activity of hepatocytes and secondly to

the accumulation of fats within this organ, caused by non-esterified fatty acids. More so, the increase in the weight of the aorta could be justified by the hypercholesterolemia, which is susceptible to cause a deposition of lipids on the walls of the aorta and thus, increasing its weight while narrowing its lumen[19]. This narrowing leads to an increase in the peripheral resistance of the aorta and will cause an increase in blood pressure at the level of the left ventricle which hypertrophies to compensate the cardiac rate during effort downstream[20].

Concerning triglycerides and cholesterol, their increase could be due to an inhibition of the enzyme HMG- CoA (3-hydroxy-3-methyl glutaryl- coA) activity and an increase in VLDL, LDL]. . HMG- CoA synthezises *in vivo* cholesterol[21]. A reduction in the level of HDL cholesterol could be due to the stimulation of Lecithine cholesterol acyl transferase (LCAT) which induces the esterification and sequestration of cholesterol in HDL molecules, or by the stimulation cholesterol ester transfer protein (CETP) which assures the transfer of cholesterol esters from HDL to chylomicrons, VLDL and LDL, thereby increasing the plasma level of the latter[22, 23].

Diets rich in fatty acids increase the atherogenic index by inducing oxidative stress (enzymatic and non-enzymatic) in rats, thus, increases the oxidation of low density lipoprotein (LDL) which plays a key role in the genesis of atherosclerosis. The increase in the level of triglycerides, LDL cholesterol, atherogenic index and the decrease in the level of HDL cholesterol characterise what is called atherogenic dyslipidemia[7, 24].

The rise in the arterial blood pressure and the increase in the weight of the heart, the aorta and the left ventricular could be the resultant of the reduction in the activity of NO induced by the different oils administered. The NO produced by the endothelium of the aorta and other arteries possesses vaso-dilating and anti-mitogenic effects on cardiovascular muscles through inhibition of growth and proliferation of its cells[25, 26]. A reduction in the production of NO by the endothelium is thus susceptible to cause an increase in the arterial blood pressure and hypertrophy of the concerned organs[27, 28]. Moreover, high cholesterol diet induces endothelial dysfunction, atherosclerosis and increase oxidative stress by increasing the expression of sensitive-oxidation genes, such as Elk-1 and p-CREB[29]. This finding was contradictory to the other studies that had reported a significant reduction in the BP[30]. This may be due to the different types of animals and palm oil used in the experiments. For the present study, FPO was fed to normal rats instead of spontaneously hypertensive rats or Dahl-Salt sensitive rats. Secondly, the animals in previous studies were fed with industrialised palm oil while we used locally made palm oil. Conclusively, a high intake of locally made fresh or refined palm oil could probably cause retardation in growth, atherogenic dyslipidemia and arterial hypertension even though the effects are lesser with RPO as compared to FPO.

Table 1: Characteristics of diet intake in case and control groups

Types of diet	Standard diet (g)	High fat diet (g)		
Ingredient	Standard diet	10%	15%	20%
Corn flour	668.70	600.84	562.15	532.15
Soya beans flour	205.80	185.22	180.10	165.10
Bone flour	10.30	10.30	10.30	10.30
Kitchen salt	10.30	10. 30	10.30	10.30
Fresh or refined palm oil	1.10	100	150	200
Fish flour	102.70	92.13	84.95	79.95
Vitamin	1.10	1.10	1.10	1.10

Table 2: Effect of fresh and refined palm oil at the dose of 10% on the lipid profile

