Metabolic Disorders in Poultry

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CONTEXT

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INTRODUCTION

Good health is clearly an important parameter for the efficient production of the large volumes of poultry meat and of eggs which are currently required, and the even greater volumes which will be required in the future. Poultry in poor health will not be as productive as healthy birds and consequently will yield lower volumes of food products for the human population. Therefore, health maintenance and disease avoidance in the global poultry industry is a major challenge. Poultry health impacts greatly upon the security of an important food supply, upon human health and upon international economics (Adams, 2007).

Some of the health and disease problems in poultry are undoubtedly related to the demands of a modern society for large volumes of poultry meat and eggs for human consumption. This requires intensive production, raising large numbers of birds in relatively small areas. The combination of large population numbers with high productivity inevitably means that birds are exposed to considerable stress during their productive period. Extensive preventive medication and vaccination is also a major contributor to stress. This stress, which arises from exposure to external forces and conditions, disturbs the homeostasis of the body and leads to the development of various diseases. An important group of stress-related diseases are metabolic or non-infectious diseases.

In poultry there is a whole host of metabolic diseases such as sudden death syndrome, ascites, and joint and leg problems. Metabolic disease have a set of initiating factors that activate specific patterns of gene expression that in turn act to tip biochemical equilibria to non-homeostatic states. If prolonged, these non-homeostatic states produce tissue degeneration and loss of function of one or more organs and ultimately produce signs and symptoms that lead to a clinical diagnosis of disease. Gene expression studies are beginning to show differential expression of sets of genes in tissues from metabolic diseases as compared to healthy tissue (Kornman, *et al.*, 2004). A metabolic disease only develops if there is an altered expression of a specific set of genes. This altered pattern of gene expression may be the result of a combination of factors such as environment, genetic origin and nutrition.

Metabolic diseases in poultry however, are certainly not a new phenomenon. Aldrovandi, (1600) quoted Varro (100 BC) with a very early recognition of a

Table 1.1. Chronology of metabolic disorders

Metabolic disease	*Major recognition*
Gizzard erosion	1940
Round heart	1942
Aortic rupture	1952
Cage layer fatigue	1955
Proventricular hypertrophy	1955
Fatty liver haemorrhagic syndrome	1972
Fatty liver and kidney syndrome	1974
Gout, urolithiasis	1974
Oily bird syndrome	1975
Broiler skeletal deformities	1975
Electrolyte imbalance	1978
Pectoral myopathy	1980
Heat distress	1980
Sudden death syndrome	1982
Spiking mortality	1985
Ascites	1986
Erratic ovulation	1990

which they affect the bird are greatly influenced by productivity. Fatty liver and kidney syndrome (Table 1.1) is perhaps best described as the consequence of a simple deficiency of biotin, although its onset in the early 1970s proved difficult to resolve. The condition was eventually traced to a lower-than-expected bioavailability of biotin in wheat. Fatty liver and kidney syndrome is therefore a classic example of an apparent metabolic disorder that is ingredient-specific. To some extent, oily bird syndrome is another ingredient- or ingredient class-specific problem, being related to the level and saturation of dietary fats.

Unfortunately metabolic disorders will always occur to some degree in most poultry species. Since over the last 50 years or so, we probably have identified all the nutrients required by poultry, current metabolic disorders are rarely the result of overt deficiencies or excesses of any nutrient or group of nutrients. Skeletal disorders can obviously arise due to deficiencies of key nutrients such as calcium, phosphorus or vitamin D_3 and as such cannot really be categorized as metabolic disorders. The situation of induced deficiencies, when diets are apparently adequate in the total level of all nutrients, is sometimes described as a metabolic condition. However, in this book, with a few notable exceptions, conditions that are precipitated by the deficiency of nutrients are not considered as classical metabolic disorders.

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SUDDEN DEATH SYNDROME

Other names: Acute cardiac death, Acute-death syndrome, Flip-over Disease

Species: Broilers, Broiler breeders, Turkey breeders

Compendium

Sudden death syndrome (SDS) is a condition afflicting fast growing broiler chickens, especially males. It frequently afflicts 1-5% of the flock and from 21-35 days it will usually be the major cause of death. Afflicted birds appear healthy, are well fleshed and invariably have feed in their digestive tract. Death occurs within 1-2 minutes, the birds most frequently being found dead on their backs. There are few changes in gross pathology. The heart may contain blood clots, that are likely post-mortem in origin, and the ventricles are usually empty. Diagnosis is usually by exclusion of other diseases. The lungs are often oedematous, although this usually occurs when birds spend time on their backs and fluid drains to the lung region by gravity. There are no specific changes in the tissue or blood profile that can be used for diagnosis. The condition is precipitated by fast growth rate, and so conversely it can be prevented by varying degrees of nutrient restriction. There are no clear relationships between any diet nutrients, ingredients and/or environmental factors that correlate with the onset or incidence of SDS. The condition seems to be more prevalent when ionophore anticoccidials are used (perhaps because of associated better growth rate) or if the diet contains a readily available carbohydrate source such as glucose. Sudden death syndrome can be artificially induced by intubating with lactate, although the timing of onset can be modified by diet.

The condition can best be prevented or reduced in incidence by inducing a period of initial slow growth. This can be achieved by reduction in day length, physical feed restriction and/or the use of low-nutrient dense diets. Economics will dictate the degree of early growth suppression to be implemented. The condition has been reduced in recent years with increased vigilance in genetic selection of meat birds.

seen in sudden death in horses which seems to relate to exercise induced atrial fibrillation or asphyxiation from breath-holding or bronchospasm related to subclinical respiratory disease (Gunson *et al.,* 1988).

Potential treatment and prevention

It is obvious that there is no one treatment or preventative system for the control of SDS in young broilers. The condition is undoubtedly related to fast growth rate, and as such, management techniques to reduce the early maximum genetic potential for growth offer the best preventative scenario.

Reduced growth rate

One of the most successful techniques used to temper early growth rate, is a step-down lighting programme as detailed in Table 2.1.

In all programmes, broilers are subjected to a reduced day length during the 5 - 18 day period of growth. This effectively reduces feed intake and so tempers early growth rate. Birds will learn to eat in the dark period, but there may be problems of skin-tearing *etc*., when the stocking density is very tight, because birds clamber over each other to get to the feeders. However with more normal densities (28-30 kg/sq m), the system works very well, reducing the incidence of SDS (and also skeletal problems) by up to 40% in male birds.

In extreme situations, feed restriction can also be practised, and this will virtually eliminate SDS. Feed restriction programmes are similar to those described for ascites (Chapter 4). If feed restriction and reduced day length is used, then it seems illogical to use high-nutrient dense diets. A more practical approach is to use diets with 5-7% reduction in nutrient density, and obviously there is little need for growth promoting compounds in these specialized starter

Table 2.1. Lighting programme for broilers to temper early growth rate

Age of birds	*Blackout housing*		*Open-sided housing*
(days)	*Option 1 (hrs)*	*Option 2 (hrs)*	*(hrs)*
0-4	23	23	23
5-10	8	8	Natural day length
11-14	10	8	Natural day length
15-18	14	23	Natural day length
19-23	18	23	18
23+	23	23	23

3

ROUND HEART DISEASE AND AORTIC RUPTURE IN TURKEYS

Other names: Angiopathy, Cardiomyopathy, Dilated cardiomyopathy, Spontaneous cardiomyopathy, Dissecting aneurysm, Angiorrhexis

