Effects Of Sodium Acetate Dietary Supplementation On The Serum Lipid Profile Of Gallus Domesticus

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Abstract: The effect of sodium acetate concentration on some blood serum lipid profiles was evaluated. The aim was to determine the changes in serum lipid profile levels in broiler birds receiving sodium acetate treatment. Sixty five one-month-old broiler chicks, Gallus domesticus were randomly assigned to five dietary treatments supplemented with sodium acetate (3 replicates of 5 birds per treatment replicate, besides the control). The experimental birds were fed with commercially broiler starter (for 2 weeks) and broiler finisher (remaining experimental period). The blood serum samples were collected from the birds' branchial wing vein, and were examined enzymatically using the standard procedure for estimation at weekly intervals from the zero day. Results showed significantly reduced (p<0.05) total cholesterol, low-density lipoproteins and total lipids when compared with the control values from apparently all treatment groups. There was an overall significant linear increase (p<0.05) in the concentrations of high-density lipoproteins when compared with the control value. Although there was an overall decrease in the concentrations of triglycerides and very low-density lipoproteins, there was a part decrease and increase observed within treatments. The results suggest that sodium acetate significantly (p < 0.05) reduces serum cholesterol, low-density lipoproteins, and total lipids levels but increased high-density lipoproteins linearly. Triglycerides and very low-density lipoproteins levels reduces to a level with time with a subsequent rise in level. There is therefore positive commendation to the effect seen by the sodium acetate concentration on the serum lipid parameters examined. In conclusion, it is conceivable therefore that the present study explained a satisfactory importance of sodium acetate in public health as biomarker against dyslipidemia, hypercholesterolemia, and atherosclerosis.

Keywords: Sodium acetate, broilers, cholesterol, lipoproteins, triglycerides, lipids

I. INTRODUCTION

Chicken meat is healthier than other meat sources for human consumption because of its low cholesterol and fat content (Ponte *et al.*, 2004). The quality and quantity of lipids and their fatty acid composition in meat are influenced by internal (age, gender, genotype and castration) and external (temperature, feeding) factors (Masek *et al.*, 2013). The efficiency of fatty acid conversion in liver, which varies according to the age of the animal (Bourre *et al.*, 1990), could

modify the differential lipid deposition. In recent years, much prominence has been given to the association of abnormal levels or values of lipid profile with atherosclerosis and ischemic heart disease (Fox, 2002; Bhatnagar et al., 2008). Vertebrates synthesize sufficient amount of cholesterol unlike other animals. At very high level of cholesterol synthesis, hypercholesterolemia occurs in the blood. This is a metabolic derangement that causes many diseases. notably cardiovascular diseases. Longstanding elevation of serum cholesterol can lead to atherosclerosis (Bhatnagar et al., 2008). Helpful measurement makes it possible to determine if the balance between the high-density lipoprotein (good cholesterol) and low-density lipoprotein (bad cholesterol) is within acceptable limits. While the presence of good cholesterol is beneficial to maintaining organ health and energy provision of body, the presence of bad cholesterol can lead to blockages that can lead to problems with the heart and lungs. Cholesterol and triglyceride, produced by the liver are combined with other apoproteins and secreted into the blood as very low-density lipoproteins (VLDL) which delivers triglycerides to different organs. Sodium acetate is the sodium salt of acetic acid. Acetates are normal components of the diet of humans and animals and are produced in molar quantities in the gastrointestinal tract. They are fully metabolized and so do not pose a risk to the environment. Acetic and its salts have the potential to act as preservatives in feeding stuffs and water for drinking (European Food Safety Authority, 2012). There is dearth of published work on effect of sodium acetate administration on blood lipid profile of vertebrates. Therefore, the aim of this investigation was to study the lipid metabolism of chicken broiler fed with sodium acetate dietary supplement. over 28 days advancement of age and fattening period.

