Nutritional factors in broiler bone problems

Colin Whitehead

Roslin Institute
Edinburgh, Scotland

Outline of talk

- Introduction
- Mechanisms of bone growth
  - Endochondral bone growth
  - Intramembranous ossification
- Problems of endochondral bone growth
  - Bone necrosis
  - Rickets
  - Tibial dyschondroplasia
  - Calcium/phosphorus
  - Vitamin D
- Problems of intramembranous ossification
  - Black bone syndrome
  - Vitamin D

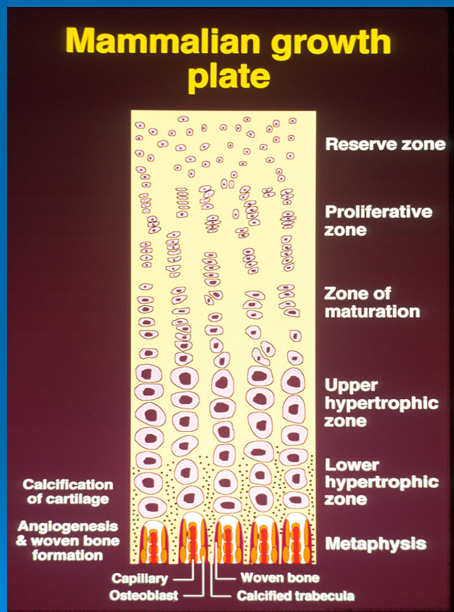
Introduction

- Broiler leg problems severe 15 years ago
- Improvements in breeding, management
  - Fewer non-specific leg problems

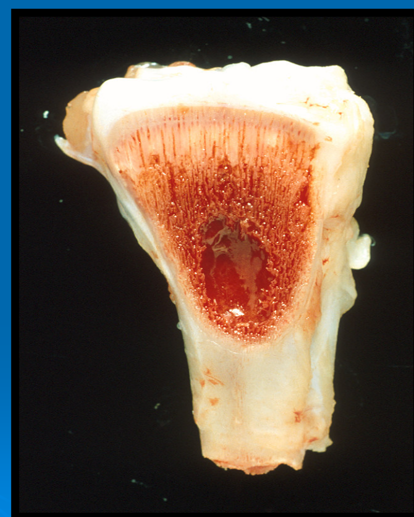
    (e.g. valgus/varus)
- Bone problems still occur
  - changing pattern, new problems
- Continued selection leading to genetic changes in bone structure
- New nutritional requirements

Mechanisms of bone growth

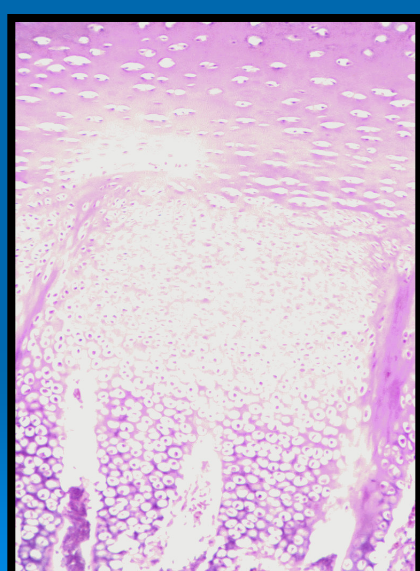
- Endochondral bone growth
  - Elongation of long bones
  - Growth plate chondrocytes
    - Proliferate (multiply)
    - Differentiate (change character)
    - Hypertrophy (enlarge)
    - Mineralise (bone formation)