Species: Turkeys

Compendium

Round heart disease and aortic rupture are two unrelated conditions occurring in turkeys, and especially fast-growing toms. Round heart is most prevalent in 2-4 week old toms where high mortality is associated with the enlargement and rounding of the apex of the ventricles. The condition is worse when stresses are involved and there may be a genetic component. Microscopic lesions consistently show myocardial congestion and degeneration, haemorrhage and epicardial fibrosis. Round heart is sometimes associated with ascites. Affected poults show reduced serum protein and deficiency of α-globulins. The condition seems to be worse when there is high salt/sodium intake, and round heart can be induced by feeding high levels of furazolidone. Aortic rupture most commonly affects older turkeys, and again males seem more susceptible. Affected birds appear normal immediately prior to death, the onset of which is very sudden. At necropsy, the musculature is often pale, presumably due to loss of blood, while the body cavity contains massive haemorrhage. The aorta is invariably split longitudinally, at close proximity to the heart. Aortic rupture can be induced by feeding a copper deficient diet. The role of copper is as a co-factor in monoamine oxidase enzyme, necessary for normal elastin production in the aorta. Birds dying from aortic rupture have reduced aorta elastin, and so the tensile strength of this vessel is affected. Feeding high levels of copper, however, does not totally resolve the problem. Lathyrism, as induced by feeding ß-aminopropionitrile (BAPN), also leads to aortic rupture, and again this toxin results in reduced aorta elastin anabolism.

Related factors

Round heart

Dietary salt

Morrison *et al.* (1975) found evidence of myocardial distension of poults fed high levels of salt, while Mohanty and West (1969) observed cardiac dilation in chicks receiving up to 1.5% salt in the drinking water. There are conflicting reports of the toxic effects of sodium for turkey poults. Dewar and Siller (1971) demonstrated that 0.57% sodium caused extensive 0-18-day mortality associated with marked cardiac enlargement and ascites being evident post-mortem. Morrison *et al.,* (1975) also suggested that high levels of salt caused ascites and oedema in poults. In a more extensive study, Leeson *et al.,* (1976) failed to show any round heart in poults fed up to 5% dietary salt, or up to 1.5% dietary sodium. Field reports do not confirm any obvious relationship between dietary salt and incidence of round heart. However a diet containing low sodium (0.10-0.12%) and high chloride (0.38-0.40%) significantly improved liveability of turkey poults by reducing round heart mortality without adverse effects on poult body weight or feed conversion ratio (Table 3.1), (Frame *et al*., 2001).

Table 3.1. The influence of dietary sodium and chloride on growth performance and round heart- attributable mortality in turkeys from 0-21 days.

Dietary treatment	*Body weight (g)*	*Feed conversion ratio*	*Total mortality(%)*	*Roundheart mortality(%)*
High Na (0.24%): High Cl (0.40%)	905	1.64	5.9	1.02[a]
High Na (0.24%): Low Cl (0.16%)	900	1.63	4.6	1.16[a]
Low Na (0.14%): High Cl (0.40%)	891	1.67	4.6	0.19[b]
Low Na (0.14%): Low Cl (0.14%)	917	1.62	4.5	0.84[a]

[a,b] Means with no common superscript differ significantly (P< 0.01)
(Adapted from: Frame *et al.*, 2001).

A possible explanation of the beneficial effect of high chloride relative to sodium is that the chloride anion may help in the excretion of the relatively high levels of potassium coming from soyabean meal common in turkey diets.

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ASCITES

Other names: Right ventricular hypertrophy, Pulmonary hypertension syndrome, Water belly

Species: Broiler chickens, Turkeys, Ducks

COMPENDIUM

Ascites emerged in the 1970s and is related to body weight in fast growing broilers. It is characterized by accumulation of fluid in the abdomen which is caused by a cascade of events related to the need to supply high levels of oxygen to the tissues. The condition was initially most prevalent in fast growing male broilers maintained at high altitude and where there is a degree of cold stress, but nowadays the problem can occur at any altitude. In extreme situations up to 8% mortality is seen, although 1-3% mortality is currently more common.

The lungs of birds are rigid and are moulded into the thoracic cavity. They cannot expand like mammalian lungs. Also the capillaries can only expand a little to allow for increased blood flow. The lungs of broilers grow less rapidly than the rest of the body and frequently lung capacity does not keep up with the very rapid growth of muscle in modern broilers. Nevertheless broilers have a high demand for oxygen necessary to fuel metabolic processes. When such demand is increased by very fast growth rate, or by cold conditions, then the lungs must oxygenate increased quantities of blood. At high altitude the situation is made worse by low oxygen tension in inhaled air. In order to meet the demands for metabolism, the bird attempts to pump more blood through the lungs and so this places extra stress on the right ventricle of the heart. Under normal conditions, the right ventricle is relatively small, but in the situation of ascites this ventricle becomes grossly dilated and its size doubles. This weakened ventricle creates back-pressure to the various supply systems, a consequence of which is leakage of plasma from the liver, commonly referred to as ascitic fluid (water belly). Ascites is effectively caused by hypoxia. Upon necropsy, the bird is identified

broilers. However there is not a good correlation between hypoxaemia and the risk of ascites. Many fast-growing broilers can be hypoxaemic without developing ascites. Probably inadequate lung perfusion, an inability to raise cardiac output in the face of oxygen demand and increased oxygen utilization are the key factors for the development of hypoxaemia. Hypercapnia in broilers however, may be significant in the aetiology of pulmonary hypertension.

Table 4.3. Arterial and mixed venous blood pO_2 and pCO_2 values, in Leghorn chickens, feed-restricted slow-growing broilers, *ad libitum*- fed fast-growing broilers, and broilers with ascites

Type of chicken	
Leghorns	
pO_2 mmHg (arterial blood)	85.76
pO_2 mmHg (central venous blood)	42.88
pCO_2 mmHg (arterial blood)	19.94
pCO_2 (central venous blood)	23.82
Slow-growing broilers	
pO_2 mmHg (arterial blood)	77.9
pO_2 mmHg (central venous blood)	42.9
pCO_2 mmHg (arterial blood)	27.5
pCO_2 (central venous blood)	40.62
Fast-growing broilers	
pO_2 mmHg (arterial blood)	58.9
pO_2 mmHg (central venous blood)	28.8
pCO_2 mmHg (arterial blood)	35.84
pCO_2 (central venous blood)	44.8
Broilers with ascites	
pO_2 mmHg (arterial blood)	34.1
pO_2 mmHg (central venous blood)	17.5
pCO_2 mmHg (arterial blood)	53.3
pCO_2 (central venous blood)	59.4

(Adapted from: Olkowski, 2007)

The onset of ascites in broilers is clearly associated with pulmonary hypertension which is an important factor in failure of the right ventricle of the heart. However there is also evidence that broilers dying from ascites also have changes in the left side of the heart (Olkowski, 2007). Left ventricular failure could also cause pulmonary hypertension. Therefore it may be that chronic heart pump failure is a major factor in the pathogenesis of hypoxaemia,

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HEPATIC HAEMORRHAGE

Other names: Liver haemorrhagic syndrome

Species: Laying hens

Compendium

Hepatic haemorrhage will often occur in laying hens fed diets containing appreciable quantities of high glucosinolate rapeseed meal. Birds die of massive liver haemorrhage caused by a loss in the reticulin structure of the hepatic tissue. These changes in tissue structure are assumed to be mediated by glucosinolate compounds either directly and/or through the alteration of thyroxine output. There is no major infiltration of lipids in the liver, and so this differentiates the diagnosis relative to Fatty Liver Haemorrhage Syndrome (Chapter 6). There is a major genetic component, with some strains of White Leghorn exhibiting a 50% mortality over a laying cycle when 15-20% rapeseed is used in a diet. Thyroid glands will also be enlarged, although this is a usual response to glucosinolates in rapeseed and should not be considered specific for hepatic haemorrhage. Birds often appear normal and have functional ovaries at the time of death. It is possible that the increased blood pressure associated with oviposition is ultimately responsible for the rupture of the liver capsule, and so the cause for the characteristic massive haemorrhage. The condition can be prevented by removing the high-glucosinolate rapeseed from the diet. When it is necessary to use this ingredient, its inclusion should be limited to 5% of the diet.