II. MATERIALS AND METHODS

A. STUDY BIRDS

A total of 65 broiler one-month-old chicks with mean weight $640\pm30g$ were collected from a local commercial flock. They were caged in 5 big partitions containing 3 cages each. Birds were reared in accordance with University of Nigeria, Nsukka guidelines for animal experimentation. Each partition housed 15 birds with 5 birds per cage (except for the control groups that had 5 birds). Birds were fed with commercial broiler starter (1kg/bird) for about 2 weeks (1 week of acclimatization and 1 week of initial treatment). Subsequent experimental weeks, they were fed with commercial broiler finisher (1kg/bird), till end of the experiment. Feed and water were available *ad libitum*, and a 12-h natural light was allowed.

B. STUDY DESIGN

The methodology adopted for the study was completely randomized treatment design. The broilers were assigned to 5 treatment groups with 3 replicates (except treatment 1, control), such that each replicate housed 5 birds. The birds were treated with 5 different concentrations of sodium acetate as follows 0, 2, 4, 6, and 8g/kg/day of feed for treatment

groups T1, T2, T3, T4, and T5 respectively. The experimentation period lasted for 4 weeks study duration.

C. STUDY SAMPLES

Specimen bottles were used to collect 2 ml of blood samples from individual replicates after 12-14 hours fasting at the beginning (0 day), and subsequently at weekly intervals of the feeding period. Blood was collected from the branchial wing vein using sterilized syringes. Samples in the specimen bottles were allowed to clot and then centrifuged at 3000 r.p.m (revolution per minute) for 5 min at room temperature (28-31 °C) to obtain sera samples of the blood for enzymatic determinations. The parameters of TCHOL, TRIG, HDL, and TL composition in the serum were determined enzymatically using commercially available reagent kits (QCA for TCHOL, and RANDOX for TRIG, HDL, and TL). The parameters of LDL and VLDL were estimated using the Friedwald equation (Friedwald *et al.*, 1972).

D. STUDY STATISTICS

Data collected were entered into Microsoft Excel and analyzed with SPSS version 16 statistical software using One-Way Analysis of Variance (ANOVA) for overall significance of mean variations. Treatment intake and blood samples measures were compared using multiple comparison of Least Significant Difference (LSD). Results were expressed as mean ± standard error mean (±SEM) mg/dl.

III. RESULTS

A. TOTAL CHOLESTEROL

Table 1 shows the mean variations of the serum total cholesterol in broiler chicken, following administration of sodium acetate dietary supplementation at varying concentrations. The control group (T1) showed an increased serum total cholesterol concentration at weekly intervals from 139.46 \pm 3.95 (Wk 0) – 161.94 \pm 2.58 (Wk 4), whereas for T2, T3, T4, and T5, it was observed that there were linear reductions in the mean values of the chicken serum total cholesterol with advancement of age.

The interactions among treatment groups at weekly intervals were estimated and the following observed. At weeks 1, 2, 3, and 4, there was a linear reduction in the serum total cholesterol concentrations of the different treatments, with T1 and T5 having the highest and lowest values respectively for the weekly intervals. At week 1, it was observed that both control and T2 groups were significantly higher (p<0.05) than the rest of the treatment groups. Furthermore, it was observed that significant similarity exist for T3 and T4, which were both higher than T5. At weeks 2 and 3, the control group was significantly higher (p<0.05) than the rest of the treatment groups. Also, whereas T2 was significantly higher (p<0.05)than T4 and T5, it was not for T3, as point of intersection of similarity exist between T2 and T3. Furthermore, it was observed that significant similarity exist for T4 and T5. At week 4, it was observed that the control group was

significantly higher (p < 0.05) than the rest of the treatment groups, which showed no significant difference ($p \ge 0.05$) to one another, or put differently, are all significantly similar.

B. TRIGLYCERIDES

Table 2 shows the mean variations of the serum triglycerides in broiler chicken, following administration of sodium acetate dietary supplementation at varying concentrations. The control group (T1) showed decreased serum triglycerides concentration at weekly intervals from 136.31 \pm 3.63 (Wk 0) – 60.68 \pm 1.09 (Wk 4), whereas for T2, T3, T4, and T5, it was observed that there exists a non-linear reduction (decrease at the 1st two weeks, and increase at 2nd two weeks of the experimental period) in the mean values of the chicken serum triglycerides with advancement of age.

