Arch. Anim. Nutr., 1992, Vol. 42, pp. 263–272
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PRELIMINARY INVESTIGATION ON THE EFFECT OF DIETARY SUPPLEMENTAL BIOTIN AND PALM KERNEL OIL ON BLOOD, LIVER AND KIDNEY LIPIDS IN CHICKS

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(Received 26 April 1990)

A total of 480 day-old broiler chicks were used in two trials conducted to investigate the performance and lipid contents of blood, liver and kidneys of birds when fed varying levels of palm kernel oil (0% and 2%) and biotin (40, 80, 120, 160, 200 and 240 mcg/kg feed) in a 2 × 6 factorial experimental design. The results showed that blood, liver and kidney lipid concentrations were significantly affected by dietary biotin treatments. While total lipid, free fatty acid, triglyceride and cholesterol contents were negatively correlated with dietary biotin level, phospholipid concentrations were positively correlated. Biotin-deficient chicks had significantly higher total lipid, free fatty acid, triglyceride and cholesterol but lower phospholipid contents in their blood and the two organs. Supplementation of the diet with 2% palm kernel oil significantly elevated blood phospholipid concentration, but depressed the accumulation of the other lipid fractions in both organs and the blood of birds. Blood, liver and kidney cholesterol concentrations were not affected by 2% fat supplementation. Observation on the lipid parameters coupled with the results on feed utilisation appeared to suggest that a minimum of 120 mcg of the vitamin per kilograme of diet was required by broiler chicks for optimum performance.

KEY WORDS: Biotin: Broiler chicken: Palm kernel oil: Lipid

1. INTRODUCTION

The significance of normal metabolism of lipid among other nutrients in optimum animal performance cannot be over emphasized. Abnormally increased fatty acid synthetic rate, mobilisation of adipose fat and hydrolysis of plasma lipoprotein glycerolipids resulting in elevated plasma fatty acid level led to significantly higher synthesis and accumulation of liver triglyceride and phospholipids (Topping and Mayes, 1972; Ontko, 1972). Triglyceride and phospholipid synthesis in the isolated hepatocytes are also significantly affected by chain

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length and degree of unsaturation of the predominating fatty acid in oil (Sundler et al., 1974). Feeding of corn oil rich in linoleic acid to rat depressed activities of liver acetyl CoA carboxylase, malic enzyme, glucose-6-phosphate dehydrogenase, α-glycerophosphate acyltransferase and diacylglycerol acyltransferase levels with consequent reduction in triglyceride synthesis (Muto and Gibson, 1970; Iritani and Fukuda, 1980).

From the foregoing information, therefore, it appeared that triglyceride and phospholipid metabolism in the animal is significantly controlled by type of dietary oil; and since the use of palm kernal oil as feed ingredient for poultry ration is becoming popular in Nigeria, the need for investigating its influence on triglyceride and phospholipid metabolism is inevitable.

The involvement of biotin enzyme (acetyl CoA carboxylase) in the pathway of long-chain fatty acid synthesis (Bortz et al., 1963) as well as the role of biotin in the alteration of fatty acid patterns in adipose tissue, with a shift to unsaturated side (Roland and Edwards, 1971) reported seem to suggest a possible influence of biotin on metabolism of triglyceride and phospholipids in animals.

There have been many conflicting reports regarding effect on cholesterol metabolism of biotin (Gram and Okey, 1958; Barnes et al., 1959; Fletcher and Myant, 1960; Dakshinamurti and Desjardins, 1968) and nature of dietary fats (Alfin-Slater et al., 1954; Okey and Lyman, 1957; Klein, 1958; Reiser et al., 1963).

The research reported herein concern the performance of broiler chicks and lipid contents of blood, liver and kidney, and the information is intended to serve as prerequisites for further investigating the role of biotin and palm kernel oil in the metabolism of lipid in chicks.