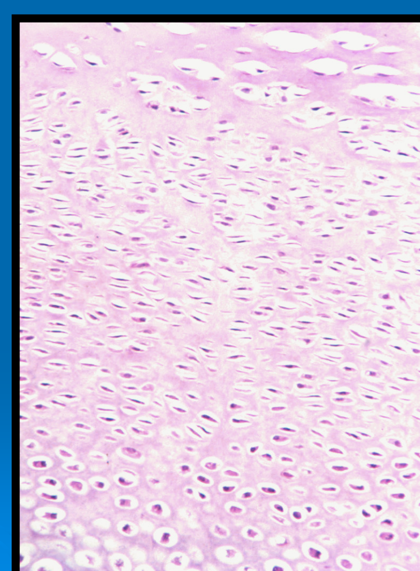


Broiler proximal tibia



Growth plate of proximal tibia in a broiler



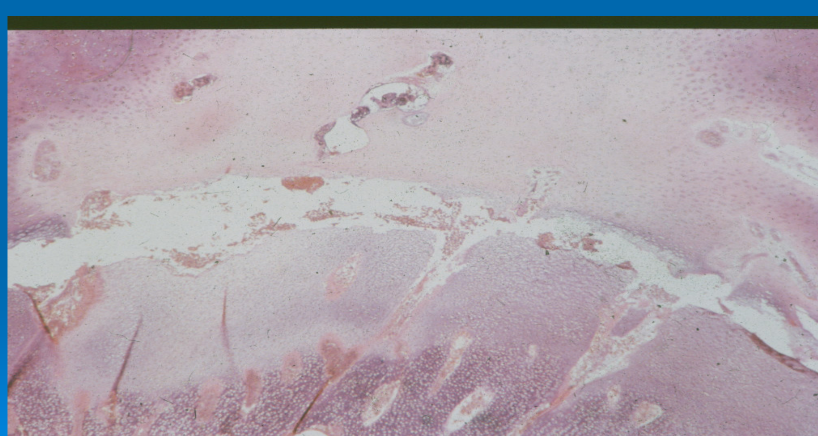


Femoral head necrosis

- Most common broiler leg problem
- Welfare problem – painful
- Separation of femoral head epiphyseal cartilage
- Fracture of femur

