

## The Role of Nutrition in Tibial Dyschondroplasia Occurrence in Turkeys

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### Introduction

A high incidence of tibial dyschondroplasia (TD) in turkeys was reported by Hirt et al 1997. As a result a large project (EU 2000) was set up involving research institutes in Germany, Netherlands, Switzerland and the UK to investigate its causes. This paper is built around the results from the project allied to associated work. Some factors covered are indirectly influenced by nutrition; others are purely nutritional.

### The Influence of Production Traits

This was investigated at Roslin Institute (EU Project 2005a). 480 turkeys of six different strains (n= 80/strain) were reared to 18 weeks. The strains differed markedly in production characteristics, i.e. unselected (unsel), slow growing - broad breasted (S-B), slow growing - narrow breasted (S-N), fast growing - narrow breasted (F-N), large, commercial (comm), fast growing, broad breasted (F-B) as seen in Table 1.

Table 1: Strain, bodyweight at 18 weeks and breast muscle mass

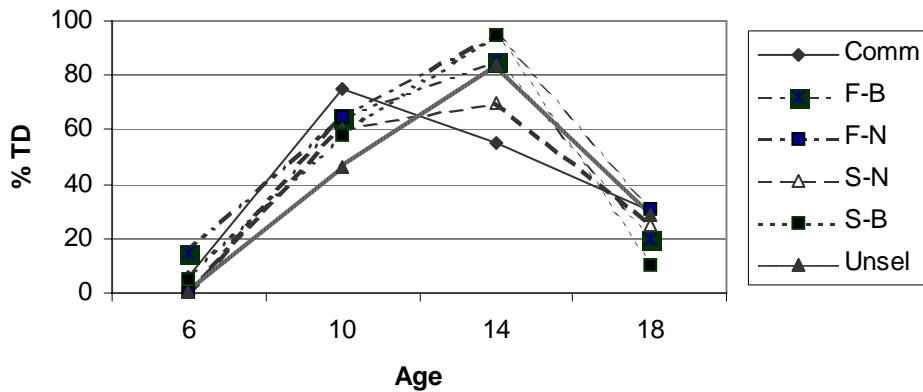
Strain	Comm	F-B	F-N	S-N	S-B	Unsel
Bodyweight (kg)	19.5	19.0	16.8	11.8	10.3	6.6
Breast muscle mass %	25	26	24	24	29	17

Figure 1 shows that the unselected turkeys with slow growth rate and small breast muscle mass had a similar peak incidence of TD as the fast growing heavy strains with large breast muscle mass. In all strains, peak TD incidence coincided with the most rapid phase of growth. The lesion also reduces sharply with age past the peak level indicating recovery.

The presence or severity of a TD lesion had no effect on skeletal morphometry, nor did it affect any gait parameters. It was concluded that TD appears not to have any primary consequences for the welfare of the animal.

This was confirmed by work at the same research institute (EU project 2005b), which found that gait and behaviour did not differ significantly between turkeys with or without TD. There was also no association with joint infection/synovitis. Osteomyelitis was observed in 10% of TD-affected birds and in no non-TD birds. The osteomyelitis appeared to be associated with the retained cartilage. While this suggests that osteomyelitis may be a secondary consequence of TD in a small proportion of growing turkeys, gait and behaviour were unaffected.

**Figure 1: The Influence of Growth and Conformation**



### The Influence of Temperature

The effect of ambient temperature on the development of tibial dyschondroplasia was studied by the Centre for Applied Poultry Research in the Netherlands (EU Project 2005c). Two strains of fast growing type male turkeys were grown at either 18 or 28°C from 42 to 140 days. At 41, 62, 83, 104 and 125 days of age, 30 turkeys per treatment (5 replicates of 6 turkeys within a treatment) were randomly chosen to score for TD.

Locomotion disorders were not observed during the entire experiment. In spite of that, the incidence of TD increased from 41 to 104 days of age and decreased from 104 to 125 days of age (Table 2.)

