Beitr. trop. Landwirtsch. Vet.med.
27 (1989) H. 3, 347–355

Effect of dietary palm kernel oil and biotin on the Fatty Liver and Kidney Syndrome in broiler chicken

RAHEEM A. OLOYO

Department of Science Laboratory Technology, Federal Polytechnic, Ilaro, Ogun State/Nigeria

B. K. OGUNMODEDE

Department of Animal Science, University of Ibadan Ibadan, Oyo State/Nigeria

(Received October 1988)

The effect of feeding biotin and palm kernel oil to broiler chicks on the appearance of Fatty Liver and Kidney Syndrome (FLKS) was investigated. A total of 480 broiler chicks was divided into two equal batches each of which was divided into 6 groups of 40 chicks per group. Each group was further subdivided into equal units of 20 chicks. Six dietary levels of biotin (40, 80, 120, 160, 200, and 240 mcg/kg feed) were given to the first batch of chicks, while the second batch had 2% palm kernel oil added to the six dietarybiotin levels. These two basic rations were supplemented with biotin in order to obtain six levels of the vitamin in the rations. The results showed that the 2% palm kernel oil forage affected FLKS mortality and the minimum biotin requirement. FLKS mortality was significantly reduced in case of palm kernel oil supplement. A lower amount of biotin (120 mcg/kg feed) was needed in case of palm kernel oil supplement – as compared with the necessary biotin (160 mcg/kg feed) – in order to prevent FLKS mortality when palm kernel oil was not contained in the rations. The biochemical analysis of the liver and kidney syndrome – coupled with the correlation and regression analysis of the data collected – showed that a minimum of 120 mcg/kg feed was needed by broiler chicks for the prevention of FLKS.

1. Introduction

Fatty Liver and Kidney Syndrome (FLKS) has been reported by several workers to be a deficiency symptom of biotin in chicken (MARTHEDAL and VELLINGE 1958, HEMSLEY 1965, RIDDELL et al. 1971, PAYNE et al. 1974, WHITEHEAD and BLAIR 1974, LOHR 1975). Some other workers demonstrated that the fat content of the feed also significantly affected the incidence of the disease. BLAIR et al. (1973) demonstrated that a high level of FLKS mortality was associated with a low level of fat in the diet. BLAIR et al. (1975) recorded mortality of 22.6% in a diet with 2% ether-extractable lipid, and of 13.7% and 5.4% respectively in isoenergetic diets containing 4.4% and 7% ether-extractable lipid. Experimenting with maize oil in chick diets, WHITEHEAD et al. (1975) observed 24% mortality when the diet contained 2% ether-extract and 4% mortality when the ether-extract was 7%. These workers further studied the effect of the type of fat on the incidence of FLKS by substituting maize oil, tallow or olive oil for starch, and it was found that these fats were equally effective in reducing FLKS mortality.

HEMSLEY et al. (1973) on the other hand reported that FLKS of young chicken was not prevented by the dietary addition of 3% tallow, maize oil, safflower oil or soya oil. It seemed therefore that there are conflicting reports on the effect of dietary fat on FLKS in chicken.

The objective of this study was to determine the effect of dietary biotin supplementation and palm kernel oil inclusion on FLKS incidence in broiler fed practical rations.

2. Material and methods

Experiment I

In this study duplicate groups of day-old broiler chicks with 20 chicks per group (40 chicks per treatment) were given basal biotin-deficient diet (table 1) supplemented with graded levels of biotin such that experimental diets had 40, 80, 120, 160, 200, and 240 mcg of the vitamin per kg of ration. The birds were housed in deep litter pens (73″ × 48″), 20 birds per pen, and they had free access to feed and water at all times. Each pen was heated with a 100-watts electric tungsten lamp that had guard support around it. Experimental birds were kept on these treatments for a period of six weeks. All birds that died were sent to the veterinary clinic of the Faculty of Veterinary Medicine, University of Ibadan, Ibadan, for *post mortem* examination. Record of FLKS mortality was kept. Four replicate samples were removed from the respective treatment groups at the end of the fourth and sixth weeks for slaughtering. The livers and kidneys were excised, drained of fluids with blotting paper and weighed. These organs were freeze-dried and kept for subsequent estimation of total lipid content by the method of FOLCH et al. (1957), and triglyceride content by the method of FLETCHER (1968).