Time (weeks)	4			8			12		
Groups	Control	FPO	RPO	control	FPO	RPO	control	FPO	RPO
TC (mg/dL)	68.40± 2.72	88.78± 5.39^{a}	82.42± 5.80	58.51± 2.84	83.52± 4.17^{c}	75.27± 3.24^{a}	52.30± 2.94	68.59± 2.76^{b}	63.50± 3.19
TAG (mg/dL)	17.92± 1.17	26.58± 1.47^{c}	24.56± 0.95^{b}	26.05± 2.73	56.75± 3.20^{c}	53.79± 2.52^{c}	37.74± 2.08	48.52± 1.78^{b}	45.14± 0.81^{a}
HDL (mg/dL)	23.23± 1.45	16.97± 0.64^{b}	18.74± 0.55^{a}	17.57± 0.18	13.23± 0.87^{c}	15.28± 0.55^{a}	16.82± 0.78	12.22± 0.78^{b}	13.31± 0.97^{a}
LDL (mg/dL)	41.17± 2.91	69.75± 5.38^{b}	56.79± 5.10	31.63± 2.75	59.18± 4.35^{c}	48.33± 3.44^{a}	24.75± 2.37	46.81± 2.61^{c}	41.16± 3.40^{b}
AI	1.780± 0.268	4.082± 0.324^{c}	3.033± 0.186^{a}	1.780± 0.268	4.900± 0.469^{c}	3.296± 0.259^{a}	1.710± 0.34	3.897± 0.3489^{b}	3.192± 0.4419^{a}

a=p<0,05 ; b=p<0,01 et c=p<0,001 significant differences versus control. All values are expressed as means ± SEM, n= 7. FPO, fresh palm oil; RPO, refined palm oil; TC, total cholesterol; TAG, triglycerides; HDL, high density lipoprotein; LDL, low density lipoprotein; AI, atherogenic index

Table 3: Effect of fresh and refined palm oil at the dose of 15% on the lipid profile

Time (Weeks)	4			8			12		
Groups	**contr**	**FPO**	**RPO**	**control**	**FPO**	**RPO**	**control**	**FPO**	**RPO**
TC (mg/dL)	68.4± 2.72	90.9± 3.51 ^{c}	84.02± 3.49 ^{b}	61.41± 5.09	93.93± 2.79 ^{c}	84.41± 2.61 ^{b}	52.30± 2.94	65.70± 1.76^{b}	60.59± 2.05
TAG (mg/dL)	17.9± 1.17	40.2± 2.98 ^{c}	33.50± 2.33 ^{b}	22.05± 1.56	43.57± 2.26 ^{c}	38.62± 2.03 ^{c}	37.74± 2.08	57.20± 1.12^{c}	50.31± 1.96^{c}
HDL (mg/dL)	22.5± 1.39	14.0± 1.25 ^{b}	14.17± 1.22 ^{b}	17.57± 0.18	15.22± 0.65 ^{b}	15.30± 0.26 ^{b}	16.82± 0.78	12.98± 0.41^{a}	13.28± 0.99^{a}
LDL (mg/dL)	42.2± 2.16	69.8± 4.22 ^{c}	63.59± 3.23 ^{c}	42.45± 3.69	70.08± 2.27 ^{c}	61.42± 2.41 ^{c}	26.08± 2.59	41.64± 1.44^{c}	38.44± 1.50^{b}
AI	1.90± 0.139	5.92± 0.576 ^{c}	4.390± 0.335^{c}	1.909± 0.139	4.680± 0.158 ^{c}	4.029± 0.121 ^{c}	1.909± 0.14	3.304± 0.15^{c}	3.00± 0.24^{b}

a=p<0,05 ; b=p<0,01 et c=p<0,001 significant differences versus control.
All values are expressed as means ± SEM, n= 8
FPO, fresh palm oil; RPO, refined palm oil; TC, total cholesterol; TAG, triglycerides; HDL, high density lipoprotein; LDL, low density lipoprotein; AI, atherogenic index

Table 4: Effect of the consumption of fresh and refined palm oil at the dose of 20% on the lipid profile