The interactions among treatment groups at weekly intervals were estimated and the following observed. At weeks 1 and 2, there was a linear reduction in the serum triglycerides concentrations of the different treatments, with T1 and T5 having the highest and lowest values respectively. However, at weeks 3 and 4, there was a linear increase in the serum triglycerides concentrations of the different treatments with T1 and T5 having the lowest and highest values respectively. At week 1, it was observed that the control group was significantly higher (p<0.05) than the rest of the treatment groups. Also, whereas T2 was significantly higher (p<0.05) than T4 and T5, it was not for T3, as point of intersection of similarity exist between T2 and T3. Furthermore, it was observed that significant similarity exist for T4 and T5. At week 2, the control group was significantly higher (p<0.05) than T3, T4, and T5, but not for T2, as point of intersection of similarity exist between control and T2. The same is true at week 3 but with significantly less (p<0.05) value of control than T3, T4, and T5, but not for T2. At week 4, it was observed that the control group was significantly less (p<0.05) than the rest of the treatment groups, which showed no significant difference (p≥0.05) to one another, or put differently, are all significantly similar.

C. HIGH-DENSITY LIPOPROTEINS

Table 3 shows the mean variations of the serum highdensity lipoproteins in broiler chicken, following administration of sodium acetate dietary supplementation at varying concentrations. The control group (T1) showed decreased serum high-density lipoproteins concentration at weekly intervals from 54.26 ± 2.06 (Wk 0) – 29.25 ± 1.11 (Wk 4), whereas for T2, T3, T4, and T5, it was observed that there were linear increase in the mean values of the chicken serum high-density lipoproteins with advancement of age.

The interactions among treatment groups at weekly intervals were estimated and the following observed. At weeks 1, 2, 3, and 4, there was a linear increase in the serum high-density lipoproteins concentrations of the different treatments, with T1 and T5 having the lowest and highest values respectively for the weekly intervals. At week 1, it was observed that both control group was significantly less (p<0.05) than the rest of the treatment groups. Furthermore, it was observed that significant similarity exist for T2, T3 and

T4, which were less than T5. At weeks 2, 3, and 4, the control group was significantly less (p<0.05) than the rest of the treatment groups. Furthermore, T2 and T3 showed significant similarity to each other in the two weeks, which were significantly less (p<0.05) than T4 and T5.

D. LOW-DENSITY LIPOPROTEINS

Table 4 shows the mean variations of the serum lowdensity lipoproteins in broiler chicken, following administration of sodium acetate dietary supplementation at varying concentrations. The control group (T1) showed increased serum-low density lipoproteins concentration at weekly intervals from 57.94 \pm 5.59 (Wk 0) – 120.48 \pm 3.50 (Wk 4), whereas for T2, T3, T4, and T5, it was observed that there were linear decrease in the mean values of the chicken serum low-density lipoproteins with advancement of age.

The interactions among treatment groups at weekly intervals were estimated and the following observed. At weeks 1, 2, 3, and 4, there was a linear reduction in the serum-low density lipoproteins concentrations of the different treatments, with T1 and T5 having the highest and lowest values respectively for the weekly intervals. Again at weeks 1, 2, 3, and 4, it was observed that the control group was significantly higher (p<0.05) than the rest of the treatment groups. Furthermore, T2 and T3 showed significant similarity to each other in the two weeks, which were significantly higher (p<0.05) than T4 and T5.

E. VERY LOW-DENSITY LIPOPROTEINS

Table 5 shows the mean variations of the serum very lowdensity lipoproteins in broiler chicken, following administration of sodium acetate dietary supplementation at varying concentrations. The control group (T1) showed decreased serum very low-density lipoproteins concentration at weekly intervals from 27.26 \pm 0.72 (Wk 0) – 12.14 \pm 0.21 (Wk 4), whereas for T2, T3, T4, and T5, it was observed that there exists a non-linear reduction (decrease at the 1st two weeks, and increase at 2nd two weeks of the experimental period) in the mean values of the chicken serum very lowdensity lipoproteins with advancement of age.