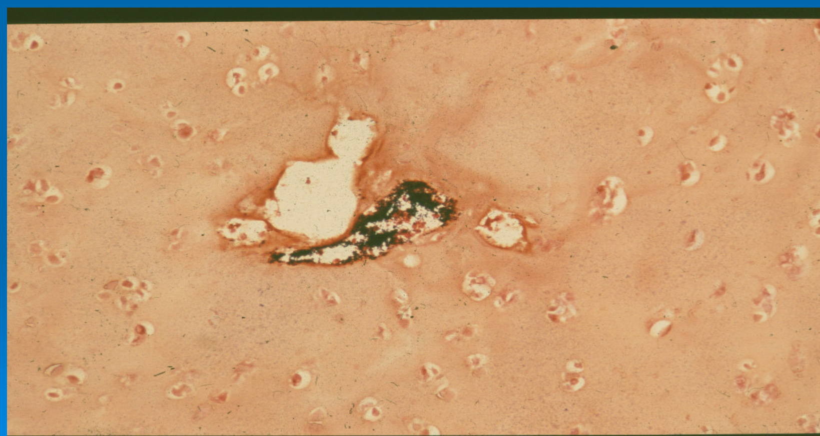
Femoral head necrosis

Cartilage lesion



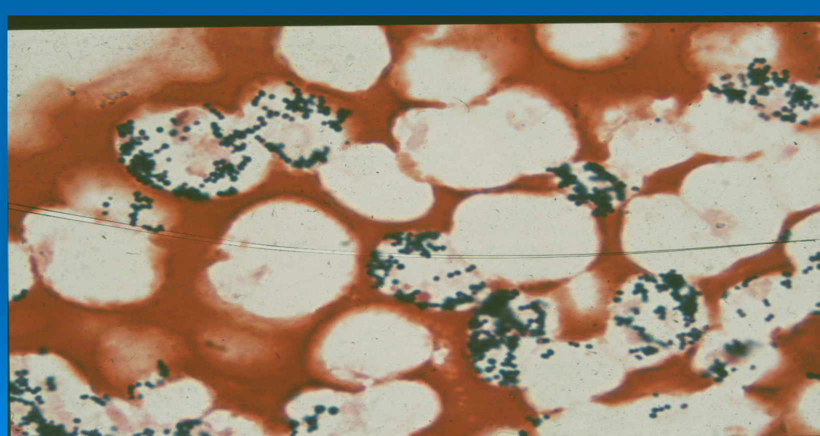
Femoral head necrosis

Bacterial invasion



Femoral head necrosis

Bacterial invasion of chondrocytes



Separation of femoral epiphysis from overlying cartilage

Femoral head necrosis

Necrosis of proximal tarsus

Femoral head necrosis

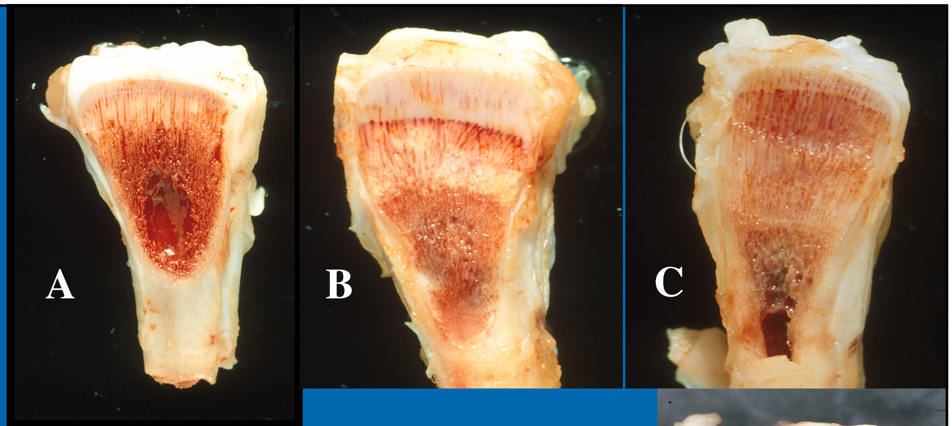
- Bacterial problem
  - Stapphylococcus aureus, E. coli
- Antibiotic treatment
  - Tylosin
- Hatchery hygiene
  - Dirty eggs
  - Human contact
- Nutrition
  - Avoid cartilage defects
- Immunosuppression
  - Genetic
  - Diseases (IBD)
  - Nutrition

Lesions of proximal tibia

- Rickets
  - Ca/P/vit D deficiency
  - Malabsorption
- Tibial dyschondroplasia
  - Genetic problem
  - Nutritional solutions

Calcium, Phosphorus, Vitamin D

- Calcium/vitamin D deficiency
  - Hypocalcaemic rickets
- Phosphorus deficiency
  - Hypophosphataemic rickets
- Calcium/phosphorus imbalance
  - Rickets, tibial dyschondroplasia (TD)



A Normal
B Ca/D deficiency rickets
C P deficiency rickets
D Tibial dyschondroplasia

Calcium deficiency rickets (field case)

Bone composition

30% Matrix   mainly crosslinked collagen microfibrils

70% Bone mineral   hydroxyapatite crystals
$Ca_{10}(PO4)_6(OH)_2$ - hexagonal rods, 200x50A

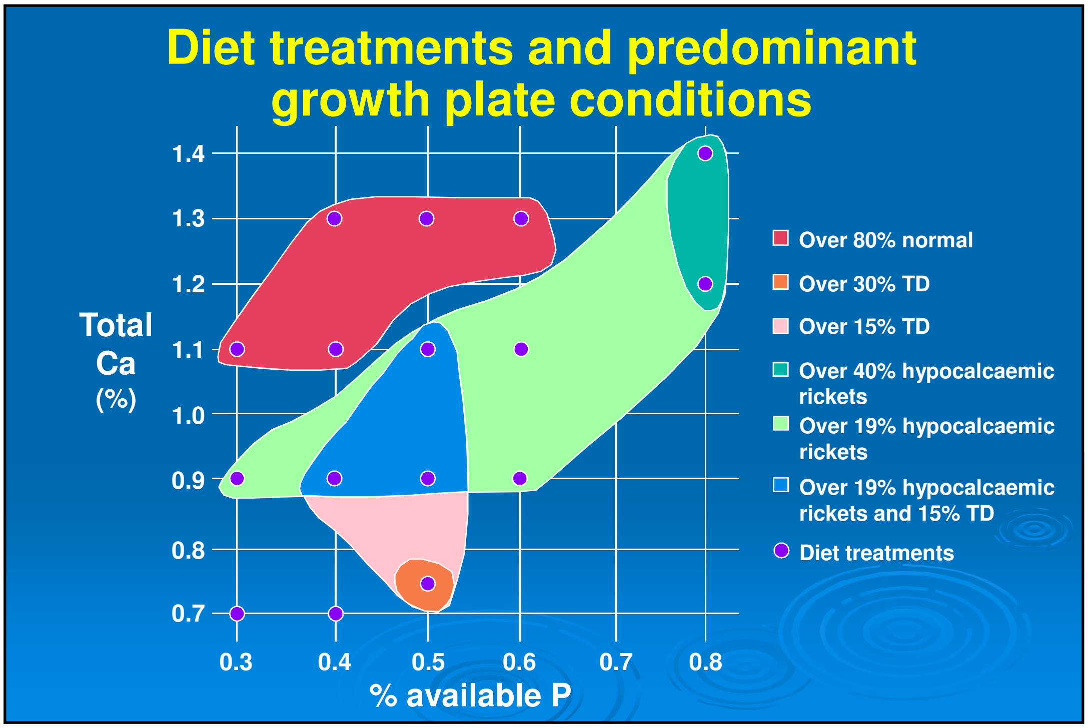
Bone mineral composition (%)