Table 2. Incidence (%) of turkeys with a TD lesion at different ages

Temp. (°C)	Strain	Age,d				
		41	62	83	104	125
18		6.7	21.7	43.3	68.3	10.0
28		6.7	11.7	38.3	53.3	1.7
	A	5.0	15.0	28.3	63.3	8.3
	B	8.3	18.3	53.3	58.3	3.3
18	A	6.7	16.7	30.0	66.7	13.3
18	B	6.7	26.7	56.7	70.0	6.7
28	A	3.3	13.3	26.7	60.0	3.3
28	B	10.0	10.0	50.0	46.7	0.0

There was also a numerical but not significant effect of temperature. Turkeys at 18°C seemed to have a higher TD incidence than 28°C at almost all ages. At 104 days of age TD lesions were more severe in turkeys at 18°C than in turkeys at 28°C. However the temperature effect is confounded by different growth rate and feed consumptions on the two temperature regimes as seen in Table 3.

Table 3: Growth rate and food consumptions on two temperature regimes.

Temperature (°C)	Age interval,d		
	28 - 63	63 -105	105 - 140
a) Body weight gain (g/d)			
18	141.4	171.8	191.8
28	119.8	141.9	150.4
b) Food consumption (g/d)			
18	246.2	394.0	429.5
28	206.9	321.0	345.6

It could be interpreted that the incidence of TD interacts with body weight gain within the same genetic strain of turkey, as opposed to growth rate per se.

### The Growth Pattern

The effect of restricting early growth rate on development of tibial dyschondroplasia was investigated at the same institute (EU project 2005d ). The growth rate for each age period and the percentage of TD through the growth period is shown in Table 4.

The results indicated no benefit from reducing early growth rate. There was an indication that the TD incidence was associated with the peak speed of growth.

**Table 4: Growth rate and % TD incidence**

#### a) Growth rate (g/d)

Age period (d)	1-22	23-57	58-85	86-107	108-147
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#### Diet

L90-8wk	25	114	186	154	146
L90-10wk	26	114	187	144	159
L9 - 12 wk	25	114	188	156	153
Control	30	123	191	134	152

#### b) % TD incidence

Age (d)	44	64	77	107	128
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#### Diet

L90-8wk	0	8.3	25	29.2	12.5
L90-10wk	4.2	12.5	25	16.7	8.3
L90-12wk	8.3	20.8	41.7	20.8	12.5
Control					

**L90 = 95% of NRC (1994) Lysine requirement followed by 105%.**

**Control 100% of NRC (1994) Lysine requirement.**

### **The Influence of Sex**

**It is a field observation supported by scientific evidence (Glass, 1971; Buffington et al, 1975) that male turkeys have a higher incidence of leg problems, typically long bone distortion than females.**

**The Centre for Applied Poultry Research in the Netherlands carried out an investigation into the incidence of TD in male and female turkeys, (EU project 2005e) using the fast growing BUT Big 6 commercial strain. The TD incidence is shown in Table 5.**

**Table 5: Incidence (%) of turkeys with TD in each sex.**

	<b>Age,d</b>				
<b>Sex</b>	<b>42</b>	<b>63</b>	<b>84</b>	<b>105</b>	<b>126</b>
<b>Male</b>	<b>8.3</b>	<b>36.7</b>	<b>60.0</b>	<b>36.7</b>	<b>6.9</b>
<b>Female</b>	<b>1.7</b>	<b>33.9</b>	<b>65.5</b>	<b>3.5</b>	<b>N/A</b>

**As will be seen, the TD incidence increases from 42 days of age onwards to a peak at 84 days, after which the TD incidence decreased in both sexes, almost disappearing in females by 105 days. The incidence approximate to the growth pattern in both sexes,**

**with recovery occurring after the growth rate slows down.**

**The higher incidence of leg problems in male turkeys than female turkeys does not therefore correlate with the incidence of TD seen in the sexes at the ages when leg problems tend to manifest themselves i.e. around 12 weeks of age.**

### The Influence of Minerals and Vitamins

Sometimes interesting information comes to light arising from an unplanned occurrence during an experiment. Such an occurrence happened during an experiment at the Centre for Applied Poultry Research in the Netherlands (E.U. project 2005f). This experiment planned to repeat the experiment outlined earlier where breeds of different growth rates and breast shape were compared. Unfortunately, the feed mill omitted the vitamin and mineral premix from the second diet fed from 14 to 35 days. This came to light when severe locomotion problems were seen at four weeks of age; to the extent that all birds were showing locomotion problems and sitting down. On changing to the next diet, most birds recovered quickly. Those that did not were culled. The experiment was continued to investigate the effects on subsequent TD development. The results are shown in Table 6.

