THE GENETICS OF LEG ABNORMALITIES IN POULTRY, WITH PARTICULAR REFERENCE TO DYSCHONDROPLASIA

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ABSTRACT

The genetics of dyschondroplasia in the broiler chicken was investigated with particular reference to the relationships between this condition, other leg problems and live-weight.

A quick and simple method of scoring growth plates by dissection is presented. It is concluded that radiological techniques are accurate in their diagnosis of lesions, but that otherwise live assessment is of little practical value. Definite strain differences were found amongst British broilers. A purified diet incorporating FP950, a soy-protein isolate, was formulated which consistently gave a high incidence of dyschondroplasia in birds reared on wire floors. Evidence for the existence of a diet x floor type interaction is presented, higher incidences being recorded when birds were reared in cages, as opposed to on litter floors.

The incidence of dyschondroplasia was increased in a selection line from 10% in G0, to 31% in G1 to 38% in G2, using information from full-sib dissections. Heritability estimates for dyschondroplasia obtained using an analysis of variance technique were high, averaging 0.66. No evidence for the sole involvement of only one gene arose from the use of test crosses.

Analyses were performed on field records of live birds from three pure-bred male-lines, each involving over 200 sires. Methods of calculating genetic parameters were compared. Analysis of variance and proband methods were both shown by simulation to be robust,

except when the incidence of the condition is low ($p<0.01$). The former is preferred for the estimation of genetic correlations, and the latter for heritabilities. Methods of predicting responses to selection in 0-1 traits are presented, which also appeared to be reliable from the use of simulation studies. All of the leg problems considered were seen to be inherited. These had largely positive genetic correlations with each other and with live-weight, and could be considered as a single trait. Dyschondroplasia appeared to have negative phenotypic and positive genetic correlations with live-weight, and was seen to be related to other leg defects. It is suggested that the various leg problems have arisen as a consequence of rapid growth rates in the modern broiler.

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CHAPTER 1. INTRODUCTION

In any flock of commercially reared broilers, up to 5% of the birds produced may die as a result of leg weakness. This, of course, represents a serious source of economic loss to the grower and retailer. It has been estimated that leg weakness costs the poultry industry of the USA over $28m per annum (Craig, 1978). Of these weaknesses, dyschondroplasia is perhaps second in importance to the various forms of twisted leg. Although it is extremely difficult to assess accurately the losses attributable to dyschondroplasia, in 1975 the Australian Chick Meat Federation offered an unverified estimate of $A0.5m per annum (Burton et al, 1981).

Many different factors can be seen to affect the incidence of the various leg problems. It is widely felt that leg weaknesses, which are not obviously the result of nutritional imbalance, are the consequence of the high growth rates found in various types of meat poultry. Weight is being gained in these birds at a rate which is faster than that with which the physiological development of the skeleton can cope. This leads to abnormal stress upon the legs and the development of defects. The concept presented above will be dealt with later. If this is the case, then the various different leg problems may be interrelated, and it may not be justifiable to separate them. However, in this study dyschondroplasia has been treated as a discrete problem.

The aim of this thesis was to investigate the genetic aspects of dyschondroplasia. As will be seen, it is impossible to achieve this

without considering various other factors which influence the condition. In particular the relationships of dyschondroplasia with other leg problems and with body weight are examined. This has been achieved by both experimental work and data analysis. The greater part of this study concerns dyschondroplasia, and therefore this is the subject of the following review.

Dyschondroplasia

Dyschondroplasia is typified by the presence of an uncalcified plug of cartilage found at the end of certain long bones. Usually the proximal end of the tibiotarsus is affected, and occasionally the proximal epiphysis of the tarsometatarsus. In a more thorough investigation of the occurence, Poulos et al (1978) have also reported the presence of abnormal cartilage in both the proximal and distal femur, the distal tibiotarsus, and the proximal humerus. An illustration of the parts of the leg normally affected is shown in Figure 1.1. Clinical symptoms are only developed in a minority of cases, and when these do occur, no signs of manifestation can be noted in broilers before 4-5 weeks old. Severely affected birds adopt unusual postures and show a tendency to squat. These birds eventually become lame, and are reluctant to move, enforced activity seemingly causing pain (Siller, 1970). On close examination, enlargement of the stifle joint can be detected, which may be indicated by tufts of feather protruding in that region. (see Figure 1.2)

Abnormal ossification may take place around the cartilage plug

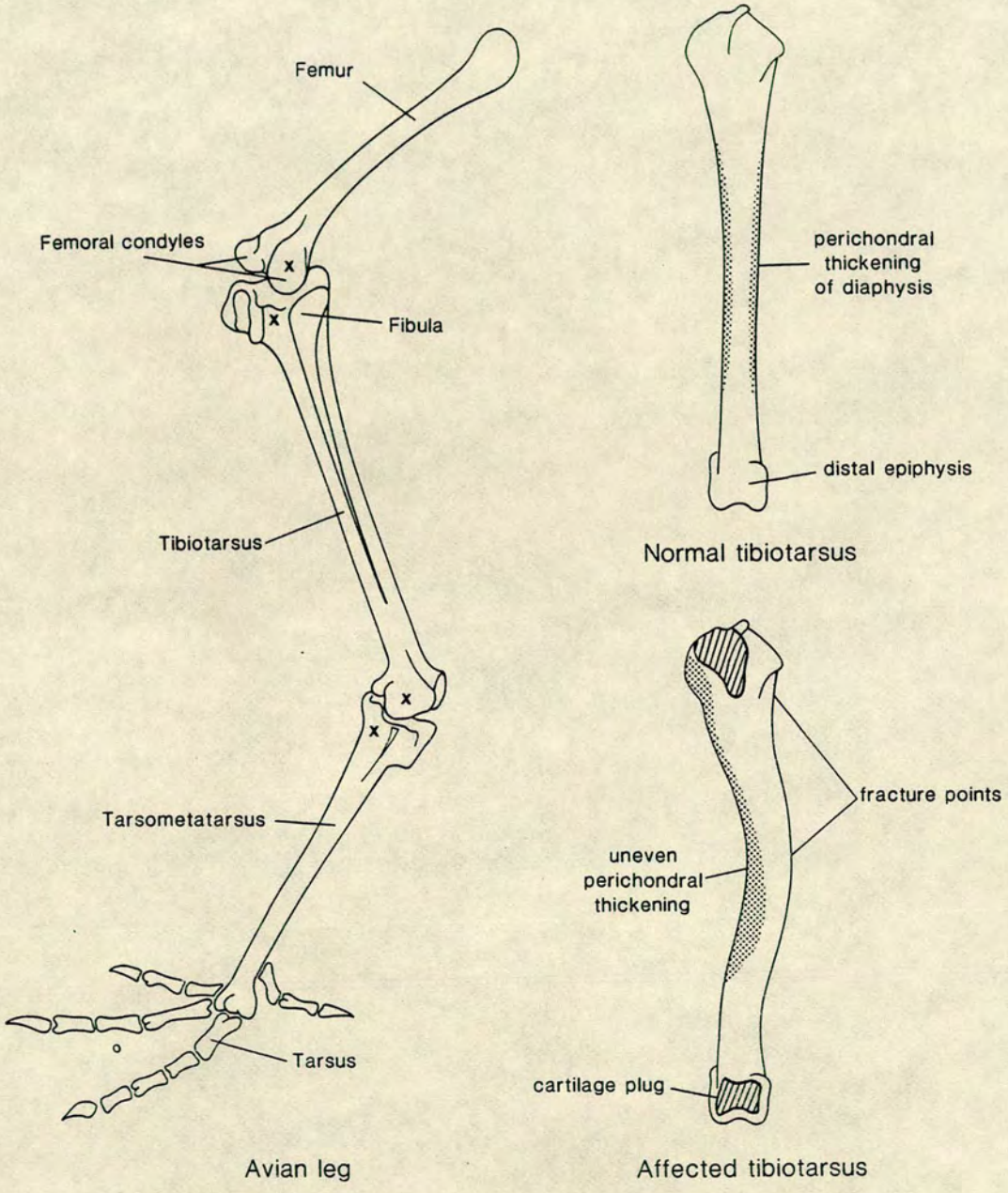

Femur
Femoral condyles
Fibula
Tibiotarsus
Tarsometatarsus
Tarsus
Avian leg
perichondral thickening of diaphysis
distal epiphysis
Normal tibiotarsus
fracture points
uneven perichondral thickening
cartilage plug
Affected tibiotarsus
x - site of dyschondroplasia

Fig.1.1

Fig 1·2

8 week old broiler affected by overt dyschondroplasia.

within the proximal epiphysis of the tibiotarsus. This can cause an uneven protruberance, sometimes leading to medial displacement of the whole epiphysis. One consequence of the cartilage plug is uneven endochondral ossification of the tibiotarsal diaphysis, the caudal aspect often becoming thicker in relation to the cranial, this leading to bowing. The relationship between bone dysplasia and dyschondroplasia has been examined by Itakura et al (1973). Such bending and bowing lead to abnormal stress on an already weakened tibiotarsus, which may fracture below the proximal epiphysis as the bird puts on weight. Poulos et al (1978) were able to demonstrate a correlation between dyschondroplasia and fibular fracture. Obviously, a broiler cannot be used if fracture occurs whilst it is still in the growing stage, but the main economic losses occur during transit to the production factory, and chiefly in the factory itself where birds are forcibly hung up by the shanks on a processing line.

The first major examination of dyschondroplasia was carried out by Leach and Nesheim (1965). They looked at a cartilage abnormality in broilers in the USA, where an incidence of 7-25% was found in four commercial 'meat' strains. Cartilage abnormalities were reported in British broilers by Laursen-Jones (1970) and by Siller and Duff (1970), who recorded incidences ranging from 0.5-2.0%. Hemsley (1970) corroborated the occurence of the abnormality in American and British flocks, and also observed its presence in Canada, South Africa and Australia. In his initial trials on various Australian flocks, clinical incidences of 0.0-0.45% and subclinical incidences of 1.0-5.5% were indicated. Siller (1970) subsequently introduced

the name tibial dyschondroplasia.

A similar condition seen in turkeys, and now thought to be the same phenomenon, was named osteochondrodystrophy by McCapes (1966), asceptic necrosis by Nairn (1968), and focal osteodystrophy by Steinke (1971). Incidences of 3-30% have been seen in turkey flocks by Nairn, and Steinke records incidences as high as 33-48%. Wise and Nott (1975) reported the occurrence of dyschondroplasia in meat strains of duck, where although relatively small numbers were examined, very high incidences up to 100% were recorded amongst different strains.

In broilers, Riddell et al (1971) reported that dyschondroplasia was widespread as a subclinical entity in Western Canada. Itakura et al (1973) recorded a 5-10% incidence in one commercial flock of hybrid White Rock x White Cornish birds in Japan. In the United States, Prasad et al (1972) have recorded a morbidity in affected flocks of 30-40%. Commercial strains have been found in Australia with incidences ranging from 13-35% (Burton et al, 1981). In contrast, the condition has not been reported so far in normal White Leghorn chickens.

Poulos et al (1978) believe that dyschondroplasia is merely one manifestation of a general condition of retarded ossification of the long bones, known as osteochondrosis in mammals. They suggest that the general term osteochondrosis be adopted. The term dyschondroplasia is preferred here as this more correctly describes the cartilage abnormality. The prefix 'tibial' should be dropped,

because, as has been seen, the condition is by no means restricted to the tibiotarsus. A grouping of dyschondroplasia with osteochondrosis is invalid, bearing in mind the structural differences of the long bones in meat poultry and mammals. Differences are found in the composition of the epiphyses, the vascularisation, ossification and developmental processes (Wise and Jennings, 1973). For clarification it is possible that the term avian dyschondroplasia should be applied.

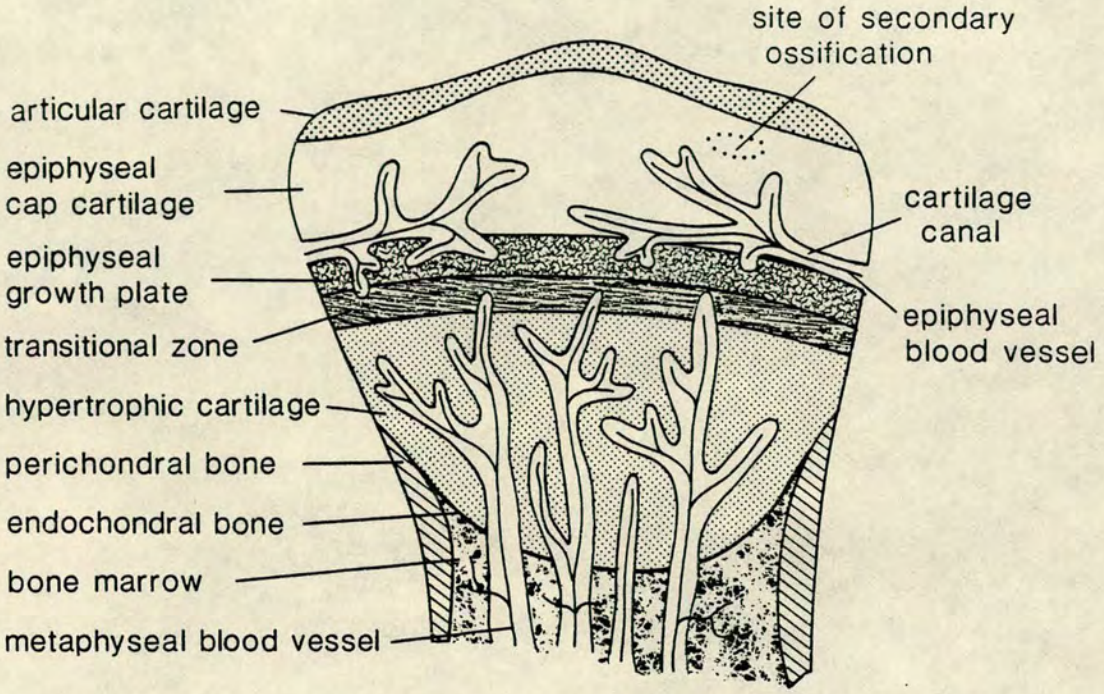
Morphology and Development

For a better understanding of dyschondroplasia it is necessary to consider the developmental process of the long bones in normal 'meat-strains' of poultry. It has been demonstrated by Wise and Jennings (1973) that the growth plates developing most rapidly in the long bones of the turkey are the proximal growth plates of the tibiotarsus and tarsometatarsus. This has also been affirmed for broilers by Riddell (1975b). In a repeat of an experiment performed by earlier workers, involving the surgical insertion of pins, he demonstrated that the proximal growth plate of the tibiotarsus is 4% and 33% more rapid in its respective growth rate than those of the tarsometatarsus and humerus respectively. He also noted that within these bones the proximal growth plates grew more rapidly than the distal growth plates. These sites of rapid development are most frequently affected by dyschondroplasia.

An embryonic cartilage cone is present in the end of the tibiotarsus when the chicken is one day old. This reaches a maximum

diameter of 3-4mm (Siller, 1970). The cone becomes eroded by invading loops of blood vessels, and has disappeared by the time the development of the medullary cavities in the long bones is complete, after about 14 days. Lutfi (1967) has demonstrated that the cone differentiates into the growth plate and epiphyseal cap by the 11th day. It is no longer thought that retention of the embryonic cone as such is responsible ~~responsible~~ for the initiation of dyschondroplasia. A similar cone is found in turkeys (Poulos, 1978), which, as development of the long bones is slower, persists until 20-25 days. Epiphyseal growth is thought to cease in the chicken by 16-18 weeks.

Howlett (1979), comparing broilers with White Leghorns, describes five cellular layers in the growth plate: a germinal layer, a proliferative zone of flattened cells, a prehypertrophic zone of transitional cells, a hypertrophic zone with large rounded cells occupying prominent lacunae, and a degenerative zone. He concludes that there are no ultrastructural differences in the cells of the growth plates of the two types of hen. Reiland et al (1978), however, do report structural differences in the arrangement of the zones between Leghorns and broilers. Egg layers have a better defined columnar arrangement of cells within the proliferative zone, this extending somewhat into the prehypertrophic zone, which is narrower than in broilers. Also in the Leghorn there are clearer defined transitions between the various cell layers, which break down in the broiler. Development of the metaphysis in layers shows a more even calcification front, together with better calcification. Details of the cellular zones of the avian tibiotarsal epiphysis are



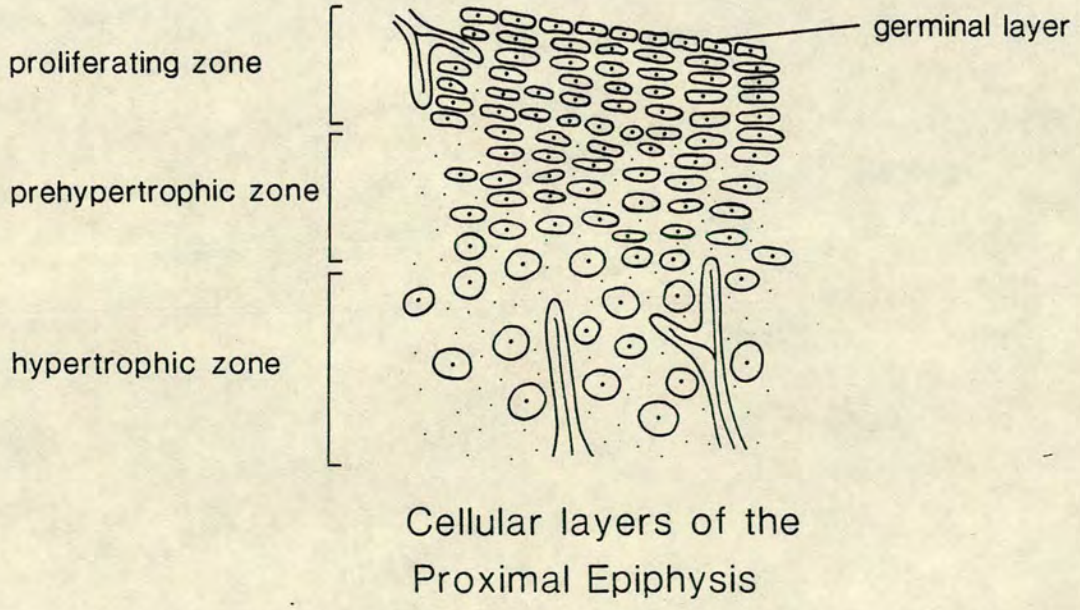


Fig. 1. 3 Morphological features of the Tibiotarsus in a normal chicken.

presented in Figure 1.3, together with the gross morphology.

An important feature of ossification is the presence of a correct blood supply. In the tibiotarsus of the chicken, a vascular groove leads into a nutrient foramen. This is situated on the caudo-lateral surface, distal to the fibular crest and adjacent to the interosseous space (Riddell, 1977). Epiphyseal arteries in vascular channels arise as branches of an epiphyseal circumferential artery, supplied in turn by the ischiatic artery (Poulos, 1978). These supply the proximal side of the growth plate, penetrating the proliferating zone of chondrocytes. They are thought by some authors to be responsible for the continued development of the growth plate. There are no anastomotic links between the vessels, which terminate in capillary loops (Wise and Jennings, 1973). The metaphyseal blood vessels are twice as numerous in the normal bird. These ascend the tibiotarsal diaphysis as terminal branches of the nutrient artery (Wise and Jennings, 1973; Riddell, 1977). Vessels penetrate the hypertrophic zone of chondrocytes and are probably responsible for degeneration of the cells, lysis occurring immediately in advance of, and lateral to, the advancing metaphyseal tunnels (Wise and Jennings, 1973). The capillary tunnels are lined with osteoblasts which will eventually deposit osteoid on the surface. The normal vascularisation process is regular and orderly. A thin avascular region separates the epiphyseal and metaphyseal blood vessels, and any tunnels passing through this region are seen to be constricted. Reiland et al (1978) report that the above process is not as orderly in broilers as in Leghorns, and that the blood supply appears poorer in the broiler.

Dyschondroplastic plugs, i.e. plugs of retained cartilage found in birds affected by dyschondroplasia, are usually described as occurring simultaneously in both tibiotarsi, but unilateral plugs have been reported by various authors. The first signs of dyschondroplasia occur after 14 days in the broiler (Riddell, 1975a; Poulos et al, 1978) and 39 days in the turkey (Poulos, 1978). A similar course of development would appear to occur in ducks (Wise and Nott, 1975), although this is not well documented.

The layers of cartilage cells within the epiphyseal growth plate become disrupted, and the plate itself increasingly thickened. The zone of proliferative cells lacks normal columnar arrangement (Siller, 1970) and becomes slightly thickened (Itakura et al, 1972). The transition into a zone of prehypertrophy breaks down. Initially focal thickening may occur (Reiland et al, 1978), cells become retained, the progression into a hypertrophic zone prior to normal ossification being disrupted.

The retained abnormal cartilage cells are thought to be prehypertrophic chondrocytes (Riddell, 1975a; Poulos, 1978). These appear mainly as nuclear bodies surrounded by shrunken cytoplasm present in otherwise vacant lacunae (Poulos, 1978; Siller, 1970; Itakura et al, 1973). There is an abrupt transition between the cartilage plug and the medullary cavity of the diaphysis, only a thin layer of spongy bone separating the two (Siller, 1970). Cytoplasmic strands, possibly capillary precursors, are seen to cross the cartilage plug (Poulos, 1978). As the abnormal chondrocytes are smaller, the cartilage matrix is more abundant

(Siller, 1970). Trabeculae below the cartilage plug are normal in size but lack the usual arrangement parallel to the axis of the bone (Riddell et al, 1971). A thin layer of ossification begins to take shape around the edges of the plug. This increases unevenly along the diaphysis, hyperplasia of the bone on one side together with reduced ossification on the other leading to bending of the tibiotarsal end (Siller, 1970). Bone dysplasia may become marked in the cortex of the diaphysis, which may become three to four times thicker than normal in birds affected by dyschondroplasia (Itakura et al, 1973). Necrosis of abnormal cells normally only occurs in the distal part of large plugs. Occassionally a necrotic band may be seen at the apex of the plug. This constitutes a point of fracture and condylar separation (Randall and Mills, 1981).

The cartilage plug reaches a maximum size of about 15-20mm in chickens (Poulos et al, 1978). This occurs at between 6-8 weeks of age (Poulos et al, 1978; Riddell, 1975a), although in certain cases degeneration of the plug may have already started by then. Randall and Mills (1981) examined a flock of broilers for clinical symptoms at 3 and 6 weeks. They found that of birds affected at 3 weeks old 43% became worse, 37% remained the same, and 17% had legs that had improved at 6 weeks old. Wise and Jennings (1972) considered that if dyschondroplasia started to develop at a time later than 6 weeks, this was usually as a sequel to another leg weakness such as rickets or perosis. Degeneration begins in turkeys after about 14 weeks, penetration by blood vessels and normal ossification gradually taking place until the plug has completely disappeared. In broilers resorption of the plug may occur at any time between 8 and 20 weeks,

but usually by 12-14 weeks (Riddell, 1975a). Sequestra of cartilage have been found further down the diaphysis, surrounded by bony spicules (Riddell et al, 1971; Itakura et al, 1973). This has been reported in birds of 30 weeks by the former authors. The gross effects on the skeleton remain, however, after all cartilage has gone.

It is possible that dyschondroplasia arises because of irregularity of the blood supply to the developing growth plates. Examination of this blood supply in affected birds has caused some discrepancy in the literature. All authors agree that blood vessels do not penetrate the abnormal cartilage plug to any extent. However, there is a question as to whether the surrounding blood supply is adequate enough to cause normal degeneration of the zone of hypertrophy. Siller (1970) reports that the epiphyseal and metaphyseal areas of affected birds appear to be less well vascularised than in normal birds. Riddell (1977), using standard histological staining techniques, as did Siller, examined two strains of chickens, one bred for a high incidence of dyschondroplasia, and the other for a low incidence. He observed that there was a statistically significant reduction of metaphyseal blood vessels present in the high incidence strain in birds younger than 5 weeks (the time by which the condition is usually well established). Indeed, Riddell (1975b) also experimentally induced a condition almost identical to dyschondroplasia by surgical blockage of the blood supply from the metaphysis to the growth plate. Unfortunately, his results are open to question, largely because of small numbers involved and a doubt as to the exact effect of trauma

attributable to the surgical technique. In contrast, Poulos (1978), using a detailed perfusion technique to produce angiograms of the turkey tibiotarsus, was unable to detect a deficiency of the blood supply in affected birds, when compared with normal birds. Possibly the reduction of metaphyseal blood vessels in Riddell's high incidence line was incidental, or maybe dyschondroplasia is not a manifestation of a reduced blood supply.

Alternatively, the condition may be be caused by defective degeneration of cartilage, prior to penetration by the metaphyseal vessels. This would allow proliferation of an abnormal amount of cells, which would accumulate in the transitional zone. Walser et al (1982) report a reduced number of chondroclasts in birds affected by Fusarium-induced dyschondroplasia. They suggest that defective lysis and resorption of hypertrophic cartilage resulting from this lack may be responsible for the plug, chondroclasts being a prerequisite for vascular penetration and ossification.

A further possibility is that development of a cartilage plug is a function of the cartilage itself, some biochemical property possibly preventing the penetration of capillaries or the action of degenerative agents in the vascular tunnels. Interestingly, Howlett (1974) reports that in birds affected by dyschondroplasia, calcification of the cartilage matrix occurs after calcification of the hypertrophic chondrocytes, a situation which is the reverse of normal! This points to a possible biochemical difference in the dyschondroplastic cartilage matrix. Lilburn and Leach (1980) demonstrated that dyschondroplastic cells had a depressed capacity

to utilise glucose, reduced metabolic activity resulting in a lowered rate of cytochrome oxidase and citrate synthesis. This could indicate either an impairment of mitochondrial function or a reduction in the actual number of mitochondria. Lack of evidence for the latter in ultrastructural examination (Howlett, 1979) would suggest impairment.

Lowther et al (1974) examined the matrix components of cartilage plugs in affected birds and compared these with cells from normal growth plates. There was no evidence found for a reduction of proteoglycan in dyschondroplastic cartilage. Indeed, there was possibly slightly extra proteoglycan in the proximal 2mm of the growth plates of affected birds. The ratios of proteoglycan to collagen in normal and affected birds were also similar, as were the chemical compositions of the extracted proteoglycan subunit. The collagen fibrils were not seen to be deficient in cross-linking. No differences were noted in the rate of biosynthesis of proteoglycan between hyaline articular cartilage and epiphyseal cap cartilage (the area above the growth plate) of normal and affected birds. However, there was a 92% decrease in synthetic ability of dyschondroplastic cartilage compared to growth plate cartilage. Biochemically, the abnormal cartilage had a uniform composition resembling that of the growth plate cartilage during growth. Lowther concludes that the abnormal plug results from an increased proliferation of growth plate cartilage followed by a slowing-down and cessation of biosynthesis of cartilage proteoglycan (hence the uniform composition). Proteoglycans are normally destroyed before or during the remodelling of cartilage to bone, but the inhibition of

this step may be related to the absence of capillary tunnelling.

It may be unreasonable to assume that the whole problem is vascular, and other factors concerning hormonal or nutritional imbalance may be critical. However, in the light of there being no evidence to suggest this, it seems that a reason to explain the failure of epiphyseal and metaphyseal vessels to perform the normal ossification process must be sought.

Poor vascularisation of the epiphysis, failure of the resorption process and biochemical deficiencies are not necessarily mutually exclusive. Possibly there may be a critical period of development, at which, in affected chickens, the cartilage cells of the proliferating zone undergo an abnormal period of division. Cells are produced at a mitotic rate greater than that with which the normal ossification process is able to cope. A plug of cartilage cells subsequently forms, which forces advancing blood vessels periphally around its mass. Normal degenerative processes are unable to resume until this period of abnormal cell division has ceased or slowed down sufficiently for the blood vessels to penetrate the plug, which is then eventually resorbed. It is suggested that this critical period occurs at between 2-3 weeks, and that normal development may occur unless a threshold level with respect to rate of proliferation is reached. The increased thickness of the zone of proliferation noted by Riddell (1975a) in high incidence strains supports this theory.

A possible modification to the idea of restricted blood supply was proposed by Wise and Jennings (1972), who thought that abnormal pressure caused by increased body weight and postural effect may be restricting the metaphyseal blood supply. Randall and Mills (1981) also suggest that small postural differences caused by the crowding of heavy, commercial broilers may be sufficient to cause dyschondroplasia. This will be considered further, later.

Effect of nutrition on dyschondroplasia in the chicken

An obvious explanation of dyschondroplasia would be that it occurs as a result of some nutrient deficiency or imbalance. When first noted, it was compared with the condition caused by a copper-deficiency (Leach and Nesheim, 1965). It has since been established (Lilburn and Leach, 1980) that a genetic defect in copper metabolism is not in fact responsible for dyschondroplasia. In their initial trials, Leach and Nesheim (1965) were unable to detect differences in the blood content of calcium, phosphorous, magnesium, and alkaline phosphatase between normal and affected birds. Further experiments involved feeding birds purified control diets lacking in only one of the following components: vitamin A, vitamin D, calcium, phosphorous, magnesium, manganese, zinc, choline, folic acid and niacin. Of these, none resulted in dyschondroplasia, but diets deficient in calcium or vitamin D caused rickets. A toxicity effect was looked for using diets with an excess of vitamin A, and β-aminoproprionitrile, with no success. Diets with added minerals were then fed to chicken strains with high and low incidences of dyschondroplasia. No effect on reduction of incidence

was noted with calcium, phosphorous, magnesium, selenium, iron, copper, copper plus iron and zinc. Neither were effects evident in diets both lacking in and replete with vitamin D3, vitamin D2, niacin, pyridoxine and inositol. Severity of the condition was increased in the high incidence strain, however, and both severity and incidence increased in the low incidence strain in birds fed either added fluoride, or chloride.

Similar sets of experiments have since been performed. Leach and Lilburn (1980) managed to double the incidence of dyschondroplasia in birds given a purified basal diet by feeding a wheat-based assay diet. Ferguson et al (1978) fed birds corn-soybean diets deficient in one of synthetic biotin, choline, folacin, manganese, and niacin respectively, choline and niacin deficiencies increasing the incidence of dyschondroplasia. Beirne and Jensen (1981) demonstrated that the incidence of dyschondroplasia in chickens was not related to influences in the maternal diet. Veltmann and Jensen (1981) used diets containing an excess of one of the following: monensin, sodium chloride, copper sulphate, magnesium sulphate, roxarsone, gentian violet, erythromycin, vitamin K, vitamin D, fluorine and ammonium chloride. Of these, only the latter resulted in an increase in the incidence of dyschondroplasia. Other workers, including Leach and Nesheim (1972), Riddell (1975a), Sheridan et al (1974), and Mongin and Sauveur (1977) have been able to increase the incidence by raising the level of chloride (or more specifically, ammonium chloride) in the diet. Merkeley (1979) increased the incidence with added fluoride.

On a more practical level, Leach and Nesheim (1965) found that the incidence of dyschondroplasia could be reduced to zero by continued feeding of a 50-100% chick starter diet. An incidence of 58% was also reduced to zero by continued feeding of a corn-soybean meal. However, the workers were unable to isolate an active ingredient causing the change. The further implications of these experiments upon growth rate are considered later. Veltmann and Jensen (1981) failed to reduce the incidence of dyschondroplasia by feeding extra yeast or other distillation products. Conversely, Edwards and Veltmann (1981) reduced the incidence of dyschondroplasia by including a low calcium and high phosphorous mixture in a practical-type diet. Similar results are reported by Leach (1982), who also showed, however, that there were no observable changes in total calcium, ionic calcium or total phosphorous associated with dyschondroplasia in a comparison of the blood content of affected and non-affected birds.

Dyschondroplasia can be induced, or the incidence lowered, by altering the acid-base balance of the blood. Leach and Nesheim (1972) were able to reduce the incidence by increasing the bicarbonate level of the blood, and were able to demonstrate a highly significant correlation between the two ($r=-0.85$). They also showed that different strains of chickens have the same response in blood pH to added chloride. Riddell (1975a) was able to correlate an increased chloride content in the diet with an increased chloride content of the blood serum, together with decreased pH and bicarbonate levels. However, he was unable to demonstrate a relationship between incidence of dyschondroplasia and the level of

blood bicarbonate. This latter result is surprising, as Mongin and Sauveur (1977) were also able to show a significant correlation between incidence of dyschondroplasia and blood bicarbonate ($r=-0.975$).

Mongin and Sauveur (1977) demonstrated that the effect of dietary chloride was dependent upon the levels of cations balancing the serum chloride, high chloride levels in relation to sodium and potassium levels (in terms of milliequivalents) causing an increased incidence. When chloride levels were high, an excess of sodium to potassium resulted in a high incidence of dyschondroplasia, but the incidence was not affected when potassium was in excess to sodium. A simple ratio of minerals was proposed of (Na+K-Cl), a maximum weight gain for the chicken occuring when fed a diet containing Na+K-Cl = 25mE per 100g.

Mongin and Sauveur (1977) were also able to reduce the incidence of acidosis-induced dyschondroplasia by increasing the levels of acetate and sulphate. The effect of these anions is explained by Sauveur and Mongin (1978). Sauveur (1969) showed that sulphate ions reduce acidosis by increasing the urinary excretion of free hydrogen ions by the renal tubules in laying hens. Richet et al (1966) showed that acetate ions consume free hydrogen ions during their catabolism and that this annuls ammonium loading. A possible effect of increased chloride levels is suggested. Vitamin D3 has to be transformed into the metabolically active 1,25-dihydroxycholecalciferol in the kidney before it becomes capable of the uptake of calcium from the gut. This process has been

seen to be affected as much as 40% by metabolic acidosis in rachitic chickens. Sauveur and Mongin suggest that a similar relationship may be operating in the case of acidosis-induced dyschondroplasia. It has yet to be seen whether dyschondroplasia actually impairs the formation of 1,25-DHCC.

To summarise, no single nutrient or mineral mixture so far examined has been shown to be directly responsible for initiating dyschondroplasia in the chicken. It would appear that this is sensitive to nutrient balance; acid-base balance, cation-anion balance and calcium : phosphorous ratios all affecting the incidence (Leach, 1982). Steinke (1971) reports that a similar situation exists for the turkey. Obviously, different nutritional environments can alter the susceptibility of birds to the condition, and this will be considered further in relation to the effect on growth rate and body weight. It is unlikely, however, given the wide range of incidences amongst broiler stocks, together with the failure of experimental work to pinpoint specific causes, that nutrition plays the most important role in the condition.

Effect of growth rate

The theory, proposed by Wise and Jennings (1972), that dyschondroplasia may be caused by increased body weight and abnormal posture reducing the metaphyseal blood supply to the growth plate of the proximal tibiotarsus, has been broadened a little by Bergmann and Scheer (1976). They conclude that the condition is based upon a disproportionality between weight, pressure and skeletal growth. The

mode of development of the cartilage plug has already been discussed, and it has been suggested that rapid early growth may be important in the development of dyschpondroplasia.

Riddell (1975b) looked at the measurements of long bones at different stages of growth in chickens of high and low incidence strains. The high incidence strain birds were seen to be slightly heavier, and to have a more rapid rate of bone growth than low incidence strains over a period of 2-8 weeks after hatching. The individual data, however, did not show any correlation between this rate and incidence of dyschondroplasia. This may be explained by the fact that affected individuals are more likely to suffer retarded growth as a consequence of their growth plate abnormalities. The average growth rate of individual growth plates was more rapid in 'high incidence' birds.

Riddell (1975b) also carried out a series of interesting experiments in an attempt to relate body weight directly to dyschondroplasia, by surgically severing the gastrocnemius tendon of one leg, and thus forcing the weight onto the other leg. Unfortunately, the results were very inconsistent, and must be regarded with scepticism, for several reasons. The numbers examined were very small, and the actual effect of the surgical technique on the individual chickens' performance and behaviour largely unknown. More importantly, it may be that in order for the blood supply to the growth plate to develop properly, the bird has to be able to exercise its leg. In birds crippled at 10 days old, a greater number of cartilage plugs developed in the operated leg, whereas in birds

crippled at 19-22 days more plugs developed in the unoperated leg. The result at 10 days was unexpected, but may be tentatively interpreted as pointing to the possibility that the blood supply to the growth plate is more critical for the prevention of dyschondroplasia at 10 days. To maintain a correct flow the leg has to be exercised. After the critical period of development, it may be that increased body weight comes more into play, the operated leg already having had the chance to develop a normal growth plate.

Most work performed on growth rate has been concerned with using dietary changes to alter body weight as an indicator of growth rate. Leach and Nesheim (1972) were able to intimate that early growth rate has a significant effect on increasing the incidence of dyschondroplasia later in life. This was achieved by feeding practical chick starter, to a 'high incidence' strain of chickens for varying periods of time. Birds were fed a purified basal diet for the rest of the trial period. When the starter was used only in the initial week of the chickens' life, a 92% incidence was recorded. If the starter was used continuously, however, from 1-4 weeks or 2-4 weeks, then incidences of 17% and 29% were recorded respectively. Feeding of starter from 0-3 weeks and then returning the chicks to the basal diet resulted in an incidence of 62%, but with the lesions being of reduced severity. These results suggest that in order to prevent the formation of dyschondroplasia, it is necessary to restrict growth rate during the time of actual catilage development at 2-4 weeks.