Occurrence and general signs

Liver haemorrhages are known to occur in laying hens fed rapeseed or mustard seed meal that contain high levels of glucosinolates. The condition seems

to be strain specific, because in some flocks the mortality can reach 50%, whereas other strains show only a 5-10% mortality. Affected birds appear normal, and there are no outward signs of morbidity preceding death, other than occasional situations of anaemia. Olomu *et al.,* (1975) fed layer diets containing up to 10% rapeseed meal for 330 days. Mortality was little affected with up to 5% inclusion of rapeseed, but at 10% there was increased mortality. However these authors showed that the rapeseed meal inclusion level had no effect on the liver fat content which averaged 35% across all treatments. On this basis, Olomu *et al.,* (1975) concluded that although the lesions at first appear typical of Fatty Liver Haemorrhagic Syndrome (FLHS), (Chapter 6), there is no major increase in the liver fat content, and so the term "hepatic haemorrhage" is more appropriate. Unaffected birds appear normal, and there seems to be no loss in egg production for these birds. Campbell, (1979) observed a marked strain difference in incidence of hepatic haemorrhage in White Leghorn birds. Feeding up to 10% high-glucosinolate rapeseed meal, resulted in a 48% incidence in one strain of bird, while no mortality occurred in another strain. The average mortality was around 5%.

Pathology and metabolic changes

Gross pathology

Hall, (1972) described a condition in the UK in the early 1970's that seemed to relate to rapeseed toxicity. Birds died in good condition, often with an egg in the oviduct, and with a functional ovary. In some instances, the birds were anaemic with pale combs and wattles. At post-mortem examination, there was invariably a massive haemorrhage in the liver, which itself was pale yellow and friable. Hall, (1972) observed little fat infiltration in the liver, and so this precluded FLHS. Jackson, (1969) observed up to 50% mortality in one strain of bird fed 20% rapeseed meal, with all affected birds showing a large haemorrhage in the liver. In addition to the single large haemorrhage that probably caused death, there were often smaller haemorrhages that appeared older, and were bordered in some cases by necrotic tissue. Yamashiro *et al.,* (1975) observed moderate to severe fatty degeneration, focal necrosis and moderate to severe haemorrhage, in layers fed 10-20% of Span rapeseed meal (high glucosinolate, low erucic acid cultivar) or 20% rapeseed oil.

6

FATTY LIVER HAEMORRHAGIC SYNDROME

Other names: Fatty Liver Syndrome

Species: Laying hens, conditions reported in broiler breeders, turkey breeders

Compendium

Fatty liver haemorrhagic syndrome (FLHS) was first described by Couch, (1956) as excessive fat in the liver associated with varying degrees of haemorrhage. The condition is almost universally confined to caged birds fed high-energy diets, and is most often seen in summer months. The liver is usually enlarged, a "putty colour", and is very friable. The abdominal cavity usually contains large amounts of oily fat. A number of workers have suggested that the affected birds have pale combs. The ovary is usually active, and the metabolic and physical stress as associated with oviposition may be the factors that induce the final fatal haemorrhage. Fatty liver haemorrhagic syndrome only seems to occur when birds are in a positive energy balance, and so the monitoring of body weight is a good diagnostic tool. Through force-feeding techniques, it is shown that FLHS is caused by a surfeit of energy rather than being specific to an excess of any nutrient such as fat or carbohydrate. Experimentally the condition can be induced in layers and even male birds by the administration of oestrogen. This reinforces the concept that FLHS occurs more frequently in high-producing birds that presumably are producing oestrogen from very active ovaries.

Numerous attempts have been made to prevent or treat the condition through diet modification. Substituting carbohydrate for supplemental fat, while not increasing the energy content of the diet, seems to be beneficial. Presumably such modification means that the liver needs to synthesize less fat for yolk. Replacement of corn with other cereals, such as wheat and barley, is often beneficial. However this substitution may involve a reduction in diet

or purified safflower phospholipids than in the safflower and palm oils group (reduced by 57% and 52%, relative to the oils) respectively.

Table 6.2. Effect of dietary safflower phospholipids on the contents of various lipid fractions in the liver in laying hens

Quantity in liver (mg/g)	*Treatment*			
	Tallow (Control)	*SP-oil**	*Saf-PL crude*	*Saf-PL*
Total cholesterol	3.56[a**]	3.05[b]	2.98[b]	3.11[b]
Triglyceride	27.79[a]	9.31[b]	5.32[b]	4.83[b]
Phospholipid	35.97	36.18	33.94	37.47

*SP-oil: safflower oil and palm oil; Saf-PLcrude: crude safflower phospholipid; Saf-PL: purified safflower phospholipid.
** Means in the same row with no common letter differ significantly ($P < 0.05$).
(Adapted from: An *et a*l., 1997)

The decrease in triglyceride content in the liver of hens fed diets containing phospholipids might be responsible for reducing the activities of enzymes related to hepatic fatty acid and triglyceride synthesis. It is recognised that any reduction in FLHS is associated with reduction in liver fat. Therefore, crude safflower phospholipid could be used as an ingredient for layer rations, and may be a valuable alternative for reducing FLHS in layers.

Co-product ingredients

There has been considerable research conducted on the role of ingredients such as fish meal, alfalfa meal, and various distillery co-products as they affect FLHS. In most of these studies, there is the confounding effect of fat inclusion as detailed previously. Jensen *et al.,* (1976a) formulated isoenergetic diets at 12.14 MJ/kg (2900 kcal ME/kg) and 14.3% crude protein to contain a number of cereals including corn, wheat or barley. Incidence of FLHS followed the ranking corn > wheat > barley. Fish meal-alfalfa *versus* corn diets were shown by Akiba *et al.,* (1982a) to reduce liver lipid content, while Akiba *et al.,* (1982b) showed reduced liver lipid in birds fed diets containing fish meal or distillers grains. In this latter situation, the fish diet contained less total dietary fat. Jensen *et al.,* (1974) substituted distillers grains in wheat and corn based diets while maintaining constant diet fat levels. While distillers grains did result in reduced liver weight, there was no change in g fat/liver.

Maurice and Jensen, (1978a) attempted to reduce plasma lipid levels through the inclusion of various ingredients. Using a conventional corn-soyabean meal

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FATTY LIVER AND KIDNEY SYNDROME

Other names: None

Species: Young broiler chickens

Compendium

Diets deficient or marginal in biotin can lead to fatty liver and kidney syndrome (FLKS) in young broilers. The condition is most commonly seen in 2-4 week old birds fed wheat-based diets, although it seems as though some environmental stress is necessary to trigger the condition. Onset of symptoms occurs quickly, with apparently healthy birds exhibiting lethargy and general reluctance to move. Prior to death, birds may lay prone on the litter with necks outstretched. Mortality of 5-20% can occur. Gross pathology reveals enlarged and pale kidneys and liver, both of which have extensive fat accumulation. This fat is usually mono-unsaturated, and there are unusually high levels of palmitoleic acid rather than stearate. Staining of the liver reveals depleted glycogen reserves. Death is ultimately caused by hypoglycaemia due to failure of hepatic gluconeogenesis which in turn is triggered by inadequate levels of the key biotin-dependent enzyme pyruvate carboxylase.