Table 6 : Incidence (%) of turkeys with a TD lesion at different ages after early mineral and vitamin deficiency.

	Age,d				
	46	65	84	105	126
<b>Growing Strain</b>					
Fast	92.3	88.9	82.6	76.5	26.8
Slow	29.8	56.1	62.5	66.1	0.0
Breast strain					
Broad	52.0	50.0	64.8	66.7	15.0
Narrow	70.8	89.0	78.5	74.6	14.1
Growing strain * Breast Strain					
Fast * Broad	82.8	75.0	83.3	75.0	25.0
Fast * Narrow	100.0	100.0	82.1	77.8	28.1
Slow * Broad	9.5	16.7	41.7	58.3	0.0
Slow * Narrow	41.7	78.6	75.0	71.9	0.0

**These show that the occurrence of the TD lesion was initiated much earlier than in previous experiments and was also adversely related to the speed of growth. This was particularly so if the strain was narrow breasted, which is also longer in the leg than the broad breasted strain.**

**The requirements for minerals and vitamins might be expected to be higher in fast growing strains than in slow growing strains. The experiment demonstrated that early mineral and vitamin nutrition, if deficient early in life, could have an effect on TD occurrence subsequently.**

**The timing of any mineral deficiency may be important. Hocking et al (2002) investigated 4 concentrations of calcium (6, 10, 14 and 18g/kg) and available phosphorus (3,5,7 and 9g/kg) in a 4 x 4 factorial experiment commencing at 4 weeks of age. There were no gross TD lesions at 4 weeks of age and at 7 weeks of age only 2% had a lesion. The TD incidence at 10 and 13 weeks is shown in Table 7.**

**Table 7: Mean prevalences (%) of turkeys with tibial dyschondroplasia at 10 and 13 weeks of age and mean area (cm<sup>2</sup>) of lesion at 13 weeks in male turkeys fed on diets with different concentrations of calcium and available phosphorus.**

Dietary calcium (g/kg)	Dietary available phosphorus (g/kg)			
	3	5	7	9
Prevalence of TD at 10 weeks; SED 9.8				

6	17	42	17	42
10	58	67	50	25
14	92	58	58	42
18	42	67	58	67
Prevalence of TD at 13 weeks; SED 9.8				
6	83	58	75	25
10	75	75	75	75
14	83	83	75	67
18	75	67	67	75
Mean area of TD lesion (cm <sup>2</sup> ); SED 1.56				
6	1.46	1.01	1.22	1.81
10	1.19	3.33	4.10	0.97
14	2.49	4.42	2.41	1.94
18	1.26	1.09	1.96	2.20

**The lesion was localised in the caudal aspect of the proximal tibiae. This is consistent with the other work reported in this paper. Dietary calcium and available phosphorus did not affect the prevalence of the lesion except in turkeys on the diet containing 6g calcium/kg where body weight and the incidence of TD were low.**

**Despite the wide range in calcium and available phosphorus levels, histological investigation showed no evidence of rachitic changes.**

Vitamin D<sub>3</sub> or its Metabolites

**Dietary supplementation with Vitamin D metabolite has shown to alleviate tibial dyschondroplasia in broilers. 1,25- dihydroxyvitamin D<sub>3</sub> (1,25-D) is the most effective metabolite and can completely prevent the condition (Edwards, 1990, Rennie et al, 1993). Another metabolite, 25- hydroxyvitamin D<sub>3</sub> (25-D) can also be effective but with less reproductibility (Rennie and Whitehead, 1996). Sanders and**

**Edwards (1991) found little evidence that 1, 25-D had any effect on TD in turkeys.**

**An experiment was carried out at Roslin Institute (EU project 2005g) to further investigate the effect of Vitamin D<sub>3</sub> and its metabolites in turkeys. Day old BUT Big 6 male turkey poults were used and reared to ten weeks of age and fed on one of four experimental diets. The dietary treatments were:**