Huff (1980) found that, by restricting the amount of food

available for the chickens to eat, he could lower the incidence of dyschondroplasia, although the body weight of the birds was also reduced. Riddell (1975b) also showed that by diluting the energy value of the diet a reduction in incidence could be achieved, this occurring at a 33% dilution, but not at a 17% dilution. A similar type of experiment was carried out by Poulos et al (1978), who fed broilers two different energy level feeds. A significant weight difference was noted between birds fed the high and low plane diets. A higher incidence of dyschondroplasia was recorded in birds fed the high plane diet, and it was concluded that the condition is related to rapid growth. Interestingly, other skeletal deformities showed no such correlations to the two diets. Poulos et al suggest that in broilers, growth and remodelling of the skeletal features is very rapid, heavy weight in the main body of the birds putting stress upon the legs. Dyschondroplasia only affects growth itself when the plug is sufficiently large to cause dysfunction of bone development. Unfortunately, these results must be treated with caution as an unintended difference in the calcium levels of the diets has since been discovered, possibly having an effect on development.

Steinke (1971) reported that the heavier of two strains of turkeys examined had the greater incidence of dyschondroplasia. The incidence in turkeys could be lowered by reducing the amount of feed, with a corresponding reduction in growth rate. Wise and Nott (1975), working with ducks, noted that severity of dyschondroplasia could be related to the mean body weight of the strain of duck, but could not find a correlation with body weight within strains. Experiments were performed in which ducks were fed restricted diets

for varying lengths of time (at the start of the experimental period, at the end of the period, or continuously). Ducks were presented feed ad libitum, or an amount calculated to represent two-thirds of the ad-libitum diet. Lower incidences of dyschondroplasia were recorded in birds started off on a restricted diet. This corresponds to the period of early growth rate, which may be very rapid in strains of duck bred for the table, high incidences of dyschondroplasia ensuing.

As yet, there has been no success in showing a positive phenotypic correlation between growth rate and development of dyschondroplasia. No real attempt has been made to examine this relationship in the early, critical period of development, with the possible exception of ducks. Various different indices of growth rate must be considered. Body weight is only one possibility, and it may after all turn out to bear no relation to dyschondroplasia. One major advantage in relating the condition to growth rate is that this can be used to explain the results obtained by various authors in breeding strains with high and low incidences of dyschondroplasia. It is just as likely that high growth rates at a critical period of development are being selected for as is dyschondroplasia per se. More is said about genetic correlations between incidence and weight in the next section.

Genetic influence

There is a general agreement that strain differences exist amongst chickens, ducks and turkeys with respect to incidence of

dyschondroplasia. In an examination of three major broiler strains on the processing line in Australia, Burton et al (1981) recorded incidences of dyschondroplasia of 35%, 27% and 13% respectively. Veltmann and Jensen (1981) found no strain difference in a trial in which broilers were reared in wire cages, but in this instance, only one case of dyschondroplasia was seen in total.

The first workers to attempt to select for the condition were Leach and Nesheim (1965) as part of their comprehensive treatment of the subject. By taking sire and dam combinations from progeny test data, two strains of chickens were bred with incidences of 41% and 16% respectively, in the first generation. The parent population (White Plymouth Rock x Vantress) had an initial incidence of 10-15%. A marked sex difference was noted in both high and low incidence strains of progeny, females having a lower incidence of dyschondroplasia. Continuing the selection programme, incidence in chickens of the 'high' line fed a purified diet reached 68%, with an exceptional year producing an incidence of 80% (Leach and Nesheim, 1972). The 'low' line incidence dropped to 0%. The previous sex difference is thought to have been caused by a difference in the time of development, females having an optimum time of development 1-2 weeks later than males. Females are, of course, also lighter. To overcome this difference, only males were used by the workers in their nutrition experiments. Family selection was used, all matings being progeny tested, and within family crossing was avoided to prevent inbreeding.

Riddell (1976) also developed a low incidence strain in which

dyschondroplasia had entirely disappeared after three generations of progeny testing, together with a high incidence strain of chickens with over 50% of the birds affected. The original commercial population had incidences of 10% in males and 7.6% in females, birds being selected for the presence or absence of cartilage plugs at 6-8 weeks by means of radiography, and being bred like to like. The selection procedure was seen to lower the growth rate of the birds (Riddell, personal communication).

The genetic component of dyschondroplasia has been more thoroughly investigated by Sheridan et al (1974, 1976, 1978). Initially, two pedigreed breeding populations were examined, and the incidence of dyschondroplasia was seen to vary considerably among 20 sire families within each population (Sheridan, 1974). Two 'high' incidence strains of broilers were subsequently established (one being later discontinued), all progeny being pedigreed to the dam family, and incidence in sire family being taken as the selection criterion.

A brief summary of the results taken from the breeding programme (Sheridan et al, 1974, 1976 and 1978) may be considered here. A change in mean incidence was reported from 10% in the first generation of selected progeny (F1) to 24.3% in F2, to 37.4% in F3 to 80% in F4. Population sizes for each generation ranged from 741 in F2 to 1891 in F4. Sire component estimates of heritability were calculated (pooled sexes) which were shown to change from 0.43 to 0.57 to 0.52 to 0.18 over the four generations of progeny respectively. The dam component heritability estimates of F2 and F3

were seen to be half those of the sire component heritabilities, but in F4, the dam component heritability was greater than that of the sire component. This is possibly attributable to maternal and/or dominance effects in this generation. There was an overall bimodal distribution of sire family incidence of dyschondroplasia, and in particular a clear bimodality amongst female offspring in F3. The heritability of body weight was seen to be greater in F4 than in F3, and the genetic correlation between body weight and dyschondroplasia was negative in F3, but positive in F4. The F3 result is surprising as it implies that selection for increased body weight would lower the incidence of dyschondroplasia.

In addition, a linkage test was carried out to try and relate dyschondroplasia to the presence of a sex-linked gene. Two sires in the trial were shown to be segregating for a sex-linked gene controlling rapid versus slow feathering. If dyschondroplasia was related to the feathering gene, then the incidence of the condition in the female progeny groups would differ from an expected 1:1 ratio. This was seen to be the case with the groups from one of the sires.

Sheridan et al (1978) suggested that if a lower genetic variance with respect to incidence of dyschondroplasia was indeed present in dams relative to sires, then this would indicate the presence of a major sex-linked recessive gene. This hypothesis is supported by the bimodality shown between the sire families, and the results of the linkage test. The F4 results were explained in terms of the gene becoming fixed within the experimental population, this leading to

the reduced sire component heritability estimate, and the lowered overall heritability estimate for dyschondroplasia. It was also proposed that the change from a negative to a positive genetic correlation between dyschondroplasia and body weight in F4 represented the occurrence of gene fixation. It was suggested that a major sex-linked gene could be overdominant in its effect upon body weight. The realised heritability estimates were calculated (method not given) to be 3.61, 1.19 and 2.44 for F2, F3 and F4 respectively. These ridiculously high figures are possibly explained by the fact that various different changes in husbandry practices took place over the experimental period (Sheridan, personal communication). No single factor has been pinpointed. Sheridan et al suggest that if a high maternal or dominance effect was operating in F4 then the maternal female parent line can substantially influence the incidence of dyschondroplasia within the flock.

A statistical problem arises in the estimation of genetic parameters for defect traits such as dyschondroplasia. These traits are usually classified as present or absent, and are therefore binomially distributed. Most quantitative genetical theory assumes normality, and allowances must be made for this discrepancy. This issue is dealt with in some detail in Chapter 6 and Chapter 7, where further reference to Sheridan et al (1978) is made.

It may be accepted that dyschondroplasia can be selected for. Unfortunately, Sheridan's conclusions are drawn from insufficient evidence, and there is room for doubt as to whether these are all valid, particularly the idea of gene fixation. The sex difference in

incidence has not always been found by other authors, such as Leach and Nesheim (and indeed, may be partially or wholly attributable to differences in developmental rate). Certainly more attention could be focussed on maternal effects. Various other environmental factors may play a large role in determining incidence, particularly hatch effects. The environment may also determine the penetrance of any single gene responsible for dyschondroplasia, which is likely to be variable. It would be convenient to attribute the condition to the effect of a major sex-linked recessive gene, but a more rigorous breeding trial would have to be performed before this became acceptable. Further attempts should also be made to correlate dyschondroplasia genetically with growth rate. The fact that the incidence of dyschondroplasia has not been accidentally raised commercially by inadvertant selection, points to the fact that the genetic mechanisms involved are not merely as simple as the involvement of a single gene. It is far more likely that selection trials have somehow also affected an aspect of growth rate or susceptibility with which dyschondroplasia is associated.

Miscellaneous influences

Various environmental factors not previously mentioned can be seen to have an effect on the incidence of dyschondroplasia. Ferguson et al (1978), in nutritional experiments, found that the incidence was greater on litter and plastic floors, as opposed to wire floors, in chickens. Similarly, Veltmann and Jensen (1980) reported lower incidences of dyschondroplasia when they reared broilers in wire cages as opposed to on litter. Incorporation of 5% raw or autoclaved

broiler litter in the diet fed to birds reared in cages made no difference to the incidence. Leach (1982) also reports higher incidences on litter. Generally, leg disorders in poultry are exacerbated by wire flooring.

Ross breeders (Gristwood, personal communication) demonstrated that incidence will increase with temperature. Batches of birds reared at temperatures of 15.5°C, 22.5°C and 29°C had incidences of 1.71%, 6.41% and 10.63% respectively. Interestingly, it was noted that the highest incidence was recorded in a pen which had a particularly high humidity. It was thought that this increase in incidence might be related to the activity of the chickens at these temperatures. Exercise trials were subsequently carried out and, surprisingly, enforced activity (birds were actively moved around the pen for five minutes every three hours of a working day, with light at 100%) doubled the incidence of dyschondroplasia. This result is difficult to explain. In another trial, however, where the feeding and watering troughs were at opposite ends of the pen, the incidence actually decreased. Activity would be expected to reduce the incidence if dyschondroplasia is caused by poor blood circulation. A further temperature trial failed to have any effect on dyschondroplasia. Caution must be taken, however, when considering the above results, as temperature may not only depress general activity, but also specifically feeding behaviour, causing birds to feed less, and thus possibly having an effect on growth rate. For comparison, Simons (1979) demonstrated that activity and lighting regimes could influence the incidence of twisted legs.

It has been suggested that dyschondroplasia arises as a result of the action of pathogens. Lowther (1971) found mycoplasma-like bodies in the dyschondroplastic matrix of broilers. Howlett (1974) noticed nuclear aggregations having a central dense core of 50nm and diameter of 90nm present with the prehypertrophic chondrocytes of a cartilage plug. He thought these were associated with chondrocytic degeneration, but did not mention the possibility that they represented pathogenic activity. Other authors, particularly Poulos (1978), who specifically looked for it, have found no evidence of pathogenicity. Lowther's results may well have been incidental, and indeed, have not been repeated (Sheridan, personal communication).

Although no pathogens have been found which are directly involved, attempts have been made to induce dyschondroplasia using fungus mould in the feed. These have met with mixed success. Huff (1980) actually saw a slight reduction in incidence (together with a lowering of body weight) when he fed Aspergillus flavusparasiticus to chickens. However, Walser et al (1982) induced an incidence of 90% by feeding low levels of Fusarium roseum. Veltmann and Jensen (1981) report a seasonal change in incidence of dyschondroplasia in broiler flocks, and suggest that susceptibility to the condition may be altered periodically by the presence of pathogens.

Probably stress, as a generalised phenomenon, increases the incidence of dyschondroplasia (Sheridan, personal communication). Leach and Nesheim (1965) considered the possibility that the condition may arise as the result of a hormone imbalance. Testosterone proprionate or diethylstilbestrol were fed respectively

to chickens in sufficient quantities to alter their secondary sex characteristics. This was not seen to have an effect on incidence of growth plate abnormalities.

To conclude, it is unlikely that any one environmental factor is directly responsible for dyschondroplasia. However, many factors may contribute to the susceptibility of a bird. These probably include hatch effects and other variables relating to husbandry which have not been considered above, such as incubation temperature, stocking density and disturbance.

Relation to general leg weakness

The leg weaknesses referred to in this section are skeletal abnormalities which cannot be attributed specifically to nutritional disorders. Much confusion exists in the literature over terminology. Perosis is a generalised term covering a number of weaknesses, particularly slipped tendon, and is usually nutritional. Wise (1975) suggested that 'perosis' be dropped from the literature and replaced by 'chondrodystrophy'. This is distinct from twisted leg, which is the commonest of the leg defects observed in the field.

Twisted leg in broilers is the most studied leg problem with the exception of dyschondroplasia. Simons (1979) reported field incidences of 5-6% in the Netherlands. He states, as does Hartmann (1979) that the incidence is greater in males than females. Simons also found that more cases of twisted leg arose in birds kept on wire floors than in birds reared on plastic floors. Wise (1975)

states that the incidence of twisted leg and bowing is greater when birds are grown in wire cages as opposed to on litter. Intermittent light regimes which increased the amount of exercise per hour produced a lower incidence of twisted leg than did continuous light regimes, although the latter produced a greater total daily activity (Simons, 1979). No relationship was found between weight on litter and incidence. The incidence was reduced by restriction of feed over the first 3 weeks.

Hartmann (1979) investigated the genetics of twisted leg. Both he and Simons (1979) founded selection lines in which the incidence of the condition was increased. Hartmann found no evidence of a correlation between the final weight of full-sib progeny groups and their mean incidence (r=-0.03), and was able to increase the body weight but reduce the incidence of twisted leg in the same birds. However, while severely affected birds were seen to be 10% smaller than unaffected on average at 8 weeks, birds with only a slight twist were 3% larger. This indicates that the condition may in fact be a consequence of a rapid growth rate. Twisted leg was found to be highly heritable. Sire component estimates of h^2=0.51 in pullets and 0.40 in cocks were obtained from a set of pure-line data, and heritability estimates of 0.10-0.30 were found in progeny from commercial crosses. Dam component estimates of heritability were seen to be higher than sire component estimates, suggesting the presence of maternal effects. Hartmann found large differences in the incidence of male progeny groups from female broiler parents resulting from reciprocal crossing of two commercial dam lines. He cites this as evidence for a sex-linked major gene, together with

bimodal distributions of incidence in both maternal grandsire and full-sib families. Hartmann compares these findings with those of Sheridan et al (1978) for dyschondroplasia, and concludes that the two conditions are probably closely related. The data presented by Hartmann are insufficient for this to be categorically accepted.

Taking all leg weaknesses in broiler roasters, and classifying them together, Hulan et al (1980) saw that the incidence was influenced by genotype, and could be increased by feeding diets with a high protein content. Poulos et al (1978) suggest that crooked toes are a result of abnormal posture in the chicken, with increased angulation of the hock joint attributable to large body weight and a heavily muscled breast causing uneven pulling on the tendons. Somes (1969) describes a condition involving twisting of the tarsometatarsus and slipping of the gastrocnemius tendon, which he calls perosis. As a result of test crosses he concluded that this was the consequence of an autosomal recessive mutant with variable penetrance, linked to the sleepy-eye locus.

Several authors believe that leg abnormalities are all a consequence of a generalised phenomenon resulting from rapid growth rate. It is suggested that dyschondroplasia is merely one manifestation of this growth condition, and should therefore not be separated from other problems (Poulos et al, 1978; Reiland et al, 1977). Summers et al (1978) have calculated that the modern broiler only consumes 60% of the vitamins and minerals per kilogram of weight gained as did birds 20 years ago. This implies that various leg deformities may arise as a consequence of poor mineralisation

caused by rapid growth. On examination of an egg-laying strain of chickens not selected for growth (White Leghorn) Reiland et al (1978) found very few leg weaknesses, in addition to an absence of dyschondroplasia.

Sheridan (1974) reports that an apparant relationship between incidence of dyschondroplasia and other leg weaknesses was noted by the Australian chicken meat industry. Randall and Mills (1981) found that 62% of chickens examined with leg deformities also had growth plate lesions, as opposed to 40% examined which had no gross leg deformities. Riddell (1976), however, was unable to find any correlation between dyschondroplasia and twisted leg or sponylolisthesis (a spinal defect). The same situation was again reported for twisted leg by Veltmann and Jensen (1981). In nutritional experiments which lowered the incidence of dyschondroplasia Poulos et al (1978) did not observe any corresponding reduction in the prevalence of various other skeletal deformities. These observations suggest that there is no direct association of dyschondroplasia with other leg problems.

It is possible that a relationship between growth plate and other leg abnormalities arises through cause and effect. Ferguson et al (1974) conclude that slipped tendon (perosis) is often a consequence of twisted leg and bow, but makes no reference to dyschondroplasia. Riddell (1976) suggests that twisted leg and bowing may be a sequel to, but not necessarily a result of dyschondroplasia. Randall and Mills (1981) report that twisting almost invariably occurs if dyschondroplasia is present in the distal end of the tibiotarsus.

The incidence of dyschondroplasia begins to decrease after 6 weeks, while that of other leg weaknesses increases from 5-13 weeks in chickens (Poulos et al, 1978). This is probably a consequence of cartilage plugs being resorbed, and a number of the other weaknesses may in fact be a consequence of growth plate abnormalities.

The evidence for association of dyschondroplasia with other leg problems in broilers appears to be conflicting. There seems to be no doubt that such abnormalities are more numerous in rapid growing strains. However, as for dyschondroplasia, little evidence has been found yet to demonstrate a positive genetic correlation between incidence of other abnormalities and live-weight. Restriction of diet has been found to lower the incidence of other leg defects, but this apart, nutritional experiments which alter the incidence of dyschondroplasia have not been seen to affect that of other abnormalities. Sex-linked single genes have been proposed for both dyschondroplasia and twisted leg. If this was the case then it would be expected that the two be closely associated. The evidence for their existence, however, is as yet circumstantial. Certainly both dyschondroplasia and twisted leg may be increased by selection. Whilst the incidence of dyschondroplasia has been reported to be higher when birds are reared on litter, that of other leg defects seems to be greater on wire floors.

Taken together, the above points indicate that there is no primary correlation between growth plate and other leg abnormalities. Above all, the cartilage plug, characteristic of dyschondroplasia, is unique to that condition, and for this reason it is possible to

treat dyschondroplasia as a discrete entity. This does not negate the possibility that the various different conditions share similar etioligies. Indeed, it is fair to say, as do Poulos et al (1978), that it is likely that skeletal deformity and ensuing leg weakness is the price that must be paid, at present, for rapid growth amongst broilers.

Conclusions

1. The condition involving the formation of cartilage plugs at the epiphyses of long bones in strains of poultry bred for meat is widespread, and should be referred to as avian dyschondroplasia.
2. The cartilage plug is composed of a mass of undifferentiated prehypertrophic chondrocytes arising from a failure of the epiphyseal growth plate to develop properly.
3. No known nutrient or nutrient mixture has been shown to be responsible for the condition, but a change in the acid-base balance of the blood serum may increase the incidence of dyschondroplasia - nutritional effects being contributory rather than primary causes of development.
4. A phenotypic correlation between growth rate and dyschondroplasia has not yet been established. It is suggested that early growth rate, as a critical factor, be examined. Although the condition is common in broilers it is absent in lighter egg-laying strains.
5. It is possible to produce both high and low incidence strains of dyschondroplastic birds by selection, but no definite genetic correlation with growth rate has been found.
6. A major recessive sex-linked gene has been proposed as the underlying cause of dyschondroplasia, but the evidence presented to

support this hypothesis is inadequate.

7. No definite involvement of pathogens has been shown.

8. Dyschondroplasia can be seen to be affected by various environmental factors, none of which are thought to be solely responsible for the condition.

9. Other leg weaknesses probably also arise as a result of rapid growth, although the evidence for this is no better than that for dyschondroplasia. The latter is sufficiently different from the other leg problems to be considered as a separate entity.

CHAPTER 2. METHODS OF EXAMINING DYSCHONDROPLASIA

Dyschondroplasia is a relatively scarce condition. It is therefore a problem to find suitable material with which to work. As the field incidence is very low, examination of large numbers of birds would be necessary to make any statistical conclusions concerning affected individuals. It is advantageous, therefore, to work with a population having a higher incidence than normal. There are three options for establishing a high incidence flock. As each can be expected to yield valuable information in its own right, all methods were utilised in the present study. These were as follows:

1. Searching existing populations for naturally occurring high incidence breeds or strains.
2. Using artificial selection for presence of dyschondroplasia, and breeding an experimental flock with a high incidence.
3. Manipulating the environment to produce conditions which stimulate a higher incidence than would be found under standard rearing conditions.

Details of each of the above are presented in later chapters.

Before attempting any experimental work, however, it was necessary to survey various techniques of actually examining birds, and to develop methods of assessing dyschondroplasia. Although dyschondroplasia may be clinically assessed in severely affected birds, many cases may have abnormal cartilage plugs and yet not show any overt symptoms, particularly when young. For this reason, dissection has usually been carried out, to be certain of detecting

the presence or absence of the condition. This was investigated in a survey to discover just which parts of the skeleton needed to be examined to detect dyschondroplasia; at what age were birds most likely to have developed the condition; and just how could the condition be quantitatively measured by dissection. Secondly, the question of how to assess dyschondroplasia in live birds, for purposes of selection, was considered. To this end, radiographic techniques were examined in conjunction with clinical assessment.

Dyschondroplasia in spontaneous mortalities and culls

An initial survey was carried out on the spontaneous mortalities and culls in male broilers reared on a commercial growing farm to a roaster weight. These birds were therefore reared for longer and obtained heavier weights (with attendant leg problems) than would be the case in normal broilers. Dead birds of known age were looked at as a whole carcass, and their legs assessed for bow-leg, twisted-leg, slipped tendon, and presence or absence of a leg abnormality. Both legs were then excised and the growth plates of the distal femur, proximal and distal tibiotarsus, and proximal tarsometatarsus assessed for abnormalities. Finally, the length of the tibiotarsi was recorded, and measurements of the growth plates taken.

It was felt that little bias would be introduced into the survey by the use of dead birds, as before the age of five weeks , very little of the mortality would comprise of culls attributable to leg problems. Thus, before this age, the sample would be drawn from

birds dying for many different reasons, and the distribution of dyschondroplasia can be assumed to be random. After this age, a few more individuals with leg problems would be seen, and the incidence would become higher than one would expect from a random sample.

Examination of the growth plate of the proximal tibiotarsus was achieved by taking several successive sagittal sections through the head of the tibiotarsus using a sharp scalpel. Sections of the other growth plates were taken in a coronal plane, but it is felt that providing several sections are taken in the same growth plate, it is unlikely that anything larger than a very small plug of abnormal cartilage will be missed, whichever plane is dissected. Growth plates were awarded a score of 1-5 according to the amount of abnormal cartilage present. Although this scale is to a certain extent subjective, the scores are consistent within categories, given that the same person carried out all the examinations. The categories are illustrated in Figure 2.1, and were as follows:

1. - normal growth plate, regular prehypertrophic zone of cells.

2. - thickened growth plate, even expansion of prehypertrophic zone, so that it is now thicker than usual.

3. - uneven thickening of the growth plate leading to a plug of cartilage cells not larger than 5mm in depth at any one point.

4. - medium size cartilage plug which may or may not extend across the whole growth plate, but rarely exceeding 10mm in depth.

5. - large plug greater than 10mm in depth and usually extending over the entire growth plate.

Figure 2.1. Sections of the heads of proximal tibiotarsi of 4 week old broilers.

(a) Normal growth plate - Grade 1.

(b) Thickened growth plate, very small cartilage plug - Grade 2.

(c) and (d) Notable cartilage plug, larger at one side of growth plate - Grade 3.

(e) Large plug extending across width of growth plate - Grade 4.

(f) Plug occupying whole of tibiotarsal head - Grade 5.

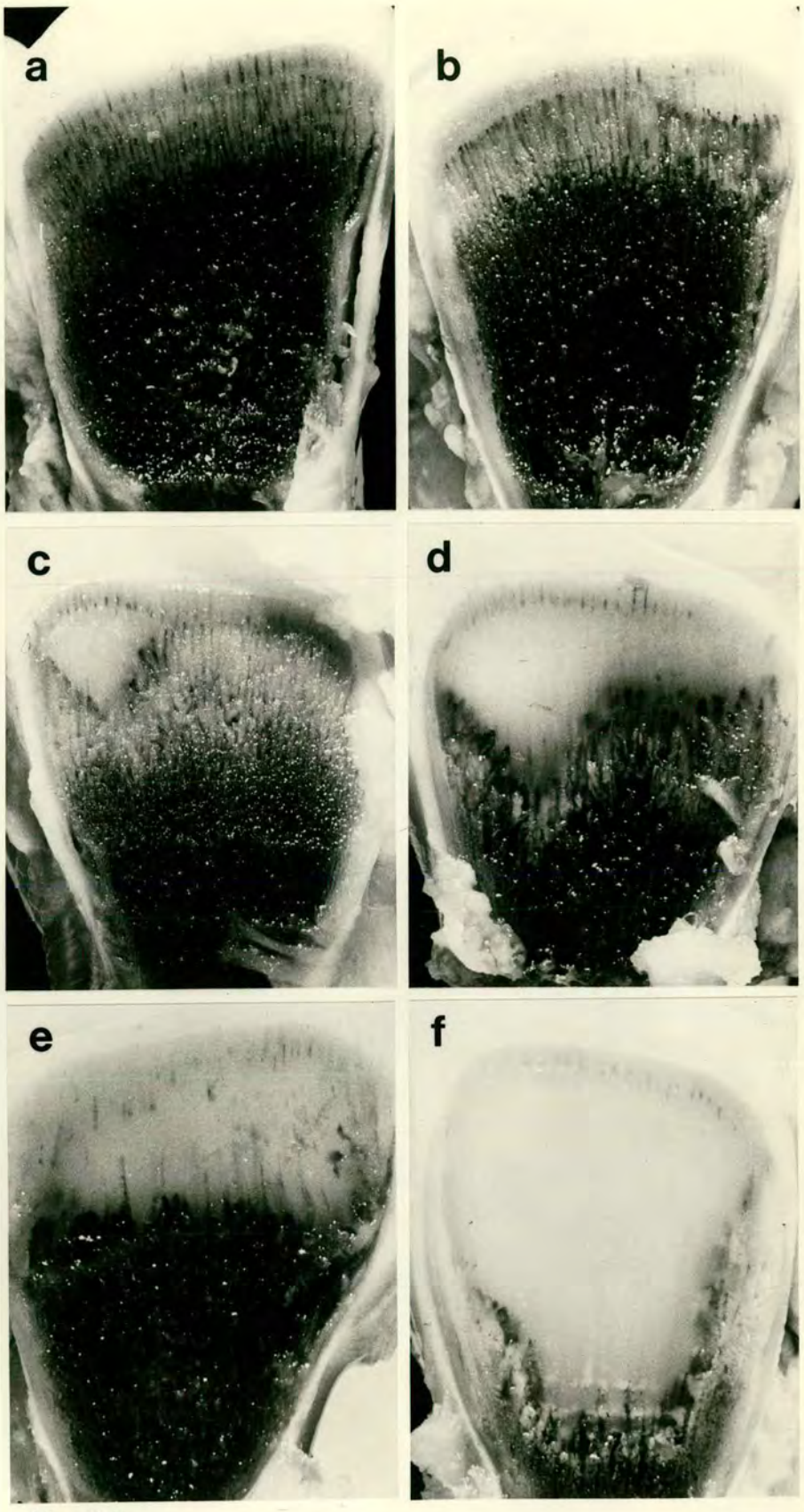


Fig 2·1

A total of 534 birds were examined out of a flock total of approximately 10800, mortality being collected from 10 days until slaughter age at 57 days. The overall incidence of growth plate abnormalities was 20.59%; the incidence of dyschondroplasia ignoring thickened growth plates being 14.79%. The location of growth cartilage lesions is presented in Table 2.1, and the change in incidence with age in Figure 2.2.

The survey shows that dyschondroplasia is most commonly found in the proximal tibiotarsus, and is also frequent in the proximal tarsometatarsus, but is rare in the distal tarsometatarsus and the distal femur. This pattern corresponds to the respective growth rates of the leg bones involved, the tibiotarsus developing more rapidly (Riddell, 1975b). It is interesting to note that a greater number of cases in which the tarsometatarsi alone were seen to have dyschondroplasia were recorded when the birds were older (7-8 weeks). It is possible that in these birds the lesions were healing, and that the cartilage plugs had been resorbed in the more rapidly devloping proximal tibiotarsus.

An examination of the age distribution shows a large number of young birds with thickened growth plates. In most broilers growth plates subsequently develop normally, an abnormal plug being derived from this thickening in the rest. However, the phenomenon was not observed in later studies, and may be partly the result of a mycotoxin infection known to be present at an early age in the flock. (cf Walser et al, 1982) No cases of true dyschondroplasia were seen in birds younger than 2 weeks old, but cartilage plugs

Table 2.1

(a)Distribution of growth plate abnormalities found in the initial survey of spontaneous mortalities and culls.

AGE(DAYS)	DF	PT	DT	PM	PT+PM	PT+PM+OTHER	N[1.]	TOTAL	%I
10-14	0	1	0	4	2	3	28	10	36
15-21	0	3	1	3	8	3	27	18	67
22-28	1	6	0	4	5	1	48	17	35
29-35	0	5	0	0	1	1	42	7	17
36-42	0	7	1	0	6	5	132	19	14
43-49	0	13	0	9	9	0	184	31	17
50-57	0	8	0	1	4	1	73	14	23
TOTAL	1	43	2	21	35	14	534	116	22

(b)Distribution of dyschondroplasia

AGE(DAYS)	DF	PT	DT	PM	PT+PM	PT+PM+OTHER	N	TOTAL	%I
10-14	0	0	0	0	0	0	28	0	-
15-21	0	3	0	1	0	0	27	4	15
22-28	0	5	0	2	3	0	48	10	21
29-35	0	5	0	0	1	1	42	7	17
36-42	0	5	1	0	7	4	132	17	13
43-49	0	12	0	9	8	0	184	29	16
50-57	0	8	0	1	2	1	73	12	16
TOTAL	0	38	1	13	21	6	534	79	15

DF = distal femur
PT = proximal tibiotarsus
DT = distal tibiotarsus
PM = proximal tarsometatarsus

Figures refer to the number of affected birds found in each category

1. The following headings are consistent throughout the rest of the tables presented:
N = number of birds examined.
I = incidence of dyschondroplasia.
%I = % incidence of dyschondroplasia.
LWT = live-weight.

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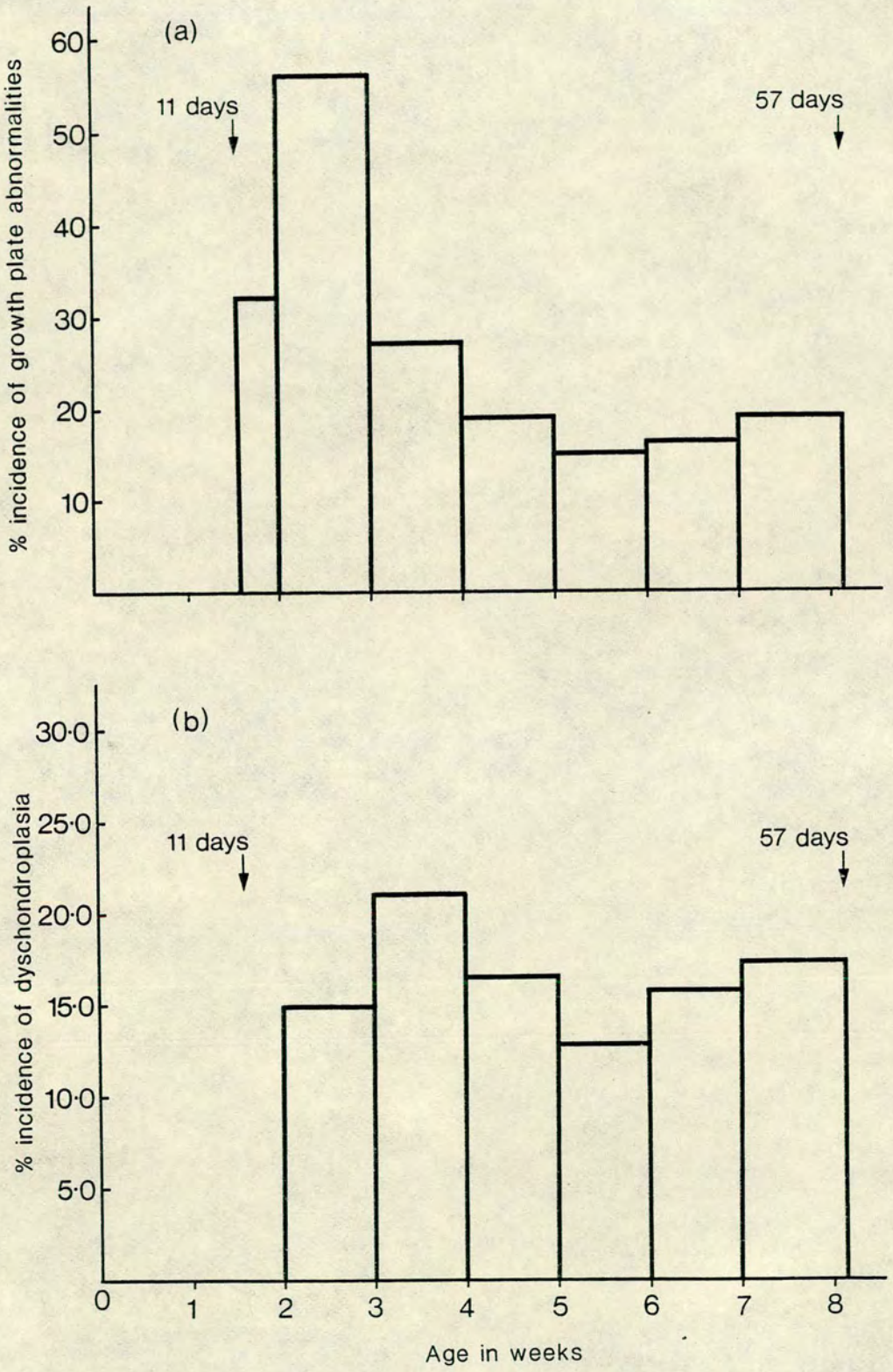


Fig. 2.2 Incidence of (a) growth plate abnormalities and (b) dychondroplasia amongst mortality in initial survey

have been found in birds only 10 days old in subsequent studies presented in this thesis. There appears to be a peak in the incidence of dyschondroplasia at 3-4 weeks, followed by another rise at 7-8 weeks. This may be explained by the fact that after 3 weeks a certain amount of resorption of the cartilage plugs takes place, as was found by Randall and Mills (1981). The second peak in incidence is probably caused by the inclusion of culled birds in the mortality.

The phenotypic correlation in incidence of dyschondroplasia (scored 1-5) between both legs is very high (r=0.96); however, the correlations between growth plate abnormality scores within individual growth plates are not quite as large (r=0.88, 0.92 0.91 and 0.87 for distal femur, proximal tibiotarsus, distal tibiotarsus and proximal tarsometatarsus respectively.) All correlations presented in this section have been corrected for age of bird. These differences may be considered to be outweighed by the overall agreement, and indicate that dyschondroplasia is largely bilateral.

Severity of cartilage abnormalities can be seen to be worse as birds become older, reaching a peak after 6-7 weeks. (Figure 2.3) There seems to be good agreement between the changes in severity in the tibiotarsus and the tarsometatarsus. After 7 weeks the slight decrease in the severity of the lesions may also be caused by resorption of the plug. The low mean severity of plugs in young birds is affected to some extent by the high incidence of thickened growth plates.