Stress is a major contributor to the severity of FLKS, and under experimental conditions onset is most often induced by sudden change in temperature. Stress likely induces an epinephrine induced catabolism of the already low glycogen reserves. The condition is prevented by ensuring adequate levels of available biotin in the diet, and that broiler chicks hatch from eggs with adequate biotin carry-over. Both broilers and breeders should be fed diets containing at least 0.2 mg available biotin/kg diet. This level will ensure adequate biotin reserves, even when low-protein diets are used, or if sulpha drugs are used to treat other infections.

may need to be increased when low protein diets are used, if sulpha drugs are administered, or if unstabilized fat is included in the diet.

Palm kernel oil seems to be able to spare biotin and reduce the incidence of FLKS (Oloyo and Ogunmodede, 1989). Addition of 2% palm kernel oil significantly reduced FLKS mortality and also reduced the minimum biotin requirement. A lower amount of biotin,0.120 mg/kg feed, was needed in case of palm kernel oil supplementation as compared with a biotin requirement of 0.160 mg/kg feed in order to prevent FLKS mortality when palm kernel oil was not contained in the rations.

Bibliography

Balnave, D. and Pearce, J. (1976). Aspects of lipid and carbohydrate metabolism, dietary biotin and fatty liver and kidney syndrome in broilers. Br. Poult. Sci,. 17: 627-636.

Bannister, D. W. (1976). Hepatic gluconeogenesis in chicks. Effect of biotin on gluconeogenesis in biotin-deficiency and fatty liver and kidney syndrome. Comp. Biochem. Physiol., 53B: 575-579.

Bannister, D. W., Evans, A. J. and Whitehead, C. C. (1975). Carbohydrate absorption by chicks affected with the fatty liver and kidney syndrome. Res. Vet. Sci., 19: 90-92.

Bannister, D. W., Sales, D. I. and Lee, A. (1985). Biotin deficiency and susceptibility to fatty liver and kidney syndrome in broiler chicks: reduced 6-phosphofructokinase (EC 2.7.1.11) activity but normal fructose 2,6, bisphosphate content in birds with hepatomegaly. Br. J. Nutr., 54: 535-543.

Blair, R. and. Whitehead, C. C. (1974). An assessment of the factors associated with fatty liver and kidney syndrome in broilers. *in* Proc. Abstr. XV World's Poult. Congr., New Orleans, LA. World Poult. Sci. Assoc., US Branch, Storrs, CT. pp 380–384.

Blair, R., Whitehead C. C. and Teague, P. W. (1975). The effect of dietary fat and protein levels, form and cereal type on fatty liver and kidney syndrome in chicks. Res. Vet. Sci., 18: 76-81.

Broz, J. and. Ward, N. E. (2007). The role of vitamins and feed enzymes in combating metabolic challenges and disorders. J. Appl. Poultry Res., 16: 150-159.

Bryden, W. L. (1990). Inclusion of low metabolisable energy wheat in broiler diets and the incidence of fatty liver and kidney syndrome in broilers. Res. Vet. Sci., 49: 243-244.

Bryden, W. L. (1991). Tissue depletion of biotin in chickens and the development of deficiency lesions and the fatty liver and kidney syndrome. Avian Path.,

8

GOUT AND KIDNEY UROLITHIASIS

Other names: Visceral gout, Articular gout, Baby chick nephropathy, Blue comb, Acute toxic nephritis, Renal gout, Kidney stones, Nutritional gout, Nephrosis.

Species: All birds

Compendium

Kidney dysfunction often leads to either visceral or articular gout, or urolithiasis. In all situations, increased substrate load to the kidney eventually leads to precipitation of insoluble products within the kidney itself or in other regions of the body. Gout describes the condition in which high plasma uric acid leads to precipitation of monosodium urates either in the synovial fluid and tendon sheaths of various joints, especially the hock joint, or on the serous surface of various visceral organs. There seems to be a genetic basis for gout, although interestingly the articular and visceral forms are rarely seen together. Baby chick nephropathy and blue comb disease may be forms of gout. Articular gout is most commonly seen in birds fed excess levels of protein, and/or where the balance of K+Cl:Na is <1. Urolithiasis most commonly occurs in Leghorn birds, and is induced by feeding high levels of calcium for many weeks prior to sexual maturity. Feeding diets containing 3-5% calcium after 8-10 weeks of age invariably leads to a proportion of the flock developing insoluble uroliths in the kidney tubules. These uroliths are usually composed of calcium-sodium-urate. Often just one kidney is affected, and due to compensatory hypertrophy of the normal kidney, then asymmetry in size and weight can be used during diagnosis. There does not seem to be any problem with urolithiasis if pullets are fed high calcium layer diets for a more conventional period of 2-3 weeks prior to sexual maturity. Mycotoxins such as oosporein and citrinin can also adversely affect kidney function, while urolithiasis at least is made worse when birds are infected with infectious bronchitis. Urine acidification can reduce urolith formation and solubilize

were the dilation and the impaction of the kidney collecting ducts in the medulla, with the accumulation of cell debris, inflamed cells and urates. These changes are accompanied by severe interstitial fibrosis and compression of other medullary tissues. In the cortical tissues vitamin A deficiency resulted in hyperuricemia and areas of tubular degeneration and necrosis. Deficiency of vitamin A probably leads to the keratinization of tubular epithelium, since this is common to other epithelial tissues in the body. Chandra *et al*., (1984) suggested that hyperuricemia and nephritic lesions occurred only after vitamin A levels in the liver were below 24 IU/g tissue.

Potential treatment and prevention

Urolithiasis seems to be most problematic in laying hens fed high levels of calcium well in advance of sexual maturity. Although the situation is often confounded with IBV infection, it seems obvious that no more than 1% calcium should be fed to Leghorn birds prior to maturity. Feeding pre-lay (2% Ca) or layer diets containing 3% calcium for 2-3 weeks prior to first egg is not problematic, and surprisingly, uroliths rarely form in adult male breeders fed high calcium diets. Visceral and articular gout both seem to have a genetic component, since the occurrence is sporadic within a flock. High levels of crude protein will increase plasma uric acid levels, and so potentially provide conditions conducive to urate formation. Certainly numerous mycotoxins influence kidney function, and so general mill management regarding quality control and/or use of feed additives to suppress their harmful effects would likely be beneficial.

Most forms of kidney dysfunction are associated with an increased loss of water and electrolytes, and so electrolyte therapy is often considered. Condron and Marshall, (1985) suggest the addition of potassium and to a lesser extent sodium salts, especially citrates and bicarbonates. However, such therapy is not straightforward because feeding additional sodium will exacerbate problems of water loss. Also death from acute renal failure is frequently related to cardiac arrest caused or induced by high levels of plasma potassium.