Table 1: Composition of the basal biotin-deficient diets

	Experiment I %	Experiment II %
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 (%)	21.26	21.25
Metabolizable energy ME (Kcal/kg)	2741.92	2751.82
ME/CP	128.73	129.48
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.

Experiment II

In this study palm kernel oil was included in the biotin-deficient basal diet of experiment I at 2% level (table 1) such that both diets had similar metabolizable energy value and protein content. Biotin-deficient diet with 2% oil inclusion therefore served as basal diet for this experiment. Graded levels of biotin were added to the basal diet with 2% oil inclusion such that experimental diets had 40, 80, 120, 160, 200, and 240 mcg of the vitamin per kg of feed. This arrangement therefore completed a 2 × 6 factorial experimental design. These rations were fed to duplicate groups of day-old chicks with 20 birds per group (40 birds per treatment) for a period of six weeks. The birds were reared in deep-litter pens as described in the first experiment. Four replicate samples were removed from the respective treatment groups and the analysis of the liver and kidney were as described in experiment I. Results obtained in the two trials were subjected to analysis of variance in accordance with procedures of STEEL and TORRIE (1960). Significantly different treatment means were separated by the multiple range test of DUNCAN (1955).

3. Results and discussion

In order to study the FLKS condition experimentally, the development of a diet which would reproduce the syndrome becomes important. The reproduction of the syndrome could be undertaken with the aid of synthetic diets deficient in the causative agent, but mixing of the experimental ration with the actual feed ingredients lends the results obtained to practical application. In this study biotin-deficient diets used to produce FLKS condition were the two basal rations shown in table 1. Total available biotin in the basal diets (40 mcg per kg feed) was about half the value estimated as the requirement by WAGSTAFF et al. (1961), about a third of the value estimated by OGUNMODEDE (1978), and a quarter of the value estimated by WHITEHEAD and BANNISTER (1980). The highest supplement level of 200 mcg biotin per kg feed was five times the lowest. Thus a wide range of dietary biotin level was tested.

FLKS, a metabolic disorder, is characterized by morbidity followed by death in young chicks, usually in the age-range of 3 to 5 weeks, but it can occur as early as 10 days and as late as 56 days of age. The syndrome is variable in its occurrence and mortality can be as high as 20% in occasional outbreaks but even when mortality is not apparent there is often a low background incidence (BANNISTER 1976). Its incidence can be reduced

Table 2: FLKS mortality of chicks fed graded levels of biotin and palm kernel oil

Dietary biotin levels (mcg/kg feed)	Dietary oil levels (%)	Mortality (%)	
		4 weeks	6 weeks
40	0	10.0 a*	10.0 a
80	0	7.5 b	10.0a
120	0	5.0c	5.0c
160	0	0.0e	0.0d
200	0	0.0e	0.0d
240	0	0.0e	0.0d
40	2	7.5b	7.5b
80	2	2.5d	2.5d
120	2	0.0e	0.0d
160	2	0.0e	0.0d
200	2	0.0e	0.0d
240	2	0.0e	0.0d

* Values denoted by different subscripts in a column were significantly different at P (0.05)

Table 3: Liver and kidney weight of chicks fed graded levels of biotin and palm kernel oil