Calcium	26.7	Phosphate	12.5
Magnesium	0.4	Carbonate	3.5
Sodium	0.7	Citrate	0.9
Potassium	0.06	Chloride	0.08
Strontium	0.04	Fluoride	0.07

Ca/P in bones of broiler strains

Age (d)	Old strain	Latest strain
4	2.16	1.85
11	2.09	2.43
18	1.90	2.56
25	2.36	1.81
32	1.70	2.15
39	2.08	2.20

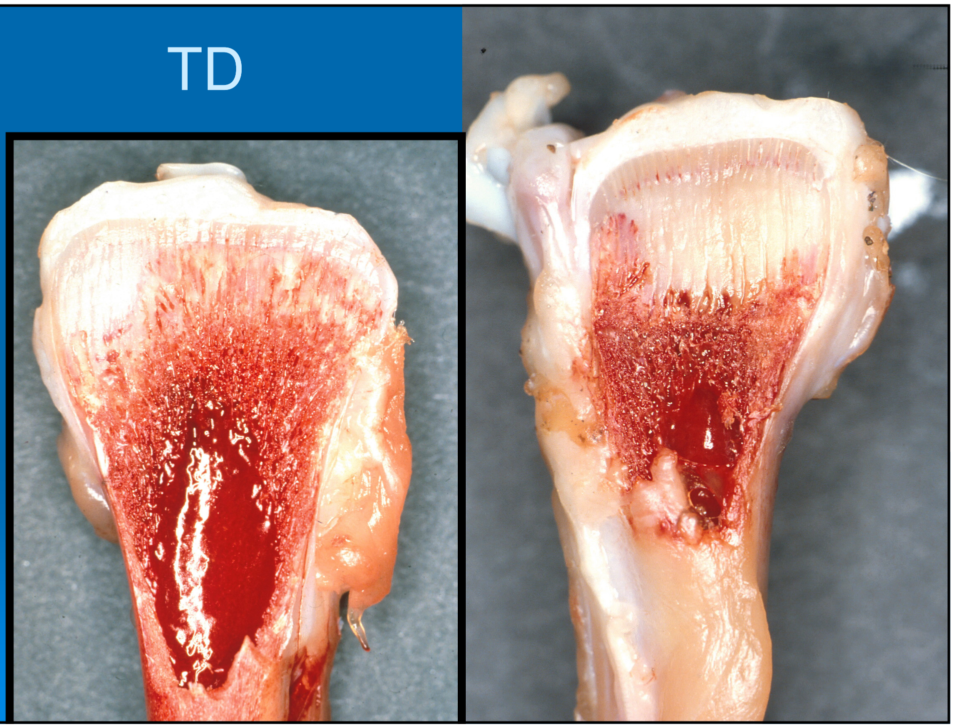
(Williams et al, 2000)