In humans at least, urolith formation can be prevented or treated through the use of urine acidifiers. Glahn *et al*., (1988b) studied this possibility in pullets intentionally fed high-calcium diets prior to maturity. At 32 weeks of age, birds were fed either 1% NH_4Cl or 1% $NaHCO_3$, and performance monitored for 20 weeks. Birds fed NH_4Cl developed increased blood H^+ and reduced HCO_3^- content. At 52 weeks these birds had a more acidified urine, and none of these birds had uroliths in the kidney. Urolithiasis (8%) did occur in the birds fed the untreated diet while 13% of birds fed the alkaline diet developed

this problem. Unfortunately all treatments resulted in an increased water intake (20%) and manure moisture was increased by 10-14%. Stevens and Salmon, (1989) fed up to 3% NH_4Cl to young turkey poults. Diet acidification resulted in a 30% increase in kidney weight together with increased faecal moisture content. A concomitant reduction in tibia ash led Stevens and Salmon, (1989) to conclude that the acidified urine resulted in increased calcium solubilisation in the urine. Wideman and co-workers have shown that urine acidification can reduce urolith formation in immature birds, and have also shown that such treatment can solubilize uroliths already formed in mature birds. One of the potential problems in using NH_4Cl in laying hens, is that it induces a metabolic acidosis and this is detrimental to eggshell quality especially under conditions of heat stress. Such treatment also assumes the kidney can clear the increased load of H^+, and for a damaged kidney, this may not always occur. As a potential urine acidifier without such undesirable side effects, Wideman *et al*., (1989) investigated the role of methionine hydroxy analogue (MHA) free acid. From 5-17 weeks of age, pullets were fed diets containing 1 or 3% calcium in combination with 0, 0.3 or 0.6% MHA. Birds fed the untreated high-calcium diet excreted alkaline urine containing elevated calcium concentrations together with urolith formation and some kidney damage. Feeding 0.6% MHA acidified the urine, but did not cause a general metabolic acidosis. Methionine hydroxyl analogue therefore reduced kidney damage and urolith formation without there being acidosis or increased water consumption. In subsequent studies Wideman *et al.,* (1993) also showed supplemental DL-methionine to be effective in preventing kidney damage.

It appears that urine acidification can be used as a prevention or treatment of urolithiasis, and that this can be accommodated without necessarily inducing a generalized metabolic acidosis. From a nutritional viewpoint, kidney dysfunction can be minimized by not oversupplying nutrients such as calcium, crude protein and electrolytes.

Bibliography

Anasuya, A. (1983). Urinary calculus disease: role of nutrition, A Review. Nutr. Rep. Int., 27: 1095-1100.

Austic, R. E. and Cole, R. K (1972). Impaired renal clearance of uric acid in chickens having hyperuricemia and articular gout. Amer. J. Physiol., 223: 525-530.

Campbell, M. L., Doerr, J. A. and Wyatt, R .D. (1981). Immune status in broiler chickens during citrinin toxicosis. Poultry Sci., 60: 1634.

Chandra, M. (1985). Occurrence and pathology of nephritis in poultry. Acta-

Veterinaria, 35: 319-328.

Chandra, M., Singh, B., Soni, G. L. and Ahuja, S. P. (1984). Renal and biochemical changes produced in broilers by high-protein, high-calcium, urea containing and vitamin A deficient diets. Avian Dis., 28: 1-11.

Cole, R. K. and Austic, R. E. (1980). Hereditary uricemia and articular gout in chickens. Poultry Sci., 59: 951 - 960.

Condron, R. J. and A. T. Marshall. (1985). Pathogenesis of infectious bronchitis nephritis. 2. Studies of water and electrolyte balance in colostomised chickens. Avian Path., 14: 509-520.

Cowen, B. S., Wideman Jr., R .F., Rothenbacher, H. and Braune, M. O. (1987). An outbreak of avian urolithiasis on a large commercial egg farm. Avian Dis., 31: 392-397.

Davies, N. M. and Anderson, K. E. (1997) Clinical pharmacokinetics of diclofenac. Therapeutic insights and pitfalls. Clin. Pharmacokinet. 33:184–213.

Glahn, R. P., Wideman Jr., R. F. and Cowen, B. S. (1988a). Effect of gray strain infectious bronchitis virus and high dietary calcium on renal function of single comb white leghorn pullets at 6, 10 and 18 weeks of age. Poultry Sci., 67: 1250-1263.

Glahn, R. P., Wideman Jr., R. F. and Cowen, B. S. (1988b). Effect of dietary acidification and alkalinization on urolith formation and renal function in single comb White Leghorn laying hens. Poultry Sci., 67: 1694-1701.

Gustavson, S. A., Cockrill, J. M., Beasley, J. N. and Nelson, T. S (1981). Effect of dietary citrinin on urine excretion in broiler chickens. Avian Dis., 25: 827-830.

Halvorson, D .A., Walser, M. M. and. Newman, J. A. (1982). Calcium carbonate toxicity in Leghorn pullets. J. Amer. Vet. Med. Assoc., 181: 287.

Hnatow, L. L. and Wideman Jr., R. F. (1985). Kidney function of Single Comb White Leghorn pullets following acute renal portal infusion of the mycotoxin citrinin. Poultry Sci., 64: 1553-1561.

Hocking, P. M. (1989). Effect of dietary crude protein concentration on semen yield and quality in male broiler breeder fowls. Brit. Poultry Sci., 30: 935-945.

Hocking, P.M. (1990). The relationships between dietary crude protein, body weight, and fertility in naturally mated broiler breeder males. Brit. Poultry Sci., 31: 743-757.

Huff, W. E. and Doerr, J. A. (1981). Synergism between aflatoxin and ochratoxin A in broiler chickens. Poultry Sci., 60: 550-555.

Hussain, I., Khan, M. Z., Khan, A., Javed, I. and Saleemi, M. K.(2008). Toxicological effects of diclofenac in four avian species. Avian Pathol., 37: 315-321.

Jain, T., Koley, K. M., Vadlamudi, V. P., Ghosh, R. C., Roy, S., Tiwari, S. and Sahu, U. (2009). Diclofenac-induced biochemical and histopathological changes in white leghorn birds (Gallus domesticus). Indian J. Pharmacol., 41: 237-241.

Julian, R. J. (1982). Water deprivation as a cause of renal disease in chickens. Avian Path., 11: 615-617.

Koutsos, E. A., Smith, J., Woods, L. W. and Klasing, K. C. (2001). Adult cockatiels (Nymphicus hollandicus) metabolically adapt to high protein diets. J. Nutr., 131: 2014–2020.

Mallinson, E. T., Rothenbacher, H., Wideman Jr., R. F., Snyder D. B.and Russek, E. (1984). Epizootiology, pathology, and microbiology of an outbreak of urolithiasis in chickens. Avian Dis., 28: 25-43.

Mars, Y. W. H. M., Lemmens, A. G. and Beynen, A. C. (1988). Dietary phosphorus and nephrocalcinosis in female rats. Nutr. Rep. Int., 38: 249-258.

Martindale, L. (1975). The effect of high dietary sodium chloride on the renal function in chicks. Brit. Poultry Sci., 16: 577-581.

Martindale, L. and Lee, D. J. W. (1976). Renal function changes in laying hens fed on dried poultry manure. Brit. Poultry Sci., 17: 195-197.

McNabb, R. A. and McNabb, F. M. A. (1975). Urate excretion by the avian kidney. Comp. Biochem. Physiol., 51A: 253-258.

Mollenhauer, H. H., Corrier, D. E., Huff, W. E., Kubena, L. F., Harvey R. B. and Droleskey, R. E. (1989). Ultrastructure of hepatic and renal lesions in chickens fed aflatoxin. Amer. J. Vet. Res., 50: 771-777.

Naidoo, V. and Swan, G. E. (2009). Diclofenac toxicity in Gyps vulture is associated with decreased uric acid excretion and not renal portal vasoconstriction. Comp. Biochem. Physiol. C Toxicol. Pharmacol., 149: 269-74.

Niznik, R. A., Wideman, R.F., Cowen, B.S. and Kissell, R.E. (1985). Induction of urolithiasis in SCWL pullets: effect on glomerular number. Poultry Sci., 64: 1430-1437.