Dietary biotin levels (mcg/kg feed)	Dietary oil levels (%)	Liver weight (g)		Kidney weight (g)	
		4 weeks mean ± sd	6 weeks mean ± sd	4 weeks mean ± sd	6 weeks mean ± sd
40	0	9.56a* ± 0.7	16.06 ± 4.3	3.56 ± 0.3	5.76 ± 0.4
80	0	9.11ab ± 0.9	13.97 ± 1.8	3.51 ± 0.4	5.63 ± 0.9
120	0	8.29bc ± 0.2	15.54 ± 3.9	3.09 ± 0.1	5.47 ± 0.9
160	0	7.90bc ± 1.0	15.71 ± 2.4	3.14 ± 0.4	5.60 ± 1.6
200	0	8.00bc ± 1.4	15.74 ± 2.8	3.13 ± 0.1	6.02 ± 1.3
240	0	7.65c ± 0.9	15.59 ± 3.1	2.96 ± 0.7	5.56 ± 1.9
40	2	7.34cd ± 0.4	13.62 ± 2.8	2.86 ± 0.3	4.79 ± 0.6
80	2	6.50d ± 0.8	14.27 ± 1.4	2.81 ± 0.6	5.17 ± 0.7
120	2	7.50cd ± 0.6	15.27 ± 1.9	3.46 ± 0.2	5.75 ± 0.2
160	2	7.34cd ± 1.0	15.21 ± 1.8	3.15 ± 0.2	5.71 ± 1.1
200	2	7.48cd ± 0.6	14.99 ± 0.4	3.02 ± 0.1	5.21 ± 0.1
240	2	7.50cd ± 0.8	14.80 ± 4.1	3.05 ± 0.2	5.41 ± 0.9

* Values denoted by different subscripts in a column were significantly different at P (0.05)

by increasing the level of dietary fat (BLAIR et al. 1975, WHITEHEAD et al. 1975) and eliminated by biotin supplementation in the diet (PAYNE et al. 1974, WHITEHEAD et al. 1976). In this study, groups of birds given diets without oil had FLKS mortality as early as the first week of age in those given 80 and 120 mcg biotin per kg feed (table 2) and as late as the fifth week in those given 80 mcg of the vitamin; whereas the mortality occurred only in the second and third weeks of study in birds given 40 and 80 mcg of the vitamin with added 2% palm kernel oil. Percentage FLKS mortality showed that up to 120 mcg of the vitamin did not prevent the incidence of the syndrome in birds given feed without added oil. When oil was added, 120 mcg of the vitamin seemed to prevent mortality due to FLKS. Throughout the period of study, mortality recorded in groups of birds fed 0% and 2% oil inclusions were 4% and 1.7% respectively. Histologial studies by WHITEHEAD et al. (1973), and WIGHT and SILLER (1975) showed that the liver and kidneys may be markedly involved in fatty infiltration. The analysis of livers and kidneys of FLKS-affected chicken by WHITEHEAD (1975) confirmed that these organs enlarged and could contain a two-fold increase in lipid content. Higher liver weight and lipid contents of liver and kidney in birds fed 40 and 80 mcg biotin without added oil (tables 3, 4, and 5) reflected deposition of fat in these organs as a result of biotin deficiency, hence higher incidence of FLKS mortality was recorded. Triglyceride concentration and triglyceride (% of total lipid) in liver and kidneys were significantly higher in birds given 40 and 80 mcg of the vitamin with and without added oil than those given other dietary biotin levels (tables 4 and 5). This observation is in agreement with those of JOHNSON et al. (1972) and WHITEHEAD (1975). They observed that the extra lipid in liver and kidney was mainly triglyceride.

Total lipid, triglyceride and triglyceride as percentage of total lipid in the organs were significantly affected by and were negatively correlated with the dietary biotin level (table 5). The significant negative correlation of these lipid fractions with biotin indicated that a higher level of dietary biotin reduced the chances of FLKS incidence in broiler chicks. The result is therefore in agreement with those of PAYNE et al. (1974), WHITEHEAD and BLAIR (1974) and WHITEHEAD et al. (1976), who reported that biotin supplementation prevented FLKS in chicken.