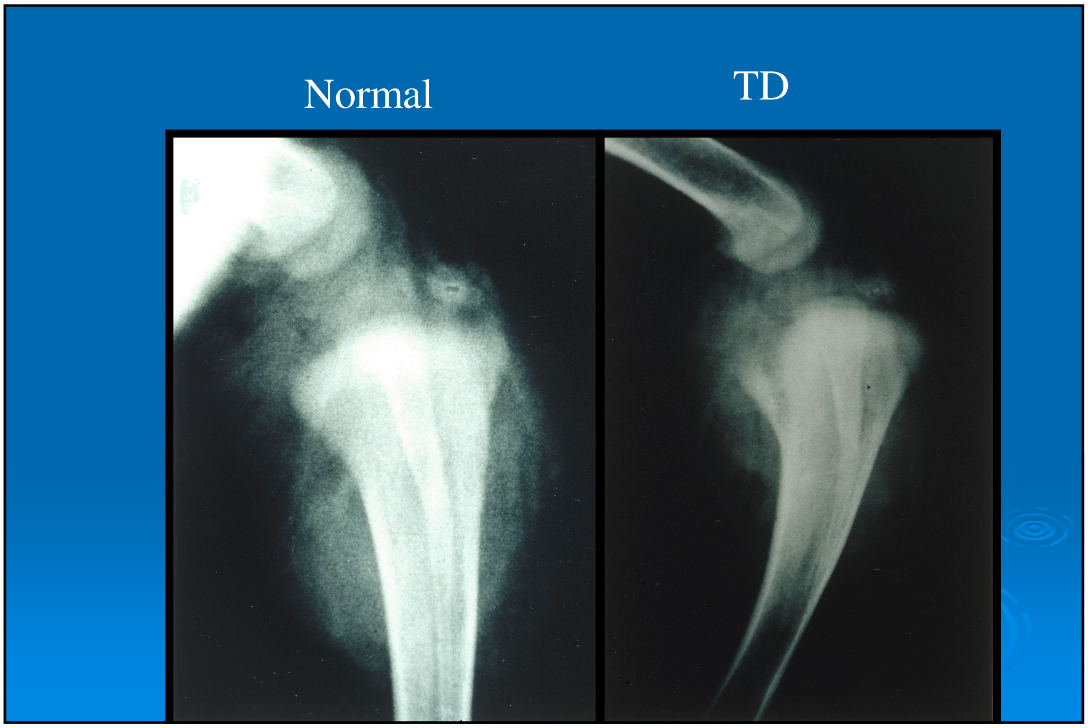
Conclusion Ca/P

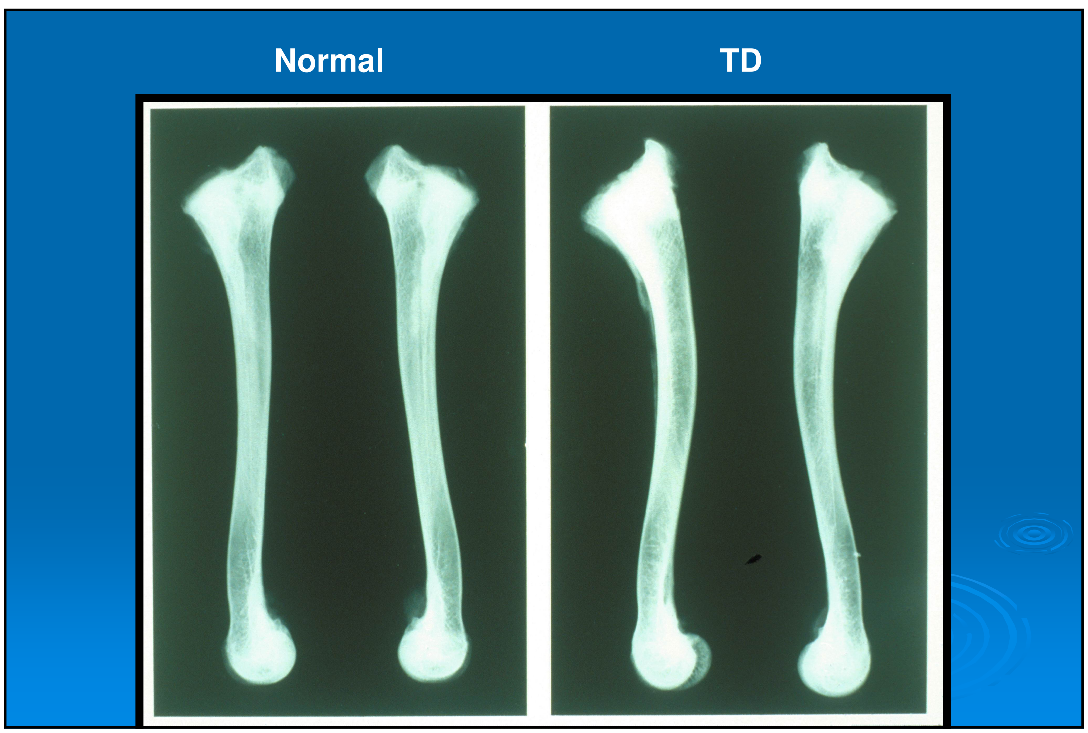
- Ca requirement may be higher for starter diets: 1.1-1.2%
- P requirement unchanged: 0.45%