The interactions among treatment groups at weekly intervals were estimated and the following observed. At weeks 1 and 2, there was a linear reduction in the serum very lowdensity lipoproteins concentrations of the different treatments, with T1 and T5 having the highest and lowest values respectively. However, at weeks 3 and 4, there was a linear increase in the serum very low-density lipoproteins concentrations of the different treatments with T1 and T5 having the lowest and highest values respectively. At weeks 1 and 4, it was observed that the control group was significantly higher (p<0.05) than the rest of the treatment groups, but in weeks 2 and 3, the control was not significantly higher (p<0.05) than T2, as point of intersection of similarity exist between control and T2. Furthermore, it was observed that significant similarity exist for T2, T3, T4, and T5, in weeks 3 and 4.

F. TOTAL LIPIDS

Table 6 shows the mean variations of the serum total lipids in broiler chicken, following administration of sodium acetate dietary supplementation at varying concentrations. At week 4 (end of experimental period), there was decreased serum total lipids concentration in relation to treatment groups from 421.57 ± 9.81 (Control), having the highest concentration to 333.33 ± 9.80 (T5), having the lowest concentration.

The interactions among treatment groups at the week 4, was estimated and the following observed. It was observed that control group was significantly higher (p<0.05) than T3, T4, and T5, but was not for T2, as point of intersection of similarity exist between control and T2. As observed, T5 had the least values with a significantly less (p<0.05) mean than the rest of the treatment groups.

Weeks	Treatment groups					
	T1 (0g/kg)	T2 (2g/kg)	T3 (4g/kg)	T4 (6g/kg)	T5 (8g/kg)	
0	139.46±3.95	141.72±2.85	142.65±3.05	138.88±3.38	137.89±1.75	
1	138.33±0.40	138.60±0.33	132.30±1.27	131.07±0.66	126.10±0.93	
2	141.94±2.39	134.00±2.38	130.42±1.10	127.00±0.75	125.28±0.48	
3	150.00±1.28	132.05±2.20	127.83±1.85	126.35±1.61	125.02±1.78	
4	161.94±2.58	139.98±10.98	127.47±0.44	126.21±1.65	125.21±1.27	

All values expressed as mean \pm standard error mean (\pm SEM) mg/dl.

Table 1: Effects of sodium acetate treatments on serum total cholesterol in broiler chicken

T1 (0g/kg)	T2 (2g/kg)	T3 (4g/kg)	T4 (6g/kg)	T5 (8g/kg)
136.31±3.63	134.56±2.85	136.00±2.52	134.58±2.48	135.68±3.21
113.45±6.62	82.16±8.26	68.09±3.32	54.30±5.13	52.37±0.97
88.94±5.23	76.79±8.28	62.36±3.45	47.07±5.34	40.38±0.84
68.64±2.33	76.90±4.79	79.24±3.79	82.72±1.89	83.14±2.18
60.68±1.07	92.23±5.95	101.37±1.05	101.42±0.29	101.62±3.25
	136.31±3.63 113.45±6.62 88.94±5.23 68.64±2.33	T1 (0g/kg) T2 (2g/kg) 136.31±3.63 134.56±2.85 113.45±6.62 82.16±8.26 88.94±5.23 76.79±8.28 68.64±2.33 76.90±4.79	136.31±3.63 134.56±2.85 136.00±2.52 113.45±6.62 82.16±8.26 68.09±3.32 88.94±5.23 76.79±8.28 62.36±3.45 68.64±2.33 76.90±4.79 79.24±3.79	T1 (0g/kg) T2 (2g/kg) T3 (4g/kg) T4 (6g/kg) 136.31±3.63 134.56±2.85 136.00±2.52 134.58±2.48 113.45±6.62 82.16±8.26 68.09±3.32 54.30±5.13 88.94±5.23 76.79±8.28 62.36±3.45 47.07±5.34 68.64±2.33 76.99±4.79 79.24±3.79 82.72±1.89

All values expressed as mean \pm standard error mean (\pm SEM) mg/dl.