Significant differences in liver weights and the lipid fractions in liver and kidney were observed at the fourth week age. This therefore confirms the report of BANNISTER

Table 4: Lipid content of liver of chicks fed graded levels of biotin and palm kernel oil

Biotin (mcg/kg)	Oil %	Total lipid (mg/g) 4 weeks mean ± sd	Total lipid (mg/g) 6 weeks mean ± sd	Triglyceride (mg/g) 4weeks mean ± sd	Triglyceride (mg/g) 6 weeks mean ± sd	Triglyceride (% of total lipid) 4 weeks mean ± sd	Triglyceride (% of total lipid) 6 weeks mean ± sd
40	0	299.1a* ± 8.3	179.7 ± 33.6	210.2a ± 7.1	86.4 ± 11.5	70.3a ± 1.7	48.5 ± 3.1
80	0	254.1b ± 7.7	169.1 ± 20.3	165.4b ± 5.4	81.1 ± 11.0	65.1b ± 1.1	47.9 ± 1.5
120	0	169.9cd ± 10.0	171.2 ± 38.4	79.9d ± 3.7	80.4 ± 19.3	47.0d ± 0.6	46.9 ± 2.4
160	0	160.9d ± 9.6	169.8 ± 15.0	73.9d ± 6.1	78.3 ± 8.1	45.9d ± 1.5	46.1 ± 2.7
200	0	163.0cd ± 23.2	169.3 ± 13.8	75.3d ± 11.7	81.1 ± 9.9	46.1d ± 0.8	47.8 ± 2.3
240	0	159.5d ± 19.0	165.4 ± 17.9	74.7d ± 9.5	77.7 ± 11.6	46.8d ± 0.9	46.8 ± 2.2
40	2	188.5c ± 16.6	175.8 ± 27.5	100.9c ± 2.9	83.9 ± 12.4	53.8bc ± 3.6	47.8 ± 0.7
80	2	183.2cd ± 23.3	168.3 ± 13.7	96.5c ± 12.2	78.4 ± 8.5	52.7c ± 0.9	46.5 ± 1.6
120	2	167.7cd ± 12.8	164.8 ± 14.0	76.4d ± 6.8	74.1 ± 2.9	45.5d ± 0.9	45.1 ± 2.5
160	2	165.8cd ± 19.9	166.3 ± 12.9	75.8d ± 8.4	73.7 ± 8.3	45.7d ± 0.7	44.2 ± 1.6
200	2	166.0cd ± 11.1	168.5 ± 2.3	77.5d ± 5.4	75.1 ± 1.7	46.7d ± 0.4	44.6 ± 0.8
240	2	166.8cd ± 15.8	174.7 ± 36.7	78.6d ± 7.6	80.0 ± 16.4	47.2d ± 0.3	45.9 ± 0.3

* Values denoted by different subscripts in a column were significantly different at P (0.05)

Table 5: Lipid content of kidney of chicks fed graded levels of biotin and palm kernel oil

Biotin (mcg/g)	Oil %	Total lipid (mg/g) 4 weeks mean ± sd	Total lipid (mg/g) 6 weeks mean ± sd	Triglyceride (mg/g) 4 weeks mean ± sd	Triglyceride (mg/g) 6 weeks mean ± sd	Triglyceride (% of total lipid) 4 weeks mean ± sd	Triglyceride (% of total lipid) 6 weeks mean ± sd
40	0	349.5a* ± 37.0	201.6 ± 10.3	247.9a ± 22.4	62.7 ± 3.7	70.9a ± 1.3	31.1 ± 0.5
80	0	332.5a ± 29.0	203.8 ± 28.9	225.1b ± 21.0	57.9 ± 8.7	67.7a ± 0.5	28.4 ± 0.2
120	0	189.7bc ± 13.9	211.3 ± 36.0	60.0e ± 5.3	62.0 ± 10.4	31.6c ± 1.4	29.4 ± 0.4
160	0	196.3bc ± 26.1	215.5 ± 50.1	63.1e ± 9.4	61.2 ± 17.7	32.1c ± 0.5	28.1 ± 1.6
200	0	187.4bc ± 11.4	198.5 ± 48.1	56.0e ± 2.8	56.8 ± 14.8	29.9c ± 0.4	28.6 ± 0.8
240	0	196.0bc ± 36.2	219.7 ± 66.2	60.5e ± 11.7	66.4 ± 21.3	30.8c ± 0.6	30.1 ± 0.7
40	2	212.5b ± 23.4	213.2 ± 15.1	143.3c ± 19.1	63.4 ± 7.7	67.4a ± 4.9	29.7 ± 1.9
80	2	204.5bc ± 35.0	195.8 ± 20.0	104.6d ± 15.4	58.9 ± 8.1	51.4b ± 1.5	30.1 ± 2.7
120	2	168.7c ± 9.5	189.8 ± 7.3	49.9e ± 2.3	56.9 ± 0.6	29.6c ± 0.4	30.0 ± 1.4
160	2	186.6bc ± 15.7	177.3 ± 28.6	61.4e ± 5.2	51.9 ± 7.7	32.9c ± 0.5	29.4 ± 0.5
200	2	191.5bc ± 6.2	192.5 ± 22.7	54.7e ± 1.2	58.1 ± 1.6	28.6c ± 0.9	30.5 ± 3.3
240	2	189.4bc ± 13.7	182.2 ± 21.6	56.8e ± 3.2	56.5 ± 9.3	30.0c ± 0.9	31.2 ± 4.8