TD



Field cases of severe TD


Normal
TD


Normal
TD

Nutritional Factors in TD

Ca/P
Ionic balance (Na + K -Cl)
Cysteine
Thiuram, disulphiram
Mycotoxins
Trace minerals (Zn, Cu, Mo)
Vitamin D metabolites

Vitamin D metabolites

Prevention of TD by 1,25-dihydroxyvitamin D

	Diet content (µg/kg)		TD (%)
	Vit D	1,25-D	
Expt 1	25	0	47
	75	0	51
Expt 2	25	0	36
	25	2.5	19
	25	5	0
	25	10	0

Vitamin D metabolites

Prevention of TD by 25-hydroxyvitamin D

	Diet content (µg/kg)		TD (%)
	Vit D	25-D	
Expt 1	75	0	64
	0	75	10
Expt 2	75	0	28
	0	75	19
	0	250	2.5

Vitamin D Metabolites and TD

- 1,25-Dihydroxyvitamin D

  Most effective (3-5 µg/kg)
  Interaction with Ca (hypercalcaemia)
  Safety margin x2

- 25-Hydroxyvitamin D

  Variable effectiveness (70 µg/kg)
  Growth benefit ?
  Safety margin x10
  Commercially available (HyD)

Incidences (%) of tibial dyschondroplasia

	Dietary		Dietary vitamin D_3 (IU/kg)			
	Ca (%)	avP (%)	200	800	5000	10000
Experiment 1						
	1.00	0.45	78[a]	39[b]	4[c]	4[c]
	0.80	0.35	88[a]	51[b]	6	8[c]
Experiment 2						
	0.80	0.35	78[a]	84[a]	22[b]	0[b]
	0.80	0.50	79[a]	78[a]	52[b]	20[b]
	1.30	0.35	4	0	4	0
	1.30	0.50	40[a]	39[a]	8[b]	0[b]

Values followed by different letters in a row differ significantly ($P<0.05$)

Conclusions – vitamin D

- Vitamin D requirements can be high for the young broiler – above 5000 IU/kg in starter diets
- This may be related to the requirements of fast growing broiler strains for higher calcium levels in starter diets

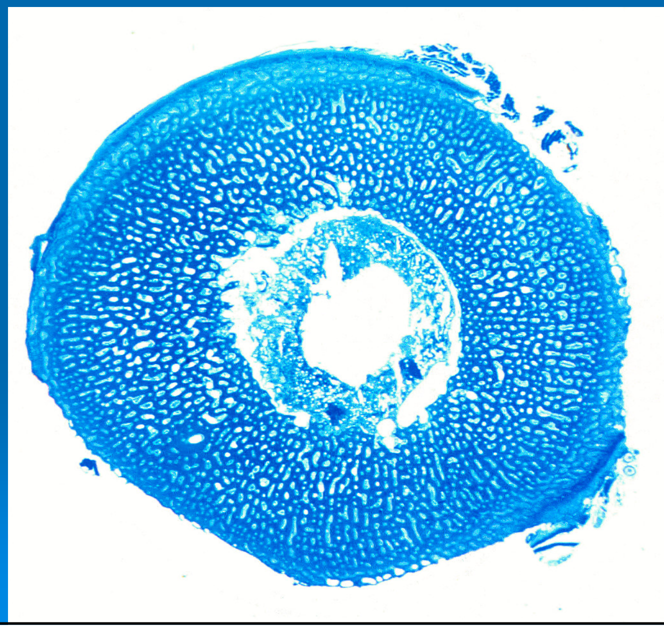
Vitamin D limits

- Max diet content - 5000 IU/kg
- Toxic threshold – 40000 IU/kg
- New legal limit – 10000 IU/kg?
- Starter diets only?