2. MATERIAL AND METHODS

2.1. Trial 1

A total of two hundred and forty day-old commercial broiler chicks were randomly assigned to six treatment groups, the dietary treatment being 40, 80, 120, 160, 200 and 240 mcg biotin/kg feed. Each treatment group had two replicates consisting of 20 birds each. Experimental diets were prepared by formulating basal biotin-deficient diet without palm kernel oil supplementation (Table 1) as described by Oloyo and Ogunmodede (1989), and then supplemented with feed — grade biotin (ROVIMIX H-2, a ROCHE product) such that six graded levels of the vitamin were obtained. Chicks were raised in 12 floor pens, each of 4.2 m^2 floor area and contained dry wood shavings litter. Birds were maintained on the respective treatments for a period of 28 days during which feed and water were provided at all times and routine vaccinations administered. Body weights and feed intake were recorded weekly. Four replicate samples were randomly selected from the respective treatment groups at the end of the feeding trial for blood collection and subsequent slaughtering. 2 cm^3 of blood sample was taken from the wing veins of each bird into heparinized containers and kept for

estimation of total lipid by the method of Folch et al. (1957), free fatty acid by method of Pearson (1976) and cholesterol as described by Kim and Goldberg (1969). Triglyceride and phospholipid were determined in accordance with the procedures of Fletcher (1968) and Tietz (1976) respectively. Selected bird samples were slaughtered, the liver and kidneys were excised, drained of fluids with blotting paper and weighed. These organs were freeze-dried and kept for lipid analysis as in blood samples.

2.2. Trial 2

In this study, palm kernel oil was included in the biotin-deficient basal diet of the previous trial at 2% level such that both diets had similar metabolizable energy value and protein content (Table 1). Biotin-deficient diet with 2% oil inclusion therefore served as basal diet for this trial. Graded levels of biotin were added to this basal diet such that experimental diets had 40, 80, 120, 160, 200 and 240 mcg of the vitamin per kilogramme of feed. These rations were fed to duplicate groups of day-old broiler chicks with 20 birds per group (40 birds per treatment) for a period of 28 days. Experimental birds were reared in 12 floor pens as described in the first trial. Four replicate samples were removed from each of the treatment groups for analysis of blood, liver and kidney as described in trial 1.

Results obtained in the two trials were subjected to statistical analysis in accordance with the procedures of Steel and Torrie (1960). Significantly different treatment means were separated by the multiple range test of Duncan (1955).

Table 1 Composition of the basal biotin-deficient diets used in trials 1 and 2

	Trial 1 %	*Trial 2* %
Yellow maize	54.0	49.5
Palm kernel meal	18.0	18.0
Blood meal	10.0	10.5
Fish meal	2.5	2.5
Brewer's grain	12.2	12.2
Oyster shell	1.0	2.0
Bone meal	2.0	3.0
Vitamin/Mineral premix (UNIT-VIT 15)*	0.1	0.1
Salt	0.2	0.2
Palm kernel oil	0.0	2.0
Total	100.0	100.0
Calculated analysis		
Crude protein (%)	21.3	21.2
Metabolizable energy, ME (MJ/kg)	11.48	11.52
Fat (%)	3.5	5.5
Linoleic acid (%)	1.40	1.33
Biotin (mcg/kg)	38.9	36.2
Crude fibre (%)	5.61	5.52

* UNIT-VIT 15 supplied the following vitamins and mineral elements per kg of feed: Vitamin A 8000 i.u; Vitamin D_3 1500 i.u.; Vitamin E 3 i.u; Menadione sodium bisulphite (Vitamin K) 1.5 mg; Vitamin B_2 2.5 mg; Calcium d-pantothenate 3 mg; Nicotinic acid 8 mg; Vitamin B_6 0.3 mg; Vitamin B_{12} 0.008 mg; Iron 15 mg; Manganese 25 mg; Copper 2.5 mg; Zinc 10 mg; Iodine 0.3 mg.

3. RESULTS AND DISCUSSION

In order to study the effect of biotin on lipid contents of blood, liver and kidney in chicks, it is important to start from deficiency to more than sufficient level of the vitamin. Consequently, basal biotin-deficient diets (Table 1) that were used by Oloyo and Ogunmodede (1989) to reproduce fatty liver and kidney syndrome (FLKS), a deficiency symptom of biotin in chicken, constituted basal diets that were supplemented with the vitamin in this study. The highest dietary biotin level of 240 mcg/kg feed was six times the lowest, and was more than estimated requirement values of 90 mcg/kg (Wagstaff et al., 1961), 120 mcg/kg (Ogunmodede, 1978) and 160 mcg/kg (Whitehead and Bannister, 1980; Oloyo and Ogunmodede, 1989).