- 2000 IU vitamin D<sub>3</sub>/kg (control)**
- 5000 IU vitamin D<sub>3</sub>/kg**
- 125 µg 25-D/kg with no vitamin D<sub>3</sub>**
- 5 µg 1,25-D/kg with 2000 IU vitamin D<sub>3</sub>/kg**

**The birds were euthanised at 10 weeks of age and both tibiotasi were dissected out. The right tibiotassus was subjected to destructive three point bending test and the left tibiotassus was used to assess the incidence and severity of TD.**

**The results are shown in Tables 8 and 9 below:**

Table 8: Liveweight, tibia dimensions and biomechanics in turkeys at 10 weeks of age fed diets containing vitamin D<sub>3</sub> or its metabolites.

	2000 IU Vitamin D <sub>3</sub>	5000 IU Vitamin D <sub>3</sub>	125 µg 25-D	5 µg 1,25(OH) <sub>2</sub> D <sub>3</sub>	sed	Significance of effect
Liveweight (g)	7200	7311	7060	7314	176.5	NS
Tibia weight (g)	79.8	80.3	81.4	81.5	1.98	NS
Tibia	18.9	19.2	19.0	19.0	0.14	NS

length (cm)						
Tibia	1.61	1.59	1.63	1.62	0.033	NS
width (cm)						
Breaking strength (N)	1098	1003 <sup>a</sup>	117 <sup>b</sup>	1187 <sup>b</sup>	54.4	*
Stiffness (N/m)	484701 <sup>b</sup>	477394 <sup>b</sup>	485213 <sup>b</sup>	533142 <sup>a</sup>	17724	**

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**Table 9 : Incidence and mean dimensions of lesions of tibial dyschondroplasia in the proximal tibiae of turkeys at 10 weeks of age fed diets containing vitamin D<sub>3</sub> or its metabolites.**

	<b>2000 IU Vita min D<sub>3</sub></b>	<b>5000 IU Vita min D<sub>3</sub></b>	<b>125 µg 25-D</b>	<b>5µg 1,25(OH)<sub>2</sub> D<sub>3</sub></b>	<b>sed</b>	<b>Signifi cance of effect</b>
<b>Incidence of TD (%)</b>	<b>85</b>	<b>70</b>	<b>76</b>	<b>81</b>		<b>NS</b>
<b>Lesion Area (mm<sup>2</sup>)</b>	<b>15.0</b>	<b>7.4</b>	<b>17.3</b>	<b>9.0</b>	<b>5.27</b>	<b>NS</b>
<b>Lesion length (mm)</b>	<b>3.81</b>	<b>2.45</b>	<b>4.14</b>	<b>3.14</b>	<b>0.90 3</b>	<b>NS</b>
<b>Lesion width (mm)</b>	<b>6.07</b>	<b>4.26</b>	<b>5.92</b>	<b>5.52</b>	<b>0.98 3</b>	<b>NS</b>

There were no significant effects of dietary treatment on bird or tibia weights or tibia dimensions. The tibia breaking strength did not differ significantly between the control and other treatment groups but tibia stiffness was significantly (P<0.01) greater in the birds fed 1,25-D than in all other treatments.

**The overall incidences of TD were very high in all treatments but there was no difference between the treatments in incidences or mean sizes of lesions.**

**The results for 1,25-D confirm the previous observation of Sanders and Edwards (1991) that dietary supplementation with this metabolite does not affect the occurrence of TD in turkeys. The results also show that 25-D is similarly ineffective. The contrast between these observations and those in broilers strongly suggests that TD has different aetiologies in these two species.**

**Other possible causes**

**To date in turkeys, only the more obvious possible causes have been investigated. These investigations have shed little light on the cause of TD in turkeys. They have however shown it to be occurring at high incidence, albeit that the lesion is mild and disappears with age.**

**In broilers it has been shown to be induced by Fusarium infected feedstuff (Chu et al, 1996). Various dithiocarbamates such as thiram have been used to induce very high incidence of TD (Edwards, 1987; Rath et al, 2004). These are used as fungicides and seed fumigants. Their effect can be prevented by copper supplementation which opens up the possibility that a trace mineral deficiency is a causal**