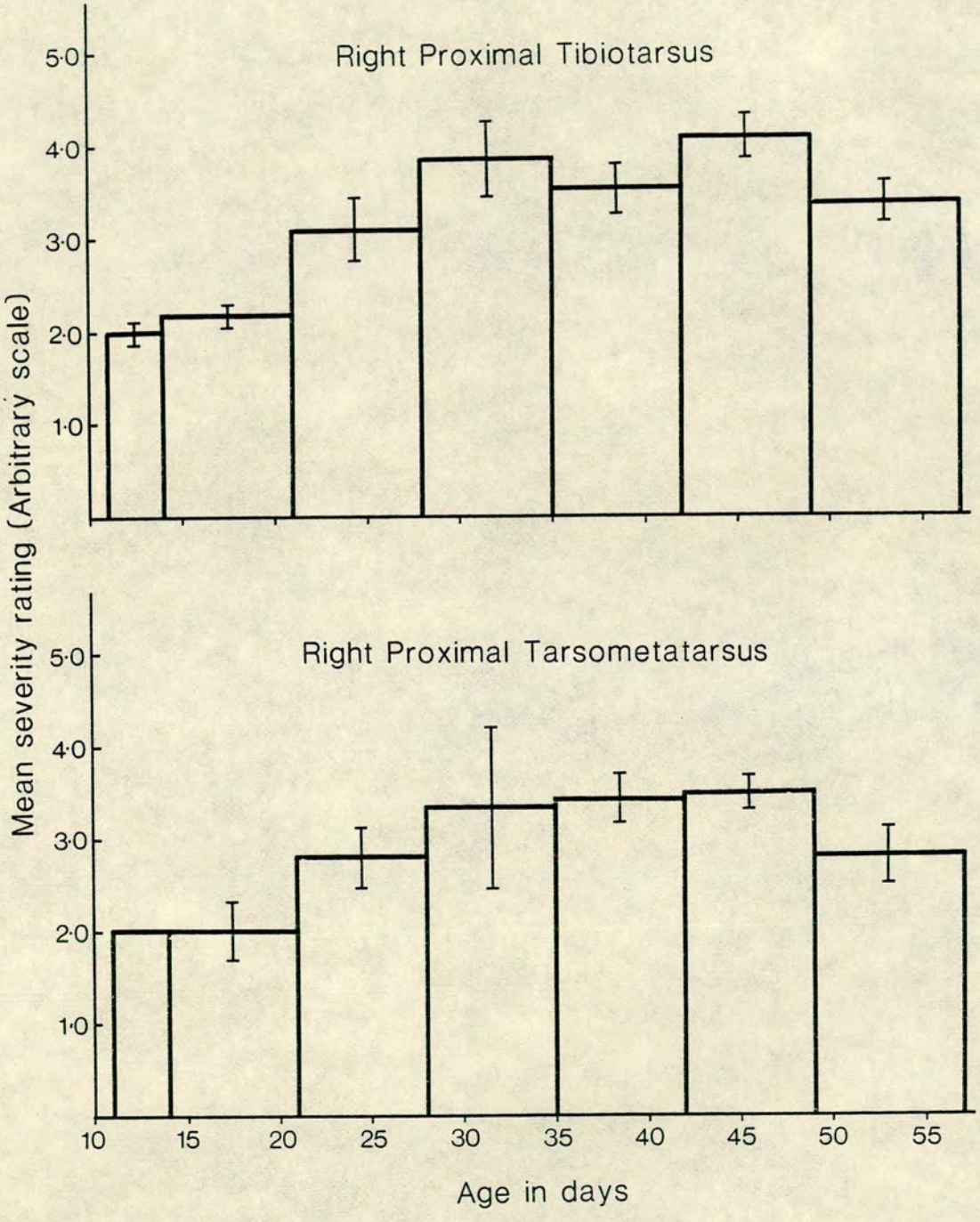


Fig. 2.3 Change in severity of growth plate abnormality in affected birds

Dyschondroplasia may be the underlying cause of loss attributable to other leg defects. As a consequence, it is important to consider the relationship of growth plate abnormalities with leg defects. In this study bow leg was considered as a bending of the tibiotarsal or tarsometatarsal diaphysis resulting in a 'bandy-legged' appearance. Twisted leg encompassed all valgus and varus deformities of the tarsometatarsus and hock joint, including true leg-twist and splay, as described in Chapters 4 and 6. True leg-twist was invariably accompanied by slipping of the gastrocnemius tendon at the hock joint. The incidence of leg abnormalities overall was quite high (47.19%), and the distribution of incidence over time similar to that of the growth plate abnormalities (Figure 2.4). Of the individual leg defects, the incidence of bow, twisted leg and slipped tendon were 12.21%, 37.21% and 3.62% respectively. The distribution of individual leg defects over time also reflects that found with dyschondroplasia, in that there is an early and a later peak in incidence. Bow is an exception to the above, development being delayed somewhat.

Twisted leg was also awarded a graded score from 1-5 depending on the degree of deviation at the hock joint. The phenotypic correlation between overall scores for twisted leg and overall scores for cartilage abnormalities was small but positive ($p=0.03$). In the growth plates of the proximal tarsometatarsi, however, this correlation is slightly larger ($p=0.08$). Dyschondroplasia seems to have little influence on the development of twisted leg, with the possible exception of dyschondroplasia in the hock joint, which corroborates Randall and Mills (1981).

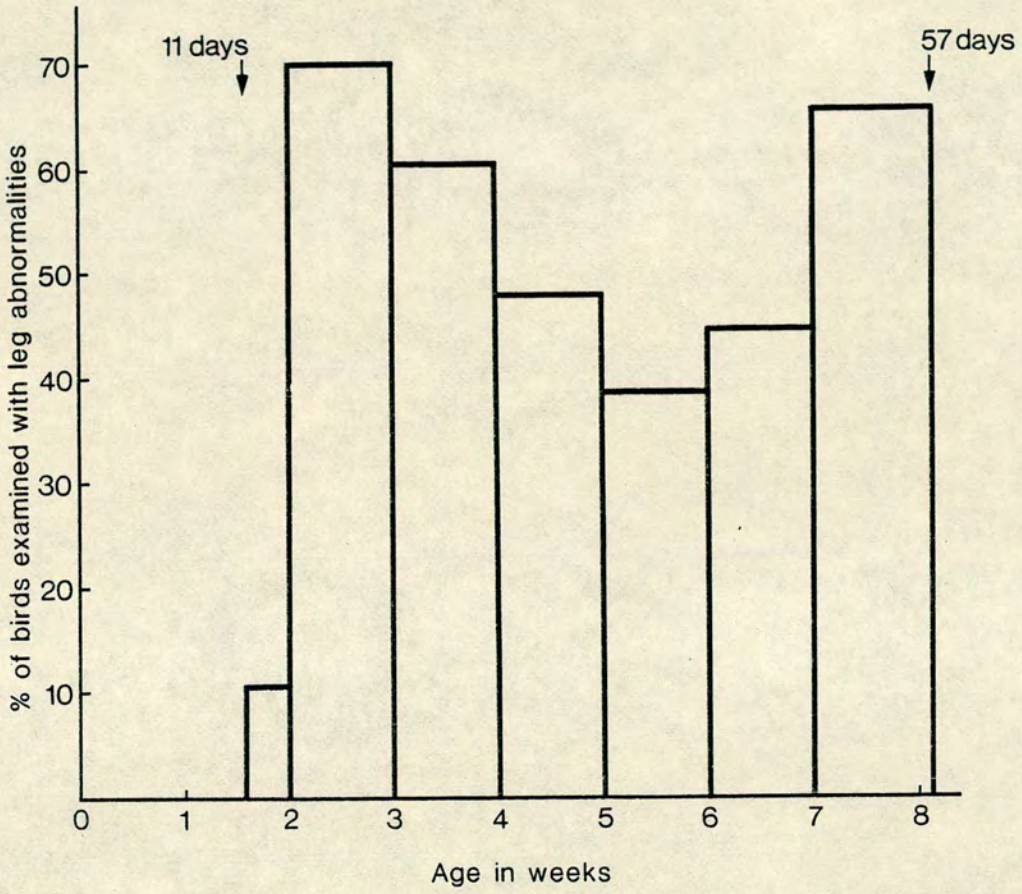


Fig. 2.4 Incidence of leg abnormality amongst mortality in initial survey

Table 2.2 gives an idea of the relationship between leg abnormalities and dyschondroplasia in the form of 2x2 contingency tables. Although no association between twisted leg and dyschondroplasia is shown, there is a significant association between leg abnormalities as a whole and dyschondroplasia (χ^2=18.62 1df). Indeed, 71% of birds with dyschondroplasia were seen to be affected by another leg problem. There is a significant association of bow with dyschondroplasia (χ^2=32.75 1df), which is not surprising, considering that one of the consequences of dyschondroplasia is bowing of the tibiotarsus in either an anteroposterial or lateral plane. No direct evidence was found of an association of dyschondroplasia with slipped tendon. Dyschondroplasia was often found, however, in the distal tibiotarsus and proximal tarsometatarsus of birds with slipped tendons, and was present overall in 47.5% of birds affected by slipped tendon. Taken with the slight positive correlation between dyschondroplasia and twisted leg, which may be attributable to cases of true leg-twist , this gives some indication that slipped tendon is sometimes concomitant with dyschondroplasia in the hock joint. The study here makes no allowance for the fact that leg weaknesses were found in some other birds that showed traces of having had cartilage plugs. These had subsequently healed and were detected by the presence of either sequestra of prehypertrophic cartilage or abnormal ossification in the diaphysis of the tibiotarsus. Therefore, there may be a greater degree of association than was shown here between growth plate abnormalities and other leg abnormalities, particularly in the case of bow. The implication of this is that dyschondroplasia may be responsible for economic loss not only through the culling of

Table 2.2

Association of dyschondroplasia (DY) and all growth plate abnormal-ities (GPA) with other leg abnormalities found in mortalities from the initial trial.

All leg abnormalities (LA)

	+DY	-DY	
+LA	56	196	252
-LA	23	249	272
	79	445	524

χ^2 = 19.37 1df P<0.005

	+GPA	-GPA	
+LA	73	179	252
-LA	37	235	272
	110	414	524

χ^2 = 18.62 1df P<0.005

Twisted leg (TW)

	+DY	-DY	
+TW	32	163	195
-TW	47	282	329
	79	445	524

χ^2 = 0.43 1df 0.50<P<0.75

	+GPA	-GPA	
+TW	46	149	195
-TW	64	265	329
	110	414	524

χ^2 = 1.27 1df 0.25<P<0.50

Bow (B)

	+DY	-DY	
+B	25	39	64
-B	54	406	460
	79	445	524

χ^2 = 32.75 1df P<0.005

	+GPA	-GPA	
+B	26	38	64
-B	84	376	460
	110	414	524

χ^2 = 18.53 1df P<0.005

Slipped tendon (ST)

	+DY	-DY	
+ST	5	14	19
-ST	74	431	505
	79	445	524

χ^2 = 1.96 1df 0.10<P<0.25

	+GPA	-GPA	
+ST	5	14	19
-ST	105	400	505
	110	414	524

χ^2 = 0.34 1df 0.50<P<0.75

1. Unless otherwise stated, statistical methods have been taken from Snedecor and Cochran (1967).

severely dyschondroplastic birds, but also by contributing to other leg problems which necessitate the culling of birds. The above relationships are, of course, all phenotypic; further consideration is given to the genetic correlations between traits in later chapters.

A final purpose of this study was to look at measurements of the legs and examine their relationship with dyschondroplasia. Obviously, live assesment could be greatly helped if there were, for example, large positive correlations between length of the tibiotarsus and presence of a cartilage abnormality. Measurements taken of a number of birds at various ages can also be examined to see if the presence of dyschondroplasia had any effect on the growth curves, or indeed, if the birds examined in the survey showed much deviation from a normal population in terms of growth rate.

Tibiotarsal length was measured from proximal to distal condyle. Growth plate width was measured to give an indication of variation in overall size of the tibiotarsal head. In the proximal tibiotarsus the width of the metaphyseal growth plate junction was measured after sagittal splitting of the bone extremity, in the other growth plates, this distance was recorded after coronal splitting of the extremity. Measurements of pairs of legs were compared, resulting in the conclusion that the left tibiotarsus is the same length as the right ($r=0.99$). It is felt that procedural errors would have been low, although this was not tested, and that differences in measurements are therefore real. Overall, the width of growth plates is almost the same in each leg ($r=0.87$), however, the correlations

within individual growth plates are not quite as high as this (r=0.87, 0.40, 0.74, 0.68 in the distal femur, proximal tibiotarsus, distal tibiotarsus and proximal tarsometatarsus respectively). In the proximal tibiotarsus, the low correlation between legs was corroborated by the fact that the mean width for each day of age was consistently greater in the right leg than in the left, and suggests that the right leg may be dominant in the growing chicken. The growth curves of both tibiotarsal length and growth plate width (Figure 2.5 and 2.6) suggested an almost linear increase in size at this stage of development, as would be found in normally developing chickens. These curves are similar to those presented by Reiland et al (1978), and add some justification to the concept of examining spontaneous mortalities and culls.

Looking at the relationship between growth plate abnormalities and measurements, a significant negative correlation between score and mean tibiotarsal length was found of r=-0.14. This implies that birds affected by dyschondroplasia are likely to have shorter legs than unaffected birds, shortened legs are probably a consequence of retarded growth attributable to dyschondroplasia. Conversely, there is a small, significant, positive correlation between overall growth plate width and growth plate score of r=0.12. This correlation was slightly larger in the proximal tibiotarsus and the proximal tarsometatarsus. Possibly birds with a faster growing growth plate are more susceptible to the development of dyschondroplasia, or possibly the presence of dyschondroplasia causes a diametric increase of the growth plate. The above correlations are not sufficiently large, however, to be of any practical use in the

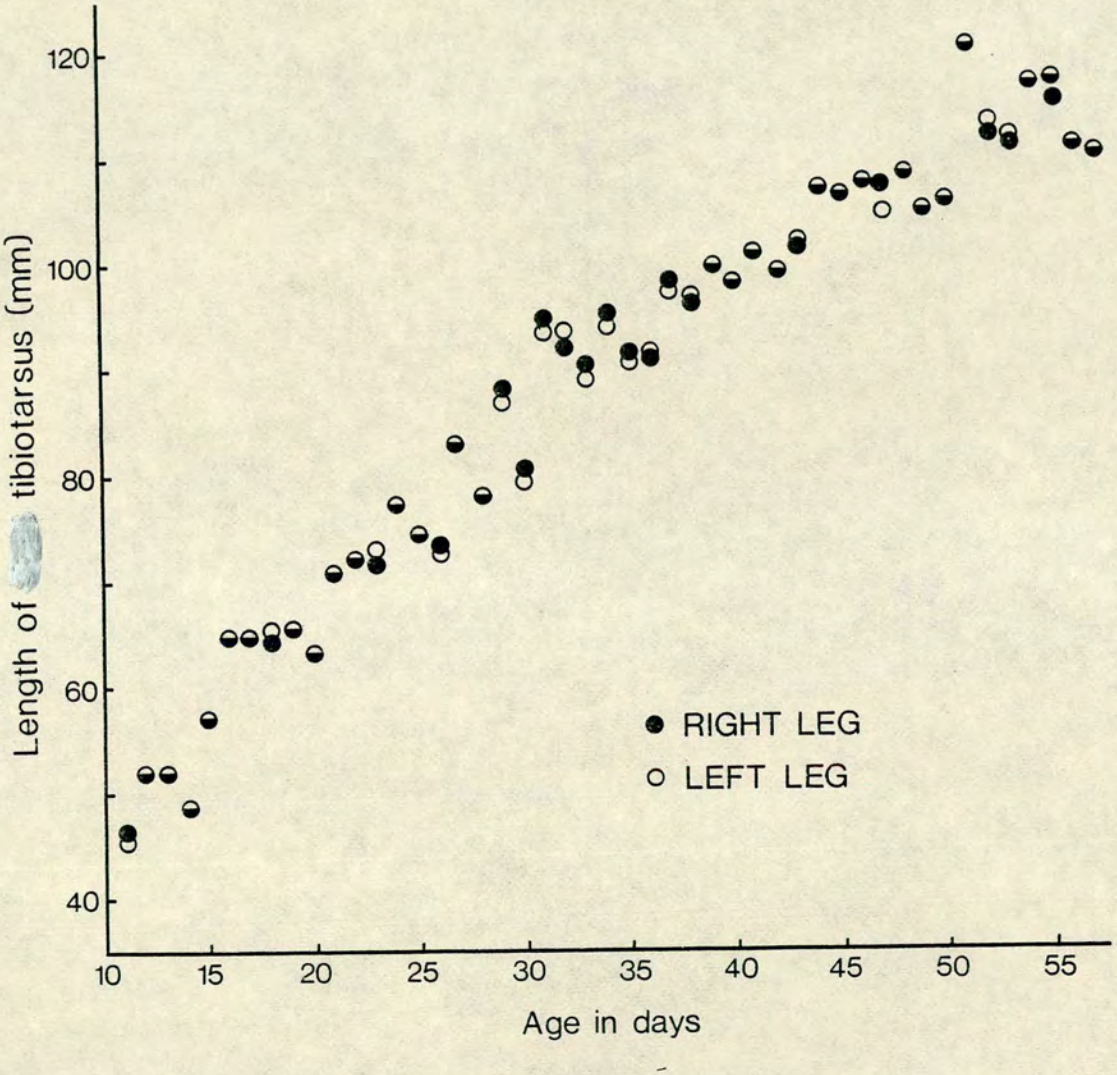


Fig. 2.5 Growth of right and left tibiotarsus amongst mortality in initial survey

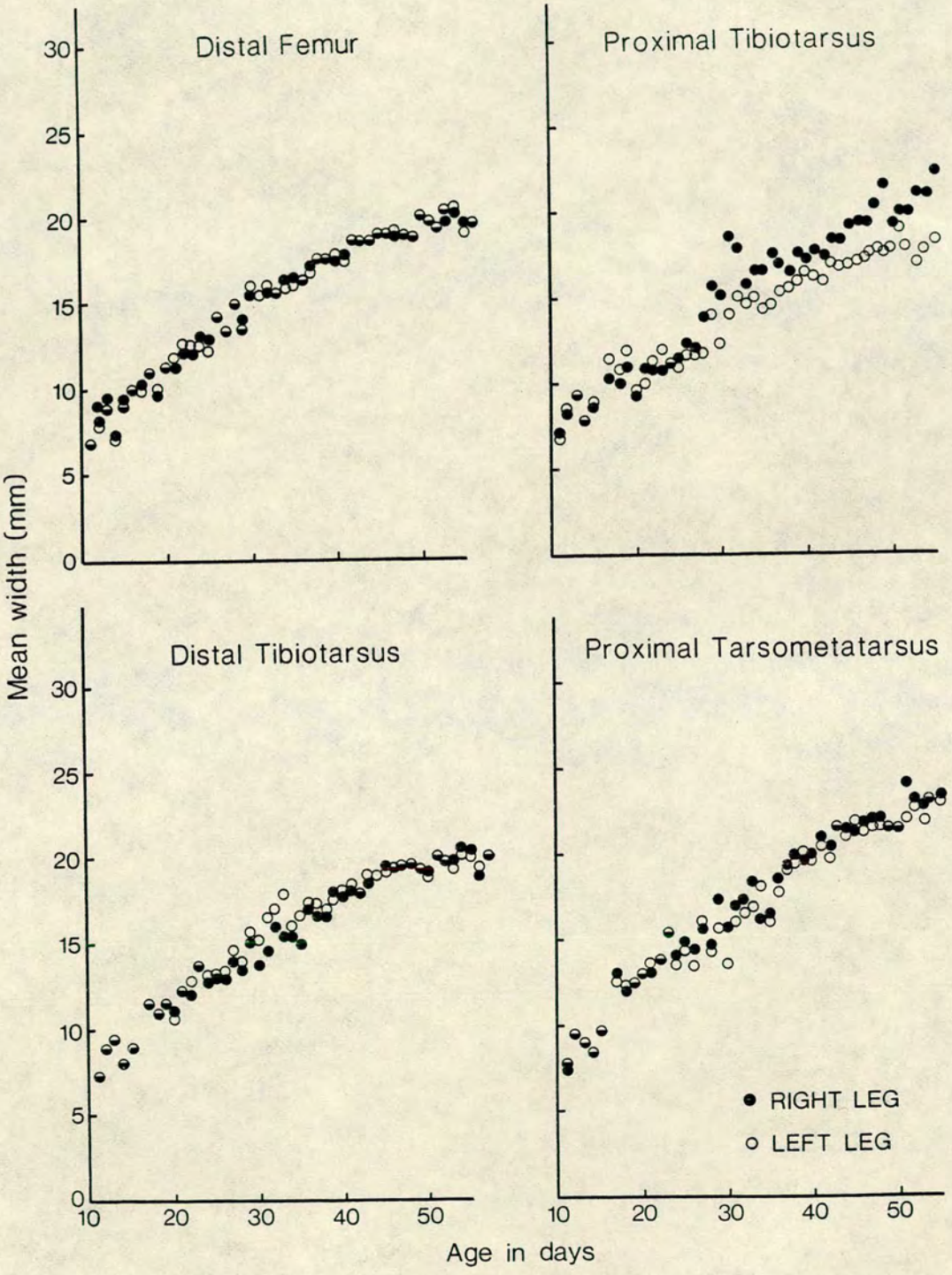


Fig. 2.6 Development of growth plates amongst mortality in initial survey

diagnosis of dyschondroplasia in live birds. No correlations were found between any of the measurements and the score for twisted leg.

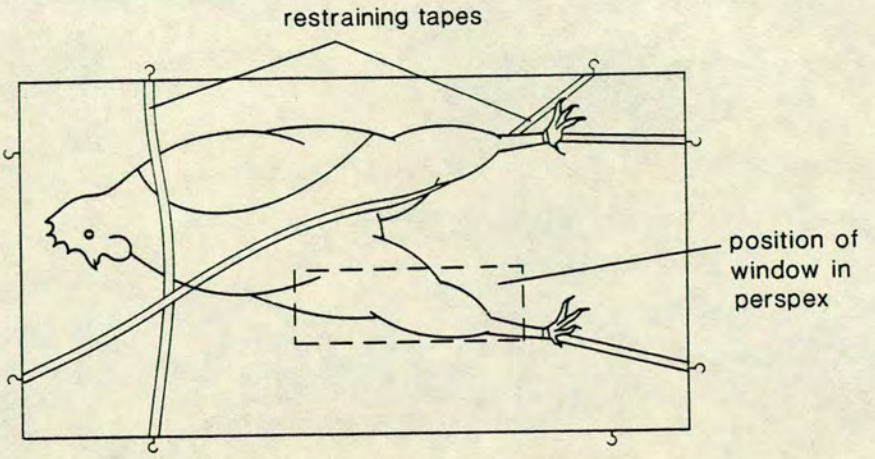
Several decisions could be taken on the basis of the survey described. It is sufficient to examine one leg only per bird, as little information will be lost by ignoring the other. Within this leg only the most rapidly developing growth plates (ie the proximal tibiotarsus and proximal tarsometatarsus) need be examined for cartilage abnormalities, as it is unlikely that others will be affected without these. The best time to examine birds by dissection is when they are 4 weeks old, before small cartilage abnormalities have time to disappear during the growth process. The exact nature of the relationship of growth rate to the presence of defects has not been clarified by this survey, and the inclusion of information on physical measurements on the leg does not aid diagnosis. More information on the association of other leg abnormalities is needed, however, to confirm that true phenotypic and genetic relationships do exist.

Radiological techniques

Obviously, it would be advantageous to be able to diagnose the presence of dyschondroplasia in live birds to a high degree of accuracy. Selection against the condition could then be practised much more successfully than it is at present, and affected birds chosen for experimental matings. Perhaps the surest way to achieve this is to use a radiographical procedure. Several authors have used radiographic techniques in the past (Burton and Howlett, 1979;

Poulos, 1978; and Riddell, 1975a). Cartilage shows up as a radiolucent area, as opposed to bone which is radiodense. Presence of a cartilage defect may be indicated by a radiolucent zone where one would expect to see bone. The identification process may be aided by the presence of intra-articular fat, by which the outline of the joint surface can be discerned. However, there is some doubt concerning the accuracy with which metaphyseal defects can be detected. One purpose of this study was to see whether or not it was possible to establish a quick and harmless method of radiographing birds, which would not only tell if a bird was affected, but give an idea of the degree of severity of any metaphyseal lesions.

Burton and Howlett (1979) describe a rapid technique of radiographing birds involving the use of a perspex table to which birds were strapped. A similar table was designed which was essentially a box in which an x-ray cassette could be placed, with a large perspex lid. (Figure 2.7) This was covered by lead except for a window through which x-rays were allowed to pass. After the discovery that the perspex was slightly radiodense, resulting in poor contrast radiographs, a hole was cut in the perspex directly below the area on which the birds' legs were to be placed. Conscious birds were positioned on top of the perspex and secured by a series of tapes and bands attached to hooks, in order to minimise movement and achieve accurate radiographic positioning. The whole table was then placed inside a sealed lead box into which x-rays were emitted from a Watson MX2 veterinary x-ray unit across a distance of 90cm. Two projections of each leg were taken at KV selector 3, 15-20ma for



Positioning of bird on table to obtain an oblique projection on the radiograph

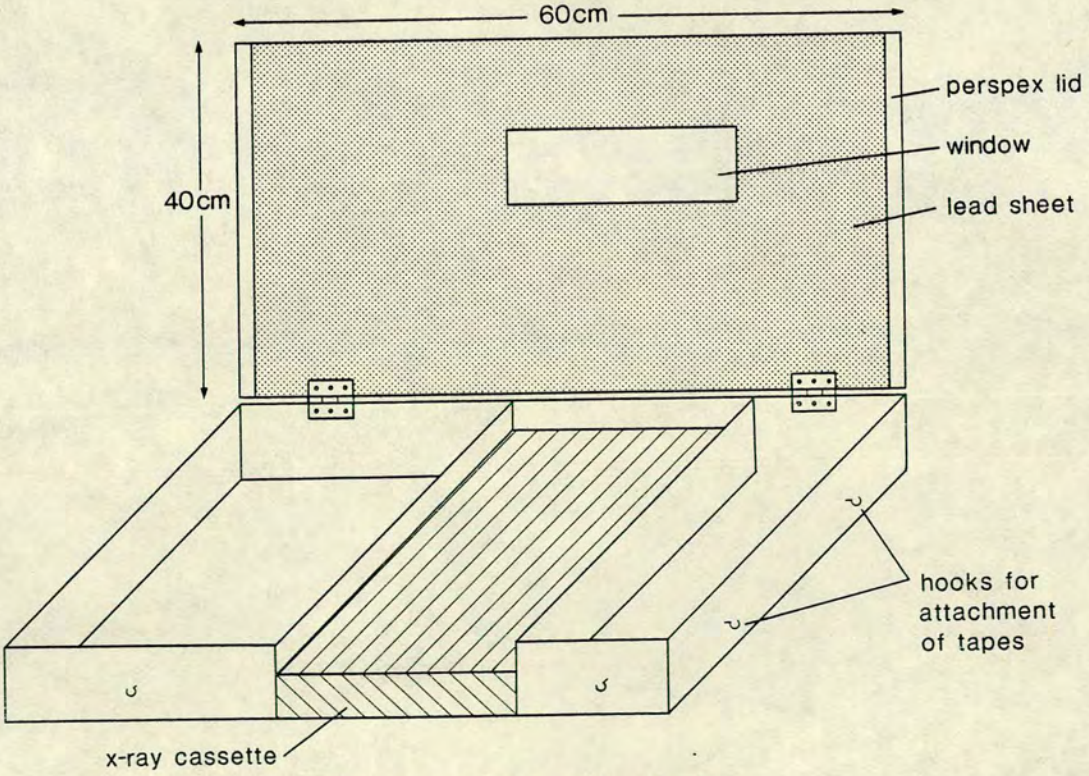


Table used for radiography

Fig. 2.7

0.25 seconds on a 18x24cm Kodak XRP1 cassette plate, which was then manually processed.

Positioning of the bird is crucial to the quality of the radiograph of the relevant growth plates. However, it was difficult to ensure that the legs were placed at exactly the same angle each time, and also to ensure that the birds did not move. Where possible, an antero-posterior view was taken of the whole leg by placing the bird directly on its back. This was followed by rotating the bird slightly so that an oblique view of the leg was taken, in which the anterior aspect of the tibiotarsus was turned outwards. This is similar to the position recommended by Riddell (1975a). In both cases the legs were fully extended to present a clear view of the joints, and every effort was made to prevent wing feathers from obscuring the picture.

The birds used for this study were taken from an experiment in which a soy-bean isolate, Ardex-R, was fed to broiler chicks in a purified diet developed by Dewar and Downie (1981, unpublished) at the ARC Poultry Research Centre, Roslin (PRC), this diet giving a high incidence of dyschondroplasia. Two samples of size 30 and 15 respectively, were taken from different experiments; birds being radiographed at 4 weeks old, then killed by cervical dislocation, and their legs dissected. Scores for severity of lesions were awarded on a 1-5 scale, as previously described, for both radiography and dissection, assessments referring to the proximal tibiotarsus only.

Figure 2.8. Radiographs of stifle joints in birds affected and unaffected by cartilage abnormalities.

(a) Normal 4 week old live broiler.

(b) Normal 6 week old live broiler.

(c) Excised leg of 4 week old broiler - moderate lesion, lateral projection.

(d) Antero-posterior view of same bones shown in (c).

(e) Small lesion in live 4 week old bird - note position of fibula.

(f) Extremely large lesion in 4 week old bird - note large amount of abnormal bone deposition.

(g) Moderate lesion in 6 week old broiler.

(h) Severe lesion in 6 week old broiler - again note abnormal bone deposition below lesion.

For further details of interpretation of radiographs see text.

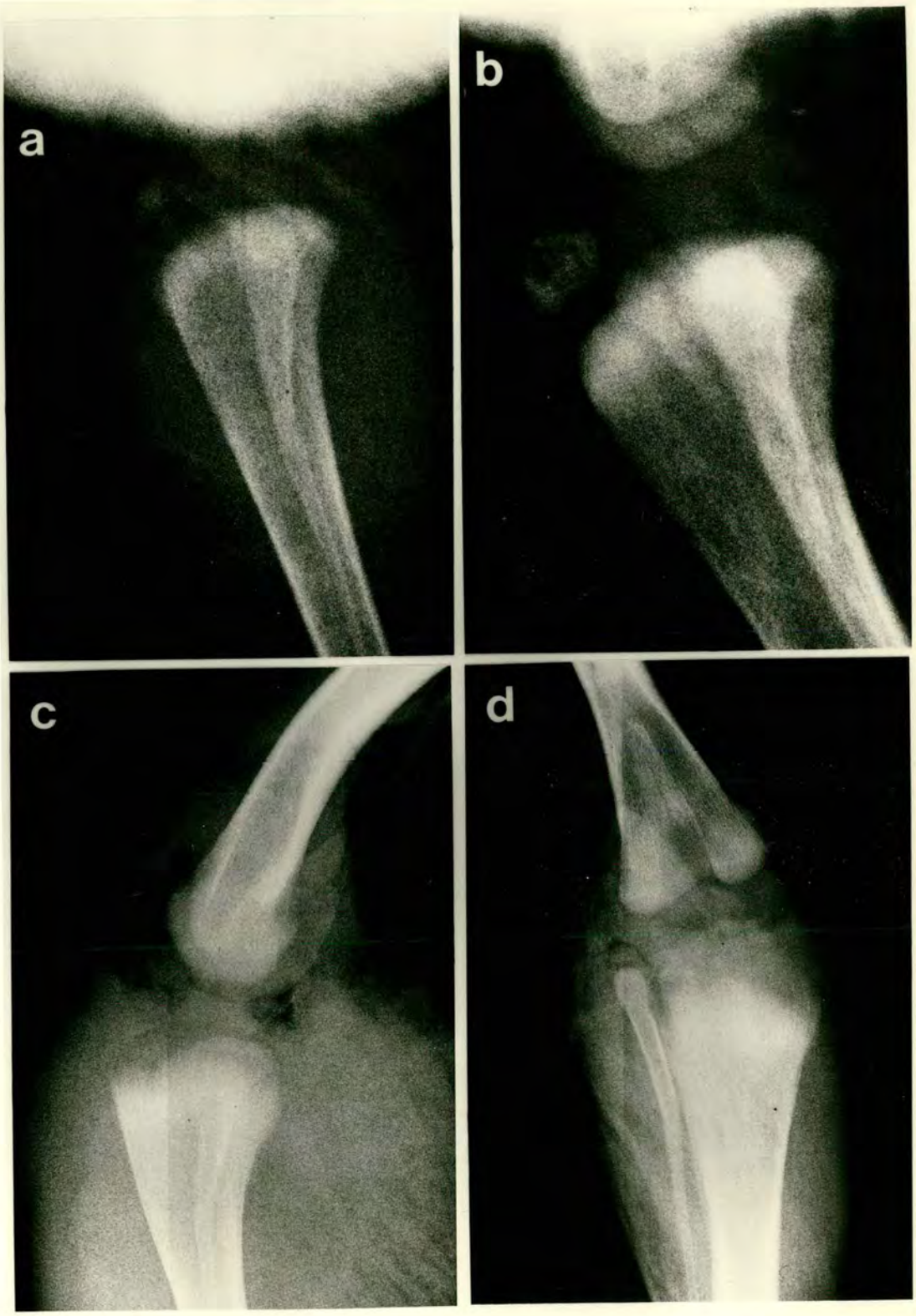


Fig 2·8

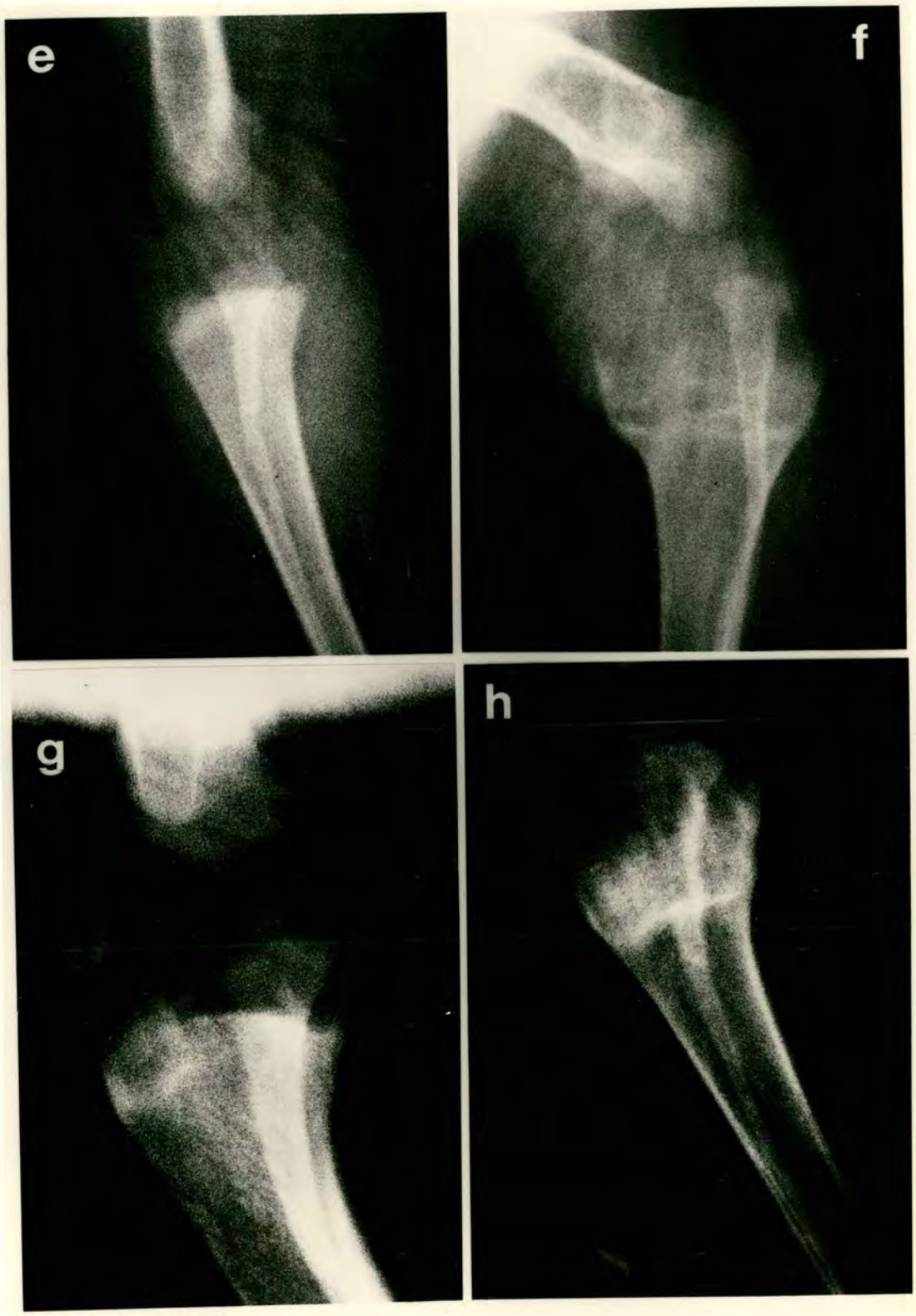


Fig 2·8 cont

Examples are shown in Figure 2.8 of 4 week old birds with lesions of varying severity, and also the legs of 6 week old birds from a later trial, normal birds of both ages being included for comparison. In the normal bird the outline of the metaphyseal growth plate junction of the proximal tibiotarsus is convex and regular, the same area of the fibula coming up to, or just below this level. Clear lines of demarcation exist between the radiodense areas at the articular junction of the proximal tibiotarsus and distal femur. In some cases the outline of the bone extremities may be seen. The lines of striation superimposed across the joint surface, visible in the 4 week old birds are caused by wing feathers.

Metaphyseal defects are normally caused by abnormal cartilage plugs which appear as radiolucent areas within the metaphysis on the radiograph, and are most obvious in severely affected birds. Other signs of abnormal cartilage development are concave, straight or irregular surfaces at the growth plate junction; protrusion of the visible part of the fibula above the visible part of the tibiotarsal metaphysis, and greater distances between the developing bones in the joint than would be expected. Severely affected growth plates often appeared to have radiodense bands representing areas of abnormal ossification adjacent to, or directly below a plug. The presence of these bands may indicate abnormal cartilage formation even though the plug itself may not be obvious, or has already been resorbed. Abnormal curvature in all planes of the tibiotarsal diaphysis may also be noticeable in birds with large dyschondroplastic plugs. An oblique projection was found to result in better radiogaphs with respect to the detection of metaphyseal

defects.

On a scale of 1-5, scores from live and post-mortem examination agreed remarkably well. (see Table 2.3) Only twice in 45 birds did a discrepancy in scores between the two methods of two points occur. A good range of scores from birds with all degrees of severity was found, birds in the first trial having an incidence of dyschondroplasia of 70% (80% for all growth plate abnormalities), and in the second trial the incidence of dyschondroplasia was 80%. A comparison of the two methods for each of the trials using a paired t-test showed no significant difference between the scores awarded on examination of radiographs and those awarded for dissection by each of the two investigators ($t=0$, $p=1.0$). It was felt, as a consequence, that providing care is taken in assessing radiographs, and by using more than one view of a joint where necessary, that radiographical techniques are reasonably accurate in detection of dyschondroplasia in the proximal tibiotarsus.