* Values denoted by different subscripts in a column were significantly different at P (0.05)

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

Parameter	r-value	t-value	Regression line+
Liver weight (g)	0.072 n. s.	0.145 n. s.	Y = 11.4195 + 2.51339296E-04X
Liver total lipid (mg/g)	–0.854*	–3.282*	Y = 209.227867 — 0.218071964X
Liver triglyceride (mg/g)	–0.837*	–3.064*	Y = 118.946275 — 0.214025625X
Liver triglyceride (% of TL)	–0.810*	–2.758*	Y = 55.077 — 0.0449629464X
Kidney weight (g)	–0.111 n. s.	–0.224 n. s.	Y = 4.35058334 — 0.112053563E-04X
Kidney total lipid (mg/g)	–0.796 n. s.	–2.632 n. s.	Y = 244.360817 — 0.255576295X
Kidney triglyceride (mg/g)	–0.835*	–3.035*	Y = 129.982083 — 0.364084821X
Kidney triglyceride (% of TL)	–0.845*	–3.159*	Y = 49.943125 — 0.100834821X

n. s. = not significant at P (0.05); * = significant at P (0.5)
+ Y = Biotin (mcg/kg feed); X = parameter

(1976) that FLKS is a metabolic disorder of young chicken. The consistently and significantly lower levels of liver weight and lipids in liver and kidney in groups of birds fed diets with added oil indicated reduced lipid deposition in these organs. HILL et al. (1958) reported that as little as 2.0% fat in the diet resulted in measurable depression of lipogenesis in rat livers as measured by the incorporation of acetate into fatty acid. Depression in hepatic lipogenesis in rat by fat feeding was identified to be enzymatic in nature and thus indicated that the block was localized at the step involving the carboxylation of acetyl CoA (BORTZ et al. 1963). WHITEHEAD et al. (1978) reported that liver pyruvate carboxylase, a biotin-dependent enzyme, activity was low in birds fed a diet causing a high incidence of FLKS, but the addition of fat to this diet reduced the metabolic need for lipogenesis; this in turn reduced the activity of another biotin-dependent enzyme, acetyl CoA carboxylase. This enzyme seemed able to sequester biotin preferentially, thus a decrease in its activity allows more biotin to become available for pyruvate carboxylase. This explains why the oil inclusion in the ration reduced the dietary biotin requirement of the chicken. Consequently 120 mcg biotin/kg is needed in the presence of 2% dietary palm kernel oil, whereas 160 mcg of the vitamin is needed when no supplementary oil is added to the ration. The use of oil and the level of biotin to be fed will depend on the relative cost and availability of both of them.

Acknowledgement
The authors gratefully acknowledge the donation of feed grade biotin by F. HOFFMANN LA-ROCHE Co. Ltd., Switzerland.