Oaks, J. L., Gilbert, M., Virani, M. Z., Watson, R. T., Meteyer, C. U., Rideout, B. A., Shivaprasad, H. L., Ahmed, S., Chaudhry, M. J., Arshad, M., Mahmood, S., Ali, A. and Khan, A. A. (2004). Diclofenac residues as the cause of vulture population decline in Pakistan. Nature, 427: 630-633.

Oldroyd, N. O. and Wideman, R. F. (1986). Characterization and composition of uroliths from domestic fowl. Poultry Sci., 65:1090-1094.

Pegram, R. A. and Wyatt, R. D. (1981). Avian gout caused by oosporein, a mycotoxin produced by *Chaetomium trilaterate*. Poultry Sci., 60: 2429-2440.

Rattner, B. A., Whitehead, M. A., Gasper, G., Meteyer, C. U., Link, W. A., Taggart, M. A., Meharg, A. A., Pattee, O. H. and Pain, D. J. (2008). Apparent

tolerance of turkey vultures (Cathartes aura) to the non-steroidal anti-inflammatory drug diclofenac. Environ. Toxicol. Chem., 27: 2341-2345.

Shirai, J., Nakamura, K., Narita, M., Furata, K. and Kawamara, H. (1990). Avian nephritis virus infection in chicks: virology, pathology and serology. Avian Dis., 34: 558-565.

Siller, W. G. (1981). Renal pathology of the fowl - a review. Avian Path., 10: 187-262.

Slemons, R. D., Locke, L. N., Sheerar, M. G., Duncan, R. M., Hinshaw, V. S. and Easterley, B. C. (1990). Kidney lesions associated with mortality in chickens inoculated with waterfowl influenza viruses. Avian Dis., 34: 120-128.

Sonmez, G. (1991). Organic distribution and interrelationships of lesions occurring in laying hens suffering from gout and urolithiasis. Adv. Exp. Med. Biol., 309A: 247-250.

Stevens, V. I. and Salmon, R. E. (1989). Effect of chronic acid load as excess dietary protein, ammonium chloride, sulfur amino acid or inorganic sulfate on the incidence of leg problems in turkey. Nutr. Rep. Int., 48: 477-485.

Uphoff Meteyer, C., Rideout, B. A., Gilbert, M., Shivaprasad, H. L. and Oaks, J. L. (2005). Pathology and proposed pathophysiology of diclofenac poisoning in free-living and experimentally exposed oriental white-backed vultures (G*yps bengalensis*). J. Wildlife Dis., 41: 707-716.

Ward, J. M., McNabb, R. A. and McNabb, R. M. A. (1975). The effects of changes in dietary protein and water availability on urinary nitrogen compounds in the rooster. *Gallus domesticus*. I. Urine flow and the excretion of uric acid and ammonia. Comp. Biochem. Physiol. 51A: 165-169.

Wideman Jr., R. F. (1985). Poultry kidney damage: some new perspectives. Poultry Digest. August, pp. 334.

Wideman Jr., R. F., Closser, J. A,. Roush, W. B. and Cowen, B. S. (1985). Urolithiasis in pullets and laying hens: role of dietary calcium and phosphorus. Poultry Sci., 64: 2300-2307.

Wideman Jr., R. F., Ford, B. C., Leach, R. M., Wise, D. F. and Robey, W. W. (1993). Liquid MHA free acid and DL-methionine attenuate calcium-induced kidney damage in domestic fowl. Poultry Sci., 72: 1245-1258.

Wideman Jr., R. F. and Glahn, R.P. (1987). Nutrition, kidney dysfunction in poultry examined. Feedstuffs Dec. 18, pp. 14.

Wideman Jr., R. F., Mallinson, E. T. and Rothenbacher, H. (1983). Kidney function of pullets and laying hens during outbreaks of urolithiasis. Poultry Sci., 62: 1954-1970.

Wideman Jr., R .F., Roush, W .B., Satnick, J. L., Glahn, R. P. and Oldroyd, N. O. (1989). Methionine hydroxy analog reduces avian kidney damage and urolithiasis induced by excess dietary calcium. J. Nutr., 119: 818-828.

9

OILY BIRD SYNDROME

Other names: None

Species: Broilers

Compendium

Oily bird syndrome (OBS) results in broiler carcasses that are oily and greasy to the touch, and often have pockets of water accumulating in regions beneath the skin. The condition is most prevalent in warm climates and is accentuated by harsh processing conditions and especially harsh scald temperature and pick-time. The syndrome is most noticeable in female broilers. However, OBS is not due to the incorporation of unsaturated oils into the diet, and in fact there is an indication of a greater incidence when tallow is fed. Characteristics of OBS are caused by changes in skin collagen structure. The various skin layers separate more easily and oil and/or chilled water accumulates in the discreet pockets, especially in the back region. The skin of affected birds shows a deficiency in collagen crosslinkage that has been described as an immature development of this important structural bonding. Collagen maturation can be adversely affected by a deficiency of copper or an excess of vitamin A in the diet. However, because broiler performance is often normal when outbreaks of OBS occur, then diet imbalance *per se* is not likely a major factor.

Occurrence and general signs

As its name implies, carcasses with OBS have skin that is oily or greasy to the touch. Garrett, (1975) was one of the first to document the condition, indicating the subcutaneous fat, especially in the back region, to be very oily. At that time, Garrett, (1975) suggested that OBS was observed most frequently in older broilers and especially those fed high energy diets in the warmer

10

WATER IMBALANCE

Other names: Wet Manure, Dehydration

Species: Mainly laying hens, but all species can be affected.

Compendium

This chapter deals with the expectations of, and factors influencing normal water balance in poultry. The most common consequence of water imbalance is wet manure, which is problematic in modern poultry houses. Due to increased water:feed intake, wet manure is most commonly associated with elevated levels of sodium and potassium in the diet. Diet sodium levels should not exceed 0.15-0.20% of the diet, and where high-salt water is suspected, both feed and water should be assayed for sodium and chloride levels independently. A number of diet ingredients can stimulate water intake, the most common being barley, molasses and soyabean meal. When ingredients such as wheat or barley are necessarily used due to economics, and wet (sticky) manure is a problem, then consideration should be given to use of various xylanase and glucanase enzymes. Laying hens also seem to produce a wetter manure, and especially under hot weather conditions when high calcium layer diets are introduced 2-3 weeks prior to maturity. Wet manure due to high-calcium intake can be prevented by the use of appropriate pre-lay diets that contain intermediate (2%) levels of calcium.

Dehydration most commonly occurs due to equipment failure on the farm. Depending upon the degree of water restriction, the effect on poultry is usually not too severe. For birds at peak egg production, an unintentional period of water deprivation will dramatically reduce egg output. Evidence shows almost total cessation of production in a flock subjected to an unintentional 48 hours period of water deprivation. Under these conditions however, a pause-in-lay is induced, and birds will resume normal production in 4-6 weeks. Young turkey poults are most susceptible to water deprivation, where death often occurs when water is reintroduced.

Occurrence and general signs

Excessive drinking occurs periodically in poultry flocks as a consequence of various environmental stresses, while dehydration can result from mechanical failure of water supply systems. In most instances, birds will modify their water input/output mechanisms in order to obtain a physiological balance, although this sometimes results in management problems at the farm. With intensive mechanized housing systems, the major problem of water imbalance is wet manure, and this occurs most frequently in laying hens. Excessive water intake, for whatever reason, results in increased manure water content, and this can lead to problems of manure handling, odour and fly control, dirty eggshells and increased barn humidity. The manure of layers will normally contain around 75% water, although it seems that even quite small changes in the water content can have a dramatic effect on its physical appearance, and handling characteristics. Environmental temperature, diet composition, feed texture and drinker design are all known to influence water consumption by poultry. Knowledge of water intake under various conditions is also important due to the fact that vaccines and other medications are now frequently administered via the drinking water. Prior to the discussion of specific problems of water imbalance, it is pertinent to consider the normal physiological systems of water intake and output, and how these can be influenced by the commercial farm environment.