Live assessment of birds

Without radiography, there is some doubt about the value of live assessment as a means of detecting dyschondroplasia. Tibial dyschondroplasia (TD) is diagnosed commercially in birds by palpation of the stifle joint, and is thought to be present if this appears to be swollen and accompanied by curvature of the tibiotarsus, often with protrusion of feathers at the level of the stifle joint surface. Particularly at 4 weeks, no obvious signs of leg weakness or locomotory disturbance were noted in the majority of

Table 2.3 Comparison of scores awarded to growth plates assessed by dissection and radiography.

TRIAL A		TRIAL B	
DISSECTION	RADIOGRAPHY	DISSECTION	RADIOGRAPHY
5	5	5	4
3	3	4	4
2	2	4	2
4	3	1	1
4	4	1	1
3	2	3	3
3	4	4	5
4	5	4	3
4	4	5	4
4	4	4	4
4	4	4	4
2	4	4	3
3	3	4	3
4	5	1	1
1	1	2	1
4	5		
3	3		
4	4		
4	4		
3	3		
1	1		
3	4		
3	4		
4	4		
2	3		
4	4		
1	2		
1	2		
1	1		
1	1		

mean of scores awarded by two investigators

birds reared experimentally at the PRC and found subsequently to be affected by dyschondroplasia on dissection. In the selection experiment described in Chapter 4, very few birds were assessed as having TD by the regular selection team examining the birds. In the first generation of progeny which were looked at, a total of 22 out of 306 birds were scored positive for TD, of which only 9 were seen to have dyschondroplasia on dissection. Table 2.4 demonstrates that there was no significant association of TD with true dyschondroplasia (χ^2=1.08 1df). The scores awarded by the selection team here, may be a reflection of the variation in the size of the head of the proximal tibiotarsus, as commented on earlier in the chapter.

A comparison was made of three different methods of assessing birds for leg defects used on cocks in the first trial (1) described in Chapter 3, in which five different breeds were reared. Firstly, 6 week birds were made to walk in front of an observer and a score awarded according to the facility or reluctance with which they moved. This gives an indication of severity of locomotory problems caused by leg abnormalities. Secondly, freshly killed birds at a slaughter age of 45 days were assessed on two separate occasions for overt leg deformities as they passed along a processing line hung up by their shanks. The observer in this instance had only time for a cursory glance at each bird (200 cocks from each breed being examined), and the method is only intended to give a rough estimate of the severity of leg problems. Finally the incidence of dyschondroplasia in spontaneous mortality and culls ascertained by dissection was recorded. Results are presented in Table 2.5. Such a

Table 2.4 Relationship between TD and true dyschondroplasia found in G1 selected progeny

	+TD	-TD	
+DY	9	86	95
-DY	13	198	211
	22	284	306

Table 2.5 Comparison of different methods of assessing leg defects (males only) in five broiler strains (A-E)

1. Percent of birds with locomotory problems

	SEVERE	MODERATE
A	28	61
B	26	54
C	30	60
D	25	57
E	22	42

2. Percent of birds with leg defects assessed on the processing line

	1st ASSESSMENT		2nd ASSESSMENT
	SEVERE	MILD	SEVERE
A	7.0	12.5	18.0
B	4.0	13.5	11.5
C	4.5	16.0	16.0
D	7.5	13.0	17.5
E	3.5	14.0	12.5

3. Percent of birds affected by dyschondroplasia (dissection)

A	7.7
B	14.3
C	0.0
D	10.7
E	0.0

comparison should give an indication of whether breeds with more obvious leg deformities were also likely to be those most affected by dyschondroplasia.

Breeds were ranked on presence of locomotory problems, appearance on the processing line, and their incidence of true dyschondroplasia, in males only:

Locomotory problems:- 1.C 2.A 3.B 4.D 5.E

Processing line (a):- 1.D 2.A 3.C 4.B 5.E

Processing line (b):- 1.A 2.D 3.C 4.E 5.B

Dyschondroplasia:- 1.B 2.D 3.A 4.C=E

There was only moderate agreement between the three different indicators of leg problems in terms of rank, although generally, breeds were either high, or low ranking overall. This, together with the findings concerning assessment of TD, point to the fact that live assessment is of little value in itself to the detection of true dyschondroplasia. If used in conjunction with radiographic techniques, however, it may help in cases where the diagnosis is uncertain. Unless stated otherwise, further references to dyschondroplasia concern 'true dyschondroplasia' and not 'TD'.

It would appear, therefore, that in order to assess dyschondroplasia, it is best to use post-mortem dissection or radiography for screening of live birds. Live assessment without radiography is not sufficiently accurate to be used as a means of diagnosis by itself. Although phenotypic correlations between measurements of the leg with the presence of dyschondroplasia do

exist, these are of little practical use in the diagnosis of a lesion. Examinations carried out by the two former methods are able to judge the severity of cartilage lesions on a five point scale. One leg only need be examined, and the best time to do this seems to be when the chicken is 4 weeks old. The growth plates of the proximal tibiotarsus and the proximal tarsometatarsus only need to be considered.

CHAPTER 3. STRAIN DIFFERENCES AND THE EFFECT OF THE ENVIRONMENT

Three options are described at the beginning of the last chapter, by which the incidence of dyschondroplasia may be increased. Of these, two are dealt with here. These involve looking for a naturally occuring flock of birds with a high incidence of dyschondroplasia, and also searching for a particular set of environmental conditions which would produce a high incidence. The condition is multifactorial in origin, and many different management policies could contribute to its presence. The title of the chapter is to some extent misleading, in that only a very limited number of strains and environmental effects are considered. These were largely factors which were being tested in trials, either commercially or at the PRC, from which material and data were made available for this study. Indeed, a full-scale study of the environmental effects upon dyschondroplasia could prove to be a very lengthy task, and is beyond the remit of this thesis. It was possible by looking at the mortality from the experimental rearing trials to obtain an idea of the relationship of various husbandry variables with dyschondroplasia.

Search for a high incidence strain

A commercial breeding company (X) kindly allowed an examination for the presence of dyschondroplasia in the various strains of their birds most affected by leg problems. Two strains were considered, both male line broilers. The one with reputedly the highest incidence of leg problems was an imported 'heavy' strain, kept under

keep up with physical increases in the skeletal system, and thus prevent the onset of dyschondroplasia. A less likely option is that in the latter two trials, the birds chosen for examination had a slower growth rate than the flock average. If, however, either of the previous suggestions is true, the trial illustrates a further instance of the necessity to consider a whole range of environmental stimuli which may be influencing the presence of dyschondroplasia. More will be said about the effects of restriction and activity later in the chapter.

Mortalities from rearing trials

Another commercial breeding company (Y) offered the chance of examining the spontaneous mortalities and culls from a series of consecutive rearing trials. These trials were executed under standard commercial rearing conditions, approximately 10,000 birds being placed in a hut at any one time, the only restriction being that this was subdivided into a number of experimental pens. Most of these trials involved both a strain comparison and a further experiment to test the effect of different variables, such as stocking density or feed-type on growth rate and feed conversion efficiency. From this, the incidence of dyschondroplasia in certain strains could be compared over consecutive trials, to see whether or not it remained constant within strains.

Mortalities were collected by the farm staff and sent with identification of strain, sex and test, together with the date, to the laboratory, where the legs were removed and stored in a

deep-feeze, ready for examination. As discussed in Chapter 2, it is felt that the use of such material is justified, as the mortality is thought to represent a virtually random sample from the population. Any upwards bias in incidence found to be attributable to the inclusion of older birds, culled because of leg problems, should be the same whatever the treatment or strain, and therefore comparisons are still valid. Scores for growth plate abnormalities of 1-5, as described in Chapter 2, were awarded on dissection of the right leg.

No pedigree information was available on the birds in the trial, and the numbers involved were low. The incidence and severity of dyschondroplasia were examined statistically using an analysis of variance option of the GENSTAT package (Talbot, 1981). Scores were assumed to be normally distributed in this instance. Differences between means were tested as a t-statistic. Birds were also awarded a 0-1 score, according to whether dyschondroplasia was present or absent, and these were analysed as binomials using the GLIM package (Baker and Nelder, 1978).

GLIM is an iterative maximum likelihood programme which uses a link transformation to fit data to a linear model. In this case the binomial scores are transformed to continuous probabilities using a logit link, correction for effects being additive. A deviance statistic is calculated, which is analagous to the the sums of squares in an analysis of variance. For any particular model the deviance is equal to $-2\ln(Lm/Lmax)$, where Lmax is the likelihood when all effects are fitted in the model, and Lm is the likelihood of a particular subset of the full model. The difference between the

mean residual deviance (for the full model) and the mean deviance for a model not including all the effects, is tested here as a chi-square, with degrees of freedom equal to the difference in the number of parameters fitted. The probability of the chi-square value is the significance of the effects not included in the partial model. For example, in the following trials a full model may include effects of strain, diet and an interaction between the two. To find the effect of a strain x diet interaction, a partial model leaving out this effect is fitted. The difference in the deviance of a model not including the strain x diet interaction, and the full model, is distributed as a chi-square with degrees of freedom equal to the difference between the number of degrees of freedom for the partial and full models.

The results of each trial are considered separately and the cumulative results from all the surveys are, including the initial survey , described later in the chapter.

Trial 1 (Table 3.1)

This was a strain comparison in which birds of both sexes were reared to 10 weeks. Only three of the five strains contained affected individuals, the incidence of dyschondroplasia being very low in the trial as a whole, with only one affected female being found. None of the effects examined (strain, sex, sex x strain interaction or age) were seen to have a significant influence on the incidence of growth plate abnormalities. A feature of this, and indeed, other trials, is that low numbers were examined. This

Table 3.1 Results of rearing trial 1

(a) Incidence and severity of dyschondroplasia + 45 day wt.(Kg)

	MALES					FEMALES				
STRAIN	N	I	%I	LWT	SEVERITY	N	I	%I	LWT	SEVERITY
A	13	1	8	2.10	4.00	8	0	-	1.79	-
B	14	2	14	2.07	3.00	11	0	-	1.73	-
C	11	0	-	2.11	-	9	0	8	1.79	3.0
D	28	3	11(14)[1]	1.97	3.25	12	1	-	1.65	-
E	10	0	-	2.08	-	7	0	-	1.75	-

(b)Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	7.55	4 ns[2]
Sex	2.25	1 ns
Strain x Sex	1.85	4 ns
Age	1.15	1 ns

Table 3.2 Results of rearing trial 2

(a) Incidence and severity of dyschondroplasia + 49 day wt. (Kg) - all males

STRAIN	N	I	%I	LWT	SEVERITY
E[a][3]	107	16	15(16)	1.89	3.81
F[a]	32	4	13	1.75	3.50
G	43	19	44(49)	1.83	3.70

(b) Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	12.9	2 ***
Age	0.5	1 ns

1. (% incidence of all growth plate abnormalities)
2. ns = not significant; * = P<0.05; ** = P<0.01; *** = P<0.005
3. groups with the same letter after them have not got significantly different incidences (Students' t-test, P>0.05).

probably means that only large differences in incidence will be statistically significant. The live-weights shown, as in the other trials, are flock averages, and in this case there appears to be no obvious relationship between weight of strain and occurence of dyschondroplasia.

Trial 2 (Table 3.2)

Only male birds from the strain comparison part of this trial were examined. There was a highly significant difference in incidence between strains, this being so whether 0-1 or 1-5 scored data were analysed. Strain G had the highest incidence of dyschondroplasia found in any of the rearing trials. This difference was not reflected in the severity of affected growth plates, and could not be explained by weight differences between the strains. The effect of age was not seen to be significant.

Trial 3 (Table 3.3)

No strain comparison was carried out in this trial, which examined the effects of stocking density and different levels of dietary protein. Pens were stocked with either 150 or 100 birds, and feeding regimes including 18.6% or 20.3% protein in the chick starter were given to birds reared at each stocking density. The number of birds per pen did not influence the incidence of dyschondroplasia, nor did the age or sex of the bird. Restricted birds appeared to be more affected than birds fed a diet with a higher protein content, but the severity of lesions was greater in the latter, and the effect of

Table 3.3 Results of rearing trial 3.

(a) Incidence of dyschondroplasia - all strain E

	MALES						FEMALES						TOTAL
	UNRESTRICTED			RESTRICTED			UNRESTRICTED			RESTRICTED			
DENSITY[1]	N	I	%I	N	I	%I	N	I	%I	N	I	%I	%I
0.51	18	2	11	17	3	18(24)[2]	9	0	-	2	0	-	11(13)
0.79	26	2	8	26	5	19(27)	16	1	6	15	1	7(13)	11(15)
OVERALL	44	4	9	43	8	19(26)	25	1	4	17	1	6(12)	11(14)

(b) 41 day wt. (Kg) and severity of lesions

	MALES		FEMALES		TOTAL
DENSITY	UNRESTRICTED	RESTRICTED	UNRESTRICTED	RESTRICTED	
0.51	1.69	1.64	1.53	1.45	1.58
0.79	1.79	1.69	1.59	1.50	1.64
OVERALL	1.74	1.67	1.56	1.47	1.61
SEVERITY	3.75	3.09	3.00	3.00	

(c) Analysis of deviance of 0-1 data

EFFECT	χ^2	df	
Diet	1.22	1	ns[3]
Pen Size	0.01	1	ns
Sex	2.37	1	ns
Diet x Pen Size	0.06	1	ns
Diet x Sex	0.16	1	ns
Pen Size x Sex	1.56	1	ns
Diet x Pen Size x Sex	0.00	1	ns
Age	0.65	1	ns

1. density expressed as sq. ft. per bird
2. (all growth plate abnormalities
3. ns = not significant

diet was not statistically significant. However, if restricted birds did in fact develop higher incidences of dyschondroplasia, this is contrary to the findings of other authors, in which faster growing birds were seen to have the greater incidences.

Trial 4 (Table 3.4)

Subsequent trials were split into separate parts, all birds being grown in the same houses, but in different pens. Birds in the strain comparisons were always fed a practical diet. A strain comparison and a feed test, both using males only, were included in this set of trials. Diets were made up containing 10% of either tapioca or a maize by-product in the grower, and 15% in the finisher. The performance of two strains reared on the different feed additives was compared with their performance on a standard diet. No statistically significant differences between the two strains or the three diets were seen, although the incidence in birds fed the maize by-product was lower than in birds fed tapioca or the standard diet. Age of birds was seen to have an influence on the presence of dyschondroplasia, as was found in the preliminary survey.

The strain comparison showed that differences in incidence did exist, but these were not statistically significant. Severity of the lesions did not appear to follow the same ranking as for incidence, which suggests that the degree with which strains were affected is in fact fairly homogeneous. Age effect was not important in this case.

Table 3.4

(a) Results of rearing trial 4a (all males)

1. Incidence of dyschondroplasia

STRAIN	TAPIOCA N	I	%I	MAIZE N	I	%I	STANDARD N	I	%I	TOTAL %I
B	16	2	13	17	0	-(12)[1]	17	2	12	8(12)
E	18	1	9	6	1	17	23	1	4(9)	6(9)
TOTAL	34	3	9	23	1	4(13)	40	3	8(10)	7(10)

2. Severity of lesions + 49 day wt.

STRAIN	TAPIOCA SEVERITY	LWT	MAIZE SEVERITY	LWT	STANDARD SEVERITY	LWT
B	3.0	2.23	2.0	2.12	3.5	2.26
E	4.0	2.22	4.0	2.15	2.5	2.17

3. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	0.61	1 ns[2]
Diet	5.72	2 ns
Strain x Diet	0.14	2 ns
Age	4.19	1 *

(b) Results of strain comparison trial 4b (all males)

1. Incidence and severity of dyschondroplasia + 50 day wt. (Kg)

STRAIN	N	I	%I	SEVERITY	LWT
A	51	9	18	3.22	2.09
B	22	3	14	3.67	2.24
E	35	7	20	3.25	2.25
F	36	2	8	3.50	2.02

2. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	3.8	3 ns
Age	0.5	1 ns

1. (all growth plate abnormalities)
2. ns = not significant; * = $p<0.05$

Table 3.5

(a) Results of rearing trial 5a (all males)

1. Incidence of dyschondroplasia

	DIET 1			DIET 2			DIET 3			DIET 4			TOTAL
STRAIN	N	I	%I	N	I	%I	N	I	%I	N	I	%I	%I
B	8	0	-	7	2	29	10	2	20	6	0	-	16
E	14	0	-	17	1	6	11	1	9	18	0	-	3
TOTAL	22	0	-	24	3	13	21	3	14	24	0	-	7

2. Severity of lesions + 49 day wt. (Kg)

	DIET 1		DIET 2		DIET 3		DIET 4		TOTAL
STRAIN	SEVERITY	LWT	SEVERITY	LWT	SEVERITY	LWT	SEVERITY	LWT	LWT
B	-	1.94	3.0	2.03	3.0	1.93	-	1.91	1.95
E	-	1.96	4.0	1.99	3.5	1.88	-	1.93	1.94
TOTAL LWT		1.95		2.01		1.91		1.92	1.95

3. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	2.13	1 ns[1]
Diet	8.19	3 *
Strain x Diet	0.23	3 ns
Age	0.07	1 ns

(b) Results of strain comparison trial 5b (all male)

1. Incidence, severity of lesions + 53 day wt. (Kg)

STRAIN	N	I	%I	SEVERITY	LWT
A^{a} [2.]	32	9	28	3.56	2.26
B^{ab}	21	3	14	4.00	2.39
E^{ab}	32	3	9	3.00	2.40
F^{b}	15	0	-	-	2.21

2. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	9.89	3 *
Age	6.15	1 *

1. ns = not significant; * = $P<0.05$
2. groups with the same letter after them are not statistically different (Students' t-test, $P>0.05$)

Trial 5 (Table 3.5)

This trial was again divided into two halves, and male birds examined. The same two strains involved in Trial 4a were fed four commercial diets and their performance tested. Only two of the four diets produced any affected birds, these causing similar incidences of dyschondroplasia, and being statistically different in their effect from the other two diets. Strain B had a higher incidence of the abnormality than E, but this was not statistically significant, and bore no relationship to live weight.

In the strain comparison, strains A and F had statistically different incidences of 28% and 0% respectively. Other differences in incidence were not significant, and again did not appear related to the severity of the condition. The age of the birds influenced the incidence, in contrast to the first half of the trial.

Trial 6 (Table 3.6)

In the first part of this trial birds of one strain were given either low-energy (defatted) or normal diets, in different quantities. Defatted or normal diets were fed in amounts per 1000 birds of 500Kg starter and 1000Kg grower, or 750Kg starter and 750Kg grower to cocks; plus 420Kg starter and 1080 Kg grower or 500Kg starter and 1000Kg grower to pullets. Normal diets appeared to cause a higher incidence of growth plate abnormalities than low energy diets in males, although this difference was not significant. Only one affected pullet was recorded. The ranking of incidence in cocks

Table 3.6

(a) Results of rearing trial 6a

1. Incidence of dyschondroplasia (all strain E)

	MALES				FEMALES			TOTAL
FEEDING REGIME[1]	N	I	%I	FEEDING REGIME	N	I	%I	%I
1. 500 Kg LOW ENERGY	22	1	5	1. 420 Kg LOW ENERGY	12	1	8	6
2. 500 Kg NORMAL	11	2	18	2. 420 Kg NORMAL	9	0	-	10
3. 750 Kg LOW ENERGY	16	1	6(13)[2]	3. 500 Kg LOW ENERGY	8	0	-	4
4. 750 Kg NORMAL	23	4	17	4. 500 Kg NORMAL	12	0	-	11
TOTAL	72	8	11(13)	TOTAL	41	1	2	8

2. Severity of lesions + 48 day wt. (Kg)

	MALES		FEMALES	
FEEDING REGIME	SEVERITY	LWT	SEVERITY	LWT
1.	3.0	2.05	-	1.71
2.	3.0	2.19	-	1.75
3.	2.5	2.24	3.0	1.79
4.	3.5	2.18	-	1.81

3. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Diet	2.50	3 ns[3]
Sex	3.51	1 ns
Diet x Sex	3.21	3 ns
Age	0.53	1 ns

(b) Results of strain comparison trial 6b (all males)

1. Incidence, severity of lesions + 54 day wt. (Kg)

STRAIN	N	I	%I	SEVERITY	LWT
A	26	7	27	3.33	2.33
B	19	1	5	3.00	2.47
E	34	1	3	3.00	2.41
F	17	2	12	4.00	2.23

2. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	8.63	3 *
Age	0.36	1 ns

1. see text for details
2. (all growth plate abnormalities)
3. ns = not significant; * = $P<0.05$

was not reflected by either 48 day live-weight or severity. As found, one would expect a lower incidence of leg problems in birds which were restricted at an early age.

Males from the same four strains as the previous two trials were performance tested. Strain A again had the highest incidence, which was much the same as in the last trial, and significantly different from the incidence of 2.9% in strain E. This was only statistically the case when 0-1 scores were analysed. Age did not have any effect on incidence in either of the two parts of the trial.

Trial 7 (Table 3.7)

The first half of this trial compared diets with varying amounts of soy-bean protein from two different sources. These diets therefore contained different energy contents. Standard diets had inclusions of soya-50 of 14.5% in starter, 14.4% in grower and 16.0% in finisher; high diets 24.3%, 20.3% and 17.8%, and medium diets 19.0%, 16.0% and 15.0% respectively. As in other trials, starting diets were fed until 14 days, growers from 14-28 days and then finishers up until slaughter age. Interestingly, incidences of dyschondroplasia were higher in cocks of strain E fed normal or medium soya-content diets. 48 day live-weights were slightly less in birds fed a high soya-protein inclusion, which may have been responsible for the reduced incidence. Dietary effects, however, were not statistically significant. Few pullets were affected.

The second part of Trial 7 involved somewhat different strains. E

Table 3.7

(a) Results of rearing trial 7a (all strain E)

1. Incidence, severity of lesions and 48 day wt. (Kg)

	MALES					FEMALES					TOTAL
DIET	N	I	%I	SEVERITY	LWT	N	I	%I	SEVERITY	LWT	%I
NORMAL SP1	19	4	21	3.50	2.08	10	0	-	-	1.81	14
MAXIMUM SP1	15	2	13	3.67	1.91	7	0	-	-	1.79	9
NORMAL SP2	17	4	23(29)[1]	3.33	2.10	7	1	14	3.0	1.79	21(25)
MAXIMUM SP2	13	2	15	4.00	2.01	9	2	22	3.0	1.76	18
MEDIUM SP2	13	3	23(39)	3.00	2.06	2	0	-	-	1.77	20(33)
TOTAL	77	15	20(23)		2.03	35	9			1.78	19

.2. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Diet	1.07	4 ns[2]
Sex	1.21	1 ns
Diet x Sex	7.16	4 ns
Age	0.01	1 ns

(b) Results of strain comparison trial 7b (all males)

1. Incidence, severity of lesions and 38 day wt. (Kg)

STRAIN	N	I	%I	SEVERITY	LWT
E	37	6	16	3.80	1.70
H	36	8	22	3.33	1.72
I	28	4	14	3.25	1.61
J	24	2	8	3.33	1.68

2. Analysis of deviance of 0-1 data

EFFECT	χ^2	df
Strain	2.03	3 ns
Age	8.03	1 ***

1. (all growth plate abnormalities)
2. ns = not significant; *** = P<0.005

is the same commercial hybrid as used in the previous trials, G is a pure-bred male-line, I a broiler cross from the same dam-line as E, and H a roaster cross from the same sire- line as I. However, no significant strain effect on incidence was noted, although the heavier male-line was seen to be the worst affected by dyschondroplasia. There was a highly significant effect of age of bird in this part of the trial, as opposed to the feed test.

Analysis of trials combined

Taken together, several points from the above trials are worth noting. As mentioned, the numbers involved in individual cases are low, and therefore although differences in incidence existed, they were seldom significant. A summary of the various incidences found in the strain comparisons is presented in Table 3.8. These rarely bore any relation to the severity of growth plate abnormalities seen in the tibiotarsus, nor did the ranking of incidence correspond with that for mean live-weight at around 6-7 weeks, except in Trial 7a. Overall, strain effects were more often significant than dietary effects, strains A and G having particularly high incidences. No genotype x diet or genotype x sex interactions were seen to be significant.

The results of tests in which birds were fed low or high energy content diets were confusing. Previous authors (Leach and Nesheim, 1972; Riddell, 1975b; Poulos et al, 1978; Huff, 1980) have all reduced the incidence of dyschondroplasia by using diets designed to

Table 3.8

Summary of results from the strain comparison trials (males only)

TRIAL	1		2		4		5		6		7	
BREED	N	%I	N	%I	N	%I	N	%I	N	%I	N	%I
A	13	8			51	18	32	28	26	27		
B	14	14			22	14	21	14	19	5		
C	11	0										
D	28	3										
E	10	0	107	15	35	20	32	9	34	3	37	16
F			32	13	26	8	15	0	17	12		
G			43	44								
H											36	22
I											38	14
J											24	8

restrict growth rate. This is in direct contrast to Trial 3, in which a restriction produced a high incidence, although this effect was not statistically significant. The trial (6) incorporating defatted diets did support these earlier conclusions, though, while Trial 7 showed that an increase in the energy content was not necessarily accompanied by an increase in dyschondroplasia.

Table 3.9 presents a summary of various analyses performed on data from several, or all trials. Taking strain E alone, and including the initial survey described in Chapter 2, a significant trial effect on incidence of growth plate abnormalities is shown. This merely confirms that different experimental conditions produce different incidences. If all data from all the rearing trials, including the initial survey, are considered, test, sex and strain all have significant effects on incidence of dyschondroplasia. If just the strain comparison data are considered, however, only the effect of strain is significant. This suggests strain differences are repeatable. No evidence of a genotype x environment interaction was found. In contrast to the findings of the initial survey, in which age was seen to have a linear relationship with incidence, no age effect appears in any of the above analyses. This suggests that the probability of a bird being affected by dyschondroplasia does not alter much with time. Finally, data from the four strains compared most often, were examined over four tests. No significant effects upon incidence were seen in abnormalities scored 1-5, but statistically significant effects for both strain and age were found if the birds were scored on a 0-1 scale. The 0-1 analyses are thought to be more accurate, because of the corrections using the logit transformation involved in the GLIM model. Again, as in the

Table 3.9

Analyses of rearing trials combined

(a) Analysis of deviance of 0-1 data from all rearing trials

EFFECT	χ^2	df	
Trial	16.0	1	* [1.]
strain	34.0	9	***
Sex	7.0	1	**
Trial x Strain	9.0	9	ns
Trial x Sex	1.0	3	ns
Strain x Sex	2.0	4	ns
age	1.0	1	ns

(b) Analysis of deviance of 0-1 data from all strain comparison trials (males only)

EFFECT	χ^2	df	
Strain	28.1	9	***
Trial	7.9	5	ns
Strain x Trial	13.5	9	ns
Age	0.00	1	ns

(c) Strain comparison trials 1,4,5,6 (males only) including strain F from trial 2.

1. AoV of scores

EFFECT	F-ratio	df	
Strain	2.36	3	ns
Trial	0.49	3	ns
Strain x Trial	1.08	9	ns
Age	1.03	3	ns
Residual		382	

2. AoD of 0-1 data

EFFECT	χ^2	df	
Strain	8.0	3	*
Trial	1.0	3	ns
Strain x Trial	14.7	9	ns
Age	4.5	1	*

(d) All strain comparison data on strain E (males only) including initial survey (Chapter 2)

1. AoV of scores

EFFECT	F-ratio	df	
Trial	1.77	6	*
Residual		1030	

2. AoD of 0-1 data

EFFECT	χ^2	df	
Trial	10.3	6	ns
Age	0.2	1	ns

1. ns = not significant; * = P<0.05; ** = P<0.01; *** = P<0.005

previous comparison there was no evidence of a genotype x environment interaction. As the rearing conditions were standard in the strain comparison trials, these strain differences are felt to be consistent and real, and suggest a strong genetic component in the susceptibility of birds to dyschondroplasia.

Nutritional Experiments

As indicated in the first chapter, much work has been done on the effect of diet on the incidence of dyschondroplasia. The purpose of performing nutritional trials in this study was to perfect a diet which could be used with confidence to increase the incidence of growth plate abnormalities, and thus lower the threshold above which affected birds could be selected. In the course of this work, I was involved with various other trials which were carried out at the PRC. It is necessary, before any specific ones are described, to give some background to the work being done at the PRC on dyschondroplasia (Dewar, Downie and Duff, unpublished material).

In all the experiments mentioned here, commercial broilers supplied by one company (X) were reared in wire brooder cages. Approximately eight birds were placed in compartments 94cm long x 40cm wide x 30cm in depth. Each of these compartments contained a darker part next to a central heater, with an area 36 x 40 x 30cm. Birds were reared on a light regime of 23 hours continuous light followed by one hour dark, at a temperature of approximately 21°C. Unless otherwise stated, diets were purified and fed ad libitum. Each purified diet was mixed to the same specifications, with only

the amount of the active protein ingredient being changed. Full details of a specimen diet containing FP950 as a protein source are given in Table 3.10.

Most purified diets tested seemed to produce higher incidences of dyschondroplasia than were seen in birds fed practical diets. These included egg-albumen extracts, soya-bean protein extracts and casein. A soya-bean protein isolate, Promine-R, induced incidences of dyschondroplasia averaging 65% over seven trials. Unfortunately, this was not available for later trials. In a trial in which ammonium chloride was added to both a purified and a practical diet, the incidence of dyschondroplasia was slightly increased with the latter, but decreased with the former, compared to similar diets without the addition. This is partly in contrast to the findings of Leach and Nesheim (1972). Again using Promine-R, restriction of broilers to the food intake of a layer strain of the same age reduced the incidence of dyschodroplasia. When broilers were killed at the mean 4 week weight of egg layers (14 days), there was a high incidence of thickened growth plates, but few cases of dyschondroplasia were observed. Surprisingly, one case of dyschondroplasia was noted in an egg-laying type bird, reared up to a broiler 4 week weight. The condition has not been reported in egg-laying strains previously to this (cf Reiland et al, 1978).

Feed restriction trial

The first trial in which I was directly involved was a feed restriction experiment in which broilers reared up to 4 weeks were

Table 3.10

Formulation for 100 Kg purified diet with FP950

Vitamin mix (g)[1]	
Nicotinic Acid	5.0
Ca Pantothenate	2.0
Pyridoxine HCl	0.45
Folic Acid	0.4
Menapthone	0.152
Biotin	0.30
Vitamin E25	26.4
Thiamine	1.1
Riboflavin	1.1
Vitamin B12	0.002
Vitamin AD3	1.08

Mineral mix (g)	
$MgSO_4.H_2O$	250.0
$FeSO_4.7H_2O$	33.3
$MnSO_4.H_2O$	33.3
KI	0.26
$CuSO_4.5H_2O$	1.67
$CoCl.6H_2O$	0.16
$NaMoO_4.2H_2O$	0.83
$ZnCO_3$	11.5

Major Ingredients (Kg)	
FP950	27.0
Anhydrous Glucose	58.5
Corn Oil	3.0
Cellulose	3.0
Methionine	0.07
Glycine	0.3
$CaHPO_4.2H_2O$	2.16
$CaCO_3$	1.9
KH_2PO_4	1.39
NaCl	0.6
50% Choline	0.308

1. made up to 60g with starch

kept in conditons as previously described. Approximately 25 males received each treatment. Birds were fed Promine-R, and groups were restricted from either 7-14 days, 7-21 days, 14-21 days, 7-28 days or not at all. During restriction broilers were fed the amount of feed consumed by an egg-laying strain over a similar period, as described above. Otherwise, diets were fed ad libitum. Surviving birds were killed at 28 days and their legs examined for dyschondroplasia as described in Chapter 2.

Results from this experiment are shown in Table 3.11. Weights achieved by birds restricted from 7-28 days were very similar to those in birds restricted to the weights achieved by an egg-laying strain of the same age in a previous trial. Surprisingly, the birds which were restricted during their second week achieved the best weights, these being considerably heavier than in birds which had an ad libitum diet over the whole period. Cessation of restriction resulted in a sudden increase in weight. Birds restricted from 7 days onwards had a low incidence of dyschondroplasia (13%). This compared with incidences of 48% and 43% in birds restricted for the second week, and second and third weeks respectively; and incidences of 73% and 88% in birds restricted in the third and fourth week, or unrestricted over the whole period respectively. The result in the birds with the heaviest restriction is not surprising, but this apart, the incidences bear no relation to the mean weights of each group of birds. It may be significant, however, that those birds restricted at 7-14 days all showed lower incidences. This suggests that there was a critical period of development between one and two, or possibly three weeks, at which, if growth was slowed, bones were

Table 3.11

(a) Results of PRC restriction experiment

DIETARY REGIME	4WK LWT g	N	I	%I
PrR REST. WK 2	649	25	12	48(52)[1]
PrR REST. WK 2,3	365	23	10	43(74)
PrR REST. WK 3,4	243	15	11	73
PrR REST. WK 2,3,4	214	23	3	13
ARDEX R	319	10	10	100

(b) Results of PRC Ardex R trial

DIET	4WK LWT g	N	I	%I
ARDEX R OLD	411	9	9	100
ARDEX R NEW	505	7	7	100

(c) Results of PRC dietary comparison

DIET	SEX	4WK LWT g	N	I	%I
FP950 + CASEIN	M	561	18	5	28(50)
FP950 + CASEIN	F	550	18	7	39(50)
FP950	M	700	17	7	41(65)
FP950	F	622	14	6	43(64)
CASEIN	M	96	10	0	-
CASEIN	F	120	18	0	-
CASEIN + Se	M	117	16	0	-
CASEIN + Se	F	112	11	0	-

1. (incidence of all growth plate abnormalities)

able to develop more normally, and not as many growth plate abnormalities were seen. Some supporting evidence for this conclusion may be found in the data obtained by Leach and Nesheim, (1972), in an experiment in which chick starter was fed to birds for varying lengths of time.

Dietary comparison

As the stocks of Promine-R were no longer available, an analagous soya-protein isolate, Ardex-R was tried in purified diets. A batch of 25 cocks were reared at the same time as the restriction trial, which were fed an ad libitum diet including Ardex-R. Subsequently, another trial was performed in which two separate mixes of diets containing Ardex-R were fed to groups of 25 males. In all these groups the incidence of dyschondroplasia-like lesions in surviving birds was 100%. The mortality, however, was well over 50% in every case, and the diet was seen to have a toxic effect. It was decided therefore, not to use Ardex-R further, as these losses were considered unacceptable.

As the best types of incidence-inducing purified diets were no longer available, a further dietary trial was carried out in order to try and establish another diet which would raise the level of growth plate abnormalities. Two protein sources which had previously been seen to produce a high incidence, were compared again. FP950, another soya-bean protein isolate which is used in certain practical mixes, and casein were added to purified diets; as were a FP950/casein mixture (50:50), and casein with added selenium. Groups

of 25 cocks and 25 pullets were fed each of these ad libitum, in the same rearing conditions as before. The results are also presented in Table 3.11.

Birds fed diets containing casein by itself as a protein source, or casein plus selenium, grew very poorly, had high mortalities, and did not develop any growth plate abnormalities. Indeed, many of these birds were seen still to have juvenile growth plates on dissection. This performance is in contrast with that in earlier trials, when birds fed with a purified diet containing casein grew quite well, and developed an incidence of 48%. The reason for this poor development was not explained. In contrast, birds reared on the other two diets performed quite well, and developed incidences of 42% and 34% with FP950 and FP950 + casein respectively , despite the fact that these diets were made with the same vitamin and mineral mixes. These two diets had statistically significant effects on both incidence of dyschondroplasia, and 4 week weight compared with the other diets. This was so whether birds were scored as 0-1 or 1-5 and analysed as for the rearing trials. FP950 was chosen for subsequent use as it gave the highest incidence and produced the best weights in this trial.

In conclusion, results from the various trials reported in this chapter support the idea that many different factors affect the incidence of dyschondroplasia. Perhaps the main significance of the two sets of trials discussed is that each was performed under largely the same set of conditons, and thus only the experimental

variables should be those responsible for any changes in prolificacy of lesions. The results from each of these sets are more comparable than are results from trials performed in slightly different ways by either different laboratories or companies. In a situation, as we have here, where a condition seems very sensitive to a large range of factors, standardisation of environmental conditions is crucial in the determination of precisely which of these is responsible for changes in incidence.

Of the alternatives examined, the varying incidences found between strains are supported by the existence of strain differences amongst Australian broilers (Burton et al, 1981). The strain differences here were consistant over trials, and the fact that no genotype x environment interactions were detectable either supports this, or suggests that rearing conditions are not too different from trial to trial. Important implications to the breeder may arise because of this. Stocking density appeared to have little effect on incidence in the trials, but as noted in the first section of the chapter, may be important in reducing incidence if very low. Different dietary treatments produced different amounts of dyschondroplasia in commercial broilers, incidences appearing higher when purified diets were used. No direct comparison between the company trials and those carried out at the PRC can be made, as large pens with litter floors were used in one set of experiments, and small wire cages in the other. More is said about this in Chapter 5. A purified diet containing a soya-bean protein isolate, FP950, was formulated to produce a high incidence in later experiments.

The effect of growth rate was examined in various trials, although overall, the results were inconclusive. These seem to indicate that restriction at an early age produces a decrease in the incidence of dyschondroplasia. The effect of restriction was similar to that found by Leach and Nesheim (1972), Riddell (1975b), Poulos et al (1978), Huff (1980) and Wise and Nott (1975), working on ducks. The experiment designed to produce an even higher growth rate than normal did not appear to have much effect on the incidence of dyschondroplasia.