References

BANNISTER, D. W.: The biochemistry of Fatty Liver and Kidney Syndrome: Biotin-mediated restoration of hepatic gluconeogenesis in vitro and its relationship to pyruvate carboxylase activity. Biochem. J. *156* (1976) 167–173.

BLAIR, R.; WHITEHEAD, C. C.; BANNISTER, D. W.; EVANS, A. J.: Involvement of diet in Fatty Liver and Kidney Syndrome in broiler chickens. Vet. Rec. *92* (1973) 118–119.

BLAIR, R.; WHITEHEAD, C. C.; TEAGUE, P. W.: The effect of dietary fat and biotin levels, form and cereal type on Fatty Liver and Kidney Syndrome in chicks. Res. Vet. Sci. *18* (1975) 76–81.

BORTZ, W.; ABRAHAM, S.; CHAIKOFF, I. L.: Localization of the block in lipogenesis resulting from feeding fat. J. Biol. Chem. *238* (1963) 1266–1272.

DUNCAN, D. B.: Multiple range and multiple range F tests. Biometrics *11* (1955) 1–42.

FLETCHER, M. J.: A colorimetric method for estimating serum triglyceride. Clin. Chim. Acta. *22* (1968) 393–397.

Folch, J.; Lees, M.; Sloane-Stanley, G. H.: A simple method for the isolation and purification of total lipids from animal tissues. J. Biol. Chem. *226* (1957) 497–509.
Hemsley, L. A.: The Fatty Liver and Kidney Syndrome of young chickens. Vet. Rec. *77* (1965) 124–126.
Hemsley, L. A.; Smetana, P.; Edgar, J.; Marshall, L. G.: The effect of dietary fat on the Fatty Liver and Kidney Syndrome. Br. Vet. J. *129* (1973).
Hill, R.; Linazasoro, J. M.; Chevallier, F.; Chaikoff, I. L.: Regulation of hepatic lipogenesis; the influence of dietary fats. J. Biol. Chem. *233* (1958) 305.
Johnson, A. R.; Pearson, J. A.; Fogerty, A. C.; Shenstone, F. S.; Kozuharov, S.; Pitt, J. I.; Gipps, P.: Fatty Liver and Kidney disease in young chicken. Proc. Australasian Poultry Sci. Conv. (1972) 15–16.
Lohr, J. E.: Fatty Liver and Kidney Syndrome in New Zealand chickens. N. Z. vet. J. *23* (1975) 167–174.
Marthedal, K. E.; Vellinge, G.: Lever og nyrelidelse hos Kyllinger. Proc. Nord. Vetmod. *8* (1958) 250–256.
Ogunmodede, B. K.: Biotin requirement of broiler chicks fed maize-groundnut cake or guineacorn-groundnut cake rations. African J. Agric. Sci. V (1978) 47–53.
Payne, C. G.; Gilchrist, P.; Pearson, J. A.; Hemsley, L. A.: Involvement of biotin in the Fatty Liver and Kidney Syndrome of broilers Br. Poultry Sci. *15* (1974) 489–498.
Riddell, C.; Olsen, G. V.; Grimson, R. E.: Fatty Liver and Kidney Syndrome in a broiler flock. Avian. Dis. *15* (1971) 398–405.
Steel, R. G. D.; Torrie, J. H.: Principles and procedures of statistics. New York. N. Y. 1960.
Wagstaff, R. K.; Dobson, D. C.; Anderson, J. O.: Available biotin content of barley. Poultry Sci. *40* (1961) 503–509.
Whitehead, C. C.: Tissue lipid composition in Fatty Liver and Kidney Syndrome in chicks. Res. Vet. Sci. *18* (1975) 32–35.
Whitehead, C. C.; Bannister, D. W.: Biotin status, blood pyruvate carboxylase activity and performance in broilers under different conditions of bird husbandry and diet processing. Br. J. Nutr. *43* (1980) 541–549.
Whitehead, C. C.; Bannister, D. W.; Cleland, M. E.: Metabolic changes associated with the occurrence of Fatty Liver and Kidney Syndrome in chicks. Br. J. Nutr. *40* (1978) 221–234.
Whitehead, C. C.; Blair, R.: The involvement of biotin in the Fatty Liver and Kidney Syndrome in broiler chickens. World's Poultry Sci. J. *30* (1974) 231.
Whitehead, C. C.; Blair, R.; Bannister, D. W.; Evans, A. J.: The involvement of dietary fat and vitamins, stress, litter and starvation on the incidence of the Fatty Liver and Kidney Syndrome in chicks. Res. Vet. Sci. *48* (1975) 100–104.
Whitehead, C. C.; Blair, R.; Bannister, D. W.; Evans, A. J.; Jones, R. M.: The involvement of biotin in preventing the Fatty Liver and Kidney Syndrome in chicks. Res. Vet. Sci. *20* (1976) 180–184.
Whitehead, C. C.; Siller, W. G.; Blair, R.; Bannister, D. W.; Evans, A. J.; Wight, P. A. L.: Studies on the Fatty Liver and Kidney Syndrome of chicks. Proc. Vth. World Vet. Poultry Assn. Congr. (1973) 1310–1317.
Wight, P. A. L.; Siller, W. G.: The histopathology of Fatty Liver and Kidney Syndrome in chicks. Res. Vet. Sci. *19* (1975) 173–184.