There is ample evidence in the literature to show that biotin is needed for the promotion of higher feed intake and body weight gain. Results of feed utilisation in Table 2 seemed to indicate that 120 mcg biotin/kg feed was required to promote better feed consumption and body weight whether or not palm kernel oil was included in the ration. Consistently and significantly lower feed intake and poorer body weight of birds given supplemental palm kernel oil compared with birds on the same dietary level of biotin but given no oil supplement seemed to show that 2% oil addition had adverse effect on feed utilisation of chicks. The significantly lower consumption of diets with 2% oil supplementation by broilers confirm the reports of Rand et al. (1958), Maner et al. (1962) and Mateos et al (1982). These authors reported that lower level of supplemental fat in the diet induced higher feed intake. Maner et al. (1962) and Mateos et al. (1982) reported that higher oil level in the diet increased transit time of ingesta in the gastro-intestinal tract, hence reduced intake of feed. The significant depression of

Table 2 Feed utilisation of experimental broiler chicks at 28 days

Biotin (mcg/kg)	*Oil (%)*	*Feed intake (g)*	*Body weight (g)*	*Feed efficiency**
		Mean	Mean	Mean
40	0	280c**	252de	0.20
80	0	284c	266cd	0.25
120	0	322b	310a	0.25
160	0	318b	298ab	0.22
200	0	365a	298ab	0.24
240	0	334ab	300ab	0.24
40	2	254d	229f	0.23
80	2	256d	237ef	0.24
120	2	301c	286bc	0.22
160	2	305c	270cd	0.25
200	2	304c	254de	0.22
240	2	306c	272cd	0.24
	±*SEM****	8.71	7.24	4.30E-03

* Weight gain/feed intake (21st day — 28th day)
** Values denoted by different subscripts in a column were significantly different at $P < 0.05$
*** SEM, standard error of the mean

weight gain resulting from the addition of 2% oil to the feed is contrary to the reports of Yacowitz (1953) and Bossard and Combs (1961) who demonstrated that chicks fed low fat diet had slightly lower gain than those chicks that received higher fat levels.

Leong et al. (1955), Combs and Romoser (1955) and Sunde (1956) attributed the poor performance of broiler chicks fed high fat rations to the failure to provide sufficient amino acid and perhaps other essential nutrients required in the higher energy ration. Combs and Romoser (1955) observed very rapid growth in broiler chicks fed 15% added fat by supplementing the diet with required levels of amino acids in proportion to their higher energy content. Biely and March (1954) found that the addition of fat to a 19% protein ration depressed growth and feed conversion in chicks but did not adversely affect growth and feed conversion when added to rations containing 24 to 28% protein. Marion and Woodroff (1965) reported that body weights and feed efficiency were greater at higher levels of protein when fat was added to the diet. Beneficial effects of supplemental fat in the diets of chicks on feed utilisation have been suggested by Rand et al. (1958) to be due to the improvement in the utilisation of metabolisable energy and protein. Rand et al. (1958) reported that higher protein levels were needed when fat was used in the feed formulation due to the reduction in total feed intake. Since the two basal diets used in this study (trials 1 and 2) had similar metabolisable energy values and protein contents (Table 1), the extra energetic effect of the added palm kernel oil in second basal diet (trial 2) might have raised the protein requirement of the birds for maximum gain and feed efficiency. Consequently the significantly lower body weight gain in groups given 2% palm kernel oil may be attributed to the lower dietary nutrient intake.

In fatty acid synthesis, acetyl CoA is carboxylated to malonyl CoA in the presence of a biotin-enzyme known as acetyl CoA carboxylase (Wakil and Bressler, 1962). It is therefore expected that reduced acetyl CoA carboxylase activity with consequent depression in lipogenesis will result in biotin deficiency. Results obtained in this study is contrary to this expectation. Blood, liver and kidney total lipid and free fatty acid contents were significantly affected by dietary biotin treatments. These parameters were consistently and significantly higher in birds given 40–80 mcg biotin/kg feed than in those given higher levels, that is, 120–240 mcg biotin/kg feed (Tables 3, 4 and 5). Also, they were negatively correlated with the dietary biotin level (Table 6). Since 40 and 80 mcg biotin/kg feed are below the vitamin requirement level of broiler chicks, birds given these two dietary levels of the vitamin are thus considered to be in a deficiency state.

No significant effect of biotin deficiency on liver fatty acid synthesis in rat was observed (Gram and Okey, 1958; Puddu et al., 1967). Biochemical studies on lipid metabolism in biotin-deficient broiler chicks also revealed that although plasma, liver and kidney lipid were elevated, hepatic and kidney lipogenesis was normal (Whitehead, 1975; Wight and Siller, 1975; Evans et al., 1975).