 Table 2: Effects of sodium acetate treatments on serum

 triglycerides in broiler chicken

Weeks					
	T1 (0g/kg)	T2 (2g/kg)	T3 (4g/kg)	T4 (6g/kg)	T5 (8g/kg)
0	54.26±2.06	58.21±2.59	52.81±1.37	58.80±2.42	58.26±1.77
1	41.23±3.26	60.51±2.48	62.55±1.63	68.36±3.25	98.08±2.71
2	37.58±0.63	61.26±1.40	64.50±2.72	70.40±4.18	101.21±1.34
3	33.98±0.65	63.44±2.97	65.23±2.17	73.06±3.07	103.15±2.27
4	29.25±1.11	64.10±1.14	65.92±0.91	74.79±2.89	104.14±1.76

All values expressed as mean \pm standard error mean (\pm SEM) mg/dl.

 Table 3: Effects of sodium acetate treatments on serum highdensity lipoproteins in broiler chicken

Weeks					
	T1 (0g/kg)	T2 (2g/kg)	T3 (4g/kg)	T4 (6g/kg)	T5 (8g/kg
0	57.94±5.59	56.59±4.77	62.52±3.92	53.22±5.47	52.49±2.75
1	74.41±4.64	61.65±2.42	56.13±1.44	51.88 ± 2.11	17.54±3.5
2	86.27±1.88	57.38±1.03	53.44±2.73	47.36±4.57	16.00±1.0
3	102.32 ± 2.22	53.23±2.75	46.75±2.82	36.74±1.35	5.24±1.2
4	120.48±3.50	45.79±2.06	40.22±2.65	30.14±1.98	0.27±0.0

All values expressed as mean \pm standard error mean (\pm SEM) mg/dl.

 Table 4: Effects of sodium acetate treatments on serum lowdensity lipoproteins in broiler chicken

Weeks					
	T1 (0g/kg)	T2 (2g/kg)	T3 (4g/kg)	T4 (6g/kg)	T5 (8g/kg)
0	27.26±0.72	26.91±0.57	27.32±0.50	26.86±0.49	27.74±0.64
1	22.69±1.33	16.44±1.65	13.62 ± 0.67	10.90 ± 0.99	10.47±0.20
2	17.79±1.05	15.30±1.66	12.47±0.69	9.23±1.15	8.08±0.17
3	13.73±0.47	15.38±0.96	15.85±0.76	16.55±0.38	16.63±0.44
4	12.14 ± 0.21	18.45±1.19	20.25±0.21	20.20±0.06	20.32±0.65

All values expressed as mean \pm standard error mean (\pm SEM) mg/dl.

Table 5: Effects of sodium acetate treatments on serum very low-density lipoproteins in broiler chicken

Weeks					
	T1 (0g/kg)	T2 (2g/kg)	T3 (4g/kg)	T4 (6g/kg)	T5 (8g/kg)
At					
4	421.57±9.81	392.15±9.80	372.55±9.80	362.74±9.80	333.33±9.80

All values expressed as mean \pm standard error mean (\pm SEM) mg/dl.

 Table 6: Effects of sodium acetate treatments on serum total
 lipids in broiler chicken

IV. DISCUSSION

The control group showed elevated total cholesterol, lowdensity lipoproteins, and total lipids, but reduced triglyceride, high-density lipoproteins, and very low-density lipoproteins, as one would expect following dietary feeding of animal fat diet with advancement of age, with promising potency of deleterious effects. It is against this backdrop that the findings of the present study on the beneficial supplementation of sodium acetate are of great interest.