CHAPTER 4.

USE OF FAMILY INFORMATION IN SELECTION FOR DYSCHONDROPLASIA

Selecton is the last of the three options mentioned at the beginning of Chapter 2, with which the incidence of dyschondroplasia may be increased. As live assessment performed by observation and handling of chickens is thought to be inaccurate, mass selection becomes difficult. To select on an individual basis, radiography would have to be used. This has been successfully achieved by Riddell (1976), and will be discussed further in the next chapter. Alternatively, family information must be used, and birds selected on the basis of results from relatives which have been killed and dissected. Leach and Nesheim (1965) used progeny test information to select sire and dam combinations in order to produce divergent lines with respect to incidence of dyschondroplasia. A similar approach was used by Sheridan et al (1978), birds being selected on progeny-tested sire family incidence.

If only definitely affected individuals were used for matings, the selection differential, calculated as the difference in incidence between these and the rest of the population, would be high, and therefore the selection response would be rapid. It was not feasible to radiograph large numbers of birds, however, in a commercial situation. Dissection of full-sibs was therefore chosen as the selection criterion. This has the advantage of giving a more conclusive diagnosis than radiography. The breeding value of an individual, in terms of incidence, can be taken as the mean

incidence of its full-sibs. Use of family information, however, will lead to the use of phenotypically unaffected birds (if these were to be dissected). Thus the selection responses will not be as rapid as they would be if mass selection of definitely affected birds were used. Problems arise in the present study in that birds are derived from pre-selected populations, and therefore selection responses may not be as predicted. In order to see if selection responses had in fact occured, control birds were included with the second generation of selected progeny. These were birds belonging to the male-line from which the selected population was derived.

Initial population (G0)

The selection experiment described here was executed by courtesy of a breeding company (X), who kindly provided birds, facilities and labour. Unfortunately, the experiment was constrained in the respect that it had to fit into the conditions employed by the company. Broiler hens are kept for a total of nine hatches, chicks from each of these being placed on a separate farm. Every hatch consists of eggs saved over a period of two weeks. Selection decisions are taken on the cumulative information from hatches, therefore birds whose progeny perform poorly are culled. Testing of progeny is carried out when birds are 6 weeks old, information on live-weight, conformation and presence of abnormalities being recorded. A number of selected sires and dams from the same hatch are reared together as one mating group. Within these, each sire is placed in a mating pen with approximately eight dams. These birds contribute to the replacement flock in their turn.

Selection records of a pure-bred male-line were examined over a period of time to identify birds thought to have overt symptoms of tibial dyschondroplasia. Full-sibs of these birds were kept as parents for the first generation of matings, the parents of affected birds having in many instances already been discarded. As the incidence of TD is very low ($p<0.005$), it was necessary to collect birds from a wide range of ages, mating groups and farms. The object was to collect sufficient numbers of parent birds to produce a synchronised hatch. This would result in a first experimental generation large enough to apply a reasonable selection differential. Although it was realised that a number of the G0 parents would not be affected by dyschondroplasia, it was felt that this approach was more sensible, as it utilised existing pedigree information and obviated the need for the dissection of the hundreds of birds required to identify high incidence sire families.

Having identified potential parent birds in the records, subsequent full-sibs of 4 weeks of age were collected and dissected. As these came at various different times from different farms and in rather unequal numbers, no attempt was made to use this information as a perfomance test. The incidence in full-sibs of parent birds was recorded as a whole, to represent the G0 incidence. Birds were killed by cervical dislocation, and growth plate abnormalities assessed by dissection as described in Chapter 2. The as-hatched incidence in 97 full-sibs examined was 10.31%. Altogether a total of 16 cocks and 60 pullets were taken as parents. These were reared on different farms according to age, pedigreed matings being made randomly between these birds. Not all of those originally chosen

survived to breed. By the time the youngest of the parents were producing their first viable hatches at approximately 28 weeks old, the oldest were having their last hatches. Therefore it was only possible to take three hatches of G1 progeny, which could be placed in the incubator at the same time.

Chapter 7 concerns the prediction of responses to selection on defect traits. In contrast to normally distributed traits, in which full-sib family selection produces greater responses than half-sib family selection, the converse applies to 0-1 traits. It is easier in a practical situation, however, to use full-sib family selection, as only low selection pressures may be applied. To prevent inbreeding, this was the method used. Predicted responses to full-sib family selection for dyschondroplasia in the G0 generation were made using the equations presented in Chapter 7. Assuming a heritability for dyschondroplasia of 0.50, an initial population incidence of p=0.10, and a mean full-sib family size of 6, the incidence would be expected to increase to p=0.18 in G1. This assumes that all families with affected individuals are selected. In fact, all full-sib families with visibly affected birds were chosen. For comparison, the incidence would be expected to increase to p=0.19 if half-sib family selection were used, assuming a half sib family size of 30.

G1 Generation

The second hatch of chicks from the G0 parents were reared under standard conditions until 4 weeks old, when they were all examined

for clinical leg abnormalities by one member of the regular selection team. Predefined defect traits are picked out during selection, of which five are included here. (Figure 4.1) These are :

1. Crooked Toe (CT), in which lateral deviation of the tarsi occurs giving the toes a "crab-like" appearance.

2. Bow (B), in which bowing of the tarsometatarsi and, to a lesser extent, the tibiotarsus occurs.

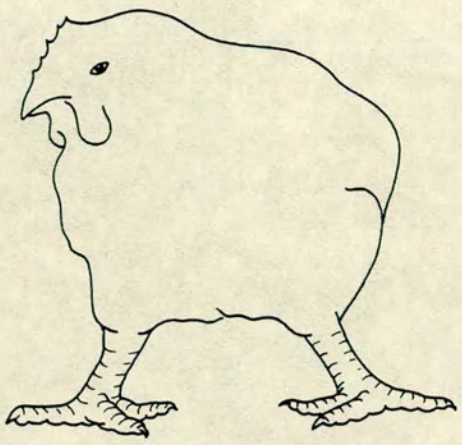
3. Splay (S), the opposite of bow, leading to a valgus deformity of the tarsometatarsi.

4. Twisted-leg (TW), which is similar to splay in that the tarsometatarsi of one or both legs adopt an unnatural angle to the rest of the limb, deviating as much as 90 degrees from the hock joint, and is often accompanied by slipping of the gastrocnemius tendon. The deviation may be caused by tilting of the condylar surface, or abnormal growth of the upper third of the tarsometatarsal diaphysis.

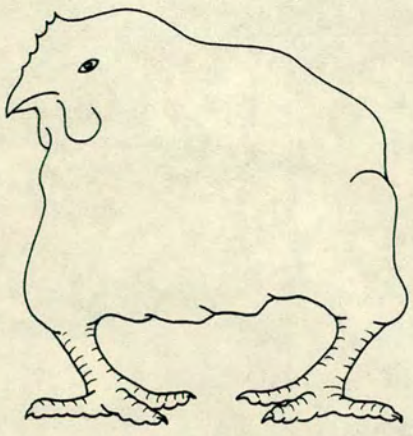
5. Tibial Dyschondroplasia (TD), here assessed on the clinical condition in which protrusion of the stifle joint and bowing of the tibiotarsus occur.

Birds were then killed and the right leg dissected. Growth plates were scored for abnormalities as described in Chapter 2.

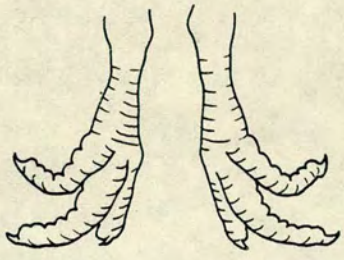
Genetic parameters for the G1 population were obtained by analysis of variance and modified proband methods fully described in Chapter 6. Analysis of variance was performed using Harvey's Mixed Model Least Squares Analysis of Variance programme (Harvey, 1975) on male and female data combined, sex being fitted as a fixed effect. This analysis was carried out on both 0-1 and 1-5 scored data, both of



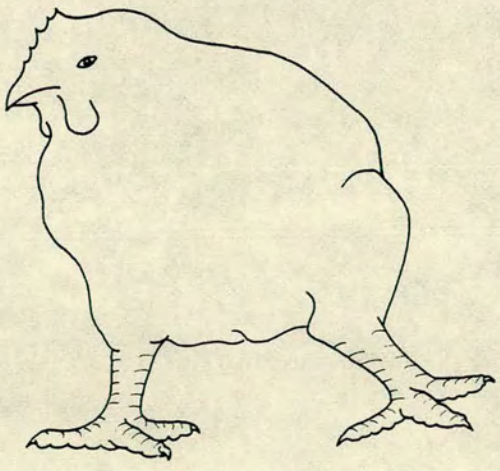
Splay (S)



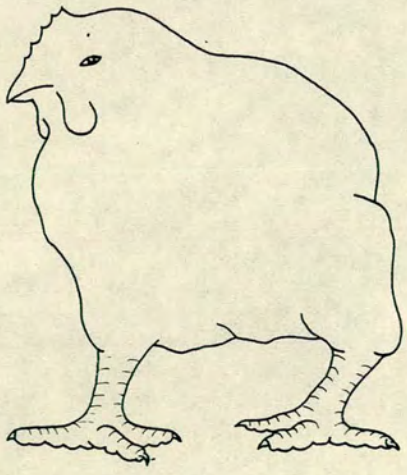
Bow (B)



Crooked toe (CT)



Twisted leg (TW)



Tibial dyschondroplasia (TD)

Fig. 4.1 Leg abnormalities selected against by company X.

which gave similar results, and therefore only the estimates obtained from 0-1 data are presented. Heritability estimates were corrected, so that these were made on the normally distributed scale using the transformation of Robertson and Lerner (1949). The estimates obtained in this chapter are from low numbers of sires and progeny, and are therefore unreliable. Full details of the methods used to calculate genetic parameters, together with their application on more substantial data sets, are presented in Chapter 6.

A total of 306 birds were examined from 52 full-sib families. The overall incidence of dyschondroplasia in the G1 generation was 31.05% (38.14% for all growth plate abnormalities). Details of the incidence in each of the 9 sire families which contributed to the flock are presented in Table 4.1, together with the mean severities of affected tibiotarsi. As can be seen, there is a large range of sire family incidence from 3.8%-71.4%, this being continuously distributed over a range of 0-40%, with one exceptional sire (18). Sires were ranked in order of increasing incidence. Severity of lesions corresponded generally to the ranking of the sires. The family with the worst defects also had the greatest incidence, the lesions of affected birds in the families with the lowest incidences not being as bad. In all sire families except 12, which had few individuals, higher incidences were observed in male as opposed to female progeny.

The overall incidence found in G1 was rather higher than that predicted (0.31 as opposed to 0.18). This could be explained if

Table 4.1

Sire family incidence of dyschondroplasia (all growth plate abnormalities) in the G1 generation

SIRE	N DAMS	MALES N	MALES I	FEMALES N	FEMALES I	TOTAL %I	SEVERITY	RANKING
10	7	32	2(3)	19	0(1)	4(8)	2.25	9
11	4	4	1	4	0	13	3.00	8
12	5	8	2	6	2	29	3.25	5
13	8	34	16(18)	19	6(7)	42(47)	3.16	2
14	7	20	5(7)	17	3	22(27)	2.80	6
15	6	24	4(5)	11	2	17(31)	2.45	7
16	3	10	4	14	3	29	3.00	4
17	6	27	10(17)	15	5	36(52)	2.36	3
18	6	28	21(24)	14	9	71(79)	3.27	1
TOTAL	52	187	65(85)	119	30(32)	31(38)	2.91	

Table 4.2

(a) Incidence of defect traits

	DY	CT	B	S	TW
G1	0.311	0.128	0.095	0.125	0.036
G2	0.380	0.176	0.102	0.097	0.014
CONTROL	0.187	0.099	0.049	0.160	0.0

(b) Phenotypic correlations between dyschondroplasia and other traits

	CT	B	S	TW	LWT
G1	-0.05	0.08	-0.07	-0.05	-
G2	-0.02	0.09	0.16	0.02	-0.10

(c) Sire plus dam component estimates of heritability ($\hat{h}^2_c$) of dyschondroplasia, and genetic correlations of dyschondroplasia with other traits (AoV).

	DY	CT	B	S	TW	LWT
G1	1.00±0.25		0.41±0.26	-0.29±0.29	0.13±0.29	
G2	0.36±0.20	-2.73±5.77	-0.09±0.46	0.82±0.34	0.76±0.74	-0.41±0.36

selected birds had a higher incidence than their full-sibs which had been dissected. Alternatively, the heritability of dyschondroplasia may be higher than 0.50. Another more likely explanation is that the environmental conditions in which G1 birds were reared favoured the development of dyschondroplasia, and that the penetrance of any genes involved with the condition was increased.

The incidences (Table 4.2a) of the various leg abnormalities were quite high, ranging from 3.6% for twisted leg to 12.8% for crooked toe. These figures are greater than is normally the case in pure-line flocks (cf Chapter 6). Phenotypic correlations between dyschondroplasia and other defects, both scored as present or absent (Table 4.2b), were all low and of the order of 5-10%. These were all negative except for that between bow and dyschondroplasia. The numbers of defects found are considered later, in conjunction with those from G2, when the significance of the phenotypic correlations between dyschondroplasia and other abnormalities is examined. Estimates of heritability and genetic correlations are also discussed in relation to the G2 results (Table 4.2c).

On the basis of the results presented above, the ranking of sire families was examined, and dam families with high incidences recorded from the best of these. No full-sib families with an incidence less than 50% were considered. Full-sibs of birds in these families from the first hatch were also being reared. These were 6 weeks old when the second hatch were killed at 4 weeks. Birds were chosen from these to be parents of the next generation. Again approximately 15 cocks and 60 pullets were selected to ensure that

there were enough birds to produce the same number of matings as in the G1 generation. The G1 parents were reared together and mated from 21 weeks as before, the first hatch being set at 28 weeks.

A mixture of full and half-sib family selection was therefore used on G1 birds. Responses were predicted as before. The average incidence in selected dam families was p=0.64, and that in families not selected was p=0.21, giving a realised selection differential of p=0.43. Assuming the incidence of dyschondroplasia in G1 to be p=0.18, rather lower than was actually expressed, a response of 0.34 would be expected from full-sib family selection. This would give an incidence of 0.52 in the next generation. Half-sib family selection would result in an G2 incidence of 0.57.

A third hatch of the G1 generation was obtained and used in the experiment described in the next chapter. It was decided not to include these birds in the present analysis of the G1 progeny as they were reared under entirely different conditions.

G2 generation

Unfortunately, only a small number of progeny were produced in the first hatch from the G1 parents, because of mortality and low viability combined. It was decided to examine these birds and also look at the next hatch in order to assess the effects of selection. Both hatches were reared in different houses on the same farm under standard conditions until the chicks were 4 weeks old, when they were killed and their right legs dissected. Birds were examined for

leg abnormalities as before, and also weighed prior to cervical dislocation. For each hatch, the incidence in contemporaneous standard broilers was assessed. These birds were of the same pure-line from which the G0 parents were taken, and were included as a control. It is felt that as very little selection pressure had been applied against leg defects in this line, a comparison between these and the G2 birds was justifiable.

Details of the incidences of growth plate abnormalities in G2 birds are presented in Table 4.3a. Overall, the incidence of dyschondroplasia was 38.43%, this being slightly higher than in the G1 generation. Again the incidence in cocks was greater than in pullets. A total af 216 progeny taken from 9 sires and 32 dam families were examined, which is slightly less than in the previous generation. Sire family incidences again varied enormously, with a continuous range from 12.5% to 76.9%. With the exception of sire family 19, in which low numbers were involved, the severity of lesions in affected tibiotarsi corresponded quite well with the ranking of the sires in terms of incidence. The overall mean severity was much the same as for the previous generation.

If the two hatches are considered separately, however (Table 4.3b), a somewhat different story emerges. Progeny reared in the first hatch had a total incidence of 18.29%, which is rather lower than was seen in hatch 2 of the G1 generation. This is offset by an overall incidence of 50.75% in the second hatch of G2 birds, which showed a substantial increase in the incidence over G1 birds in the corresponding hatch. Subsequent analyses using Harvey's programme,

Table 4.3

(a) Sire family incidence of dyschondroplasia (all growth plate abnormalities) in the G2 generation

SIRE	N DAMS	MALES N	MALES I	FEMALES N	FEMALES I	TOTAL %I	SEVERITY	RANKING
11	4	17	11	23	12(17)	58(70)	3.04	2
12	6	23	14	24	5(7)	40(45)	3.00	3
13	4	12	6(7)	11	1(4)	30(48)	2.89	6
14	3	3	0	5	1(3)	13(38)	2.33	9
15	2	7	6	6	4(5)	77(85)	3.18	1
16	3	5	1	14	3(7)	21(42)	2.50	8
17	4	12	5(6)	19	5(6)	32(39)	2.83	4
18	5	15	4(5)	11	4	31(35)	2.88	5
19	1	5	1	4	1	22	4.00	7
TOTAL	32	99	48(51)	117	35(53)	38(48)	2.95	

(b) Comparison of G2 sire family incidence (M+F) over hatches

SIRE	HATCH 1 N	HATCH 1 %I	HATCH 1 RANKING	HATCH2 N	HATCH2 %I	HATCH2 RANKING	EXPECTED[1] %I
11	14	36	1	26	69(88)	3	76
12	19	21	3	28	54(61)	4	77
13	7	0	6=	16	44(69)	5	74
14	4	0	6=	4	25(75)	9	64
15	0	-	-	13	77(85)	2	72
16	8	13(38)	5	11	27(45)	8	54
17	11	27	2	20	35(45)	7	69
18	12	17	4	14	43(50)	6	52
19	7	0	6=	2	100	1	60
TOTAL	82	18(21)		134	51(65)		

1. predicted incidences calculated as the sum of half the full-sib means of both male and female parents ignoring regression.

Table 4.4 Incidence of dyschondroplasia (all growth plate abnormalities) in control birds reared with G2 chickens

SEX	HATCH 1 N	HATCH 1 I	HATCH 2 N	HATCH 2 I	TOTAL %I
M	20	6(9)	20	5(14)	28(57)
F	20	2(3)	21	2(4)	10(17)
M+F	40	8(12)	41	7(18)	18(37)

Table 4.5 4 week live-weight (g) of G2 and control birds

	SEX	HATCH 1	HATCH 2	TOTAL
G2	M	990	976	983
	F	845	875	860
	M+F	917	925	921
CONTROL	M	1271	1059	1165
	F	948	956	952
	M+F	1110	1008	1059

with hatch as a fixed effect in the model, have shown this to have a statistically significant effect not only on the incidence of dyschondroplasia, but also on the presence of crooked toes and bow. Sire family ranking was seen to alter between the two hatches, but the overall ranking was, naturally, more influenced by the incidence in hatch 2.

Further clarification is obtained on consideration of the incidence in birds used as controls (Table 4.4). In the first hatch the incidence of control birds was overall almost identical to that of G2 birds. In the second hatch the incidence of dyschondroplasia in control birds was slightly less than in control birds from the previous hatch, and substantially lower than in G2 birds from hatch 2. The incidence in cocks was also greater than in pullets in the control population. The mean weights (Table 4.5) do not indicate any large differences in growth rate in G2 birds between hatches. The weights achieved by the G2 birds are less than those of the corresponding controls. The difference between hatches appears to be a genuine one in the G2 progeny. If a hatch effect has caused the difference in incidence then it is another variable to be considered in any analysis of birds for dyschondroplasia. This may be important, especially if looking to select birds on the presence of dyschondroplasia.

In G2 birds, the overall incidence of dyschondroplasia was less than that predicted. However, if the two hatches are considered separately, whereas birds in the first hatch had a much lower incidence than expected, those in the second hatch had almost

exactly the predicted incidence ($p=0.52$). The penetrance of genes may have been affected by the rearing environment in the first hatch. The breeding values of G2 chickens could be predicted by adding half the incidence found in the full-sib families of their sires to half that of the full-sib families from which their dams were taken. The mean breeding values predicted for G2 sire families are compared to the actual incidences obtained (Table 4.3b). To be more correct the parental incidences should have been regressed to make a more accurate prediction, but the numbers involved were not thought to warrant this. The two sets of results were compared for each hatch, and then overall using a chi-square test of heterogeneity. The incidences in G2 birds were seen to be significantly different from those predicted in each case ($p<0.01$). These results are hard to interpret because of the low numbers of progeny involved, and differing environmental conditions. It is again possible that variable penetrance of genes may explain some of these discrepancies.

The incidences of other leg problems were again quite high in the flock examined (Table 4.2a). With the exceptions of twisted leg, and observed tibial dyschondroplasia, which had low incidences, the other incidences were comparable to those seen in the G1 generation. Apart from that of splay, the incidences of leg defects were seen to be considerably higher in G2 than in control birds. The phenotypic correlations observed (Table 4.2b) were somewhat different to those found previously. A small negative correlation was again seen between dyschondroplasia and crooked toe. However, a moderate positive correlation with splay ($rp=0.16$) was noted in this

generation, together with a small positive correlation with twisted leg. Bow again had a positive correlation with dyschondroplasia. If the various leg defects are thought to be related in origin, it is probable that some birds affected with one problem will develop another. This is particularly relevant in the case of bow, which is often a consequence of dyschondroplasia. In G2, dyschondroplasia was shown to have a negative phenotypic correlation with 4 week weight. This is probably caused by the fact that the presence of a leg abnormality will have a deleterious effect on the growth rate in individual birds.

The relationship between all growth plate abnormalities and other defects is further considered in Table 4.6. Data from G1 plus G2 and control birds from both hatches are pooled in these contingency tables. The only trait which appears to be significantly correlated with growth plate abnormalities here is bow, which has a positive phenotypic correlation with dyschondroplasia in both G1 and G2. Using all the available data, there appears to be no obvious association between presence of a leg abnormality and growth plate lesions. If G2 birds from the high incidence hatch 2 are considered separately, however, a highly significant positive correlation between the two is observed. This corroborates the findings of the earlier study in Chapter 2. The numbers of birds examined here are again too low to make firm conclusions, and these relationships are treated in more depth in Chapter 6.

Genetic parameters were estimated for the G2 generation in exactly the same way as for the previous generation. The only exception to

Table 4.6 Association of observed leg abnormalities with growth plate abnormalities (G1 + G2, pooled sexes)

All leg abnormalities

	+LA	-LA	
+GPA	103	146	249
-GPA	131	223	354
	234	369	603

$\chi^2 = 1.17$ $P>0.25$

Crooked toe

	+CT	-CT	
+GPA	28	221	249
-GPA	57	297	354
	85	518	603

$\chi^2 = 2.85$ $0.05<P<0.10$

Splay

	+S	-S	
+GPA	37	212	249
-GPA	35	319	354
	72	531	603

$\chi^2 = 3.44$ $0.05<P<0.10$

Bow

	+B	-B	
+GPA	31	218	249
-GPA	26	328	354
	57	546	603

$\chi^2 = 4.45$ $0.01<P<0.05$

Twisted Leg

	+TW	-TW	
+GPA	3	246	249
-GPA	11	343	354
	14	589	603

$\chi^2 = 2.33$ $P>0.10$

Tibial Dyschondroplasia (TD)

	+TD	-TD	
+GPA	11	238	249
-GPA	14	340	354
	25	578	603

$\chi^2 = 0.08$ $P>0.75$

this was that a fixed effect for hatches was fitted into the Harvey model. Results, together with those from G1, are presented in Table 4.5c. In estimates obtained from both generations, problems were encountered with the low numbers involved, leading to high standard errors. The use of pre-selected birds is also thought to have affected the size of the estimates. An indication of the unreliability of the G2 estimates is shown by the heritability estimate for live-weight, which was 0.56 instead of in the order of 0.25-0.30. No attempt was made to calculate heritability estimates using an offspring-parent regression of G2 on G1 results, for the above reasons.

It is appropriate to leave discussion of estimates of heritability and genetic correlations until Chapter 6, for reasons given in the last paragraph. A very high heritability estimate of 1.00 was obtained in G1, and a lower estimate of 0.36 in G2. These indicate that the heritability of dyschondroplasia is high. As will be discussed in Chapter 6, the heritabilities produced by Harvey will be biassed upwards. Results, not presented, showed dam component estimates to be higher than sire component estimates, suggesting the presence of maternal effects. With the exception of twisted leg , in which genetic correlations with dyschondroplasia were positive in both generations, the genetic correlations of the latter with other defects were inconsistent from one generation to the next. No sensible estimates of correlations between dyschondroplasia and crooked toe were obtained. Indirect evidence for an association between growth plate abnormalities and other leg defects has arisen from the fact that a greater incidence of other problems was seen in

G2 birds than in the control line.

A further hatch of G2 birds was planned. Unfortunately, for some reason as yet unexplained, over 80% of these eggs were infertile. As this was the last hatch from this generation that time and resources permitted, the selection line was discontinued.

The selection experiment was a success, in spite of reservations given, in that the incidence of dyschondroplasia in selected birds was increased to twice that of a control line. The proportion of birds affected rose from 10.31% in G0 to 31.08% in G1 to 38.43% in G2. If the incidence found in the second hatch of G2 birds only is considered then the response is seen to be even greater. As the incidence in G1 is also that of progeny from the second hatch, results from this hatch in the two generations are comparable. Responses differed from those predicted, but this could have been a consequence of changing environmental conditions and the use of a pre-selected population. At this stage of the selection programme, these results compare favourably with those obtained by other authors. Sheridan et al (1974b, 1976 and 1978) report a change in mean incidence from 10% in the first generation of selected progeny (equivalent to the G0 generation used in the present study), to 24.3% in F2, 37.4% in F3 and 80.0% in F4. Similar rapid responses were also achieved by Leach and Nesheim (1965) and Riddell (1976), each initial population starting with approximately the same incidence as observed here (see Chapter 1). All these studies

indicate, as does this, that dyschondroplasia is highly heritable.

More is said about other estimates of genetic parameters for leg weaknesses in Chapter 6. It is sufficient to note here that high estimates of heritability were also found by Sheridan et al (1978) of 0.57 and 0.52 in generations F2 and F3 respectively (cf 1.00 and 0.36 in this study). These workers also found evidence suggestiing the influence of maternal effect upon dyschondroplasia. No indications of bimodality in the distributions of sire family incidences were seen in the birds examined in the present study. This is in contrast to Sheridan et al (1978), who cited this as one aspect of circumstantial evidence pointing to the existence of a single gene. Further consideration of the mechanism of inheritance of dyschondroplasia is made in the next chapter.

The selection trial suffered from being performed under conditions, and in circumstances, which were never designed for experimental purposes. The benefits of using commercial conditions, thus simulating practical outcomes, must be weighed against lack of control over environmental variables, particularly from generation to generation. If resources had been available, much larger numbers of sires should have been used per generation, or the selection continued over more generations, and an unselected control included. It is perhaps significant that despite these shortcomings, it was found that full-sib selection could still be used to successfully increase the incidence of dyschondroplasia.

CHAPTER 5. FURTHER STUDIES INVESTIGATING MECHANISMS OF INHERITANCE

It has been proposed (Sheridan et al, 1978) that a single sex-linked recessive gene may be responsible for dyschondroplasia. Only circumstantial evidence, however, from selection lines developed to increase the incidence, was presented to support this theory. Obviously, before this can be accepted, a more thorough examination of the mode of inheritance of dyschondroplasia is required. The best way to do this is to perform specific crosses and look for evidence of the involvement of a single gene.

In the previous chapters, ways of influencing the incidence of dyschondroplasia have been discussed. It is necessary to have the use of a high incidence flock of birds in order to select reasonable numbers of affected birds for experimental purposes. If such a flock were not available, then very large numbers of chickens would have to be examined to obtain the required quota. Having obtained a population with a high incidence, it should be then possible to identify several affected and non-affected individuals, and mate these in a series of test crosses. This would necessitate the use of live assessment for detection of the presence of growth plate abnormalities. The progeny would themselves be examined to see if the incidence of dyschondroplasia fitted into Mendelian ratios. Reciprocal and back crosses could then be used to confirm these results as necessary.

The present study combines information and material already mentioned in order to meet such requirements. Birds known to have a

high incidence were fed a diet formulated to increase the incidence of dyschondroplasia. As several authors (Ferguson et al, 1978; Veltmann and Jensen, 1980; Leach, 1982) have reported that incidences are usually higher when birds are grown on litter as opposed to on wire, chicks were reared in litter pens in the hope that this would further exacerbate the morbidity. Live assessment was carried out using radiography, and the subsequent progeny tests examined by dissection. As will be seen later, although the trial was designed to yield plenty of affected birds, the numbers involved in the subsequent crosses were inadequate for the determination of mechanisms of inheritance.

Rearing Trial

A hatch of G1 birds from a line (MT) selected for dyschondroplasia (see Chapter 4), with an incidence previously established as 31% was available for use. These chickens were reared at the PRC together with pedigreed broilers of the same male-line (ML) from which MT was initially derived. It was hoped that by using as many birds as could be accomodated from two closely related lines, estimates of genetic parameters for dyschondroplasia could be calculated. A total of 422 birds were examined from 14 sire families and 75 dam families. 100 chickens from an egg-laying strain (S) were also placed in the trial. S-line birds were thought to be unaffected by dyschondroplasia, and were therefore reared to verify this. If they did in fact prove to be free of the condition it was intended to mate some S-line birds to affected broilers.

Approximately 25 birds of one sex and one line were placed in each of 20 pens of 2.4m x 1.5m containing a deep litter of wood shavings. Temperature was kept at approximately 21C, and a 16-hour on, 8-hour off lighting regime used. Chicks were reared under an infra-red brooder lamp until 7 days old. All birds were fed a purified diet containing FP950 (see Appendix 1). Food was weighed into hoppers and the amount eaten per pen recorded. This was discontinued after 4 weeks when remaining birds were switched to a broiler layer starter mash. Birds were weighed weekly up to 4 weeks and those remaining were weighed again at 6 weeks.

Two hundred birds from the MT and ML lines were examined at 26 or 27 days using the radiographical technique described in Chapter 2. The rest of these lines, plus half the S-line birds, were killed at 28 days by cervical dislocation, their legs dissected, and the incidence of growth plate abnormalities recorded , also using the methodology described in Chapter 2. Radiographs were processed automatically at the Royal (Dick) Veterinary School Field Station. Birds thought to be affected were given a further radiograph at 40 days for confirmation. MT males from a single pen, and thought to be unaffected were killed at 45 days. As the number of affected individuals already found fell short of that desired, and some of the birds assessed as not having dyschondroplasia were seen to be affected, all remaining birds were again radiographed at 46 days. Confirmatory radiographs were again taken of possibly affected birds. This extra screening revealed few extra cases of abnormalities.

For comparison, 10 S-line birds were also radiographed at 27 days. Half of the birds from this line were killed and their legs assessed at 28 days. As affected individuals were found during the course of this examination the remaining S-line chickens were killed at 45 days.

All birds were subjected to a live assessment at 28 days, and survivors again at 41 days. This consisted of handling the birds, palpating the stifle joint, and awarding a score for obvious leg defects. Birds were also placed on the ground, and the facility or reluctance with which they moved noted. These indicators were considered in conjunction with radiographs when making a final assessment of whether or not a bird was affected.

Sire plus dam component estimates of heritability and genetic correlations, together with their standard errors, were computed using a Least Squares Maximum Likelihood programme (Harvey, 1975) on pooled 4 week data from ML and MT. Phenotypic correlations were calculated using the same model. Line of birds was fitted as a fixed effect, together with sex. Estimates of h^2 for dyschondroplasia were corrected for continuity using the transformation of Robertson and Lerner (1949). As discussed in Chapter 4, the estimates obtained suffer from the fact that low numbers were involved, and details of the methodology are described in Chapter 6.

Results:

Details of the incidences observed, as calculated after all

Table 5.1

(a) Incidence of dyschondroplasia (all growth plate abnormalities) in PRC rearing trial (all stages)

STRAIN		MALES				FEMALES				TOTAL
		N	I	%I	SEVERITY	N	I	%I	SEVERITY	%I
Control Sel layers	ML[1]	128	30(32)	23(25)	3.44	113	7(9)	6(8)	2.78	15(17)
	MT	92	21	23	3.43	89	9	10	3.22	17
	S	40	2	5	3.00	59	0	-	-	2

(b) Live-weights (g) of birds plus FCR from 0-4 weeks

STRAIN	SEX	WK0	WK2	WK4	WK6	FCR[2]
ML	M	43	285	841	1637	0.74
ML	F	42	277	790	1428	0.72
MT	M	41	267	766	1457	0.73
MT	F	41	261	702	1297	0.70
S	M	38	141	321	514	0.52
S	F	38	132	271	440	0.50

(c) Correlations of dyschondroplasia with live-weight (ML + MT)

	WK0	WK1	WK2	WK3	WK4
PHENOTYPIC	0.07	0.00	-0.07	-0.22	-0.22
GENETIC	0.12±0.22	0.16±0.23	0.15±0.24	0.01±0.25	0.04±0.24

(d) Sire + dam component heritability estimates (AoV pooled sexes, ML + MT)

DY	0.63±0.12
LWT WK2	0.55±0.12
LWT WK4	0.56±0.12

1. includes cock with no wing band
2. FCR = weight gained / food eaten (g)

assessments had been performed, are shown in Table 5.1a. These were disappointing, an overall incidence of dyschondroplasia of 16% resulting in ML and MT birds considered together. There were no apparent differences in either incidence or severity of affected tibiotarsi between these two lines, cocks having a greater incidence than pullets in both cases. This compares with previously recorded incidences of 31% and 42% in birds fed FP950 and reared in wire cages, and 31% in the previous hatch of MT birds reared under commercial rearing conditions.

Dyschondroplastic lesions can be induced in layer-type chickens. Thus it cannot be stated that egg-layers are genetically free of any susceptibility to the condition. For this reason it was decided that broilers assessed as being unaffected would be used in preference to egg-layers for any test matings.

Live-weights of birds are presented in Table 5.1b, and correlations of weights (excluding S-line birds) with dyschondroplasia in Table 5.1c. Although ML birds were seen to be heavier on average than MT birds, this had no obvious effect on the incidence of dyschondroplasia. ML chickens also had slightly better food conversion ratios. Phenotypic correlations at 3 and 4 weeks were seen to be quite strongly negative ($r_p=-0.22$), suggesting that birds affected by dyschondroplasia have impaired growth rates. In contrast, the genetic correlations of dyschondroplasia with live-weight at all ages were seen to be positive. At 1 and 2 weeks these were moderate ($r_g=0.16$, 0.15), but at 3 and 4 weeks the correlations were very small ($r_g=0.01$, 0.04). Care must be taken in

interpreting these figures as the numbers of birds involved is low. It appears that heavier weights during the developmental period of the skeleton are more critical to the formation of abnormalities, than is the case when the birds are older.

The sire plus dam component heritability estimates for 3 and 4 week weights are, not surprisingly, very similar, but higher than might be expected (Table 5.1d). These are also similar to the heritability estimates for 4 week weight calculated in Chapter 4. The estimate for dyschondroplasia is also high, as were estimates obtained in Chapter 4 and by Sheridan et al (1978). It should be remembered that the birds involved originally came from a pre-selected population, and that this may have had an effect on estimates obtained.

Sire family incidences (Table 5.2) were seen to range between 6% and 25% in ML and between 0% and 50% in MT. Whereas a continuous range of incidences were observed in ML, there are indications that these fall into two groups in MT. Five sires had family incidences of 14% or less, and the other three had incidences of 38%, 44% and 50% respectively. This bimodality may indicate the segregation of a single gene, although the numbers in each sire family are very low. The distribution of sire family incidences in MT can be shown to be heterogeneous by use of chi-squared ($p<0.05$). Sheridan et al (1978) also noted bimodality in the distribution of sire family incidence in their selection line.

Very few cases of other leg abnormalities were noted in the birds

Table 5.2 Sire family incidences of dyschondroplasia in PRC rearing trial

(a) ML (41 dam families)

SIRE	N	I	%I	RANKING	SEVERITY
10	34	5	15	2=	2.43
11	52	3	6	6	4.33
12	52	13	25	1	3.50
13	24	3	13	5	3.67
14	38	6	17	4	3.33
15	40	6	15	2=	3.00
TOTAL	240	35	15		

(b) MT (34 dam families)

SIRE	N	I	%I	RANKING	SEVERITY
10	45	5	11	6	2.80
12	16	6	38	3	3.33
13	52	6	12	5	3.67
14	30	1	3	7	3.00
15	7	0	-	8	-
16	16	7	44	2	3.43
17	7	1	14	4	5.00
18	8	4	50	1	3.25
TOTAL	181	30	17		

examined, with the exception of splay. Ninety-nine percent of all ML and MT birds were seen to have at least very slightly splayed legs, 53% of these being more pronounced. None of these defects were seen to affect the birds in any way. An analysis of birds with 'moderate' splay was carried out using the modified proband method (see Chapter 6). This gave heritability estimates of 0.46 for dyschondroplasia, 0.28 for splay and a genetic correlation of -0.75 between the two. The correlation is rather large and thought to be attributable to sampling variance arising as a consequence of the low numbers involved. No birds with severe locomotory disturbances were noted. Extra means of live assessment were felt to be largely unhelpful as an aid to diagnosing leg conditions in this instance. Similarly, the few birds assessed as having a swollen stifle joint were not subsequently seen to be affected by dyschondroplasia.