These authors attributed the characteristic fatty infiltration of tissues to a response to the hypoglycaemia caused by failure to hepatic gluconeogenesis,

Table 3 Effect of dietary biotin and palm kernel oil supplementation on blood lipid contents of chicks at 28 days

Biotin (mcg/kg)	*Oil (%)*	*Total lipid (g/l)*	*Free fatty acid (mmol/l)*	*Triglyceride (mmol/l)*	*phospholipid (mmol/l)*	*Cholesterol (mmol/l)*
		Mean	Mean	Mean	Mean	Mean
40	0	19.90a*	3.52a	22.32a	0.42ef	5.18
80	0	17.07bc	2.89b	19.58b	0.38f	4.97
120	0	16.20bc	2.07c	16.74c	0.50ef	4.61
160	0	15.65c	1.08c	16.00c	0.53ef	4.74
200	0	16.10bc	1.99c	16.48c	0.41f	4.97
240	0	16.60bc	1.80c	16.95c	0.61e	4.97
40	2	21.50a	3.98a	20.57b	0.67cd	4.97
80	2	19.65a	2.81b	17.42c	0.75cd	6.42
120	2	16.25bc	2.62b	16.34c	0.97ab	4.09
160	2	16.20bc	2.70b	16.39c	0.67cd	4.53
200	2	17.45bc	2.70b	17.66c	1.09a	4.33
240	2	17.20bc	2.62b	17.66c	0.83bc	4.33
	±*SEM***	0.512	0.183	0.543	0.062	0.165

* Values denoted by different subscripts in a column were significantly different at $P < 0.05$
** *SEM*, Standard error of the mean

brought about by a very low activity of pyruvate carboxylase, a biotin-dependent enzyme. Significantly higher lipid contents of blood, liver and kidney observed in birds given 40 and 80 mcg biotin/kg feed in this study was therefore due to accumulation brought about by biotin deficiency rather than lipogenesis.

Liver weight, liver total lipid and kidney total lipid and free fatty acid contents were significantly influenced by dietary oil treatment (Tables 4 and 5). The consistently and significantly lower levels of these lipids in both organs as well as

Table 4 Effect of dietary biotin and palm kernel oil supplementation on liver weight and lipid at 28 days

Biotin (mcg/kg)	*Oil (%)*	*Weight (g)*	*Total lipid (mg/g)*	*Free fatty acid (mg/g)*	*Triglyceride (mg/g)*	*Phospolipid (mg/g)*	*Cholesterol (mg/g)*
		Mean	Mean	Mean	Mean	Mean	Mean
40	0	9.56a*	299.1a	14.9a	210.2a	60.1c	12.1a
80	0	9.11ab	254.1b	7.8b	165.4b	70.0b	8.0ab
120	0	8.29bc	169.9cd	2.4c	79.9d	80.7ab	5.4b
160	0	7.90bc	160.9d	2.6c	73.9d	80.2ab	3.9b
200	0	8.00bc	163.0cd	2.8c	75.3d	79.9ab	4.1b
240	0	7.56c	159.5d	2.6c	74.7d	78.6ab	3.3b
40	2	7.34cd	188.5c	9.5b	100.9c	62.8c	10.8a
80	2	6.50d	183.2cd	13.9a	96.5c	58.0c	10.9a
120	2	7.50cd	167.7cd	3.7c	76.4d	79.7ab	6.7b
160	2	7.34cd	165.8cd	2.0c	75.8d	81.7a	4.1b
200	2	7.48cd	166.0cd	2.0c	77.5d	80.6ab	4.0b
240	2	7.50cd	166.8cd	3.0c	78.6d	80.1ab	3.4b
	±*SEM***	0.228	12.09	1.31	12.04	2.50	0.90

* Values denoted by different subscripts in a column significantly different at $P < 0.05$
** *SEM*, Standard error of the mean

Table 5 Effect of dietary biotin and palm kernel oil supplementation on kidney weight and lipid at 28 days