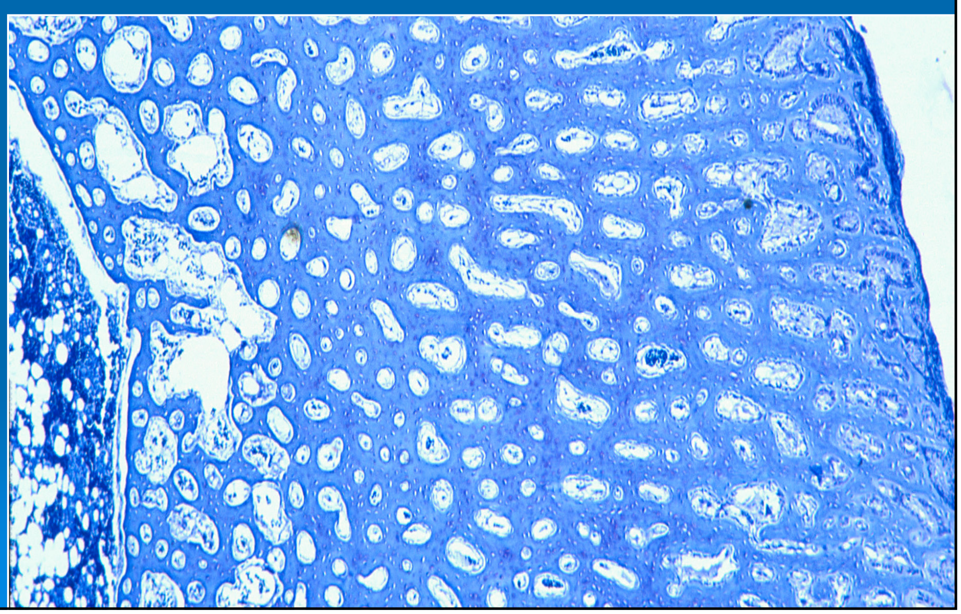
Mechanisms of bone growth

- Intramembranous ossification
  - Mechanism by which leg bone grow wider
  - Formation of matrix and mineral (cortical bone) by osteoblasts on outer (periosteal) surface
  - Incomplete mineralisation so holes left in cortical bone
  - Resorption of bone on inner (endosteal) surface
  - Bone widens as expanding ring
  - Continued genetic selection for fast growth is leading to greater porosity of cortical bone

Cross-section of tibia

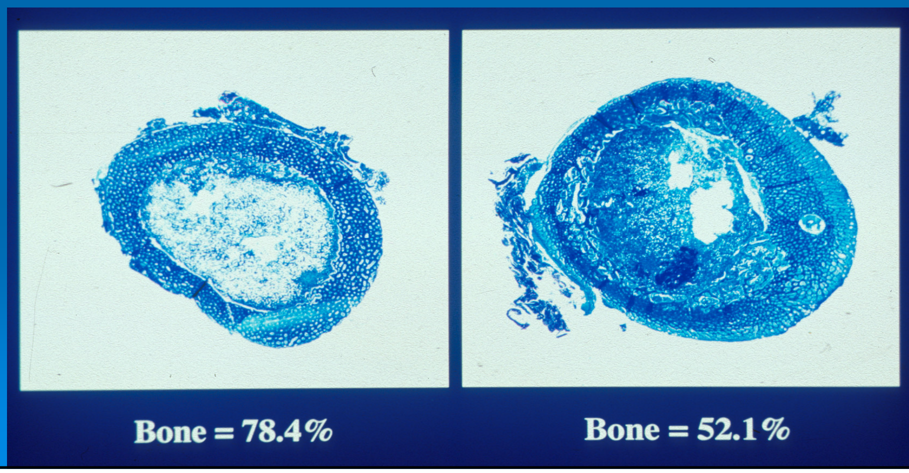


Cross-section of tibia



Cross-sections of tibias from different broiler strains

Old strain | Newer strain



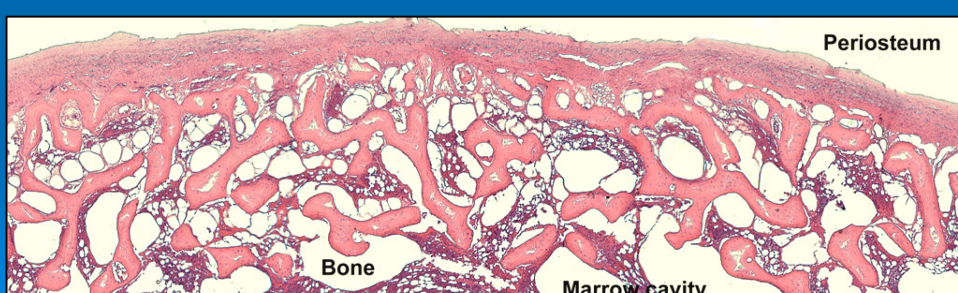
Black bone syndrome (BBS)

- Cortical bone very thin in places in proximal tibia
- Leakage of blood from marrow through bone onto periosteal surface
- More leakage after freezing and/or cooking
- Black discolouration on bone and adjacent meat
- Food quality problem, perhaps welfare in future?