Normal water metabolism

Water is the major component of the body and represents about 70% of total body weight in chickens. In turkeys total body water varies from 60 - 65% of total body weight (Riek *et al.,* 2008). Water is also the major nutrient consumed and in turkeys consumption was on average 837 mL in male and 569 mL per day in female birds. This gave a water:feed ratio ranging from 1.46 – 1.82. The water:feed ratio is always >1.0 and Feddes *et al.,* (2002) reported values from 1.7 – 1.85 for broilers. In layers subjected to heat stress the water:feed ratio increased from 1.9 during acclimation to 3.4 during the first week of heat exposure, and then declined slightly to 3.0 by end of the fourth week of heat exposure (Xin *et al.,* 2002).

Water requirements of poultry are modified by different physiological states such as egg production in layers, muscle growth in broilers, or changes in body composition with age (Leeson and Summers, 1997). Various factors have been shown to influence water intake in poultry including strain (Bessei *et al.,* 1999), sex (Ziaei *et al.,* 2007), stocking density (Feddes *et al.,* 2002),

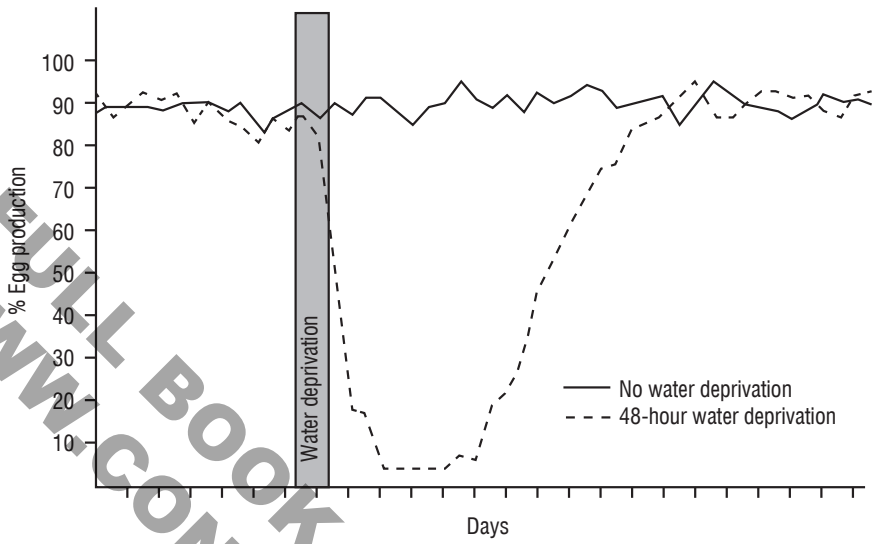

Figure 10.1. Effect of a 48 hour period of water deprivation on egg numbers.

Whatever the reason for dehydration in the bird, the intracellular and extracellular fluids and plasma to some extent share the water deficit. Depending on the severity of water deficit, circulation is affected, resulting in increased body temperature and metabolic acidosis (Barragry, 1974). In extreme cases, subsequent death is often a result of bradycardia, circulatory failure, toxaemia, damage to the nervous system or cardiac failure in cases of hyperkalemia (Barragry, 1974).

Stocking density

The effect of stocking density on water consumption (WC) (Table 10.2) demonstrated that WC increased at higher stocking densities (Feddes *et al.,* 2002). Birds housed in the lowest stocking density at 11.9 birds/m^2 consumed less water (5,093 mL/bird) than those in the other treatments. Some of this effect might have been due to lower feed consumption (FC). However, the WC:FC ratio clearly demonstrated that as the stocking density increased, WC increased independently. The amount of water consumed and the water to feed ratio was highest with a stocking density of 23.8 birds/m^2 (5,546 mL/bird and 1.85 mL/g, respectively). At high stocking densities, disproportionately high water intake may relate to satiety, imposed by failure to eat enough feed due to limited feeder space.

11

ELECTROLYTE IMBALANCE

Other names: Acidosis, Alkalosis. Also affects , tibial dyschondroplasia and amino acid metabolism

Species: All poultry

Compendium

Dietary electrolyte balance (DEB) in the body is mainly affected by the supply of electrolytes in the diet, but also by endogenous acid production and rates of renal clearance. While requirements for individual elements have been clearly defined, there is now an understanding of the need to achieve a balance between cation and anion supply. Most commonly, DEB is described by the simple formula of Na+K-Cl expressed as mEq/kg of diet. In most situations it seems as though an overall diet balance of around 250 mEq/kg is optimum for normal physiological functions. In reality, electrolyte imbalance does not occur, because the buffering systems in the body ensure the maintenance of near normal physiological pH. In extreme situations the need for buffering capacity seems to adversely affect other physiological conditions, thereby producing or accentuating potentially debilitating conditions.

In young broiler chickens, tibial dyschondroplasia (TD) can be affected by the DEB of the diet. Unusual development of the cartilage plug at the growth plate of the tibia can be induced by a number of factors, although its incidence can be greatly increased by metabolic acidosis induced by feeding products such as ammonium chloride. It seems as though TD occurs more frequently when the diet contains an excess of sodium relative to potassium when at the same time chloride levels are very high. Unfortunately much of the research involving TD and acid-base balance is confounded with a concomitant effect on body weight. For example, a certain balance of electrolytes may be claimed beneficial in reducing TD, but at the same time the body weight may be greatly reduced and this in itself will reduce TD severity. Great care must be taken in the interpretation of any research data in this area, such that any

base parameters in the blood. These authors showed the acidogenic properties of sulphate to depend on source, with values relative to mEq of chloride being approximately 58% when calcium sulphate and potassium sulphate were used, but that potency increased to 84% relative to chloride when, sulphate originated from sodium sulphate. As pointed out by Ruiz-Lopez and Austic, (1993) the failure of phosphate to influence acid-base parameters is likely a reflection of the buffering capacity of phosphate, because the pK_2 of phosphoric acid is within the range of normal physiological pH.

In a study of young pullets Moghaddam *et al.,* (2005) showed that varying the DEB from 187 to 284 had no effect upon the general performance parameters of feed intake, weight gain and FCR (Table 11.1). Also tibia ash and calcium contents were not influenced by the electrolyte balance.

Table 11.1. The effect of varying the dietary electrolyte balance on the performance of pullets from 7-35 days of age.

Performance parameter	*Dietary electrolyte balance (mEq/kg)*			
	187	*230*	*251*	*284*
Feed intake (g)	618.5	652.5	644.9	621.0
Weight gain (g)	203.3	205.1	206.7	202.0
FCR	3.04	3.18	3.12	3.08
Tibia ash (%)	46.67	48.03	48.93	46.54
Tibia calcium (%)	36.70	34.43	34.67	37.96

(Adapted from: Moghaddam *et al.,* 2005)

It seems that the birds were able to adjust their acid-base balance over a relatively wide range of DEB.

Murakami *et al.,* (2001) established dietary sodium and chloride requirements for growing broiler chickens, at 0.15 and 0.23%, respectively to give the best FCR. The best DEB for broilers was between 249 and 261 mEq/kg so generally agreeing with the original hypothesis of Mongin, (1980). These sodium, chloride, and DEB levels did not affect acid-base balance status or the incidence of TD in broilers.