Biotin (mcg/kg)	*Oil (%)*	*Weight (g)*	*Total lipid (mg/g)*	*Free fatty acid (mg/g)*	*Triglyceride (mg/g)*	*Phospholipid (mg/g)*	*Cholesterol (mg/g)*
		Mean	Mean	Mean	Mean	Mean	Mean
40	0	3.56	349.5a*	21.1a	247.9a	59.8c	16.8ab
80	0	3.51	332.5a	23.0a	225.1b	63.5c	18.6a
120	0	3.09	189.7bc	11.3de	60.0e	100.2ab	15.3b
160	0	3.14	196.3bc	14.3bcd	63.1e	102.0ab	12.7c
200	0	3.13	187.4bc	15.2bc	56.0e	100.1ab	12.2c
240	0	2.96	196.0bc	12.0cde	60.5e	107.9a	12.8c
40	2	2.86	212.5b	15.0bc	143.3c	39.7d	16.7ab
80	2	2.81	204.5bc	16.3b	104.6d	63.4c	16.2ab
120	2	3.46	168.7c	15.2bc	49.9c	85.0b	11.6c
160	2	3.15	186.6bc	14.9bc	61.4e	97.0ab	11.1c
200	2	3.02	191.5bc	10.5e	54.7e	111.3a	10.3c
240	2	3.05	189.4bc	11.4de	56.8e	107.3a	10.6c
	±*SEM***	0.068	16.29	1.05	19.32	6.56	0.78

* Values denoted by different subscripts in a column were significantly different at $P < 0.05$
** *SEM*, Standard error of the mean

low liver weights in birds fed diets with added 2% palm kernel oil indicated reduced lipid deposition in the organs. The result is in agreement with those of Hill et al. (1958) who reported that as little as 2% corn oil in the diet resulted in significant depression of lipogenesis in rat livers as measured by the incorporation of acetate into fatty acid. Depression in hepatic lipogenesis due to fat feeding was identified to be enzymatic in nature and thus indicated the block was localized at the step involving the carboxylation of acetyl CoA (Bortz et al., 1963).

Synthesis of triglyceride in the liver is enhanced by elevated plasma level of free fatty acid (Topping and Mayes, 1972); and since biotin deficiency in chicks is characterized by elevated blood free fatty acid concentration (Whitehead et al., 1973; Bannister et al., 1975), this condition enhanced accumulation of triglyceride in the liver and kidney (Johnson et al., 1972; Whitehead, 1975). Consistently and significantly higher values of triglyceride in liver and kidney of birds given 40–80 mcg biotin/kg feed than those given 120–240 mcg biotin/kg feed (Tables 4 and 5) as well as the significantly negative correlation between this parameter and dietary biotin level (Table 6) is therefore in agreement with the earlier reports of Biotin deficiency effect on triglyceride accumulation in the organs.

Liver triglyceride are precursors of triglyceride component of plasma very low density lipoprotein (VLDL), and if the production rate of plasma lipoprotein does not keep pace with the liver triglyceride synthesis, fatty liver develops (Havel et al., 1962). Accumulation of triglyceride in the liver has been associated with failure in synthesis of phosphatidyl choline component of VLDL (Mishkel and Morris, 1964; Corredor et al., 1967; Mookerjea, 1971; Limbardi, 1971). Phospholipid concentration in liver and kidney of birds were significantly and positively correlated with dietary biotin (Tables 4, 5 and 6). The significantly reduced phospholipid concentration in the organs of birds given 40–80 mcg

Table 6 Estimated regression line showing the relationship between dietary biotin and the parameter

Parameter	*r-value*	*t-value*	*Regression line*+
Blood			
Total lipid (mg/ml)	– 0.755 n.s.	– 2.301 n.s.	Y = 25.750375 – 0.128174107x
Free fatty acid (mg/ml)	– 0.854*	– 3.282*	Y = 1.28498333 – 2.59727679E – 03x
Triglyceride (mg/ml)	– 0.683 n.s.	– 1.870 n.s.	Y = 20.5412375 – 8.05504461E – 03x
Phospholipid (mg/ml)	0.741 n.s.	2.204 n.s.	Y = 0.80295 + 1.63294643E – 03x
Cholesterol (mg/ml)	– 0.510 n.s.	– 1.186 n.s.	Y = 2.37950417 – 7.18482144E – 04x
Liver			
Total lipid (mg/g)	– 0.854*	– 3.282*	Y = 209.227867 – 0.218071964x
Free fatty acid (mg/g)	– 0.883*	– 3.770*	Y = 7.83197084 – 0.0271034375x
Triglyceride (mg/g)	– 0.837*	– 3.064*	Y = 118.946275 – 0.214025625x
Phospholipid (mg/g)	0.882*	3.738*	Y = 73.1659875 + 0.0615782144x
Cholesterol (mg/g)	– 0.880*	– 3.707*	Y = 6.35368334 – 8.44803572E – 03x
Kidney			
Total lipid (mg/g)	– 0.796 n.s	– 2.632 n.s.	Y = 244.360817 – 0.25576295x
Free fatty acid (mg/g)	– 0.643 n.s	– 1.680 n.s.	Y = 16.72965 – 0.0128111161x
Triglyceride (mg/g)	– 0.835*	– 3.035*	Y = 129.982082 – 0.364084821x
Phospholipid (mg/g)	0.883*	3.759*	Y = 85.9625917 + 0.126119509x
Cholesterol (mg/g)	– 0.099 n.s	– 0.198 n.s.	Y = 13.5292167 – 1.86549108E – 03x