**factor. Little work has been carried out on the turkeys' trace mineral requirement.**

## Conclusions

**Tibial Dyschondroplasia is a widespread mild lesion in both sexes of turkeys with peak incidence around 11 to 14 weeks. It disappears with age. Gait and behaviour are unaffected. Osteomyelitis may be a secondary consequence in a small proportion of the turkeys. The incidence is not influenced by the genetic potential for growth or the conformation of the turkeys.**

**Calcium and phosphorus levels are not implicated in the problem. There is some evidence that a vitamin or trace mineral deficiency early in life can influence the subsequent occurrence of TD. Vitamin D does not appear to be implicated.**

## References

- BUFFINGTON, D.E.** 1975: The incidence of leg and foot abnormalities in Wrolstad White turkeys. *Poultry Science* **54**: 457-461.
- EDWARDS, H.M., Jr.** 1987. Effects of thiuram, disulfiram and a trace element mixture on the incidence of tibial dyschondroplasia in chickens. *J. Nutr.* **117**: 964 - 969
- EDWARDS, H.M. Jr.** 1990. Efficacy of several vitamin D compounds in the prevention of tibial dyschondroplasia in broiler chickens. *Journal of Nutrition*, **120**: 1054-1061.
- E.U.** (2000) The role of selection and husbandry in the development of locomotory dysfunction in turkeys. Project acronym. Turkey gait disorders. Contract number QLRT-1999-01549
- E.U. PROJECT** (2005a) Work package 1 of E.U. Project (2000)
- E.U. PROJECT** (2005b) Work package 2 of E.U. Project (2000)
- E.U. PROJECT** (2005c) Work package 6 of E.U. Project (2000)
- E.U. PROJECT** (2005d) Work package 7 of E.U. Project (2000)
- E.U. PROJECT** (2005e) Work package 8 of E.U. Project (2000)
- E.U. PROJECT** (2005f) Work package 9 of E.U. Project (2000)

- GLASS, S.E.** 1971. Diagnostic Laboratory observations on the incidence and significance of various leg weakness syndrome. Pages 79 - 83 inc. *Proceedings of the Symposium on Leg Weakness in Turkeys*. Iowa State University, Ames, Iowa, USA.
- HIRT, H., REINMANN, M., and H.O.** (1997) Leg problems in fattening turkeys. 5<sup>th</sup> *European Symposium on Poultry Welfare*, Wageningen, Wageningen Agricultural University and the Institute of Animal Science and Health: 80 - 82
- HOCKING, P.M., WILSON, S., DICK, L.N., ROBERTSON, G.W. and NIXEY, C** (2002) Role of dietary calcium and available phosphorus in the aetiology of tibial dyschondroplasia in growing turkeys. *British Poultry Science* **43**: 432 - 441
- RATH, N.C., HUFF, W.E., BALOG, J.M., and HUFF, E.R.** (2004) Comparative efficacy of different dithiocarbonates to induce tibial dischondroplasia in poultry. *Poultry Science* **83**: 266 - 274
- RENNIE, J.S., WHITEHEAD, C.C., and THORP, B.H.** (1993). The effect of dietary 25- and 1- hydroxycholecalciferol in preventing tibial dyschondroplasia in broilers fed on diets imbalanced in calcium and phosphorus. *British Journal of Nutrition*, **69**: 809-816.
- RENNIE, J.S., WHITEHEAD, C.C., and THORP, B.H.** (1996) The effectiveness of dietary 25- and 1-hydroxycholecalciferol in preventing tibial dyschondroplasia in broiler chicken. *British Poultry Science*, **37**: 413-421.
- VARGAS, M.I., LAMAS, J. and ALVARGENGA, V.** 1983  
Tibial dyschondroplasia in growing chicken experimentally intoxicated with tetramethylthiuram disulfide. *Poultry Science*: **62**: 1195 - 1200
- VELTMAN, J.R., Jr. and LINTON, S.S.** 1986. The influence of dietary tetramethylthiuram disulfide (a fungicide) on growth and incidence of tibial dyschondroplasia in single comb white leghorn chicks. *Poultry Science*. **65**: 1205 - 1207
- WU, W., COOK, M.E. and SMALLY, E.B.** 1990. Prevention of thiram induced dyschondroplasia by copper. *Nutrition Research* **10**: 555-567