As a consequence of the low incidence of dyschondroplasia found, fewer birds than intended were selected at the end of the trial. As opposed to 20 unaffected and 20 affected broilers of each sex, 9 affected, 3 possibly affected and 20 unaffected males were chosen together with 2 affected, 6 possibly affected and 20 unaffected females.

Test Crosses

Birds selected from the rearing trial were put onto a restricted broiler layer dietary regime used at the PRC. Cocks were placed into two pens, with subdued lighting to prevent fighting, and hens into individual wire cages. Plastic pads were provided in the cages to

prevent foot sores. Lighting was continued at 16-hours on, 8-hours off. Cocks were tested for sperm production from 18 weeks onwards.

Unfortunately, cocks did not reach full maturity until 40 weeks. Several birds died or had to be culled because of disabling leg problems before this time. In the latter instance birds were seen to develop extreme locomotory disturbances (not necessarily related to dyschondroplasia) and could no longer move around the pen to feed. Fertility was found to be poor and not all those cocks remaining produced semen. The fertility problem did not appear to be related to the size of the birds, and more possibly was a result of being reared on the same light regime right from hatching.

Time permitted only two hatches of test matings. Crosses were set up so that affected cocks were mated to affected hens, affected cocks to unaffected hens, unaffected cocks to affected hens, and unaffected cocks to unaffected hens. As the number of affected and possibly affected birds was by this time very limited, affected birds were mated to birds of 'opposite' type in the second round of crosses. Thus if an affected cock was mated to an affected hen for the first hatch, it was mated to an unaffected hen later on. All matings were perfomed using artificial insemination. For each hatch eggs were collected for 12 days and set together, matings being repeated three times at three to four day intervals. An interval of 3 weeks was left between the two sets of matings. Fertility was seen to have further decreased by the end of the second round of matings.

Progeny were reared in exactly the same way as their parents. This

was to ensure that environmental conditions were as near as possible to those in the rearing trial, and to ensure that the penetrance of any genes involved would be the same. Chicks were sexed and given a pedigreed wingband directly after hatching. FP950 was fed to birds until 4 weeks old when they were killed by cervical dislocation and their legs examined by dissection, as described in Chapter 2.

Results of the test crosses are shown in Table 5.3. Parent birds assigned a question mark were thought to be possibly affected by dyschondroplasia, but the diagnosis from the radiographs was not certain in these cases. A total of only 78 birds from both hatches were examined. Obviously, with such low numbers of progeny it is impossible to make any statistical conclusions regarding the distribution of dyschondroplasia amongst families. Table 5.3b shows that matings involving at least one affected parent were more likely to produce more progeny with dyschondroplasia than were matings between unaffected individuals. Considering the various heritability estimates already obtained for dyschondroplasia, this merely confirms that the condition is inherited.

Various interesting resuts may be pointed out. Matings between unaffected parents are capable of producing affected offspring. When one unaffected cock was mated to two different definitely affected hens, some affected progeny were produced from the first cross, but none from the second cross. No crosses produced all affected progeny, and generally no indications were found of distributions of dyschondroplasia corresponding to Mendelian ratios. All the above suggest that the inheritance of the condition is not controlled

Table 5.3

(a) Results of PRC test crosses - number of progeny affected (+) and unaffected (-) by dyschondroplasia

COCK	HEN 1	HEN 1 PROGENY MALE +	HEN 1 PROGENY MALE -	HEN 1 PROGENY FEMALE +	HEN 1 PROGENY FEMALE -	HEN 2	HEN 2 PROGENY MALE +	HEN 2 PROGENY MALE -	HEN 2 PROGENY FEMALE +	HEN 2 PROGENY FEMALE -	TOTAL PROGENY MALE +	TOTAL PROGENY MALE -	TOTAL PROGENY FEMALE +	TOTAL PROGENY FEMALE -
1+[1]	1-					7?	1	1	0	3	1	1	0	3
2+	2?	1	0	0	1						1	0	0	1
3+	3-					16-	0	1	0	0	0	1	0	0
4?	4?	0	3	0	1						0	3	0	1
5?	5-	2	2	0	1						2	2	0	1
6-	6+	2	0	1	3	1+	0	1	0	2	2	1	1	5
7-	7?	0	1	0	1	8?	2	1	0	0	2	2	0	1
8-	8?	2	3	0	0						2	3	0	0
9-	9-	0	2	0	4						0	2	0	4
10-	10-	0	0	1	2	2?	0	0	0	1	0	0	1	3
11-	11-	0	1	0	1	5-	0	4	0	1	0	5	0	2
12-	12-	0	3	1	2	9-	0	0	0	1	0	3	1	3
13-	13-	1	0	1	3						1	0	1	3
14-	14-	1	1	0	2						1	1	0	2
15-	15-	0	1	0	2						0	1	0	2
16-	16-					18-	0	2	0	1	0	2	0	1
17-	17-	0	1	0	2						0	1	0	2
	TOTAL	9	18	4	25		3	10	0	9	12	28	4	34

(b) Distribution of affected progeny according to parental type (one or both parents having the indicated category)

SIRE OR DAM	MALES +	MALES -	FEMALES +	FEMALES -	TOTAL +	TOTAL -	%I
+	4	3	1	9	5	12	29
?	6	10	0	4	6	14	30
-	2	15	3	21	5	36	12
TOTAL	12	28	4	34	16	62	21

1. + affected, ? possibly affected, - unaffected

exclusively by one gene.

It is unlikely that the environmental conditions used in the initial rearing trial and for the subsequent test crosses were sufficiently different to permit varying penetrance of a single gene. The possibility cannot be ruled out, however, that dyschondroplasia is regulated by a few genes, or one gene whose effects are modulated by others. Similarly, the data are too sparse to refute the involvement of one or several thresholds, above which the condition is expressed. By themselves, the crosses made are not sufficiently sensitive to test the above hypotheses, and a further series of matings would be necessary to make any more positive conclusions.

The lack of results from this study was disappointing, as although little evidence was found to support the hypothesis of a single gene model, the data are far from being sufficiently adequate to refute this. Two factors were particularly puzzling. The first of these was the fact that a few birds were seen to have dyschondroplasia on dissection, having been pronounced clear after radiography. Either these birds developed the condition after the initial examination, and later than would be expected, or the technique is not as reliable as indicated by the initial studies. Unfortunately, birds with an unclear diagnosis had to be used for crosses, as the number of definitely affected birds was so small. It had been hoped that these cases could have been ignored if plenty of affected birds had been found. This uncertainty would also suggest that radiography is fallible. Secondly, the incidence in the trial as a whole was much

Data were analysed in a similar way to those collected in the PRC nutrition trials described in Chapter 3. An analysis of variance was performed on birds with growth plates awarded a score of 1-5 using GENSTAT, with a model incorporating the effects of diet, floor type and sex. Phenotypic correlations were also taken from GENSTAT. An analysis of deviance incorporated in GLIM was then used on birds reclassified as being affected or unaffected.

The incidences, weights, and correlations between the two found in the experiment are presented in Table 5.4. The incidence of birds fed with FP950 was considerably higher in birds reared on wire (67%) than in those on litter (29%). In contrast, birds fed the practical diet had the same incidences on both wire and litter, these being close to that of birds fed FP950 and reared on litter. The severity of lesions in affected tibiotarsi were seen to be less in birds reared on a practical diet. In all combinations the incidence in cocks was greater than that in pullets, but these sex differences were only marked in birds fed FP950 and reared on wire. Birds showed negative phenotypic correlations with live-weight at 3-4 weeks, as was found in the rearing trial. Four week live-weights were greater in birds reared on practical diets. Within each diet, chickens were heavier when grown in cages. These weights do not appear to bear any relation to the incidence of dyschondroplasia.

The results of the analyses performed on the data are given in Table 5.5. The effects of diet, floor type, and diet x floor type interaction are all statistically significant whether birds are scored on a 0-1 or 1-5 basis. The significance levels are higher if

Table 5.4

(a) Incidence of dyschondroplasia (all growth plate abnormalities) in an experiment to test the effects of floor type and diet

1. FP950 (purified)

	MALES				FEMALES			
	N	I	%I	SEVERITY	N	I	%I	SEVERITY
WIRE	31	25(26)	81(84)	3.51	13	7	54	3.55
LITTER	20	6	30	3.67	25	7(9)	28(36)	3.00

(2) Practical

	MALES				FEMALES			
	N	I	%I	SEVERITY	N	I	%I	SEVERITY
WIRE	17	6	35	2.63	32	8(9)	25(28)	2.92
LITTER	23	7(11)	30(48)	2.64	25	7(10)	28(40)	2.70

(b) Live-weights (2 weeks) and 4 weeks (g)

	FP950				PRACTICAL			
	MALES		FEMALES		MALES		FEMALES	
WIRE	(252)	724	(278)	785	(266)	851	(266)	842
LITTER	(238)	752	(220)	678	(226)	803	(216)	749

(c) Phenotypic correlations between dyschondroplasia and live-weight

WK0	-0.01
WK1	0.08
WK2	-0.05
WK3	-0.17
WK4	-0.14

Table 5.5 Significance of effects upon incidence of dyschondroplasia in an experiment to test the influence of floor type and diet.

(a) Analysis of deviance (0-1)

EFFECT	χ^2	df	
Floor Type	6.4	1	* [1]
Diet	3.9	1	*
Sex	2.8	1	ns
Floor Type x Diet	5.2	1	*
Floor Type x Sex	2.5	1	ns
Diet x Sex	0.6	1	ns
Floor Type x Diet x Sex	0.7	1	ns

(b) Analysis of variance (scores)

EFFECT	F-ratio	df	
Floor Type	13.1	1	***
Diet	16.5	1	***
Sex	8.0	1	*
Floor Type x Diet	13.0	1	***
Floor Type x Sex	4.3	1	ns
Diet x Sex	0.7	1	ns
Floor Type x Diet x Sex	1.3	1	ns
Residual		191	

1. ns = not significant, * = $P<0.05$, ** = $P<0.01$, *** = $P<0.005$

1-5 scores are used, and sex also has a significant effect if these are analysed. These findings support the hypotheses that both diet and floor type influence the incidence of dyschondroplasia. More importantly, the presence of a significant floor type x diet interaction explains why the incidence found in the rearing trial was lower than expected. The effect of litter in reducing the incidence of growth plate abnormalities is contrary to the results presented by other authors. This is possibly explained by the fact that the chickens did not have to compete as much for food, and were able to achieve heavier weights when reared in wire cages in the experiments at the PRC.

No evidence has been found to support the theory proposed by Sheridan et al (1978) for the involvement of a recessive sex-linked gene in dyscondroplasia. Indeed, the test crosses did not result in any distribution of dyschondroplastic progeny that could be explained by Mendelian ratios. Number of crosses made and progeny produced were small, and not sufficient to make any definite conclusions about the mechanisms of inheritance for dyschondroplasia.

Incidences involved in this study were lower than expected. This was at least partly explained by a subsequent experiment which showed the presence of a diet x floor type interaction. Lower incidences were observed when birds were reared on litter as opposed to in wire cages. This is the reverse of the situation reported

previously (Ferguson et al, 1978; Veltmann and Jensen, 1980; Leach, 1982). Some doubt has been cast upon the reliability of the radiographic technique used to assess dyschondroplasia previously described in this thesis, but the method remains an efficient way of performing live assessment.

CHAPTER 6. ESTIMATION OF GENETIC PARAMETERS FOR DEFECT TRAITS

Leg problems have been a source of concern to the broiler industry for at least twenty years. Despite the fact that many selection programmes now operate against them, little is known about the genetics of such abnormalities. Indeed, no attempt has been made to estimate genetic parameters such as heritability and genetic correlations on large data sets obtained from commercial populations, although these do now exist. Such estimates as are available, as for tibial dyschondroplasia (Sheridan et al,1978), are the results of relatively small-scale selection experiments.

Collection of data on skeletal defects presents many problems, and has only been undertaken in the last decade. Often this takes the form of 'leg-problems', and the differences between individual defects are ignored. There is much confusion in the scientific literature as to which problems can be considered sufficiently distinct as to warrant independent status, and it can be argued that various conditions are merely different manifestations of the same underlying syndrome. To distinguish between various defects requires a trained selection team, and to further categorise each condition into classes of severity requires yet more skill. If leg problems are divided into discrete defects, as would be preferable for the purposes of genetic analysis, then the incidence of each condition is likely to be small, and very large data sets will be necessary in order for subsequent estimates of parameters to be meaningful.

Of necessity, data on skeletal defects is recorded as 'plus' or

'minus', or at best in three or four categories of increasing severity. Such 'all-or-none' traits are binomially distributed, and therefore present theoretical problems in the estimation of genetic parameters. Standard analysis of variance techniques used to compute intraclass correlations suffer from the fact that in all-or-none data the variances are not independent of the means (or incidences). One way round this is to assume that the trait in question has an underlying continuous distribution, and that visibly affected individuals have a value on this scale that exceeds a threshold point. In reality this is quite an acceptable hypothesis as several genetic and environmental factors are likely to operate on the so-called 'liability' of the trait. Tibial dyschondroplasia is usually recorded as present or absent on a visual assessment, but dissection of birds shows that the growth plate may show a continuous range from a discrete layer to a large plug of cartilage in severely affected individuals.

Robertson and Lerner (1949), looking at viability in poultry, proposed a transformation that would change intraclass correlations calculated on a 0-1 scale to those on the underlying scale. The arcsine square-root transformation (Cochran, 1940), may be used on group data so that the incidence in each group is independent of its variance. These methods are the ones that have been previously used to obtain estimates of genetic parameters for dyschondroplasia (Sheridan et al, 1978) and twisted leg (Hartmann, 1978). The transformation of Robertson and Lerner, however, produces biassed estimates when the true intraclass correlation is high and the incidence of the trait close to its limits of 0 or 1 (Hill and

Smith, 1977). Unfortunately this applies to the situation with leg problems which have incidences seldom exceeding 5%.

Falconer (1965) proposed an alternative means of analysing all-or-none data which assumes an underlying normally-distributed liability and considers the incidence in affected individuals in conjunction with that of their relatives. This, and the analysis of variance method proposed by Robertson and Lerner, will be considered in detail. A further possible technique is to use an iterative Maximum Likelihood procedure such as the GLIM package adopted by Cue (1982), on mortality in lambs. This fits the data to an additive linear model using a logit transformation. Cue's results, however, did not suggest that the method produces any better estimates than do analysis of variance techniques. Indeed, GLIM lacks the flexibility of mixed-model analysis of variance packages in fitting effects to a linear model. This is because of limitations in the number and type of effects that may be included in the model, and also because the iterative procedure can only handle a certain amount of data. GLIM has therefore not been considered further in this study.

Obtaining Estimates Of Heritability And Genetic Correlations

While the analysis of live-weight data is straightforward, that of the defect data is not, because of its bimodality. Four methods of analysis were considered initially in order to determine which, if any, were most applicable to the data. These could be split into two categories, two involving analysis of variance techniques, and two

the proband method.

An analysis of variance was performed on half-sib family summaries, following the methodology of Elston (1977), and compares the variance between and within family incidences. In a situation where:

r_i = number of affected birds in the ith family

n_i = number of birds in the ith family

C = number of families

N = total number of birds

the mean-square among families MSA is equal to

$$\frac{\sum_i \frac{r_i^2}{n_i} - \frac{(\sum_i r_i)^2}{N}}{C - 1}$$

and the mean-square within families is equal to

$$\frac{\sum_i r_i - \sum_i \frac{r_i^2}{n_i}}{N - C}$$

which leads to the estimate of the intraclass correlation

$$t = \frac{MSA - MSW}{MSA + (k-1)MSW}$$

with $k = (N - \sum_i n_i / N) / C - 1$

The heritability estimate obtained on the 0-1 sale is now equal to t x the inverse of the relationship between relatives, which is 0.25 for half-sibs and 0.50 for full-sibs. To transform the estimate to one on an underlying scale, one must assume that a normal distribution applies, and use the correction for continuity of Robertson and Lerner (1949) :

$$h^2 c = h^2 01 \times \frac{p(1-p)}{z^2}$$

where p = the incidence of the trait, and z is the height of the ordinate of a standardised normal at the threshold point

corresponding to p.

To overcome the problems of heterogeneity of within group variance associated with incidence level, the data may be subjected to an arcsine square-root transformation (Cochran, 1940). For each family incidence q ,the arcsine square-root is taken : $x_i = \sin^{-1}\sqrt{q_i}$ (radians). This leads to the variance of n x becoming approximately $\sum_i n_i /4$, and therefore the within family sum of squares is $n_i/4$.

$$MSA = \frac{\sum_i n_i x_i^2 - (\sum_i n_i x_i)^2 / N}{C-1}$$

$$MSW = N / 4(N - C)$$

The intraclass correlation and heritabiliy may now be computed exactly as before.

The proband method (Falconer, 1965, 1967) examines the underlying liability of individuals to a trait. The liabiltiy of affected individuals (or propositi) is compared with that of their relatives, and the difference between the two, expressed in units of normal deviates, may be considered to be equivalent to a selection response (see Figure 6.1). Likewise, the difference in mean liability between affected individuals and the population as a whole is the selection differential. The ratio of the selection response and the selection differential gives us in this case the regression of relatives on propositi, from which the heritability is simply obtained as before, by dividing by the coefficient of relationship.

$$b \text{ relatives on propositi} = \frac{R - G}{A - G} = \frac{Xg - Xrel}{a}$$

$$h^2 = b \; / \; \text{coefficient of relationship}$$

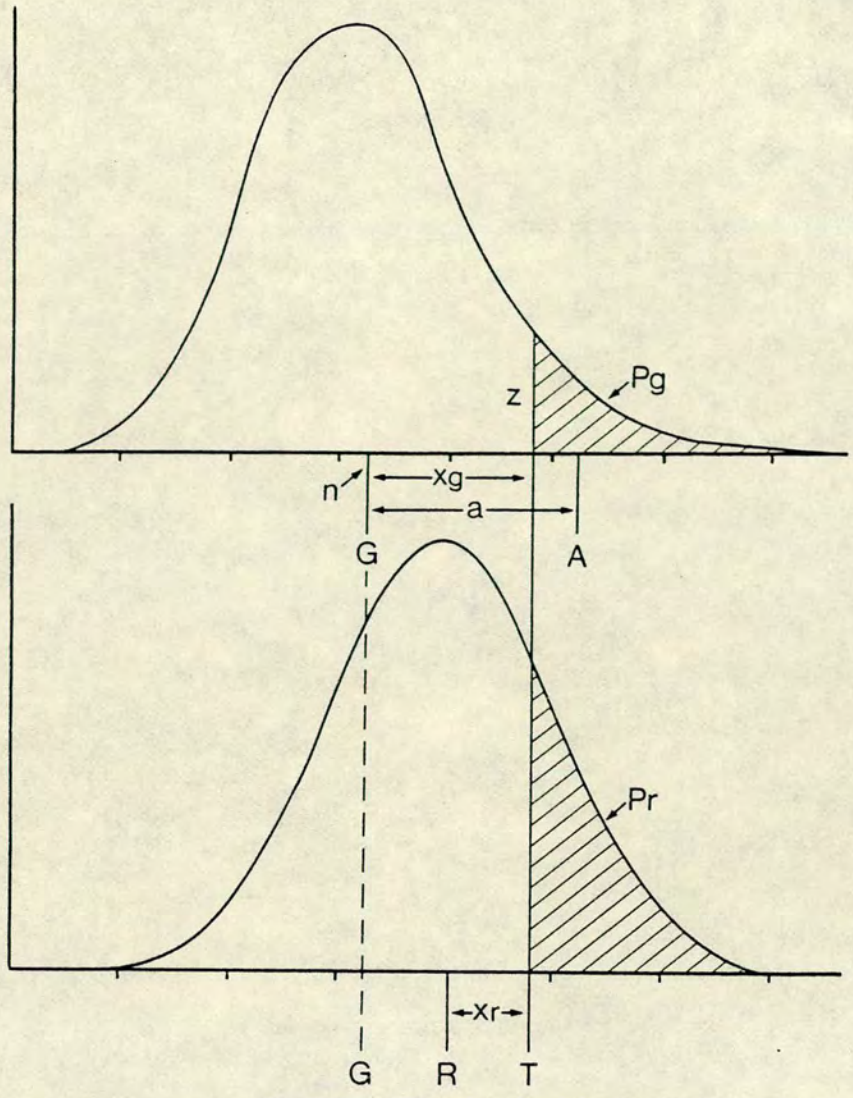


Fig. 6.1

Falconer (1965) Figure 2. Two distributions representing the general population above, and the relatives of affected individuals below, compared with reference to the fixed threshold, T.

G = mean liability of general population.
A = mean liability of affected individuals in the general population.
R = mean liability of relatives.
P = incidence, ie proportion of individuals with liabilities exceeding the threshold.
x = deviation of threshold from mean, ie the normal deviate.
z = height of the ordinate at the threshold.
a = mean deviation of affected individuals from the population mean (= z/p).
n = mean deviation of normal individuals from the population mean (= z/(1-p)).

subscript g refers to the general population, subscript r to the relatives

A modification of Falconer's proband method which takes into account the fact that the variances of the general population from which the propositi came, will be greater than the variance in the population which consists of their relatives, has been suggested by Reich, James and Morris (1972). This estimates the correlation of liability amongst relatives.

$$r = \frac{xg - xrel\sqrt{(1 - (xg^2 - xrel^2)(1 - xg/a)}}{a + xrel^2(a - xg)}$$

Before application of the techniques discussed to actual data, it was decided to examine some properties of the analysis of variance and modified proband methods. This was done by computer simulation, and also included estimation of genetic correlations. To obtain these a simple nested analysis of variance, exactly analagous to that described in the previous section, was performed using Harvey's Mixed Model Least Squares Analysis Of Variance package (Harvey, 1975), which will be simply referred to as Harvey henceforth.

For full-sibs:

$$rg = \frac{COVA\ trait1,trait2}{\sqrt{VA\ trait1\ .\ VA\ trait2}} \qquad rg = \frac{\sigma s12\ .\ \sigma d12}{\sqrt{(\sigma s1+\sigma d1)(\sigma s2+\sigma d2)}}$$

Genetic correlations may also be computed using the proband method, by considering the selection response produced in one trait from selection on another.

$$b\ \text{liability in trait2 of relatives of propositi affected by trait1} = \frac{xg\ trait2 - xrel\ trait2}{a\ trait1}$$

$$rg\ trait1,trait2 = \sqrt{\frac{b21 \times b12}{b22 \times b11}} = \sqrt{\frac{COV12 \times COV21}{V1 \times V2}}$$

This can be also modified to allow for differences in variance:

$$r \text{ liability in trait2 among relatives affected with trait2 selected on trait1}$$

$$= \frac{xg2^2 - xrel2\sqrt{1 - (xg2^2 - xrel2^2)(1 - xg1/a1)}}{a1 + xrel2^2(a1 - xg1)}$$

$$rg\ trait1,trait2 = \sqrt{\frac{r\ t2 \text{ selected on } t1 \quad r\ t1 \text{ selected on } t2}{r\ t1 \text{ selected on } t1 \quad r\ t2 \text{ selected on } t2}}$$

Simulation

A simulated population was generated with three defect traits, 150 sires each having 6 dams, with 24 progeny per dam (N=21600). Traits were assigned a fixed value of heritability (h^2 1=0.40, h^2 2=0.25, h^2 3=0.10), and a genetic correlation which was the same between all three traits, this being either 0.50, 0.25 or 0.10. The population was then truncated at various different incidences (p=0.05,0.02,0.005), which were assigned to each of the values of heritability in turn, and repeated for the three different genetic correlations. Each truncated population was subjected to a full-sib analysis using Harvey, and then the modified proband method. The estimates of heritability obtained from Harvey were corrected for continuity, and the corrected values, plus the genetic correlations, compared with those obtained from the modified proband method, and also the actual continuous parameters (Tables 6.1a,b,c). The figures presented are the means of between 18 and 25 replications for each combination of parameters, which enabled the calculation of an empirical standard error.

The results show that for a known distribution, without environmental complications, an analysis of variance produces consistently higher estimates of heritability than does the proband

Table 6.1 Comparison of mean estimates of h^2 and rg obtained (together with standard errors) from 18-25 repetitions of simulation.

(a) Incidence of T1=0.05, T2=0.02, T3=0.005

rg	h^2_{T1}	ESTIMATE	h^2_{T1}	h^2_{T2}	rgT1T2	h^2_{T3}	rgT1T3
		1.	0.40±0.001		0.50±0.004		0.45±0.004
0.50	0.40	2.	0.50±0.014	0.25	0.48±0.027	0.10	0.54±0.075
		3.	0.38±0.008		0.53±0.025		0.63±0.086
		1.	0.25±0.001		0.52±0.003		0.50±0.004
	0.25	2.	0.28±0.007	0.10	0.49±0.046	0.40	0.47±0.029
		3.	0.24±0.006		0.52±0.047		0.56±0.030
		1.	0.10±0.001		0.45±0.004		0.52±0.003
	0.10	2.	0.11±0.006	0.40	0.41±0.025	0.25	0.48±0.066
		3.	0.10±0.005		0.48±0.027		0.53±0.069
		1.	0.40±0.001		0.25±0.005		0.23±0.005
0.25	0.40	2.	0.49±0.013	0.25	0.22±0.022	0.10	0.27±0.063
		3.	0.38±0.009		0.26±0.026		0.34±0.071
		1.	0.25±0.001		0.25±0.004		0.25±0.005
	0.25	2.	0.28±0.008	0.10	0.21±0.036	0.40	0.25±0.028
		3.	0.24±0.006		0.23±0.038		0.31±0.032
		1.	0.10±0.001		0.23±0.005		0.25±0.004
	0.10	2.	0.11±0.006	0.40	0.21±0.020	0.25	0.29±0.065
		3.	0.10±0.005		0.25±0.023		0.33±0.068
		1.	0.40±0.001		0.09±0.005		0.10±0.006
0.10	0.40	2.	0.49±0.016	0.25	0.08±0.025	0.10	0.15±0.067
		3.	0.39±0.008		0.11±0.023		0.18±0.079
		1.	0.25±0.002		0.10±0.004		0.09±0.005
	0.25	2.	0.28±0.008	0.10	0.05±0.020	0.40	0.10±0.026
		3.	0.24±0.006		0.06±0.023		0.13±0.034
		1.	0.10±0.001		0.10±0.006		0.10±0.004
	0.10	2.	0.11±0.007	0.40	0.09±0.023	0.25	0.12±0.038
		3.	0.10±0.006		0.10±0.026		0.14±0.046

Estimates

1. Actual parameters of underlying distribution.
2. AoV of truncated population, with correction for continuity.
3. Modified proband method used on truncated population.

Table 6.1 Comparison of mean estimates of h^2 and rg obtained (together with standard errors) from 18-25 repetitions of simulation.

(b) Incidence of T1=0.02, T2=0.05, T3=0.005

rg	h^2_{T1}	ESTIMATE	h^2_{T1}	h^2_{T2}	rgT1T2	h^2_{T3}	rgT1T3
		1.	0.40±0.001		0.45±0.004		0.50±0.004
0.50	0.40	2.	0.55±0.018	0.10	0.41±0.025	0.25	0.53±0.060
		3.	0.37±0.009		0.48±0.027		0.59±0.058
		1.	0.25±0.001		0.50±0.004		0.52±0.003
	0.25	2.	0.30±0.014	0.40	0.48±0.027	0.10	0.67±0.110
		3.	0.23±0.008		0.53±0.025		0.73±0.106
		1.	0.10±0.001		0.52±0.003		0.45±0.004
	0.10	2.	0.11±0.010	0.25	0.49±0.046	0.40	0.44±0.059
		3.	0.10±0.008		0.52±0.047		0.52±0.064
		1.	0.40±0.001		0.23±0.005		0.25±0.005
0.25	0.40	2.	0.55±0.017	0.10	0.21±0.020	0.25	0.28±0.075
		3.	0.37±0.009		0.25±0.023		0.33±0.080
		1.	0.25±0.001		0.25±0.005		0.25±0.004
	0.25	2.	0.30±0.017	0.40	0.22±0.022	0.10	0.37±0.077
		3.	0.23±0.009		0.26±0.026		0.41±0.096
		1.	0.10±0.001		0.25±0.004		0.23±0.005
	0.10	2.	0.10±0.009	0.25	0.21±0.036	0.40	0.23±0.048
		3.	0.09±0.007		0.23±0.038		0.27±0.032
		1.	0.40±0.001		0.10±0.006		0.09±0.005
0.10	0.40	2.	0.55±0.098	0.40	0.09±0.023	0.25	0.10±0.054
		3.	0.37±0.009		0.10±0.026		0.10±0.067
		1.	0.25±0.002		0.09±0.006		0.10±0.004
	0.25	2.	0.31±0.018	0.40	0.08±0.025	0.10	0.12±0.103
		3.	0.24±0.010		0.11±0.023		0.14±0.117
		1.	0.10±0.001		0.10±0.004		0.10±0.006
	0.10	2.	0.11±0.008	0.25	0.05±0.020	0.40	0.10±0.042
		3.	0.09±0.007		0.06±0.023		0.10±0.058

Estimates
1. Actual parameters of underlying distribution.
2. AoV of truncated population, with correction for continuity.
3. Modified proband method used on truncated population.

Table 6.1 Comparison of mean estimates of h^2 and rg obtained (together with standard errors) from 18-25 repetitions of simulation.

(c) Incidence of T1=0.005, T2=0.05, T3=0.02

rg	h^2_{T1}	ESTIMATE	h^2_{T1}	h^2_{T2}	rgT1T2	h^2_{T3}	rgT1T3
		1.	0.40±0.001		0.50±0.004		0.45±0.004
0.50	0.40	2.	0.59±0.048	0.25	0.47±0.029	0.10	0.44±0.059
		3.	0.33±0.018		0.56±0.034		0.52±0.058
		1.	0.25±0.001		0.52±0.003		0.50±0.004
	0.25	2.	0.35±0.034	0.10	0.48±0.066	0.40	0.53±0.060
		3.	0.23±0.017		0.53±0.069		0.59±0.058
		1.	0.10±0.001		0.45±0.004		0.52±0.003
	0.10	2.	0.10±0.016	0.40	0.54±0.075	0.25	0.67±0.110
		3.	0.05±0.019		0.63±0.086		0.73±0.106
		1.	0.40±0.001		0.25±0.005		0.23±0.005
0.25	0.40	2.	0.59±0.048	0.25	0.25±0.028	0.10	0.23±0.048
		3.	0.33±0.018		0.31±0.032		0.27±0.058
		1.	0.25±0.001		0.25±0.004		0.25±0.005
	0.25	2.	0.36±0.035	0.10	0.29±0.065	0.40	0.28±0.075
		3.	0.23±0.018		0.33±0.068		0.33±0.080
		1.	0.10±0.001		0.23±0.005		0.25±0.004
	0.10	2.	0.13±0.010	0.40	0.27±0.063	0.25	0.37±0.077
		3.	0.08±0.017		0.34±0.071		0.41±0.096
		1.	0.40±0.001		0.09±0.005		0.10±0.006
0.10	0.40	2.	0.59±0.048	0.25	0.10±0.026	0.10	0.10±0.042
		3.	0.33±0.018		0.13±0.034		0.10±0.058
		1.	0.25±0.002		0.10±0.004		0.09±0.005
	0.25	2.	0.36±0.031	0.10	0.12±0.038	0.40	0.10±0.054
		3.	0.22±0.021		0.14±0.046		0.10±0.067
		1.	0.10±0.001		0.10±0.006		0.10±0.004
	0.10	2.	0.13±0.025	0.40	0.15±0.067	0.25	0.12±0.103
		3.	0.05±0.019		0.18±0.079		0.14±0.117

Estimates

1. Actual parameters of underlying distribution.
2. AoV of truncated population, with correction for continuity.
3. Modified proband method used on truncated population.

method at the low incidences considered here. The estimates from the proband method usually agree better with the actual values than do those from Harvey, this being more applicable to an incidence of 0.05 than 0.005, and also when the correlation is 0.50 as opposed to 0.10. The level of heritability does not seem to be as important in determining the accuracy of the estimates.

Conversely, genetic correlations obtained by the proband method on the underlying scale are consistently higher than those on the 0-1 scale from Harvey. There does not appear to be much difference between the two methods in the degree to which they agree with the actual values. The level of agreement between methods, and with the actual value is generally quite good. As for the heritabilities, the estimates are not as consistent as the incidence becomes smaller and also as the true genetic correlation decreases. This can be seen by the relative magnitude of the standard errors relative to the size of the estimates. These findings support the conclusions of Olausson and Rönningen (1975), who said that the estimate made on 0-1 data are acceptable, given that the bias is decreasing as the heritability increases.

The conclusions from the simulation seem to be that where possible, heritability estimates should be taken from the proband analysis, and the genetic correlations from either method. In practice we would wish to include a continuous trait in the analysis, namely live-weight. Olausson and Rönningen (1975) also showed by simulation that genetic correlations between a continuous trait and a 0-1 trait will be almost the same as if both traits were estimated on an

underlying distribution, except at low heritability and low incidence. This, together with the fact that mixed-model packages are more flexible in their ability to correct the estimates for other factors, points to the use of analysis of variance techniques for the estimation of genetic correlations.

Analysis of Pedigree Data

The data used in the analyses presented here were kindly supplied by the breeding company (X), which provided the material and facilities described in Chapter 4. These consist of selection records from three pedigree male-line broiler strains. At any one time, nine mating groups of each selected line are contributing replacements, nine hatches being taken from each mating group at fortnightly intervals. Within each mating group birds are contemporaneous, but each mating group is two weeks older than the next. Chicks from any one hatch week are sent to the same rearing farm, and are therefore hatched from eggs laid at different times of the laying period. Birds are selected in the first instance on various growth and conformational traits at 6 weeks old. The leg abnormalities are described in Chapter 4, and are crooked toe (CT), bow (B), splay (S), twisted leg (TW), and tibial dyschondroplasia (TD). Two keel defects are also selected against. These are knobby-keel (KK), and twisted keel (TK) , and are self-explanatory defects noticeable in the breast, caused by abnormal ossification and alignment respectively. By this time a certain number of birds with acute leg problems will already have been culled. Data from 6 week old birds were collected by a trained selection team over a

period of thirteen months from 1979-1980, and do not include records from overlapping generations. Each line includes full-sib family information on approximately 30,000 progeny taken from over two hundred sires. (Details may be found for each line in Table 6.5.)

The defect traits have been considered in conjunction with 6 week live-weight (LWT), which is obviously the most important production trait. Although defect traits were scored as 0-1-2, ie not affected, mild and severe, these have been reclassified as 0 or 1, ie not affected or affected, as the inclusion of the extra category was seen to make no difference to the results (from analysis of variance). Very few birds were categorised as having severe defects. Thus the means of defect traits represent their incidences (p), and the variance can be expressed as a binomial p(1-p). Details of incidences along with means for LWT can be found in Table 6.2.

The phenotypic correlations between traits (scored as 0 or 1) were very small as can be seen from Table 6.3. The number of birds affected by more than one defect at a time was compared to an expected number using a 2x2 contingency table, in order to test the significance of these correlations. Chi-square values with probabilities less than 5% are indicated by asterisks in Table 6.3. Few of the phenotypic correlations were seen to be statistically significant. Negative correlations appeared to exist between CT and S, B and S, and CT and KK. A positive correlation was found between the two keel traits. It is possible that a certain amount of underscoring contributed to the lack of significance in these estimates, birds only being marked down on the worst of their

Table 6.2

(a) Means (= incidence in defect traits) and standard deviations of traits

Line 0

		LWT (g)	CT	B	S	TW	TD	KK	TK
M	X	1574.3	0.0557	0.0261	0.0342	0.0006	0.0021	0.0301	0.0079
	SD	192.5	0.2211	0.1573	0.1735	0.0226	0.0465	0.1683	0.0883
F	X	1312.9	0.0431	0.0188	0.0135	0.0008	0.0043	0.0029	0.0004
	SD	135.9	0.1949	0.1326	0.1132	0.0275	0.0649	0.0531	0.0179
M+F	X	1438.0	0.0492	0.0223	0.0234	0.0007	0.0033	0.0159	0.0040
	SD	212.5	0.2094	0.1461	0.1473	0.0261	0.0569	0.1243	0.0626

Line 1

		LWT (g)	CT	B	S	TW	TD	KK	TK
M	X	1717.8	0.0430	0.0203	0.0550	0.0006	0.0038	0.0346	0.0090
	SD	210.1	0.1981	0.1362	0.2217	0.0248	0.0604	0.1802	0.0937
F	X	1423.9	0.0259	0.0093	0.0220	0.0006	0.0013	0.0011	0.0011
	SD	152.9	0.1567	0.0957	0.1453	0.0235	0.0363	0.0325	0.0320
M+F	X	1564.3	0.0341	0.0146	0.0378	0.0006	0.0025	0.0171	0.0049
	SD	239.8	0.1798	0.1192	0.1890	0.0243	0.0497	0.1292	0.0697

Line 2

		LWT (g)	CT	B	S	TW	TD	KK	TK
M	X	1637.2	0.0619	0.0142	0.0408	0.0004	0.0048	0.0496	0.0123
	SD	191.0	0.2350	0.1169	0.1902	0.0205	0.0694	0.2156	0.1097
F	X	1352.7	0.0355	0.0127	0.0193	0.0015	0.0056	0.0025	0.0012
	SD	132.3	0.1811	0.1102	0.1338	0.0372	0.0733	0.0499	0.0345
M+F	X	1489.6	0.0482	0.0134	0.0297	0.0010	0.0052	0.0252	0.0065
	SD	219.0	0.2097	0.1140	0.1650	0.0304	0.0715	0.1563	0.0804

(b) Number of records examined

	LINE 0	LINE 1	LINE 2
M	13171	16930	18513
F	14349	18506	19975
M+F	27520	35436	38488

Table 6.3 Phenotypic correlations between traits (M+F)

Line 0

	LWT	CT	B	S	TW	TD	KK	TK
LWT	1.00							
CT	0.05	1.00						
B	0.04	0.01	1.00					
S	-0.03	-0.04***	-0.03***	1.00				
TW	-0.01	0.00	-0.01	-0.01	1.00			
TD	0.02	-0.02	-0.01	-0.02	0.00	1.00		
KK	0.01	-0.02**	-0.02***	-0.02	0.00	0.00	1.00	
TK	-0.01	-0.02*	0.00	-0.01	0.00	0.00	0.02***	1.00

Line 1

	LWT	CT	B	S	TW	TD	KK	TK
LWT	1.00							
CT	0.02	1.00						
B	0.02	-0.01	1.00					
S	-0.06	-0.02***	-0.03***	1.00				
TW	-0.01	0.00	0.00	-0.01	1.00			
TD	0.00	-0.01	0.00	-0.02	0.00	1.00		
KK	-0.02	-0.02*	-0.02	-0.03***	0.00	0.00	1.00	
TK	0.00	0.00	0.00	-0.01	0.00	-0.01	0.00	1.00

Line 2

	LWT	CT	B	S	TW	TD	KK	TK
LWT	1.00							
CT	0.06	1.00						
B	0.04	-0.01	1.00					
S	-0.04	-0.03***	-0.03***	1.00				
TW	-0.02	0.00	0.00	0.00	1.00			
TD	-0.01	-0.01	-0.01	-0.01	0.00	1.00		
KK	0.02	-0.03***	-0.01*	-0.02	0.01	0.03***	1.00	
TK	0.01	-0.01	0.00	0.00	0.00	-0.01	0.02***	1.00

1. * P<0.05, ** P<0.01, *** P<0.005
- all other correlations between defect traits are not significant.
Correlations between defect traits and live-weight were not tested.

defects. This precludes much comment on the correlations themselves. As previously mentioned, however, if a bird is affected by bow, it is unlikely to also have splay, these being physiologically 'opposite' conditions.