In certain situations it may also be necessary to take into account the contribution of divalent ions in the diet. For example, feeding calcium chloride will induce acidosis in birds, while feeding sodium chloride or potassium chloride has little effect (Mongin, 1981). This situation probably develops due to less calcium being absorbed from calcium than occurs with sodium from sodium chloride. Since chloride absorption remains unchanged, and since calcium is excreted as calcium carbonate, there is the potential net loss of bicarbonate and net gain of chloride. Hurwitz and Bar, (1968) also indicated

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SKELETAL DISORDERS

Other names: Tibial dyschondroplasia (osteochondrosis)
Cage layer fatigue (osteoporosis)
Rickets (osteodystrophy)
Chondrodystrophy (Perosis, Angular bone deformity, Valgus-varus bone deformity)
Spondylolisthesis (Kinky back, Scoliosis)
Femoral head necrosis (Brittle bone disease)
Foot pad dermatitis (Pododermatitis)
Turkey leg disorders

Species: All poultry

Compendium

Various skeletal disorders affect most fast-growing meat birds, both broilers and turkeys, and also laying hens. Normal bone development is discussed in relation to bone cell types, structure, and the normal growth of bone from the embryo through to maturity. A number of factors are known to influence normal bone development and these are discussed essentially in relation to growth rate. Nutritional factors impacting bone development include protein and amino acids, vitamins, minerals and electrolyte balance as well as the role of specific ingredients and mycotoxins. Genetics, the sex of the bird, and growth rate are also major factors affecting potential bone disorders. A compendium of specific leg disorders are discussed in detail.

General introduction

Most poultry are afflicted with varying degrees of skeletal disorders at some time during their productive life-cycle. This is largely due to the selection pressure for production traits in modern lines of poultry that has placed increasing demands on skeletal integrity (Rath *et al*., 2000; Angel, 2007; Dibner *et al.,* 2007). Bone health, as manifested by tibia breaking strength per unit

in bone is around 2:1 and this essentially is the reason for the maintenance of this important ratio during feed formulation. Bone is the major mineral reserve in the body, representing about 99% of calcium, 88% of phosphorus, 80% of bicarbonate, 50% of magnesium and 35% of total mineral body reserves. The bone cavity is also a major site of fat stored in the body. Skeletal structure is shown in Fig. 12.1. Most of the discussion in this chapter deals with problems associated with the three main leg bones, namely the femur, the tibiotarsus and the tarsometatarsus. As will be discussed, most problems relate to bone or ligament integrity at the articulating joints of these three bones, and particularly the proximal and distal ends of the femur and tibiotarsus. For more extensive detail of general bone biology, the reader is directed to Whitehead, (1992) and Applegate and Lilburn, (2002).

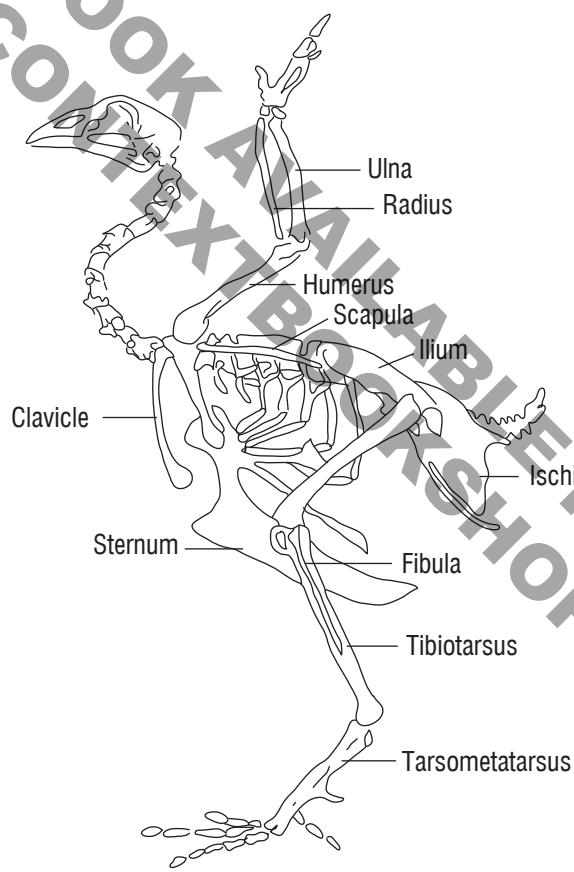

Figure 12.1 Poultry skeleton

14

MINERAL AND VITAMIN TOXICITIES

Other names: None

Species: All poultry

Compendium

There has been relatively little recent work published on the toxic effects of minerals or vitamins. Toxicities of these nutrients can affect the bird directly or more commonly cause an antagonism of other nutrient systems. Toxic levels of most minerals are tabulated, while for vitamins we have listed general upper safe levels in relation to requirement. Detailed discussions on mineral toxicities centre around copper, fluoride, magnesium, and vanadium, because these can be found at toxic levels as contaminants to conventional feedstuffs, or as in the case of copper, added as a treatment/prevention of enteric disorders. Sodium toxicity is discussed in terms of electrolyte and water balance, while an excess intake of calcium and phosphorus is described relative to skeletal and eggshell integrity.

High intakes of fat soluble vitamins seem to be problematic, and especially for vitamin A which appears to increase the birds need for vitamin D_3. Toxic effects of vitamin A can be corrected by feeding high levels of vitamin D_3. Toxic levels of vitamin E also disrupt calcium and phosphorus metabolism, while moderately high levels of vitamin D_3 and especially metabolites such as $1\alpha(OH)D_3$ cause aberrations in circulating calcium concentrations.

Trace mineral toxicities

There has been relatively little recent work published on the toxic effect of the various minerals. The summary shown in Table 14.1 is adapted from that provided by the NRC, (1994) in Nutrient Requirements of Poultry and from European Union documents on trace minerals (EC, 2004) and undesirable

substances in feeds (EC, 2002). Toxic levels shown in Table 14.1 generally relate to the lowest inclusion level currently known to produce signs. In most instances these signs are a reduced growth rate in immature birds, and have been induced when feeding as inorganic salts. In many instances, these toxicities can occur at much lower levels when the minerals are present as organic compounds. Toxicity will therefore vary with the form of mineral used, and likely be most severe for younger rather than older birds. The EU has published maximum permitted levels of trace minerals for feed use and maximum levels of various other minerals. The maximum permitted levels of trace elements are always well below any toxic level, and so these guidelines bear little relationship to the biology of the bird.

Table 14.1. Mineral toxicity from NRC (1994) and maximum permitted levels in the European Union (EU) in ppm in feed

Mineral	*Toxic level (NRC)*	*Maximum permitted level (EU)*
Aluminium	500 - 2200	
Arsenic	100	2-12
Barium	200	
Bromine	5,000	
Cadmium	20 - 40	0.5-10
Chloride	9 - 15,000	
Chromium	300	10
Cobalt	100	
Copper	250	15-175
Fluorine	400	30-2,000
Iodine	625	4-20
Iron	4,500	1,150
Lead	320	5-40
Magnesium	5,700	
Manganese	4,000	150-250
Mercury	250	0.1-0.5
Molybdenum	350	2.5
Nickel	400	
Selenium	10	0.5
Silver	900	
Sodium	8,900	
Strontium	6,000	
Tungsten	500	
Vanadium	5	
Zinc	800	0.5

(Adapted from: NRC, 1994; EC, 2002 and EC, 2004)

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