

TISSUE DEPLETION OF BIOTIN AND THE DEVELOPMENT OF DEFICIENCY
SYMPTOMS AND THE FATTY LIVER AND KIDNEY SYNDROME

W.L. BRYDEN

The fatty liver and kidney syndrome (FLKS) of young chickens is a biotin-responsive disorder, in which the liver concentration of biotin in birds succumbing to the syndrome is extremely low (Hood et al. 1976). Nevertheless, the view still persists that FLKS results from a marginal biotin deficiency and this belief appears to have arisen from the fact that many birds dying from FLKS are often the heaviest birds in the flock and do not exhibit classical biotin deficiency symptoms. In an attempt to reconcile the contradictory statements in the literature concerning the development of biotin deficiency and the incidence of FLKS, a number of experiments were conducted, in which the tissue concentrations of biotin were determined (Bryden 1982). The results of one study are presented in the table.

The influence of age and dietary biotin supplement on liver, kidney and plasma biotin concentrations (mean \pm SEM: n = 5)

Dietary biotin ($\mu\text{g}/\text{kg}$)	Age (days)	Biotin		
		Liver ($\mu\text{g}/\text{g}$)	Kidney ($\mu\text{g}/\text{g}$)	Plasma (ng/ml)
-	1	2.90 \pm 0.25	-	-
0	4	0.73 \pm 0.10	0.79 \pm 0.28	-
	14	0.53 \pm 0.21	0.67 \pm 0.12	0.85 \pm 0.14
	28	0.24 \pm 0.15	-	0.79 \pm 0.23
75	4	1.00 \pm 0.15	2.06 \pm 0.38	-
	14	1.49 \pm 0.38	2.53 \pm 0.56	1.10 \pm 0.18
	28	1.26 \pm 0.27	-	1.21 \pm 0.11

A very rapid depletion of liver biotin reserves occurs in the immediate post-hatching period but kidney and plasma levels of the vitamin remained unchanged. Biotin deficiency first became apparent when the birds receiving the unsupplemented diet were 18 d old and became severe during the following 10 d. It was not until these birds were 28 d old that their growth rate was depressed ($P < 0.05$). Three birds died during the first 14 d of the trial from FLKS. These birds did not exhibit deficiency symptoms but had mean liver and kidney biotin concentrations of $0.43 \pm 0.09 \mu\text{g}/\text{g}$ and $0.77 \pm 0.16 \mu\text{g}/\text{g}$, respectively.

These observations help explain a number of contradictions between growth rate, biotin deficiency and FLKS. Although the liver may be sufficiently depleted for FLKS to develop, it does appear that it takes a longer time for growth rate to be significantly depressed and for overt signs of deficiency to develop, whereas FLKS can occur spontaneously. The discrepancies in the literature presumably relate to the age of the bird and the period of depleted liver reserves of biotin prior to the occurrence of FLKS. The importance of kidney biotin concentrations in the pathogenesis of FLKS remains to be determined but it is worth noting that increased gluconeogenesis occurs in this tissue in birds suffering from FLKS (Bannister 1979). Finally, as a vitamin deficiency proceeds through a number of different stages which result in biochemical, functional, microscopic, anatomical and, finally, macroscopic defects, the development of FLKS, a result of biochemical and functional defects, might be expected to precede the macroscopic defects of classical biotin deficiency.

BANNISTER, D.W. (1979). *Int. J. Biochem.* **10**: 193.

BRYDEN, W.L. (1982). Ph.D. Thesis, University of Sydney.

HOOD, R.L., JOHNSON, A.R., FOGERTY, A.C. and PEARSON, J.A. (1976). *Aust. J. biol. Sci.* **29**: 429.

Dept. Animal Husbandry, University of Sydney, Camden, New South Wales 2570