Black bone

Proximal tibia: X section at 10% length

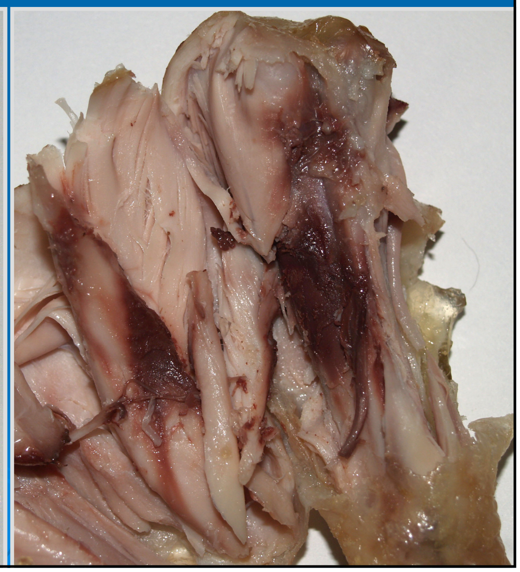
Bone is more trabecular than cortical, with possible routes leading from from marrow cavity to external periosteal layer



Black bone

Black bone

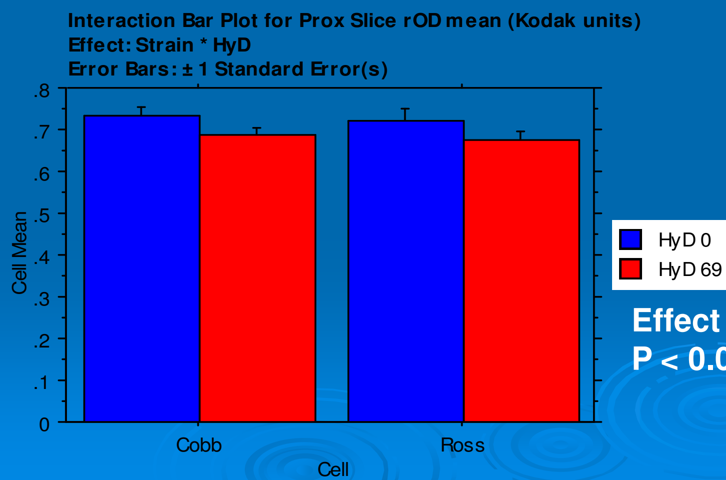
Black Bone after freezing and cooking



Black bone tibias

Mean reflected optical densities (10-15 mm from proximal end) of two strains of broilers fed diets supplemented with 0 or 69 µg

25-hydroxyvitamin D (as Hy●D®)



Effect of HyD
P < 0.043

Conclusions for Black Bone Syndrome

BBS can be seen in fresh bones but is more severe after freezing and also cooking.

It appears to be common in on-the-bone supermarket samples, particularly if frozen. There are no obvious differences between producer sources examined (so far).

The most likely cause of BBS is discolouration by bone marrow (blood) pigmentation, through highly porous bone. This highly porous bone type may also have implications for poultry welfare.

Photographic (and colourimetric) techniques are both acceptable for assessment of Black Bone Syndrome.

It appears that HyD™ can mitigate the effects of BBS, but cannot entirely prevent it in frozen samples.

Conclusions

Genetic improvement is solving some problems but bone structures seem to be changing and may be leading to new problems and changed nutritional requirements

Problems of bone necrosis are widespread and increase the importance of good hygiene

TD still occurs: vitamin D or D metabolites the best solution

BBS is a new problem seemingly related to poor bone formation mainly in proximal tibia

Higher requirements for calcium and vitamin D in starter diets to optimise bone structure (use of HyD to achieve greater vitamin D potency)