

## SULPHUR AMINO ACIDS AND TIBIAL DYSCHONDROPLASIA IN CHICKS

P. RAJADEVAN and T.L. FRANKEL

Tibial dyschondroplasia (TD) is an idiopathic condition in broiler chicks usually diagnosed by an abnormal widening at the tibial growth plate. Dietary imbalances in calcium, phosphorus and fat-soluble vitamins, genetic factors and mycotoxins may induce TD (Sauveur 1984). Experimental induction of TD is capricious but we have been able to induce TD using semi-purified diets with excess sulphur amino acids (SAA). We report here results of two experiments conducted with two strains of broiler chicks.

Day-old chicks were assigned on a weight basis to eight pens of either 10 for Experiment 1 (Chubby Chick (CC); Fantasy Farm Ltd., Warrandyte) or 22 for Experiment 2 (Inghams TM (I); Inghams Hatcheries, Pakenham). In both experiments, food and water were available ad libitum. Diets, formulated to meet National Research Council (1984) standards, consisted of glucose, maize starch, cellulose fibre, sunflower seed oil, vitamins and minerals and isolated soya protein (Ardex F, Oppenheimer Aust. Pty. Ltd.) of stated amino acid content. Three diets were prepared using added methionine to give SAA contents, relative to lysine, of 0.75X (low SAA), 1.0X (control) and 1.5X (HM) the requirement. A fourth diet (HC) contained added cysteine hydrochloride to give an SAA content equivalent to diet HM. At two and four weeks, four or five chicks from each pen were anaesthetised, bled, then killed for removal and examination of tibial bones.

Body weights, plasma calcium and inorganic phosphate were not different. At four weeks, the incidence of TD in CC birds was 38% for those fed HM and 88% for HC. Comparable rates were 30% and 40% for I birds. For controls, 13% of CC and none of the I birds were affected, and for the low SAA diet, 13% and 10% respectively had lesions. Pooled results from the two experiments are shown in the Table.

| Diet    | 2 weeks      |              |                       |   |   | 4 weeks      |              |          |   |   |
|---------|--------------|--------------|-----------------------|---|---|--------------|--------------|----------|---|---|
|         | Total Killed | No. Affected | TD Score <sup>†</sup> |   |   | Total Killed | No. Affected | TD Score |   |   |
|         |              |              | 1                     | 2 | 3 |              |              | 1        | 2 | 3 |
| Control | 16           | 1            | 1*                    | 0 | 0 | 18           | 1            | 1        | 0 | 0 |
| Low SAA | 16           | 2            | 2                     | 0 | 0 | 18           | 2            | 1        | 1 | 0 |
| HM      | 16           | 9            | 8                     | 1 | 0 | 18           | 6            | 2        | 3 | 1 |
| HC      | 16           | 8            | 7                     | 1 | 0 | 18           | 11           | 5        | 3 | 3 |

\* No. affected; † growth plate slightly (1), obviously (2), thickened or cartilaginous (3)

It seems that imbalances in SAA can lead to the development of TD and it may be possible to probe the mechanisms responsible for its development using birds in which TD has been induced by SAA imbalances.

SAUVEUR, B. (1984). *Wld. Poult. Sci.* **1**, 40:195.

NATIONAL RESEARCH COUNCIL (1984). *Nutrient Requirements of Poultry* (National Academy of Sciences: Washington).

School of Agriculture, La Trobe University, Bundoora, Victoria 3083