Proc. Nutr. Soc. Aust. (1978) 3: 57

INFLUENCES OF THE MEDIA AND THE FOOD INDUSTRY AS THEY MAY AFFECT THE PUBLIC

P. RUBINSTEIN*

This section of the Symposium is presented as an audio-visual session with audience participation and will explore five questions:

1. Why does packaged food get the largest share of Australian advertising expenditure?

2. How do advertisers change our eating habits?

3. Which consumer research methods do they use?

4. What are the lessons here for the nutrition Establishment?

5. What are the side-effects on our physical, mental and social health?

---

* St. Vincent's Community Health Care Centre, 376 Victoria Street, Darlinghurst, N.S.W. 2010.

Proc. Nutr. Soc. Aust. (1978) 3:61

MOLYBDENUM RESPONSIVE SYNDROMES IN CHICKENS

J.A. NELL and E.F. ANNISON
Department of Animal Husbandry, University of Sydney, Camden, 2570.

Two syndromes which occur sporadically in commercial poultry units have been reported to be molybdenum (Mo) responsive. These are the ginger hair, clubbed down syndrome in newly hatched chicks (Payne, 1977a) and the femoral degeneration and scabby hip syndrome in broiler chicks (Payne and Bains, 1975). These data suggested that the availability of Mo from poultry diets occasionally fell below requirement. We have attempted to produce eggs of low Mo content by feeding diets of low Mo content (0.4 µg Mo/g of diet) with and without added tungstate (WO_4), sulphate (SO_4) and copper (Cu), which have been identified as Mo antagonists in other systems. Similar diets containing added Mo (MoO_4) were also fed. The Mo status of the hens was monitored by measuring the Mo content of the eggs, and the Mo level and xanthine dehydrogenase (XDH) activity of liver tissue. The Mo content of eggs produced by hens on the low Mo diet were not significantly reduced by the antagonists, but SO_4 and WO_4 both lowered egg Mo levels on the high Mo diet (Table 1). There were no significant differences ($P<0.05$) in the XDH activities in the livers of hens fed the high and low Mo diets, and the effects on liver Mo levels were similar to those on egg levels, although the results for diets containing tungstate are not yet available.

Eggs from hens fed the low Mo diet (0.4 µg/g) with added SO_4 (3 mg/g) hatched and the chicks developed normally when fed diets containing 0.06 µg Mo/g.

In current experiments we are rearing chicks derived from hens of low Mo status in plastic isolators, using diets containing only 0.03 µg Mo/g. No evidence of Mo deficiency has been observed to date.

In other experiments, attempts to reproduce the Mo responsive loss of feathers condition in broilers observed in this laboratory by Payne (1977b) have been unsuccessful.

TABLE 1

	Mo levels			
	µg Mo/g yolk *		µg Mo/g wet liver *	
	Basal diet	Basal diet + 8 µg Mo/g	Basal diet	Basal diet + 8 µg Mo/g
No addition	0.29 a	2.67 b	0.55 a	1.80 b
0.5 mg Cu/g diet	0.40 a	2.66 b	0.61 a	1.95 b
3 mg SO_4/g diet	0.18 a	0.46 a	0.49 a	0.65 a
1.3 mg WO_4/g diet	0.16 a	0.63 a	Not available	
	($P<0.05$)		($P<0.05$)	

* Means not containing common superscripts differ significantly ($P<0.05$).

REFERENCES

Payne, C.G. (1977a). Involvement of molybdenum in feather growth. Br. Poult. Sci. 18: 427-432.

Payne, C.G. (1977b). Molybdenum responsive syndromes of poultry. Proc. 3rd Int. Symp. on Trace Element Metabolism in Man and Animals, 3.

Payne, C.G. and Bains, B.S. (1975). Femoral degeneration and scabby hip syndrome in chicks. Vet. Rec. 97: 436-437.

Proc. Nutr. Soc. Aust. (1978) 3:62

EFFECTS OF ALCOHOL AND ZINC DEFICIENCY ON TWO RAT ZINC METALLOENZYMES

I.R. RECORD* and I.E. DREOSTI*

The teratogenic effects of zinc deficiency (Hurley & Swenerton, 1966) and chronic alcoholism (Jones & Smith, 1975) have been well established. Following a report by Wang & Pierson (1975) which showed decreased levels of zinc in plasma, liver and muscle tissue of alcoholic rats, it was decided to examine the individual and combined effects of alcohol ingestion and zinc deficiency on two zinc metalloenzymes in rat foetal (20 day) and maternal tissues.

Maternal liver alcohol dehydrogenase (A.D.H.) and foetal liver and brain alkaline phosphatase (A.P.) were selected, as A.D.H. is an important enzyme in the detoxification of ethanol, and A.P. activity has been shown to parallel maturation of rat brain (Cohn & Richter, 1956).

The activity of A.D.H. in the liver fell by approximately 60% in the dams deprived of zinc throughout gestation. Zinc deficient rats receiving 20% alcohol in their drinking water over this period displayed a slightly higher level of A.D.H. Animals supplemented with 500 ppm zinc, and receiving 20% alcohol, had normal levels of the enzyme.

The levels of A.P. in the livers and brains of zinc deficient foetuses were approximately 50% of those found in the controls. When the dams received 20% alcohol, with or without 500 ppm zinc during pregnancy, the levels in both tissues returned to normal.

Zinc balance studies (Dreosti *et al*, 1978) suggest that initially the ingestion of alcohol mobilizes zinc from certain body tissues and leads to a slight elevation in plasma zinc levels. It would appear from these results that some of this plasma zinc is available for the synthesis of hepatic alcohol dehydrogenase as well as for synthesis of alkaline phosphatase in the foetus.

REFERENCES

COHN, P. & RICHTER, D. (1956). *J. Neurochem.* 1:66.
DREOSTI, I.E., BUCKLEY, R.A. & RECORD, I.R. (1978). In preparation.
HURLEY, L.S. & SWENERTON, H. (1966). *Proc. Soc. Exp. Biol. Med.* 123:692.
JONES, K.L. & SMITH, D.W. (1975). *Teratology* 12:1.
WANG, J. & PIERSON, R. (1975). *J. Lab. Clin. Med.* 85:50.

*CSIRO Division of Human Nutrition, Kintore Avenue, Adelaide, S.A. 5000.