n.s., not significant at p (0.05); *, significant at $P < 0.05$
+, Y = Biotin (mcg/kg feed); X = parameter

biotin/kg feed is also in agreement with the observation of Whitehead (1975) which indicated that there was a large increase in the proportion of triglyceride at the expense of phospholipid in the liver and kidney of biotin-deficient chicks. It therefore appeared that significantly reduced phospholipid level due to biotin deficiency was responsible for the accumulation of triglyceride in the organs.

The significant depression in the triglyceride concentration in blood, liver and kidney of bird receiving 40–80 mcg biotin/kg feed when 2% supplemental palm kernel oil was given (Tables 3, 4 and 5 indicated reduced lipogenesis in the organs, and this is in agreement with the reports of Hill et al. (1958), Bortz et al. (1963) and Iritani and Fukuda (1980). Accumulation of triglyceride in the organs of birds fed diets without supplemental 2% palm kernel oil might have been enhanced further by the inability of phospholipid concentration to cope with the abnormal infiltration of triglyceride as is the case with biotin deficiency. 2% supplemental fat significantly elevated blood phospholipid concentration (Table 3).

If malonyl CoA is an intermediate in cholesterol synthetic pathway as reported by Brodie et al. (1963), factor such as biotin deficiency that significantly affect acetyl CoA carboxylase (Martin and Vagelos, 1962) would have similar effect on cholesterogenesis. It has however been reported that biotin was not required for synthesis of cholesterol (Fletcher and Myant, 1960). Infact, addition of biotin to the medium of biotin-deficient yeast depressed cholesterol synthesis (Bloomfield and Bloch, 1960). In this study, liver and kidney cholesterol contents were significantly affected by dietary biotin treatment (Tables 4 and 5). Blood, liver and kidney cholesterol concentrations were negatively correlated with dietary

biotin level (Table 6). Significantly higher cholesterol in liver and kidney of birds given 40 and 80 mcg biotin/kg feed seemed to suggest that addition of higher levels of the vitamin to the diets of chicks significantly depressed cholesterol synthesis.

There have been conflicting reports regarding the effect on serum and liver cholesterol of nature of dietary fat. While Alfin-Salter et al. (1954) observed a significant reduction of liver cholesterol concentration due to unsaturated fat, Okey and Lyman (1957) reported no significant difference on the effect of unsaturated and saturated fat. Mead et al. (1956) and Mukherjee and Alfin-Slater (1958) reported that prior ingestion of diets containing up to 15% unsaturated fat significantly increased cholesterogenesis than saturated fat. Most reports however showed that linoleic acid is essential for both transportation as well as proper metabolism of cholesterol and hence its deficiency leads to cholesterol accumulation in the liver. In this study, blood, liver and kidney cholesterol contents were not significantly affected by dietary 2% palm kernel oil supplementation (Tables 3, 4 and 5). The result tended to suggest that a difference of 0.07% linoleic acid content between the diets supplemented with and without palm kernel oil was not sufficient to produce a significant difference in the cholesterol metabolism in the two groups of experimental birds.

CONCLUSION

Results obtained in this study tended to suggest that dietary biotin and palm kernel oil supplementation of broilers' diets had a significant controlling effect on the lipid contents of blood and the two organs examined. Since performance of broiler chicks will depend on their ability to efficiently metabolise the major nutrients, it should therefore be of interest to further investigate effect of biotin and palm kernel oil on the lipid metabolism in the birds. This is with a view to correlating the state of lipid metabolism with the establishment of their biotin requirement especially in areas where palm kernel oil is used as feed ingredient in broiler rations.

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ACKNOWLEDGEMENT

The authors gratefully acknowledge F. Hoffman-La Roche & Co. Ltd. Switzerland for supplying ROVIMIX H-2, vitamin source, used in this study.