R. A. Oloyo und B. K. Ogunmodede: Die Wirkung von Palmkernöl und Biotin in Broilerküken-rationen auf das Fettleber-Nieren-Syndrom

Untersucht wurde die Wirkung einer Ergänzung von Biotin und Palmkernöl in Broilerrationen auf Zustandekommen eines Fettleber-Nieren-Syndroms (FLKS). 480 Broilerküken wurden in 6 Gruppen à 40 Küken unterteilt und jede Gruppe in gleiche Einheiten von 20 Küken untergliedert. 6 verschiedene Futterhöhen von Biotin (40, 80, 120, 160, 200 und 240 mcg/kg Futter) wurden an die erste Gruppe von Küken gegeben, während die zweite Gruppe 2% Palmkernöl in Verbindung mit den sechs verschiedenen Höhen Biotin erhielt. Diese beiden Grundrationen wurden mit Biotin ergänzt, um sechs Höhen des Vitamins in den Rationen zu erreichen. Die Ergebnisse zeigten, daß die Nahrung mit 2% Palmkernöl die FLKS-Sterblichkeit und den minimalen Biotinbedarf beeinflußte. Die FLKS-Sterblichkeit wurde bei Gaben von Palmkernöl signifikant zurückgedrängt. Eine niedrigere Menge von Biotin (120 mcg/kg Futter) wurde bei Gaben von Palmkernöl benötigt – verglichen mit dem nötigen (160 mcg/kg Futter) Biotin – um der FLKS-Sterblichkeit vorzubeugen, wenn Palmkernöl nicht in

den Rationen enthalten war. Die biochemische Analyse des Leber- und Nierensyndroms – gekoppelt mit der Korrelations- und Regressionsanalyse der gesammelten Daten – zeigte, daß ein Minimum von 120 mcg/kg Futter Biotin als Vorbeugung gegen FLKS bei Broilerküken benötigt wird.

R. A. Oloyo и B. K. Okunmodede: Влияние масла из семян пальмы и биотина в рационах бройлерных цыплят на синдром жирной печени и почек

Изучалось влияние добавок биотина и масла из семян пальмы в рационах бройлерных цыплят на возникновение синдрома жирной печени и почек (СЖПП). Из 480 бройлерных цыплят образовывали 6 групп по 40 цыплят, а в каждой группе отделяли две единые подгруппы по 20 цыплят. Первой группе цыплят давалось 6 различных концентраций биотина (40, 80, 120, 160, 200 т 240 мкг на 1 кг корма), а второй группе — 2% пальмового масла в сочетании с 6 различными дозами биотина. В эти основные рационы добавлялся биотин для достижения 6 различных концентраций витамина в рационах. Результаты показали, что корм, содержащий 2% пальмового масла, имел влияние на падёж от СЖПП и минимальную потребность в биотине. Падёж от СЖПП достоверно снижался при скармливании пальмового масла. При скармливании пальмового масла потребность в биотине снижалась (120 мкг в 1 кг корма) по сравнению с необходимой концентрацией (160 мкг в 2 кг корма) биотина, чтобы не допустить падёж от СЖПП в случае, если в рационе не содержалось пальмовое масло. Биохимический анализ СЖПП в сочетании с корреляционным и регрессивным анализами полученных данных показали, что необходима минимальная концентрация биотина в 120 мкг/кг корма для предотвращения возникновения СЖПП у бройлерных цыплят.