A. TOTAL CHOLESTEROL

There was a reduction in the total cholesterol content of the serum which was more significant with increased concentration and accumulation of sodium acetate as depicted in Table 1. This was easily inferred owing to the fact that *ab* initio (Week 0), there was no level of significant difference $(p \ge 0.05)$ among the treatment groups, but gradually decreased linearly with increasing sodium acetate concentration per treatment per duration. Although careful observations of the treatment groups (T2-5) interactions using the least significant difference revealed not much significant differences existing among them, which explains the accumulation of sodium acetate on the serum over the experimental period of study. This work agrees with works done on two higher vertebrates, dogs and man by Francisco et al. (1960) and Port et al. (1978) respectively, that sodium acetate never increased the serum total cholesterol level. This is of health interest since hypercholesterolemia holds a key role in the development and progression of atherosclerosis and is a causative factor for coronary artery disease (Toutouzas *et al.*, 2010). Cardiovascular diseases are the leading cause of morbidity and mortality in industrialized countries (McGovem et al., 1996), wherein atherosclerosis is the underlying cause of most cardiovascular diseases.

B. TRIGLYCERIDES

On the account of the triglycerides, as shown in Table 2, the following deductions are explicit. There was a level of difference (p<0.05) noticed significant in serum concentrations of triglycerides at initial two weeks of sodium acetate administration, as there was a reduction as treatment concentrations increases linearly. Interestingly, within the 3rd week of sodium acetate administration, the serum concentrations of the triglycerides began to rise with increasing sodium acetate concentration in a linear manner. Though this was not significant ($p \ge 0.05$) initially, but gained a level of significance (p<0.05) at the 4th week of experimentation. The overall reduction of triglycerides in the birds' serum with increasing treatment concentrations over time accords with the findings of Port et al. (1978), as tested in human with sodium acetate having inhibitory potential to triglycerides upraise in blood serum. Hence, the serum level of triglyceride even though had an overall decrease when administered sodium acetate, the reduction was at its peak in the 2nd week of experimentation, but increased though not to the initial concentration in the remaining study duration.

C. HIGH-DENSITY LIPOPROTEINS

The level of high-density lipoproteins otherwise referred to as the good cholesterol is of interest, due to the fact that it has a good impact on human health. Table 3 reveals the relative interactions that transpired within the normal serum high-density lipoproteins with varied concentrations of sodium acetate over time. The deductions are that there was significant difference (p < 0.05) within the treatment groups per time, which is seen in the gradual increase of the serum highdensity lipoproteins, and is directly proportional to increased sodium acetate administration. Also, within treatments, at one point in time, the different treatment groups may be insignificant ($p \ge 0.05$) to themselves in relation to mean variations, as obtained using least significant difference, which could be explained as a result of probably due to the minute initial administration and the overtime accumulation of the sodium acetate concentrations, but it was still observed that the most accumulated serum still showed a significant difference (p<0.05) to the other groups. The high level of high-density lipoproteins observed in the present study suggests a beneficial association with a decreased risk of cardiovascular disease (Lee and Choudhury, 2010). Highdensity lipoproteins serve an anti-atherogenic function because of its ability to mediate reverse cholesterol transport (RCT), which is a major protective system against atherosclerosis (Assmann and Nofer, 2003; Rothblat and Phillips, 2010). Modulation of major macrophage mediators in RCT has been considered as promising strategies for the development of drugs aimed at the prevention of atherosclerosis (Van der Velde, 2010; Lund-Katz and Phillips, 2010; Meurs et al., 2010). High-density lipoproteins can remove cholesterol from the periphery, allowing it to be cleared by the liver and then excreted into the bile (Ragbir and Farmer, 2010).

D. LOW-DENSITY LIPOPROTEINS

Low-density lipoproteins are known as the bad cholesterol, and are of economic importance to human health as it leads to health disorders especially of the cardiovascular diseases. As depicted in Table 4, the serum concentration of low-density lipoproteins rapidly reduced with increasing concentration of sodium acetate. This simply expresses an immense inverse proportion of low-density lipoproteins to sodium acetate concentration. Interestingly, the overall reduction of the total cholesterol (Table 1) was a consequence of the low-density lipoproteins reductions, which fortunate enough, is of great health importance to humans as lowering of low-density lipoprotein and very low-density lipoprotein levels leads to a reduction in cardiovascular morbidity and mortality (Paras et al., 2010). Furthermore, existence of level of significant difference (p<0.05) seen in the treatment groups at different concentrations of the sodium acetate, and nonsignificant difference ($p \ge 0.05$) among some treatment groups could be explained in the light of the physiological and metabolic processes following initial administration and overtime accumulation, as most probable.