Time (weeks)	4			8			12		
Groups	**control**	**FPO**	**RPO**	**control**	**FPO**	**RPO**	**control**	**FPO**	**RPO**
TC (mg/dL)	68.40± 2.72	99.90± 9.97 ^{a}	97.15± 7.88 ^{a}	61.50± 5.84	96.71± 5.68 ^{a}	81.35± 6.15	52.30± 2.46	83.86± 4.36^{c}	73.77± 4.49^{b}
TAG (mg/dL)	17.92± 1.17	59.17± 3.56 ^{c}	53.76± 3.14 ^{c}	26.05± 2.73	64.88± 3.60 ^{c}	44.67± 1.86 ^{b}	37.74± 2.08	63.51± 0.87^{c}	58.40± 2.61^{c}
HDL (mg/dL)	22.51± 1.39	3.100± 1.719 ^{c}	4.533± 1.804 ^{c}	17.93± 0.39	5.936± 0.594 ^{c}	10.37± 1.18 ^{c}	16.82± 0.78	12.28± 0.22^{c}	13.45± 0.924^{a}
LDL (mg/dL)	40.77± 1.94	92.41± 6.55 ^{c}	82.14± 7.13 ^{c}	38.76± 3.00	77.50± 6.09 ^{c}	67.52± 6.21 ^{b}	26.25± 1.80	58.70± 4.17^{c}	54.67± 3.19^{c}
AI	1.710± 0.272	3.897± 0.285 ^{c}	3.192± 0.361 ^{a}	1.710± 0.272	3.304± 0.119 ^{c}	3.008± 0.183^{b}	1.710± 0.27	4.716± 0.32^{c}	3.718± 0.48^{b}

a=p<0.05 ; b=p<0.01 and c=p<0.001 significant differences versus control.
All values are expressed as means ± SEM, n= 8
FPO, fresh palm oil; RPO, refined palm oil; TC, total cholesterol; TAG, triglycerides; HDL, high density lipoprotein; LDL, low density lipoprotein; AI, atherogenic index

Table 5: Effect of the consumption of fresh and refined palm oil at the dose of 10%, 15% and 20% on some target organs and blood pressure, after 3 months

% of oil	10%			15%			20%		
Groups	**control**	**FPO**	**RPO**	**control**	**FPO**	**RPO**	**control**	**FPO**	**RPO**
Left ventricular index	627.9± 9.3	679.9± 19.1 ^{a}	685.1± 11.3 ^{b}	627.9± 9.3	704.± 21 ^{a}	697.± 11.2 ^{a}	627.9± 9.3	710.4± 23.2 ^{a}	708.9± 13.5 ^{a}
Liver (mg)	3.227± 0.368	3.301± 0.113	3.424± 0.128	3.227± 0.368	3.653±0.159	3.729±0.075	3.227±0.368	4.357± 0.251^{a}	4.748± 0.293^{b}
Aorta (mg)	23.5± 1.3	30.9± 2.1 ^{b}	38.7± 0.4 ^{c}	23.5± 1.3	32.5± 2.3 ^{a}	36.16± 2.7 ^{b}	23.5± 1.3	30.76± 2.0 ^{a}	33.66± 1.0 ^{b}
MBP (mmHg)	88.33 ± 2.36	153.3± 5.7 ^{c}	168.8± 10.7 ^{c}	88.33± 2.36	130.0± 5.6 ^{c}	158.8± 2.5 ^{c}	88.33 ± 2.36	127.5 ± 3.4 ^{c}	135.1± 5.8 ^{c}

a=p<0.05 ; b=p<0.01 and c=p<0.001 significant differences versus control.
All values are expressed as means ± SEM, n= 8
FPO, fresh palm oil; RPO, refined palm oil; MBP, mean blood pressure