(a) Preliminary analyses

Initially, the four basic methods (ie analysis of variance, analysis of variance performed on arcsine transformed data (both corrected for continuity), together with the proband and modified proband methods, were used on all the defect data available, treating them as half-sib family summaries. This ignores the fact that some full-sibs are included within these families, and makes no correction for any of the factors such as sex or farm, which may affect the results; the purpose of the comparison being to test the methods. Standard errors were calculated as indicated in Table 6.4.

The results of these analyses are shown in Table 6.4. The estimates of heritability from the two analysis of variance techniques agree quite well with each other, as do those from the two proband methods. This is probably because the variances of the traits involved are small, and therefore the effect of removing differences in variance between groups will not be great. Generally, the analysis of variance methods gave higher estimates than did the proband methods, the two agreeing rather better when the incidence of the trait was higher (eg CT, Line 1 $p=0.034$). When the incidence

Table 6.4 Comparison of half-sib heritability estimates on the underlying scale - all records (M+F)

Line 0

	CT	B	S	TW	TD	KK	TK
1	0.65±0.07	0.41±0.06	0.60±0.08	0.95±0.45	0.38±0.14	0.31±0.06	0.18±0.10
2	0.54±0.06	0.41±0.06	0.55±0.07	-	0.15±0.12	0.37±0.07	0.17±0.10
3	0.39±0.03	0.25±0.04	0.30±0.04	-	0.13±0.07	0.16±0.04	-0.01±0.06
4	0.41	0.26	0.31	-	0.13	0.16	-0.01

Line 1

	CT	B	S	TW	TD	KK	TK
1	0.43±0.05	0.40±0.06	0.36±0.05	-0.26±0.33	0.52±0.15	0.28±0.05	0.05±0.07
2	0.39±0.05	0.50±0.07	0.40±0.05	-	0.02±0.11	0.45±0.06	0.27±0.09
3	0.35±0.03	0.27±0.04	0.27±0.03	0.19±0.10	0.24±0.06	0.25±0.04	0.04±0.05
5	0.36	0.28	0.28	0.20	0.25	0.26	0.04

Line 2

	CT	B	S	TW	TD	KK	TK
1	0.33±0.04	0.33±0.06	0.74±0.08	1.68±0.36	0.83±0.13	0.18±0.03	0.14±0.06
2	0.33±0.04	0.37±0.06	0.66±0.07	-1.09±0.11	0.65±0.11	0.26±0.04	0.29±0.07
3	0.23±0.03	0.20±0.04	0.41±0.03	0.47±0.08	0.56±0.05	0.15±0.03	0.16±0.05
4	0.24	0.20	0.43	0.49	0.59	0.16	0.17

1. AoV, h^2 adjusted to continuous scale.
2. AoV of arc-sin transformed data, h^2 adjusted to continuous scale.
3. Proband method.
4. Modified proband method.
- estimate unobtainable

No appropriate method was found to calculate the standard errors of estimates obtained using the modified proband method, but these will be approximately the same as for the proband method. Standard errors for the analysis of variance methods are those described by Hill and Smith (1977):

$$SE(t) = 1-t[1+(k-1)t].\sqrt{\frac{2(N-1)}{k(C-1)(N-C)}}$$

$$SE(rc) = SE(r01).\frac{p(1-p)}{z^2}$$

was very low, as for TW in all lines, estimates were either rather 'wild' or not obtainable.

(b) Full analyses

Following these findings, the data were subjected to various analyses in which correction for the effects of hatch week and, where applicable, sex, were made. Both the modified proband and analysis of variance methods were used for comparison. The proband data were corrected for hatch week simply by ignoring all records taken from the same hatch week for any one individual. This has the effect of correcting for a farm effect, thus eliminating a common environment component, and also corrects for the actual week and season of the year in which the hatch was taken.

In this way both full-sib and paternal half-sib family summaries were analysed, the latter excluding any records in a half-sib family of full-sib individuals. Sexes were considered separately and together.

A direct comparison could then be made with hierarchical analyses of variance performed by Harvey. Details of the Harvey models used and the expected mean-squares evolved after correction for fixed effects are given in Table 6.5. Data were corrected for hatch week, as above, and sex where applicable. In order to compensate for the fact that the variance in LWT amongst cockerels and pullets is somewhat different, log-transformed data were also analysed, but as the transformation made little difference to the estimates, the

Table 6.5

(a) Model used in Least Squares AoV (Harvey)

$$Y = u + a_i + b_{j(i)} + c_k + d_l + e_{ijklm}$$

where:

Y_{ijklm}	= independent variable
u	= mean
a_i	= ith sire (random)
$b_{j(i)}$	= jth dam nested within ith sire (random)
c_k	= kth hatch week (fixed)
d_l	= lth sex - included when M + F data present (fixed)
e_{ijklm}	= error term

(b) Expectations of mean squares (after adjusting for fixed effects) - sexes pooled

Line 0

EFFECT	df	EMS
Sire	211	$\sigma^2 + 24.43\sigma^2_D + 127.28\sigma^2_S$
Dams within Sires	1101	$\sigma^2 + 20.18\sigma^2_D$
Hatch Week	27	σ^2
Sex	1	
Residual	26179	

Line 1

EFFECT	df	EMS
Sire	257	$\sigma^2 + 23.70\sigma^2_D + 135.51\sigma^2_S$
Dams within Sires	1524	$\sigma^2 + 19.17\sigma^2_D$
Hatch Week	23	σ^2
Sex	1	
Residual	33629	

Line 2

EFFECT	df	EMS
Sire	246	$\sigma^2 + 27.45\sigma^2_D + 153.30\sigma^2_S$
Dams within Sires	1354	$\sigma^2 + 23.32\sigma^2_D$
Hatch Week	28	σ^2
Sex	1	
Residual	36858	

Table 6.6 A comparison of the estimates of h^2 (diagonal) and rg obtained using AoV and modified proband methods (M+F)

(a) Line 0

		CT	B	S	TW	TD	KK	TK
CT	1.	0.29						
	2.	0.42						
	3.	0.30						
	4.	0.50						
B	1.	-0.31	0.13					
	2.	0.07	0.28					
	3.	0.01	0.18					
	4.	0.16	0.29					
S	1.	-0.17	-0.29	0.14				
	2.	0.29	-0.21	0.16				
	3.	0.17	-	0.20				
	4.	0.07	-0.11	0.62				
TW	1.	-	-	-	-			
	2.	-	-	-	-			
	3.	-	-	-	-			
	4.	0.03	0.22	0.19	3.26			
TD	1.	-0.87	-	-	-	0.00		
	2.	-	-	-	-	-		
	3.	0.08	-	-	-	-		
	4.	-0.04	0.55	0.06	0.23	0.37		
KK	1.	-0.32	-0.83	-	-	-	0.08	
	2.	0.11	-0.10	0.32	-	-	0.24	
	3.	-0.21	-0.03	0.06	-	-	0.17	
	4.	-0.08	-0.03	0.07	-0.11	-0.21	0.27	
TK	1.	-	-	-	-	-	-	-
	2.	-0.61	-0.32	-0.20	-	-	0.20	0.03
	3.	-0.13	-0.32	-0.32	-	-	-	0.07
	4.	0.01	-0.23	0.18	0.18	-0.05	0.24	0.37

1. Proband - Paternal HS Analysis (excludes records from same FS family, and same hatch week)
2. AoV - Paternal HS Analysis (Harvey)
3. Proband - FS Analysis (excludes records from same hatch week)
4. AoV - FS Analysis (Harvey)

\- estimate unobtainable

Table 6.6 (b) Line 1

		CT	B	S	TW	TD	KK	TK
	1.	0.29						
CT	2.	0.29						
	3.	0.28						
	4.	0.35						
	1.	0.40	0.15					
B	2.	0.60	0.18					
	3.	0.35	0.24					
	4.	0.39	0.51					
	1.	0.39	0.27	0.20				
S	2.	0.28	-0.07	0.18				
	3.	0.33	0.08	0.22				
	4.	0.29	0.03	0.32				
	1.	-	-	-	-			
TW	2.	-	-	-	-			
	3.	-	-	-	-			
	4.	-	-	-	-			
	1.	-	-	-	-	-		
TD	2.	0.20	0.24	-0.03	-	0.13		
	3.	-0.88	0.29	0.29	-	0.06		
	4.	0.02	-0.01	0.22	-	0.96		
	1.	-0.02	-0.19	0.38	-	-	0.16	
KK	2.	-0.12	-0.30	0.25	-	0.35	0.20	
	3.	-0.32	0.04	0.04	-	0.03	0.26	
	4.	-0.18	-0.05	0.04	-	0.10	0.24	
	1.	-	-	-	-	-0.35	-	-
TK	2.	-	-	-	-	-	-	-
	3.	-0.33	-0.31	0.13	-	0.69	0.49	0.07
	4.	0.05	0.00	-0.02	-	0.10	0.14	0.80

1. Proband - Paternal HS Analysis (excludes records from same FS family, and same hatch week)
2. AoV - Paternal HS Analysis (Harvey)
3. Proband - FS Analysis (excludes records from same hatch week)
4. AoV - FS Analysis (Harvey)

\- estimate unobtainable

Table 6.6 (c) Line 2

		CT	B	S	TW	TD	KK	TK
CT	1.	0.14						
	2.	0.23						
	3.	0.23						
	4.	0.38						
B	1.	0.55	0.05					
	2.	0.46	0.12					
	3.	0.50	0.18					
	4.	0.36	0.27					
S	1.	0.26	0.42	0.29				
	2.	0.36	-0.08	0.30				
	3.	0.22	0.15	0.26				
	4.	0.16	-0.09	0.55				
TW	1.	0.30	0.64	0.48	0.46			
	2.	-	-	-	-			
	3.	-	-	-	-			
	4.	-0.03	-0.05	0.16	6.89			
TD	1.	0.22	-	-	0.28	0.21		
	2.	-0.04	-0.06	0.37	-	0.48		
	3.	-	0.86	0.58	-	0.15		
	4.	-0.04	0.37	-0.04	0.00	0.35		
KK	1.	-	-	-	-	0.21	0.09	
	2.	0.05	0.10	0.06	-	0.44	0.20	
	3.	-	-	-	-	0.27	0.07	
	4.	0.11	-0.09	0.14	0.24	0.43	0.10	
TK	1.	-	0.99	0.23	-	-0.13	0.75	0.07
	2.	-0.06	0.47	0.41	-	0.27	0.59	0.16
	3.	-	0.15	-0.30	-	0.05	0.19	0.16
	4.	0.11	0.01	-0.19	0.28	0.30	0.78	0.08

1. Proband - Paternal HS Analysis (excludes records from same FS family, and same hatch week)
2. AoV - Paternal HS Analysis (Harvey)
3. Proband - FS Analysis (excludes records from same hatch week)
4. AoV - FS Analysis (Harvey)

\- estimate unobtainable

results have not been shown here. Estimates for full-sibs (sire+dam component), and both paternal and maternal half- sibs (sire, and dam components respectively) were obtained; again both on all the data, and the two sexes considered separately.

A comparison of the full- sib and paternal half-sib results for sexes combined, from both Harvey and the modified proband method, is shown in Tables 6.6a,b,c. All estimates of heritability have been corrected for continuity. Full details of the results from each method are not presented. Overall means taken over males, females and males plus females, and also over full and half-sib estimates, to try and summarise the parameters are presented in Tables 6.7a,b,c, these tables including LWT. The summaries have been made partly to provide overall figures for further analyses, and partly to give an indication of the magnitude of the actual parameters.

Standard errors are given for the estimates presented in Tables 6.4 and 6.9 and obtained using Harvey, details of which may be found in the User's Guide (Harvey, 1975), and should be regarded as minimal values. No simple method of calculating standard errors for the modified proband method exists. As an approximation the standard errors of heritability estimates obtained by the proband method may be used. The standard deviations of the estimates obtained by analysis of the simulated populations (Table 6.1) are analagous to the standard errors of estimates made on the real data. A comparison of these standard deviations of repetitions with the standard errors for traits with similar incidence and heritability presented in Table 6.4 shows the two to be in good agreement. For example, CT in

Table 6.7 Comparison of overall means[1] of estimates of h^2 and rg obtained by AoV and modified proband methods respectively.

(a) Line 0

(1) AoV (h^2_{01})

	LWT	CT	B	S	TW	TD	KK	TK
LWT	0.31							
CT	0.08	0.13						
B	0.18	0.19	0.06					
S	0.13	0.08	-0.13	0.10				
TW	-0.02	-0.01	0.07	0.26	0.20			
TD	0.12	0.02	0.45	0.20	-0.01	0.04		
KK	0.08	-0.06	0.04	0.15	-0.13	-0.21	0.04	
TK	-0.11	-0.12	-0.31	0.08	0.03	-0.03	0.30	0.07

(2) Modified Proband (h^2_c)

	CT	B	S	TW	TD	KK	TK
CT	0.31						
B	-0.04	0.19					
S	0.08	-0.14	0.18				
TW	-	-	-	-			
TD	-0.36	-	0.81	-	0.05		
KK	-0.39	-0.19	-0.23	-	-	0.25	
TK	0.13	-0.32	-0.26	-	-0.46	-	0.07

1. Means were taken over sire, dam and sire plus dam component estimates for males, females and sexes pooled for the analysis of variance; and over full-sib and paternal half-sib estimates for males, females and sexes pooled for the proband method.

- estimate unobtainable

Table 6.7 (continued)

(b) Line 1

(1) AoV ($\hat{h}^2_{01}$)

	LWT	CT	B	S	TW	TD	KK	TK
LWT	0.32							
CT	0.17	0.06						
B	0.32	0.49	0.06					
S	0.07	0.31	0.03	0.07				
TW	-0.07	0.04	0.30	-0.01	0.25			
TD	0.15	0.06	0.07	0.13	-	0.07		
KK	-0.06	0.17	-0.14	0.16	-0.03	0.17	0.05	
TK	0.01	0.08	-0.03	-0.10	0.00	0.04	0.11	0.11

(2) Modified Proband ($\hat{h}^2_c$)

	CT	B	S	TW	TD	KK	TK
CT	0.30						
B	0.34	0.20					
S	0.36	0.25	0.22				
TW	-	-	-	-			
TD	-0.84	-0.75	0.08	-	0.09		
KK	-0.18	-0.20	0.30	-	0.07	0.27	
TK	-0.56	-0.31	-0.01	-	0.14	0.48	0.20

(c) Line 2

(1) AoV ($\hat{h}^2_{01}$)

	LWT	CT	B	S	TW	TD	KK	TK
LWT	0.26							
CT	0.24	0.09						
B	0.20	0.36	0.03					
S	-0.01	0.19	-0.09	0.10				
TW	-0.17	0.08	-0.01	0.32	0.08			
TD	0.04	-0.07	0.29	-0.06	0.01	0.03		
KK	0.21	-0.02	0.03	-0.02	-0.08	0.24	0.02	
TK	0.09	0.05	0.21	-0.03	0.18	0.14	0.43	0.01

(2) Modified Proband ($\hat{h}^2_c$)

	CT	B	S	TW	TD	KK	TK
CT	0.19						
B	0.42	0.17					
S	0.33	0.34	0.28				
TW	0.11	0.55	0.55	0.46			
TD	-0.11	0.78	0.49	0.52	0.20		
KK	0.65	-0.18	-0.52	-	0.16	0.18	
TK	0.07	0.11	-0.10	-	0.11	0.50	0.14

Line 0 has an incidence of 0.049 and heritability of 0.39±0.03, as calculated by the proband method. The standard error of the mean of repetitions calculated for a trait with a heritability of 0.40 and incidence of 0.05 is 0.008. The standard deviation of repetitions is therefore 0.04, which is similar to the standard error of the estimate made on real data. Therefore values from either of these tables may be taken as a rough guide to the magnitude of standard errors of estimates given in the present section. Standard errors calculated by Harvey seem to be quite reliable.

The data analysed have been taken from a pre-selected population. Therefore the estimates of heritability and genetic correlations obtained will be different from those which originally applied within these strains. Similarly, these estimates can be expected to have altered marginally after the next generation of selection. As might be expected, the heritability estimates obtained using the proband method are lower than those produced by analysis of variance in most cases. The relative magnitude of estimates agrees quite well, however. Where the incidence of the a trait is quite low, as for TW, or TK in pullets, 'nonsense' estimates have often appeared. It is possible that the exclusion of data from the proband analyses has had an effect on the estimates both of heritability and genetic correlations. Thus estimates are not obtainable in a number of cases, and certain correlations are somewhat higher than expected (eg Line 0, rg B,KK, half-sibs).

LWT, as expected, has a high heritability (0.26-0.32). Taking the defect traits as analysed by the proband method, the heritabilities

of certain traits, ie CT, B, S and KK all produce mean estimates in the range of 0.17-0.31. It is noticeable that these defects are those with the higher incidences. Of the other traits, the mean heritability estimates for TK are somewhat lower and more variable (0.07-0.20), this trait having a slightly lower incidence ($p<0.01$). TD also has a somewhat varying set of estimates, and this may be for the same reasons as just stated. Genetic correlations of traits with TD also tended to be inconsistent, however. It is strongly suspected that what is actually being selected as TD bears little relationship to the actual pathological condition. Diagnosis often depends on palpation of the stifle joint, and what may be being recorded as TD could actually be only natural variation in the size of the tibio-femoral condyles (see Chapter 2). This may lead to the fact that the estimates of TD in these analyses are of little value! For comparison, Sheridan et al (1978) obtained estimates of heritability of around 0.50 for TD assessed by dissection. Very few estimates of heritability for TW were obtained, again probably because this is a very low incidence trait ($p<0.001$). The mean estimate in Line 2 of 0.46 compares favourably with those obtained by Hartmann (1978) of 0.40 in cockerels and 0.51 in pullets.

Without exception, dam component estimates of heritability for leg abnormalities obtained using Harvey were larger than their corresponding sire component estimates (Table 6.8). This was also the case for live-weight, except in pullets in Line 0, and for the keel defects in Lines 0 and 1. These results suggest the existence of maternal or dominance effects and indicate that the incidence of defect traits

Table 6.8 Comparison of sire, dam and sire plus dam component estimates of h^2_c obtained using AoV

Line 0

		LWT	CT	B	S	TW	TD	KK	TK
	S	0.28	0.38	0.09	0.23	-	-	0.26	0.20
M	D	0.31	0.78	0.43	1.47	94.53	-	0.48	0.23
	S+D	0.28	0.58	0.26	0.85	47.26	-	0.37	0.22
	S	0.38	0.56	0.47	0.03	-	-	1.28	-
F	D	0.34	0.73	1.11	1.50	5.21	2.09	-	66.05
	S+D	0.36	0.65	0.79	0.77	2.55	1.05	0.66	32.93
	S	0.26	0.42	0.24	0.16	-	-	0.24	0.03
M+F	D	0.30	0.58	0.34	1.07	6.42	0.73	0.30	0.68
	S+D	0.28	0.50	0.29	0.62	3.21	0.36	0.27	0.37

Line 1

		LWT	CT	B	S	TW	TD	KK	TK
	S	0.27	0.27	0.29	0.26	-	-	0.26	-
M	D	0.35	0.61	1.75	0.50	-	3.93	0.34	0.97
	S+D	0.31	0.44	1.02	0.38	-	1.95	0.30	0.48
	S	0.30	0.29	0.22	0.13	-	-	-	-
F	D	0.39	0.33	0.04	0.38	45.10	-	8.71	35.47
	S+D	0.35	0.31	0.13	0.26	22.48	-	4.93	17.74
	S	0.25	0.29	0.18	0.18	-	0.12	0.20	-
M+F	D	0.34	0.42	0.83	0.45	-	1.78	0.30	1.60
	S+D	0.30	0.35	0.51	0.32	-	0.95	0.24	0.80

Line 2

		LWT	CT	B	S	TW	TD	KK	TK
	S	0.19	0.29	0.19	0.37	-	1.33	0.25	0.19
M	D	0.33	0.50	0.32	0.89	22.78	-	0.03	0.18
	S+D	0.26	0.39	0.26	0.63	11.30	0.66	0.14	0.18
	S	0.23	0.23	0.13	0.26	0.38	0.02	0.79	0.00
F	D	0.33	0.75	1.03	1.37	11.01	1.70	-	1.82
	S+D	0.28	0.49	0.58	0.82	3.71	0.85	0.37	0.91
	S	0.18	0.23	0.12	0.30	-	0.48	0.20	0.16
M+F	D	0.30	0.53	0.41	0.79	13.22	0.18	-	0.00
	S+D	0.24	0.38	0.27	0.55	6.61	0.35	0.10	0.08

- estimate unobtainable

may be altered by manipulation of the environment in which the hens are kept.

At first glance the genetic correlations obtained by the proband method seem to bear little relationship to those from analysis of variance, particularly in Line 0. The disagreement shown by the methods may be a reflection of the ways in which the data were corrected for the effects of hatch week and sex, particularly in the exclusion of records for analysis using the proband method. It is possible, however, to pick out certain 'trends' amongst the estimates. On consideration of the mean estimates, the leg weaknesses seem to be positively correlated with each other, with the notable exception of B and S in Lines 1 and 2. A comparison of the estimates from the two methods for CT and S, and of CT and B in Lines 1 and 2, respectively point to quite substantial positive genetic correlations of about 0.30 in the first case, and 0.40 in the second. These estimates again are ones that have been made of correlations between relatively high incidence traits; for reasons already outlined for the estimates of heritability, the correlations of traits with TW and TD are inconsistent.

Where estimates were obtainable, the correlation between the two keel traits was consistently quite strongly positive. Correlations with other traits are inconsistent, though there seems to be generally negative correlations with the leg weaknesses, as shown particularly between B and TK in Line 0, and B and KK in Line 1.

Genetic correlations between the defect traits and live-weight were

only estimated using Harvey, although it would be feasible to hypothesise a truncation point above which birds would be selected, and analyse the population as a 0-1 trait; treating it exactly the same as for the defects. With the exception of TW, all the defect traits were positively correlated with body-weight overall, in at least two of the three male-lines. The correlations were often quite significant, as in the case of CT, B, and KK in Line 2, all three being greater than 0.20.

(c) Analysis of pooled data

As an additional analysis, defects were considered together, to investigate whether or not it is justifiable to estimate parameters for leg problems or keel problems as a whole. All leg defects were therefore grouped into one trait, with the exception of TD, and a second trait was composed of the two keel defects. Both full-sib and half-sib analyses using Harvey and the modified proband method were carried out on male and female data grouped together as before. Results are shown in Table 6.9 and 6.10, together with incidences.

The prevalence of leg and keel problems seems to be much the same over all three lines, the overall incidence of leg abnormalities being high (p=0.09). Cocks have more defects than pullets, few females having keel abnormalities. Results suggest that both leg problems and keel problems are heritable, the former having a higher heritability (about 0.25) than the latter (about 0.18). Keel defects had a lower heritability in Line 2. A quite substantial positive

Table 6.9 Sire, dam and sire plus dam component estimates of rg and h^2_c (diagonal) for leg (LP) and keel (KP) problems from M + F data - AoV

Line 0

		LWT	LP	KP
	S	0.26±0.03		
LWT	D	0.30±0.02		
	S+D	0.28±0.02		
	S	0.24±0.08	0.22±0.03	
LP	D	0.13±0.06	0.38±0.04	
	S+D	0.18±0.05	0.30±0.02	
	S	-0.14±0.12	0.05±0.13	0.17±0.04
KP	D	0.17±0.10	-0.06±0.13	0.30±0.08
	S+D	0.04±0.07	-0.02±0.08	0.24±0.03

Line 1

		LWT	LP	KP
	S	0.25±0.02		
LWT	D	0.34±0.02		
	S+D	0.29±0.01		
	S	0.33±0.07	0.19±0.03	
LP	D	0.14±0.05	0.36±0.03	
	S+D	0.21±0.05	0.28±0.02	
	S	-0.03±0.11	0.10±0.13	0.13±0.03
KP	D	-0.06±0.08	-0.15±0.11	0.32±0.06
	S+D	-0.05±0.06	-0.07±0.07	0.22±0.03

Line 2

		LWT	LP	KP
	S	0.18±0.02		
LWT	D	0.30±0.02		
	S+D	0.24±0.01		
	S	0.33±0.07	0.20±0.03	
LP	D	0.11±0.05	0.39±0.03	
	S+D	0.19±0.05	0.30±0.02	
	S	0.14±0.09	0.12±0.10	0.18±0.03
KP	D	-	-	-
	S+D	0.21±0.08	0.14±0.09	0.09±0.02

Table 6.10

(a) Paternal HS and FS estimates of rg and h^2 for leg (LP) and keel (KP) problems from M + F data analysed by the modified proband method

		LINE 0		LINE 1		LINE 2	
		LP	KP	LP	KP	LP	KP
LP	HS	0.09		0.21		0.15	
	FS	0.18		0.22		0.20	
KP	HS	-1.16	0.06	0.17	0.11	-	0.09
	FS	-0.12	0.18	-0.16	0.21	-	0.06

(b) Incidences of leg and keel problems

	SEX	LINE 0	LINE 1	LINE 2
LP	M	0.115	0.117	0.116
	F	0.074	0.057	0.068
	M+F	0.094	0.086	0.091
KP	M	0.037	0.043	0.061
	F	0.003	0.002	0.004
	M+F	0.020	0.022	0.031

genetic correlation appears to exist between LWT and leg problems of about 0.20, there being positive correlations between LWT and keel problems in Lines 0 and 2, but not Line 1. The genetic correlation between the two defect traits is negative in Lines 0 and 1, but positive in Line 2. Where a comparison can be made, the two methods produce similar results.

The estimates presented in this section clarify the findings for individual traits. Leg problems considered as a whole, therefore, are unfavourably correlated with LWT. It is important that 'grouped' problems are heritable. This obviates a need for breeders to consider selection on individual traits. Instead they may merely cull birds for having bad legs, or poor keels, and by doing this will increase the selection pressure against these defects.

Obviously, the results presented here have important implications for the farmer and the breeder. With the possible exception of TK, and leaving aside the rather suspect TD, all the defects appear to have a high component of heritable variation on the underlying scale. This is almost as much as that for live-weight at six weeks, particularly in the case of CT. When taken with the fact that the genetic correlations between the defects and body-weight are positive (ie unfavourable), this suggests that selection on body-weight alone is likely to increase the incidence of these defects. This point, basically, is the subject of the next chapter. The negative correlation between body-weight and TW is consistent

over all three lines, and suggests that birds more susceptible to twisted leg are likely to be lighter than their contemporaries. Twisted leg is a fairly serious condition, in that the manifestations are extreme. Possibly the general condition of birds which are prone to the large physiological disturbance which must take place to cause abnormal growth of this extremity, is poorer than in others, this leading to a smaller size. Conversely, the other leg weaknesses require only relatively minor disruption of the ossification process during development for observable changes to take place. Thus in faster growing birds (ie those which are heavier at a given age), weight gain may be taking place more rapidly than skeletal growth. This would lead to abnormal stresses on the frame, and a tendency to develop skeletal defects, particularly of the legs. The same conclusion has also been suggested by Poulos et al (1978), and is supported by the genetic correlations found.

If birds which are heavier are more susceptible to leg weaknesses, it is not surprising to find that positive correlations exist between the various different leg conditions, so that birds from a line susceptible to, for example, crooked toes, are also likely to develop splay. A negative correlation does appear to exist between splay and bow. As these are in fact 'opposite' conditions, in that they represent different directions in which growth of the shank may deviate from that in a normal bird, it is likely that susceptibility in a line to one may make it unlikely that birds will develop the other. As there is some uncertainty still about the relationship between keel and leg defects it is difficult to comment on cause and effect. If negative correlations between the two do exist, then it

is possible that rapid growth is affecting the physiological development of birds in different ways, the one altering the shape of the breast, and the other the shape of the legs. It is possible to merely consider 'leg' or 'keel' problems as a whole for the purposes of selection.

In conclusion, it can be said that the analysis of variance and proband methods considered do seem to produce reasonable estimates of genetic correlations and heritability. The lower the incidence of the defect, the more unreliable the estimates become. However, in commercial practice, defects with incidences less than 1.0%, with a loss of birds rather less than that, may not concern the breeder too much. It would seem sensible to use estimates of heritability from the proband method, as these are less biassed, and genetic correlations from an analysis of variance, which can easily include correlations with continuously distributed traits proper. Unfavourable correlations between desired and undesired traits are a reason for worry, however, and deserve further consideration.

CHAPTER 7. IMPLICATIONS OF THE GENETICS OF SKELETAL DEFECTS IN SELECTION PROGRAMMES FOR BODY WEIGHT

Inroduction

Given the fact that skeletal defects appear to be inherited characters, and that unfavourable genetic correlations exist between these and a desired trait, several questions need to be asked about the effects of selection on defect traits. Obviously, in a commercial breeding programme, the object would be to improve body weights and reduce the incidence of abnormalities. Present selection programmes act effectively on two independent culling levels. Individual birds affected by leg defects are selected out of the population, and then the remainder assessed on weight and conformation. Varying degrees of family information are taken into consideration in both cases. The important considerations here must be whether selection on body weight increases the incidence of defects to any great extent; what effect the converse has on final weights; what are the effects of family selection; and what form of selection programme will optimise the breeders' aims?

A major problem arises in that the standard prediction equations have been derived to deal with normally distributed data, and do not apply to all-or-none traits. Two possibilities for the analysis of such data exist. Continuous parameters calculated on the underlying scale of liability may be used in conjunction with existing

prediction equations. This requires a transformation of the variance of the defect traits, as these are binomially distributed. As concluded in the previous chapter, however, it is easier to produce unbiassed estimates of genetic correlations using analysis of variance techniques. It would seem sensible, therefore, to use these estimates, made on the 0-1 scale, for prediction of selection responses, if possible. Unfortunately, little work has been published on the application of 0-1 estimates to the prediction of selection responses. The principle of selection response in a binomially distributed trait may be found in Turner and Young (1969). This has been extended by Smith (1980, unpublished) to include family selection.

Prediction of selection responses

The response of a trait to selection may be calculated as $R = h^2S$, where S is the selection differential, and for normally distributed traits would be equal to the selection intensity multiplied by the phenotypic standard deviation. In selection against a defect trait, all affected birds will be culled out, and visibly unaffected birds left. This is equivalent to the selection differential (S). If unaffected birds are assigned a value of 0, and affected birds a value of 1, then in any group selected, whether this is a family, or the population as a whole, this will mean that S is equal to the incidence of this group (p). Thus the response is:

$$R = -ph^2 01$$

where h^2 is calculated on the 0-1 scale.

Similarly, the correlated response (CR) of a character to selection on another may be expressed as:

$$CRy = b(A)yx.Rx$$

where x and y are different traits and b(A)yx is the regression of the breeding value of y on that of x. If the selection response of x is calculated on the 0-1 scale, then the correlated response becomes:

$$CRy = -ph^2x.ra\frac{\sigma Ay}{\sigma Ax}$$

$$= -phxhy.ra\frac{\sigma py}{\sigma px}$$

where ra is the genetic correlation between x and y, and $\sigma^2 p$ is the phenotypic variance.

The above formulae apply to mass selection, and may easily be extended to include family selection. The heritability of family means is:

$$h^2 f = \frac{1+(n-1)r}{1+(n-1)t} h^2$$

where r is the degree of relationship, and t is the correlation of phenotypic values of members of the families (here taken as $t = rh^2$ (Falconer, 1981). This leads to the family response of:

$$Rfx = \frac{-px.COV(Ax,\bar{P}x)}{\sigma^2\bar{p}} \qquad Rfx = -px.h^2x.\left(\frac{1+(n-1)r}{1+(n-1)t}\right)$$

$$CR_y = -px.\frac{COV(A_y,\bar{P}x)}{\sigma^2\bar{p}_x}$$

$$= -px.ra.hx.hy.\frac{\sigma py}{\sigma px}\left(\frac{1+(n-1)r}{1+(n-1)tx}\right)$$

The incidence (p) may only be used as the selection differential in family selection in cases where all families with affected individuals are selected. The change in incidence (Δp) caused by the

removal of a proportion of affected individuals should be used.

Before proceeding to apply these prediction equations to actual data, the accuracy of the predictions was examined using computer simulation. Hypothetical populations were simulated exactly as in the previous chapter, with a continuous distribution and known parameters for both a defect trait (h^2 =0.13), and live-weight (h^2 =0.23), with a genetic correlation between the two of 0.20. Each record in the data was given both a phenotype and breeding value for each trait. Individual defect traits were then assigned a phenotypic value of 0 or 1 by truncating the population at a point where the normal deviate corresponded to the desired incidence. The populations were then subjected to artificial selection, and the mean breeding value of the selected individuals calculated. For 0-1 traits the actual response (ie the change in mean breeding value) could be computed by considering the change in the threshold points, T:

T1 (after seln.) = T0 (before seln.) - R (observed change in b.v.)

As each of these thresholds corresponded to an incidence, the response in terms of incidence, which is the value computed by the 0-1 prediction equation, is the difference in incidences (p1-p0) corresponding to (T1-T0). Simulation was repeated at incidence levels of 1.0%, 5.0%, 10.0%, and 50.0%, for cases of mass selection, and both full and half-sib family selection. The hypothetical trait 'live-weight' was given a mean of 1500g and standard deviation of 250g. In instances of family selection, family means for breeding values and phenotype were calculated, and then families ranked on phenotype, which was on a 0-1 scale for the defect trait. As the

same selection intensities were used for family selection as for mass selection, the selection differential was not equal to the incidence of the defect trait, but rather to the phenotypic change in incidence for that trait. This is because not all affected individuals would be expected to be picked out in family selection.

The results of the simulation can be seen in Table 1. Expected responses and correlated responses to selection on live-weight were calculated using the prediction equations for normally distributed traits:

$$Rx = ih^2\sigma p \qquad CRy = ixhxhyra\sigma py$$

$$Rfx = ih^2\sigma p.\left(\frac{1+(n-1)r}{\sqrt{n(1+(n-1)t)}}\right) \qquad CRfy = ixhxhyra\sigma py.\left(\frac{1+(n-1)rx}{\sqrt{n(1+(n-1)tx)}}\right)$$

The direct response to live-weight represents a check as to whether the simulation was working. The closeness of expected to observed responses shows that this was in fact the case. Responses to selection against defects agreed very well with those predicted, mass selection producing better results than full-sib family selection, which proved more accurate in turn than half-sib family selection. The correlated responses in the continuous trait followed the same pattern, but were not quite as close as the direct responses to the expected values. Correlated responses in live-weight were very small. Observed responses in defects to selection on live-weight again agreed very well with predicted responses, mass selection and half-sib family selection slightly better than full-sib family selection when the selection intensity

Table 7.1

(a) Comparison of predicted responses (expected) and actual simulated responses (observed) to selection against a defect trait with variable incidence (P) and h^2=0.13, and correlated responses in a continuous trait with a mean of 1500g, h^2=0.23, rg=0.20, ns=150, nd=6, np=24[1]

TYPE OF SELECTION	%I (=P)	E[R] %P	O[R] %P	E[CR] g	O[CR] g
MASS	1.0	-0.01	-0.01	-0.05	-0.05
	5.0	-0.15	-0.13	-0.21	-0.22
	10.0	-0.44	-0.42	-0.36	-0.39
	50.0	-4.14	-4.00	-1.47	-1.52
FULL-SIB FAMILY	1.0	-0.01	-0.01	-0.06	-0.03
	5.0	-0.20	-0.16	-0.28	-0.18
	10.0	-0.63	-0.51	-0.51	-0.40
	50.0	-6.08	-5.58	-2.16	-1.64
HALF-SIB FAMILY	1.0	-0.02	-0.02	-0.12	+0.08
	5.0	-0.31	-0.17	-0.42	-0.19
	10.0	-0.63	-0.52	-0.51	-0.33
	50.0	-5.55	-5.07	-1.97	-1.38

(b) Simulated correlated responses in defects with variable incidence to selection on a continuous trait as above.