E. VERY LOW-DENSITY LIPOPROTEINS

The serum concentration of the very low-density lipoproteins decrease linearly with increase sodium acetate concentrations over the experimental period of initial two weeks, showing a level of significant difference (p<0.05) in experimental groups. But within the 3rd week of administration of sodium acetate, the serum concentrations of very lowdensity lipoproteins began to increase with increasing sodium acetate concentration in a linear manner. Though, this was not significant ($p \ge 0.05$) initially, but gained a level of significance (p<0.05) at the 4th week. Therefore, we can infer that though there was an overall decrease in the serum level of very lowdensity lipoproteins when administered sodium acetate as can be read off from Table 5, the reduction was at its peak in the 2^{nd} week of experimentation, but increased though not to the initial concentrations in the remaining period of experimentation. Physiological studies have been adapted to correlate some of the blood parameters with the degree of fatness in broiler chickens. Whithead and Griffin (1984) indicated that plasma very low-density lipoprotein was a useful parameter to infer the degree of fatness in chickens, and that decreasing plasma VLDL level by any means, causes decreasing abdominal fat in broiler chickens. Similar suggestion was reported by Grunder et al. (1987) in two strains of chickens. Hence, explaining the de-fattening potential of sodium acetate.

F. TOTAL LIPIDS

The concentrations of serum total lipids are noteworthy. It was needful to ascertain the level of sodium acetate effect on the overall lipids per treatment. Table 6, then depicts that as the serum samples were examined per treatment, there exist a level of significant difference (p<0.05) among treatments, which explained reduction in the total lipids of the serum. The relevance of this concentration reduction is predicated on the

fact that hyperlipidemia is a metabolic disorder defined by either elevated levels of plasma concentrations of low-density lipoprotein and triglycerides, or decreased levels of the atheroprotective lipid biomarker high-density lipoprotein (Paras *et al.*, 2010). More also, lipid accumulation leads to an inflammatory condition clinically causing occlusive vascular disease, myocardial infarction and stroke (Boehm and Nabel, 2003). It is helpful to posit that the total lipids consist of all fats and oils, and our earlier deductions and discussions on some of the lipid parameters like total cholesterol, triglycerides, low density lipoproteins, aside high density lipoproteins showed an overall decrease in concentrations, which explained the result we have in the total lipids.

V. CONCLUSION

The present study presented relative alterations in the concentrations of the serum lipid profile vis-à-vis sodium acetate administration at varying concentrations. This was seen in decrease (total cholesterol, low-density lipoproteins, and total lipids), increase (high-density lipoproteins), as well as partly decrease and increase (triglycerides and very lowdensity lipoproteins), of the serum concentrations. It is pertinent also to mention that in all these serum parameters examined that none showed any significant difference $(p \ge 0.05)$ to one another as per treatment groups at the week 0 (0 day). This explains that the initial concentrations of all the treatment groups were nearly same prior to sodium acetate administration. Again, it is of interest that within treatment groups there exist at one point in time non-significant difference $(p \ge 0.05)$ irrespective of the fact that there was sodium acetate administration. The explanation to this is most probable due to instances of minute initial administration which probably may not have elicited the needed biochemical change, and the overtime accumulation of the sodium acetate concentrations leading to physiological tolerance. Nevertheless, it was still evidently clear that T5, which is the most accumulated showed a significant difference (p<0.05) to the other experimental groups. To public health relevance, since cardiovascular diseases are the leading cause of morbidity and mortality in industrialized countries (McGovem et al., 1996), the results gotten from the experimentation explained a satisfactory importance of sodium acetate, not just which it helps in the prevention and control of dyslipidemia, but it reduces the risk of hypercholesterolemia which leads to cardiovascular diseases, prevents atherosclerosis which when severe and persistent leads to stenosis or occlusion of arteries (Fox, 2002; Bhatnagar et al., 2008).

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