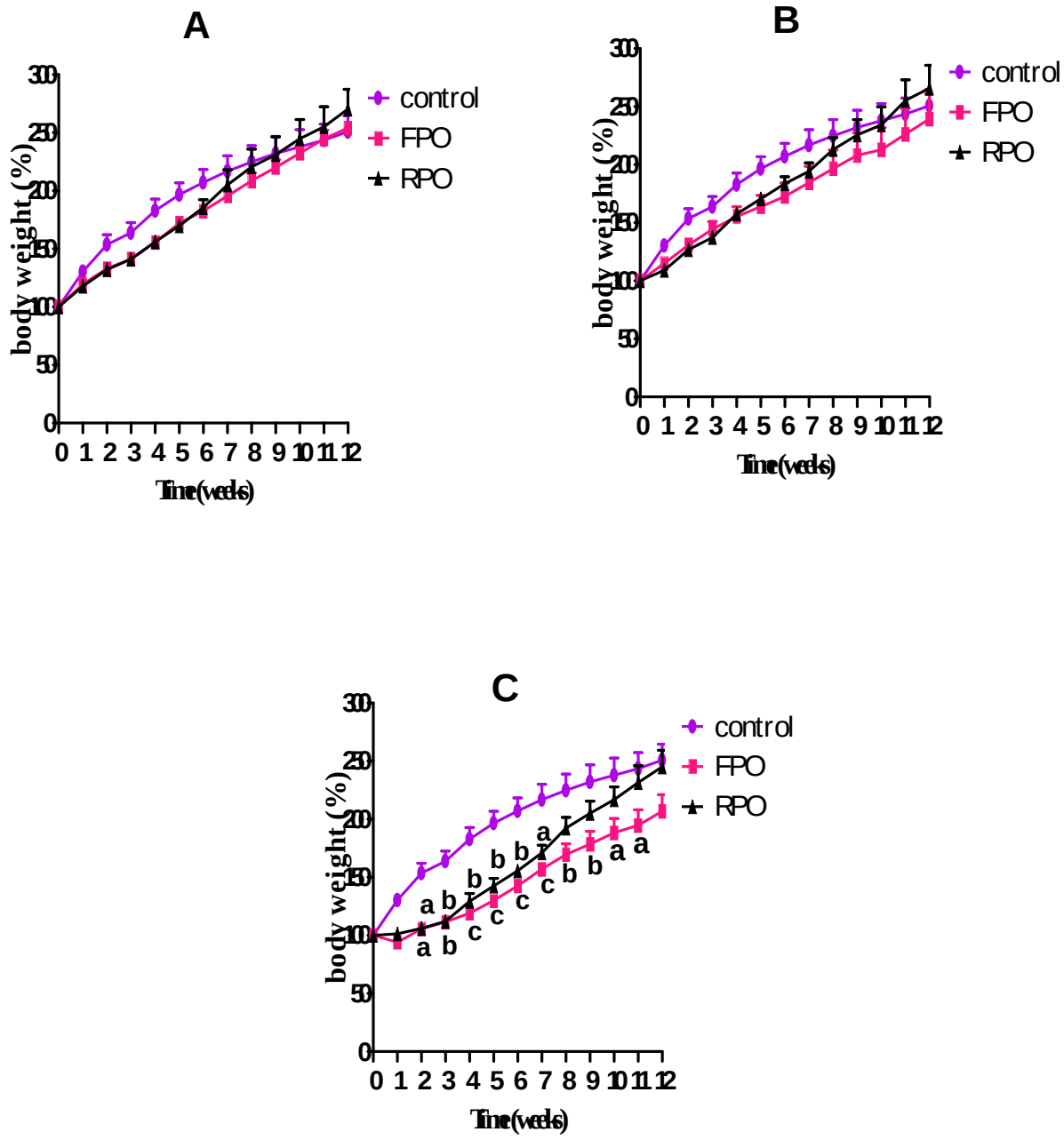

Fig. 1: Variation of body weights against time, with a ration containing 10% (A), 15% (B) and 20% (C) FPO or RPO. a=p<0.05; b=p<0.01 and c=p<0.001 significant differences as compared to the control. Each value represents the mean± SEM: n=8

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