R. A. Oloyo et S. K. Ogunmodede: L'effet de l'huile de palme et de la biotine dans des rations de poulets rôtis sur le syndrome du foie gras et des reins

A été examiné l'effet d'un supplément de biotine et d'huile de palme dans des rations de poulets, sur la naissance du syndrome foie gras-reins (FLKS). 480 poulets rôtis ont été répartis en 6 groupes à 40 poussins, chaque groupe étant réparti en unités égales de 20 poussins. 6 quantités différentes de biotine (40, 80, 120, 160, 200 et 240 mcg/kg de nourriture) ont été données au premier groupe de poussins, alors que le second groupe reçut 2% d'huile de palme, en combinaison avec 6 quantités différentes de biotine. Ces deux rations de base ont été complétées par de la biotine, pour atteindre 6 quantités de la vitamine dans les rations. Les résultats ont montré que la nourriture avec 2% d'huile de palme avait influencé la mortalité FLKS ainsi que les besoins minima de biotine. La mortalité FLKS a été remarquablement diminuée par des dons d'huile de palme. Une quantité plus faible de biotine(120 mcg de nourriture) était nécessaire avec des dons d'huile de palme – vis-à-vis de la quantité de biotine obligatoire (160 mcg/kg de nourriture) – pour prévenir la mortalité FLKS, quand il n'y avait pas d'huile de palme dans les rations. L'analyse biochimique du syndrome du fois gras et des reins, combinée avec l'analyse de corrélation et de régression des données recueillies, a montré qu'on avait besoin d'un minimum de 120 mcg/kg de nourriture de biotine pour prévenir le FLKS des poussins rôtis.

R. A. Oloyo y B. K. Ogunmodede: El efecto de aceite de pepitas de palma y de biotina en raciones para polluelos de engorde sobre el sindroma de hipertrofía del hígado y de los riñones

Se analizó el efecto de una adición de biotina y aceite de pepitas de palma a raciones para polluelos de engorde sobre la gestación de un sindroma de hipertrofía del hígado y de los riñones (FLKS). 480 polluelos de engorde fueron repartidos a 6 grupos de cada vez 40 polluelos y cada grupo fue dividido en unidades iguales de 20 animales. Se dieron 6 diferentes cantidades de biotina (40, 80, 120, 160, 200 y 240 mcg/kg de forraje) al primer grupo, aunque el segundo grupo recibió 2% de aceite de pepitas de palma combinado con seis diferentes cantidades de biotina. Ambas raciones básicas fueron completadas con biotina para obtener seis diferentes cantidades de la vitamina en las raciones. Los resultados mostraron que el forraje con 2% de aceite de pepitas de palma influyó sobre la mortalidad debida al FLKS y la mínima necesidad de biotina. La mortalidad causada por el FLKS quedó reducida de modo significativo con adiciones de aceite de pepitas de palma. Con adiciones de pepitas de palma se precisava una cantidad menor de biotina (120 mcg/kg de forraje) – comparada con el biotina necesario (160 mcg/kg de forraje) – para prevenir la mortalidad causada por el FLKS, si el aceite de pepitas de palma no era contenido en las raciones. El análisis bioquímico del sindroma de hipertrofía del hígado y de los riñones – combinado con los datos recogidos por el análisis de correlación y regresión – mostró que es preciso un mínimo de 120 mcg/kg de forraje con biotina como prevención contra el FLKS en polluelos de engorde.