TYPE OF SELECTION	%I (=P)	TOP 10.0% SELECTED E[CR] %P	TOP 10.0% SELECTED O[CR] %P	TOP 50.0% SELECTED E[CR] %P	TOP 50.0% SELECTED O[CR] %P
MASS	1.0	+0.17	+0.16	+0.08	+0.08
	5.0	+0.67	+0.62	+0.31	+0.31
	10.0	+1.14	+1.03	+0.52	+0.51
	50.0	+2.56	+2.28	+1.17	+1.16
FULL-SIB FAMILY	1.0	+0.24	+0.34	+0.11	+0.11
	5.0	+0.92	+1.30	+0.42	+0.46
	10.0	+1.55	+2.13	+0.71	+0.76
	50.0	+3.51	+4.55	+1.61	+1.64
HALF-SIB FAMILY	1.0	+0.18	+0.17	+0.08	+0.13
	5.0	+0.70	+0.68	+0.32	+0.50
	10.0	+1.18	+1.12	+0.54	+0.84
	50.0	+2.66	+2.47	+1.22	+1.87

(c) Simulated response to selection on a continuous trait as above.

TYPE OF SELECTION	E[CR] g	O[CR] g
MASS	18.95	18.80
FS FAMILY	26.35	28.78
HS FAMILY	19.66	19.82

1. ns = no. of sires, nd = no. of dams, np = no. of progeny

was high (the top 10% being selected). In contrast, mass selection and full-sib family selection agreed better than half-sib family selection when the selection intensity was lower. These discrepancies may be partly attributable to sampling variance. As a general rule, the magnitude of incidence in the defect did not influence the magnitude of deviance from the expected response.

These results indicate that the prediction equations on the 0-1 scale may be used with some confidence to determine selection responses. The next section, therefore, concerns some of the properties of the responses obtained using these methods.

Application of Prediction Equations

Various graphs were plotted by taking hypothetical values for parameters and looking at the selection responses predicted by the equations above. Results are expressed as a percentage of the mean incidence of the trait under consideration. Heritabilties and correlations are on the all-or-none scale.

Figure 7.1 shows the effect of direct selection. Under mass selection defects show a linear response with increasing heritability (exponential in the case of log h^2). The direct response to all forms of selection is proportionally the same, whatever the incidence. Family size does not seem to have much effect on the magnitude of the response, which is curvilinear when plotted against log h^2. The response is slightly greater at lower heritabilities in the case of half-sib family selection than with

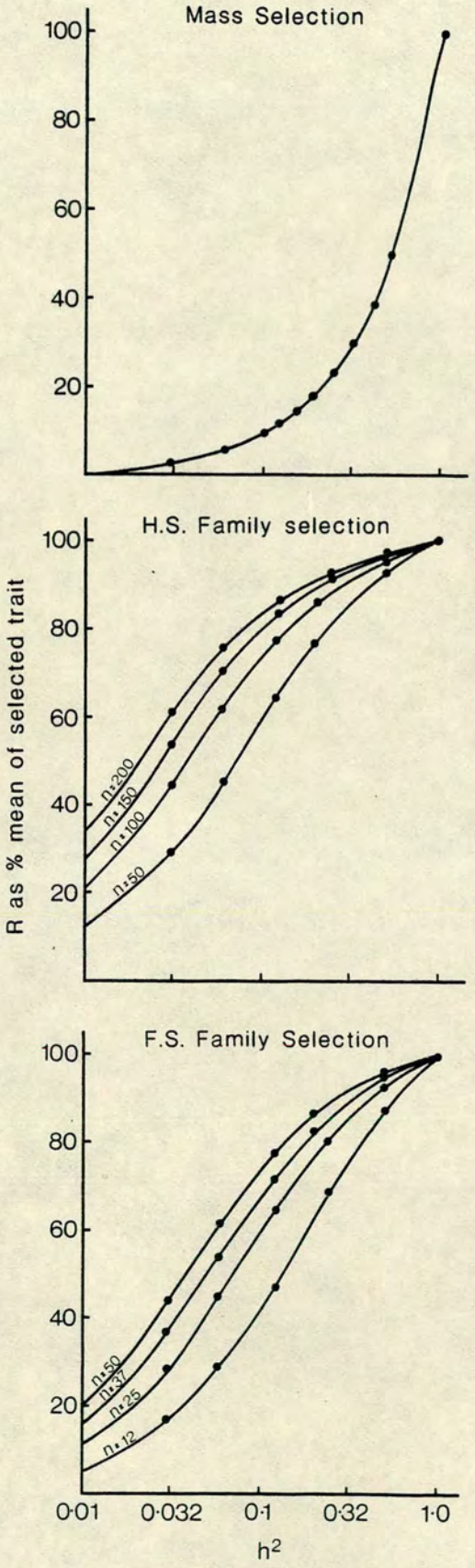


Fig. 7.1 Effect of size of h^2 on the selection response in 0-1 distributed traits. (Selection assumes that all families with affected individuals are chosen)

full-sib family selection. In all cases where family selection is applied to defects in this section, it is assumed that every family with affected individuals is selected.

Correlated responses are constrained by the initial values of parameters in both the selected and correlated traits. A simulation study was carried out to examine the correlated reponses in defect traits to selection upon both other defect traits and live-weight. A series of incidences covering broadly the range encountered in the present study were chosen. In each case the genetic correlation merely scales the magnitude of the correlated response in direct proportion to its incidence. Correlated responses to mass selection were very small, and therefore not presented here.

Family size plays very little part in determining the correlated response to selection on a continuous trait, except in cases where the families are very small. It has more effect if selection is upon another defect trait (Figure 7.2). At the family sizes considered in the present study, the responses are quite high; selection on live-weight causing a greater correlated response than selection upon a defect trait of typical incidence ($p=0.02$). A greater proportional response is obtained in traits with low incidences.

The effect of size of heritability in a correlated trait is shown in Figure 7.3, which demonstrates a simple square-root relationship of response with increasing heritability. If selection is on live-weight then greater correlated responses may be expected from using full-sib family selection than mass selection, which in turn

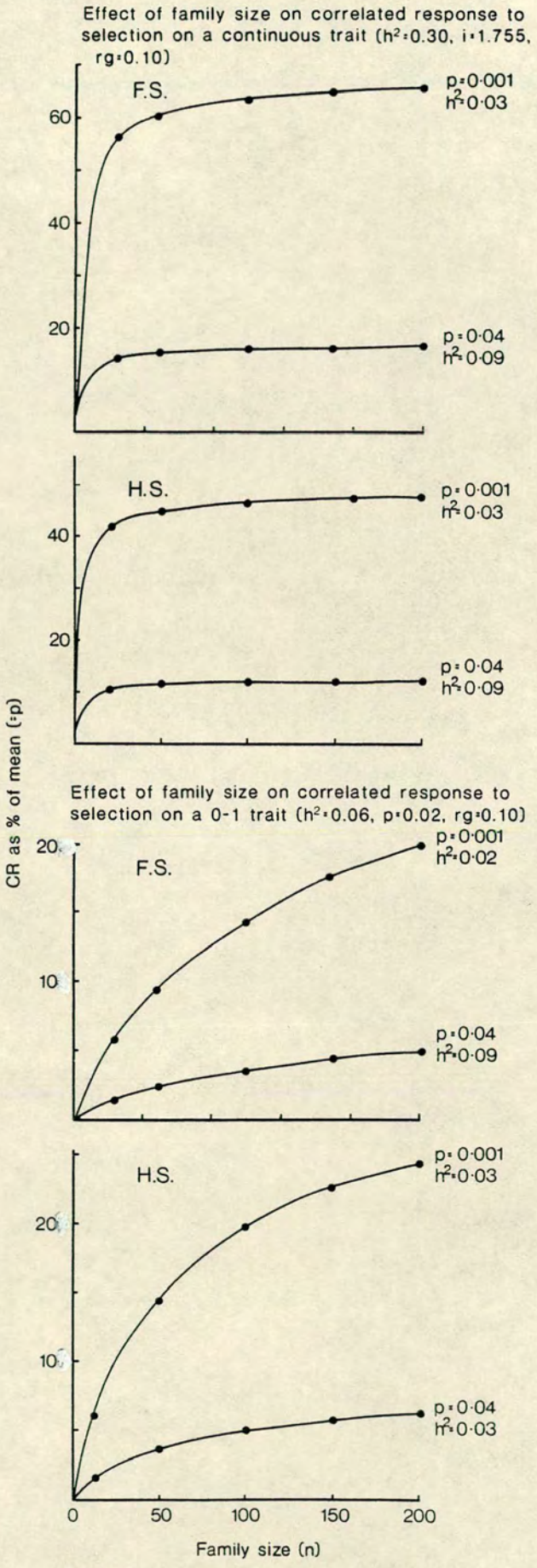
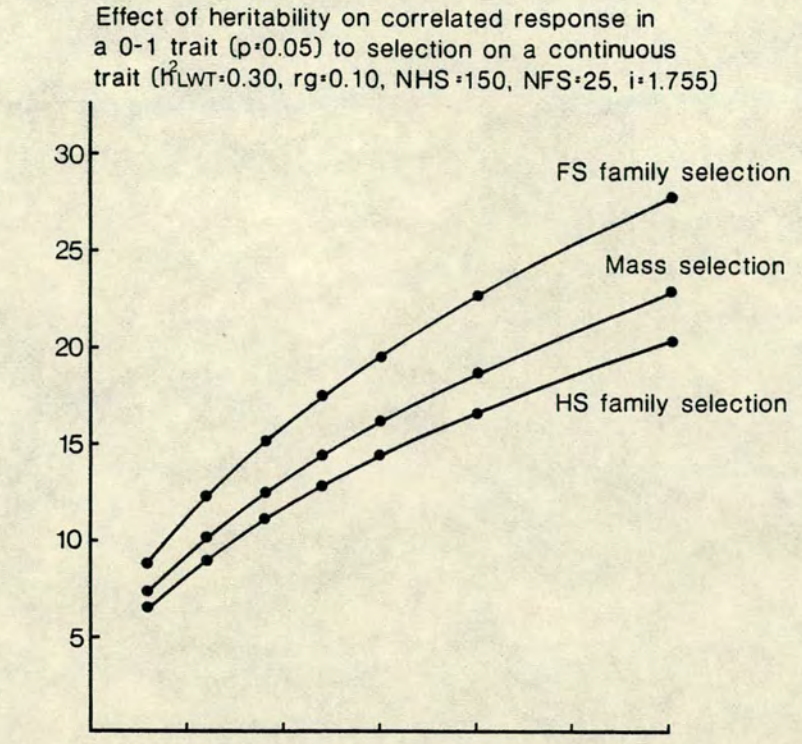

Effect of family size on correlated response to selection on a continuous trait (h^2=0.30, i=1.755, rg=0.10)
F.S.
p=0·001
h^2=0·03
p=0·04
h^2=0·09
H.S.
p=0·001
h^2=0·03
p=0·04
h^2=0·09
CR as % of mean (=p)
60
40
20
40
20
Effect of family size on correlated response to selection on a 0-1 trait (h^2=0.06, p=0.02, rg=0.10)
F.S.
p=0·001
h^2=0·02
p=0·04
h^2=0·09
H.S.
p=0·001
h^2=0·03
p=0·04
h^2=0·03
20
10
20
10
0
50
100
150
200
Family size (n)

Fig. 7.2


Effect of heritability on correlated response in a 0-1 trait (p=0.05) to selection on a continuous trait (h^2_{LWT}=0.30, rg=0.10, NHS=150, NFS=25, i=1.755)
30
25
20
15
10
5
FS family selection
Mass selection
HS family selection

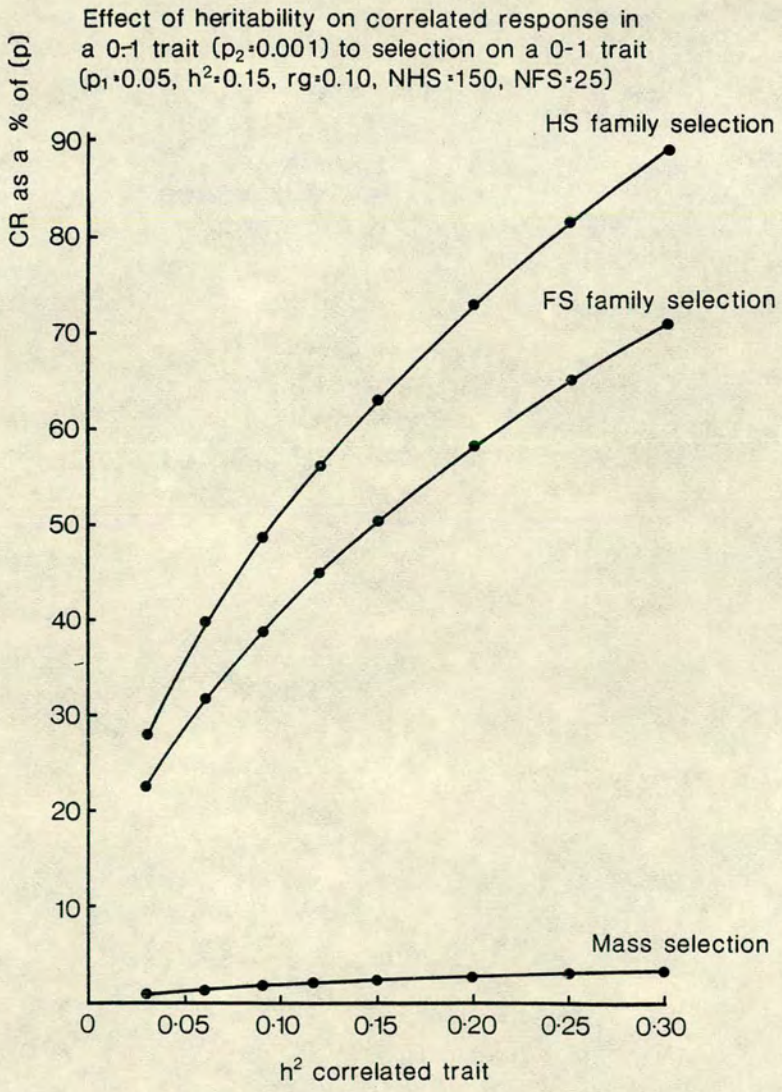
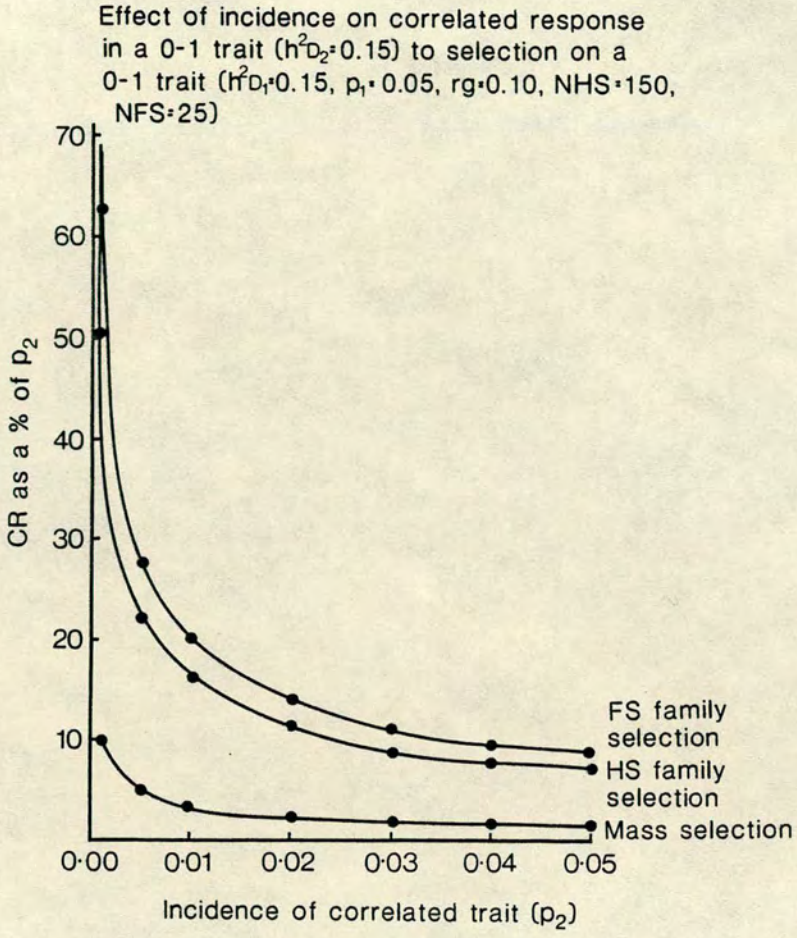

Effect of heritability on correlated response in a 0-1 trait (p_2=0.001) to selection on a 0-1 trait (p_1=0.05, h^2=0.15, rg=0.10, NHS=150, NFS=25)
CR as a % of (p)
90
80
70
60
50
40
30
20
10
0
0·05
0·10
0·15
0·20
0·25
0·30
h^2 correlated trait
HS family selection
FS family selection
Mass selection

Fig. 7.3

will produce greater responses than half-sib family selection. If selection is upon a defect trait, half-sib family selection gives a greater correlated response than full-sib family selection, which bears the same relationship to mass selection as above. A low incidence trait has been used for the purposes of illustrating the magnitude of response in the case of selection on a defect trait.

The above graphs have shown that correlated responses are proportionally dependent on the initial frequency of the trait concerned. This is seen to be the case whether selection is performed on either a continuous or an all-or-none trait (Figure 7.4). The relative magnitude of the responses in correlated traits with varying incidence to different forms of selection are the same as described for Figure 7.3.

To summarise, large correlated responses may be seen as a result of selection on live-weight, these being substantial in terms of actual changes in overall incidence, and dependent on the size (and sign) of the genetic correlation between the traits. Proportional changes in either cases of direct selection upon one defect trait, or a correlated response to selection on another, are generally smaller, as the selection intensities involved are limited by the incidence of the selected trait. For the same reason the effects of selection on a defect trait upon the correlated response in weight (not presented here) are small, and almost negligible.


Effect of incidence on correlated response in a 0-1 trait ($h^2_{D_2}$=0.15) to selection on a 0-1 trait ($h^2_{D_1}$=0.15, p_1=0.05, rg=0.10, NHS=150, NFS=25)
CR as a % of p_2
70
60
50
40
30
20
10
0·00
0·01
0·02
0·03
0·04
0·05
Incidence of correlated trait (p_2)
FS family selection
HS family selection
Mass selection

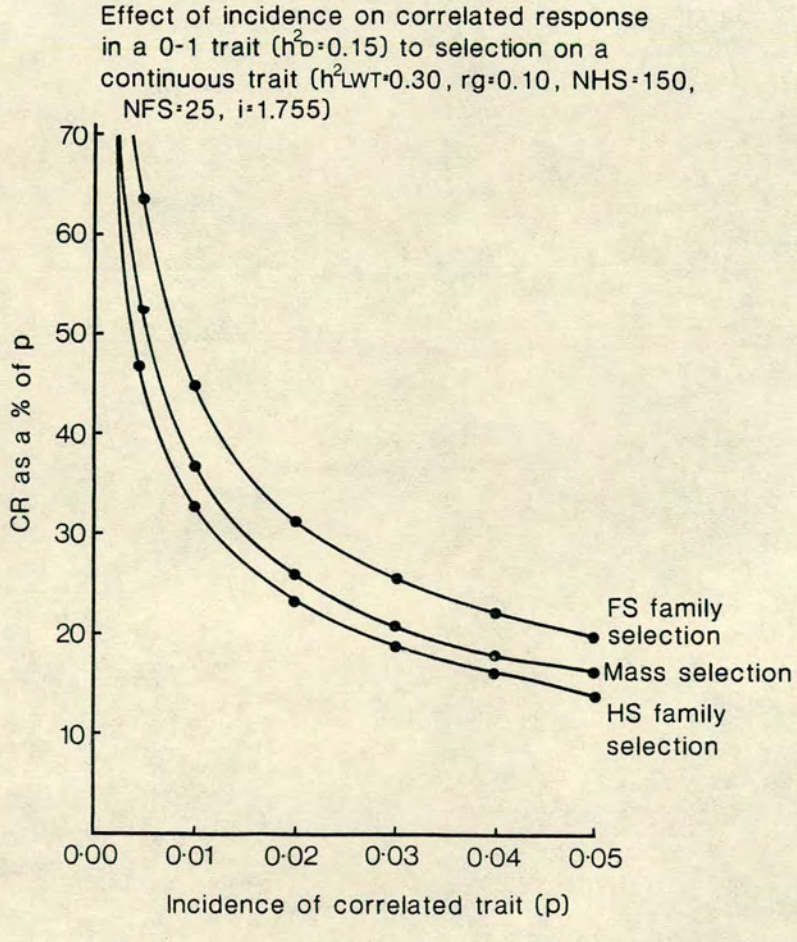

Effect of incidence on correlated response in a 0-1 trait (h^2_D=0.15) to selection on a continuous trait (h^2_{LWT}=0.30, rg=0.10, NHS=150, NFS=25, i=1.755)
CR as a % of p
70
60
50
40
30
20
10
0·00
0·01
0·02
0·03
0·04
0·05
Incidence of correlated trait (p)
FS family selection
Mass selection
HS family selection

Fig. 7.4

Independent culling levels

Having seen that selection on body weight alone is likely to have a deleterious effect on the incidence of defects, and also that conversely, selection on defects is likely to have a deleterious, if tiny, effect upon live-weight, it is necessary to consider alternative forms of selection to try and minimise these effects. An obvious, and indeed, realistic method is to use independent culling levels; that is to practice selection simultaneously on both defects and live-weight. Once each bird has been assessed, both individual and family information can be used to select on more than one trait. In reality, as defects are quite rare, this information is likely to be considered first. Selection for body size will be carried out in the population after decisions have been taken on which birds to exclude because of skeletal problems. This is more strictly tandem selection, and may be treated mathematically as such.

The total response in any one trait is the sum of its response to direct selection, plus its correlated response to selection on all other traits considered. In this way, various combinations of mass and family selection on the traits in question may be tried. Theoretically, selection on the first trait, or traits, will alter the variance of other traits in the selected population (ie those birds which are left after selection). This has the effect of reducing the phenotypic variance of remaining traits, and increasing the heritability when the original genetic correlation is positive. Equations for the prediction of such changes have been formulated by Robertson (1977). In practice, however, if selection is carried out

against the defects first, very little effect can be seen on the variance in live-weight, as the variance in the selected traits is so small. This is supported by results, not presented here, which show that it is justifiable to ignore such changes in variance, which makes the calculation of responses much more straightforward.

A simulation study was carried out to investigate the accuracy of predictions made by selection using independent culling levels, using exactly the same techniques already described for correlated responses earlier in the chapter. Mass selection on defect traits was ignored because the responses have been shown to be small. Birds were ranked on full-sib family incidence for defects with p=5.0% and p=1.0% respectively, a proportion selected out, and the remainder reranked on either family mean phenotypic live-weight, or individual phenotypic live-weight. In a practical situation it is unlikely that selection would be on a half-sib basis for defect traits because of the numbers involved. The expected response to selection could be computed as:

$$TR = R + \Sigma CR$$

Predicted responses were seen to agree well with those actually obtained from the simulation, (Table 7.2), and therefore the method may be applied to actual data. On consideration of a single defect trait only, in conjunction with live-weight, it can be seen that full-sib family selection against the defect followed by selection for live-weight leads to an overall increase in the incidence of the defect. The increase is exacerbated by the use of full-sib family selection on live-weight as opposed to mass selection. These

Table 7.2 Comparison of predicted (expected) and actual simulated (observed) responses to tandem selection on a defect trait with variable incidence (P) and h^2=0.13, and a continuous trait with a mean of 1500g, h^2=0.23, rg=0.20. ns=150, nd=6, np=24.[1]

(a) Full-sib family selection in both traits (defect + 'LWT')

%I (P)	E[R] DEFECT	O[R] DEFECT	E[R] LWT	O[R] LWT
5.00	+0.71	+0.72	+25.63	+24.48
1.00	+0.24	+0.26	+25.86	+24.75

(b) Full-sib family selection on defect followed by mass selection on 'LWT'

%I (P)	E[R] DEFECT	O[R] DEFECT	E[R] LWT	O[R] LWT
5.00	+0.47	+0.38	+18.67	+18.66
1.00	+0.16	+0.15	+18.90	+18.84

Table 7.3 Economic weights (a) used to construct selection indices - each weight is expressed in units of value corresponding to 1g.

	% MORTALITY	% DOWNGRADING	% UNIT LOSS	ECONOMIC WEIGHTS LINE 0	LINE 1	LINE 2
CT	3.0	10.0	6.23	89.6	97.5	92.8
B	5.0	50.0	20.83	299.5	325.9	310.3
S	5.0	30.0	14.50	208.5	226.8	216.0
TW	80.0	100.0	86.67	1246.4	1355.8	1291.1
TD	30.0	70.0	46.33	666.3	724.8	690.1
KK	3.0	90.0	32.10	461.6	502.2	478.2
TK	3.0	90.0	32.10	461.6	502.2	478.2

1. ns = no. of sires, nd = no. of dams, np = no. of progeny

implications will be considered further, later, when the effects of including several traits simultaneously can be seen.

Index selection

In order to optimise responses to selection, in terms of financial gain, use of an index must be made in selection. Obviously, many indices may be used on the same data, depending on the importance of one trait relative to another. Three indices are presented here, based on a suggested set of economic weights attached to each of the defect traits, combinations of family and mass selection being considered. The basic index equation is usually given in the form:

$$Pb = Ga \qquad b = P^{-1}Ga$$

where:

P is an nxn phenotypic variance/covariance matrix

G is an nxm genotypic variance/covariance matrix

a is an mxl scalar of economic weights

b is the nxl vector of index weights

n = no. of traits, m = no. of economic variables

Thus for mass selection, index weights may be calculated using the following matrices:

$$\begin{matrix} & P & & & b & & & G & & & a \\ \begin{bmatrix} covp(1,1) & .. & covp(1,n) \\ covp(n,1) & .. & covp(n,n) \end{bmatrix} & \cdot & \begin{bmatrix} b1 \\ bn \end{bmatrix} & = & \begin{bmatrix} cova(1,1) & .. & cova(1,m) \\ cova(n,1) & .. & cova(n,m) \end{bmatrix} & \cdot & \begin{bmatrix} a1 \\ am \end{bmatrix} \end{matrix}$$

where $covp(1,2) = rp\sigma p1\sigma p2$ $cova(1,2) = rah1h2\sigma p1\sigma p2$

The second index tried, examined individual plus full-sib family selection on all traits:

$$\begin{matrix} & & & P & & & & b \\ \begin{bmatrix} covp(1,1) & covp(1,FS1) & .. & covp(1,n) & covp(1,FSn) \\ covp(FS1,1) & covp(FS1,FS1) & & covp(FS1,n) & covp(FS1,FSn) \\ \\ covp(n,1) & covp(n,FS1) & & covp(n,n) & covp(n,FSn) \\ covp(FSn,1) & covp(FSn,FS1) & .. & covp(FSn,n) & covp(FSn,FSn) \end{bmatrix} & \cdot & \begin{bmatrix} b1 \\ bFS1 \\ \\ bn \\ bFSn \end{bmatrix} \end{matrix}$$

$$\begin{matrix} & & G & & a \\ \begin{bmatrix} cova(1,1) & .. & cova(1,m) \\ cova(FS1,1) & & cova(FS1,m) \\ \\ cova(n,1) & & cova(n,m) \\ cova(FSn,1) & .. & cova(FSn,m) \end{bmatrix} & \cdot & \begin{bmatrix} a1 \\ am \end{bmatrix} \end{matrix}$$

where:

$covp(1,FS2) = covp(FS1,2) = covp(FS1,FS2)$

$= Rcova + 1/n(covp - Rcova)$

$= \sigma p1 \sigma p2[Rra12h1h2 + 1/n(rp12 - Rra12h1h2)]$

$cova(1,FS2) = cova(FS2,1) = cova(FS1,FS2)$

$= ra12h1h2 \sigma p1 \sigma p2(R + (1-R)/n)$

R = relationship

A third, more realistic index assumes individual plus full-sib family selection on defect traits and mass selection on live-weight (Trait 1). This may be easily obtained from the matrix above simply by deletion of row 2 and column 2.

An alternative approach is to use an index which maximises response in desired traits whilst holding the response of the undesired traits constant. This is known as a restricted index. To compute such an index it is necessary to add a dummy variable for each restriction (Moen, 1968). In the P-matrix a new row is added which is made up of the genetic covariance of the restricted trait with the others, and the column added is the transpose of that row, with a diagonal element equal to 0. A row of zeros is added to the G-matrix. Thus,

taking the matrices already described for mass selection, a restriction of trait 2 will give the following:

$$\begin{matrix} & P & & & b & & G & & a \\ \begin{bmatrix} covp11 & covp12 & cova21 \\ covp21 & covp22 & cova22 \\ cova21 & cova22 & 0 \end{bmatrix} & \begin{bmatrix} b1 \\ b2 \\ b0 \end{bmatrix} & = & \begin{bmatrix} cova11 & cova12 \\ cova21 & cova22 \\ 0 & 0 \end{bmatrix} & \begin{bmatrix} a1 \\ a2 \end{bmatrix} \end{matrix}$$

The resulting weights in each of the above indices should be multiplied by the score for the corresponding trait, and birds selected upon the sum of these products. In making predictions from these indices, the resulting responses should be multiplied by the selection intensity to be used in the selection programme. Results from the application of these indices to the commercial data are presented in the next section.

Calculation of economic weightings used in the above depends on the fact that defect traits are a source of monetary loss to the producer. On average, the loss (L) attributable to each affected bird may be represented as:

$$L = (-m - [d(1-m)]x) \qquad a = L.LWT.V$$

where:

m = proportion of affected birds dying

d = proportion of affected birds downgraded on the processing line (d(1-m) represents the proportion of the population downgraded after mortality has occurred)

x = loss through downgrading as a proportion of each bird downgraded

LWT = mean live-weight

V = value of each unit of live-weight

a = economic weight

Each unit of response to selection for a defect trait would be therefore equivalent to loss L in terms of live-weight, and this can be taken as the economic weight assigned to that trait. Details of the economic weights used in the subsequent analyses are shown in Table 7.3. The proportions used are hypothetical, but thought to correspond to real situations, and x is assumed to be 1/3.

Results

The selection methods outlined above were applied to the data available. The parameters used were those taken from summary tables very similar to those presented at the end of the last chapter, with the exception that dam component estimates were omitted. (Table 7.4) A comparison of predicted results is shown in Table 7.5, and full details of expected direct and correlated responses can be found in Table 7.6 and Tables 7.7a, 7.7b and 7.7c. Results are expressed in terms of percent change in incidence. Therefore, if a trait with an incidence of p=0.05 shows a response of 60%, the incidence will increase to p=0.08 in the next generation.

In terms of change in incidence, selection against defect traits appears to be quite successful if family selection is used, up to 68% less birds being affected by twisted leg after full-sib family selection against this trait. Generally, responses in the order of 20-60% were found in defect traits with full-sib family selection, as opposed to less than 10% using mass selection, and 45-80% using half-sib family selection. These results, of course, would only be applicable if selection was just carried out on the defect traits.

Table 7.4 Tables of h^2_{01} (diagonal) and rg used in calculations presented in Chapter 7.

Line 0

	LWT	CT	B	S	TW	TD	KK	TK
LWT	0.31							
CT	0.09	0.12						
B	0.22	0.19	0.05					
S	0.15	0.12	-0.21	0.06				
TW	0.01	-0.02	0.12	0.25	0.13			
TD	0.09	-0.05	0.45	0.18	0.02	0.03		
KK	0.01	-0.01	0.05	0.05	0.11	-0.15	0.04	
TK	-0.10	-0.23	-0.31	0.01	-0.07	-0.01	0.21	0.04

Line 1

	LWT	CT	B	S	TW	TD	KK	TK
LWT	0.30							
CT	0.26	0.06						
B	0.29	0.49	0.05					
S	0.10	0.29	0.03	0.07				
TW	-0.05	0.04	0.30	0.00	0.17			
TD	0.21	0.11	0.12	0.07	0.00	0.03		
KK	-0.06	-0.15	-0.21	0.20	-0.03	0.23	0.05	
TK	0.00	0.06	-0.01	-0.05	-0.01	0.06	0.11	0.03

Line 2

	LWT	CT	B	S	TW	TD	KK	TK
LWT	0.23							
CT	0.31	0.07						
B	0.24	0.38	0.02					
S	-0.01	0.24	-0.08	0.08				
TW	-0.24	0.14	0.00	0.39	0.05			
TD	0.08	-0.07	0.19	0.10	0.04	0.02		
KK	0.08	0.18	0.04	-0.04	-0.08	0.24	0.03	
TK	0.12	0.02	0.20	0.11	0.16	0.32	0.34	0.01

Table 7.5 Comparison of predicted responses to different forms of selection using parameters presented in Table 7.4 (% mean).

Line 0

SELECTION	LWT	CT	B	S	TW	TD	KK	TK
1.	8	-12	-5	-6	-13	-3	-4	0
2.	7	-81	-64	-68	-83	-51	-58	-58
3.	10	-60	-37	-41	-62	-25	-31	-31
4.		13	32	24	9	29	2	-31
5.		12	28	21	8	25	1	-27
6.		16	38	28	11	34	2	-36
7.	8	0	26	16	-17	21	-3	-27
8.	9	-51	-5	-19	-147	-26	-36	-28
9.	7	-54	-11	-24	-149	-31	-36	-22
10.	8	12	10	8	-25	10	-2	-34
11.	11	10	34	25	-87	28	-8	-43
12.	8	13	33	22	-105	33	-10	-48

Line 1

SELECTION	LWT	CT	B	S	TW	TD	KK	TK
1.	8	-6	-5	-7	-17	-3	-5	0
2.	7	-71	-67	-74	-89	-54	-67	-54
3.	10	-40	-36	-44	-68	-24	-36	-24
4.		33	50	13	-48	87	-10	0
5.		29	45	11	-43	78	-9	0
6.		39	60	15	-57	105	-12	0
7.	8	23	41	3	-74	76	-15	-4
8.	8	-24	-2	-47	-189	22	-47	-31
9.	7	-31	-11	-49	-180	4	-45	-31
10.	8	38	53	17	-118	92	-13	-1
11.	11	41	60	12	-187	104	-24	-6
12.	8	45	60	12	-223	94	-28	-6

Table 7.5 (continued) Line 2

SELECTION	LWT	CT	B	S	TW	TD	KK	TK
1.	6	-7	-2	-8	-5	-2	-3	-1
2.	6	-75	-45	-78	-68	-45	-55	-29
3.	8	-48	-20	-52	-40	-20	-28	-11
4.		31	25	-1	-140	14	7	11
5.		31	25	-1	-141	14	7	12
6.		41	34	-2	-187	18	10	15
7.	6	21	20	-12	-164	9	3	8
8.	7	-30	-9	-73	-354	-28	-31	-19
9.	5	-40	-17	-72	-307	-33	-34	-23
10.	6	31	26	-8	-182	11	7	9
11.	9	36	33	-20	-284	9	5	9
12.	6	37	33	-28	-302	2	5	3

Forms of selection used

1. Direct response to mass selection
2. Direct response to HS family selection
3. Direct response to FS family selection

4. Correlated response to mass selection on LWT
5. Correlated response to HS family selection on LWT
6. Correlated response to FS family selection on LWT

7. Response to use of independent culling levels with mass selection on each trait
8. Response to use of independent culling levels with FS family selection on each trait
9. As 7 and 8 but with FS family selection on defects and mass selection on LWT.

10. Response to index selection - mass selection
11. Response to index selection - FS family + individual selection
12. As 10 and 11 but with FS family + individual selection on defects and mass selection on LWT.

Table 7.6 Predicted response[1] to selection on LWT and defect traits using parameter presented in Table 7.4 expressed as an actual change in the mean (R) and as a % change in the incidence (R%P)

Line 0

	MASS SELN		HS FAMILY SELN[2]		FS FAMILY SELN	
	R	R%P	R	R%P	R	R%P
LWT	121.5	8	106.8	7	144.1	10
CT	-0.59	-12	-4.01	-81	-2.94	-60
B	-0.11	-5	-1.40	-64	-0.81	-37
S	-0.14	-6	-1.56	-68	-0.95	-41
TW	-0.01	-13	-0.08	-83	-0.06	-62
TD	-0.01	-3	-0.15	-51	-0.08	-25
KK	-0.07	-4	-0.93	-58	-0.50	-31
TK	0.00	0	-0.23	-58	-0.13	-31

Line 1

	MASS SELN		HS FAMILY SELN		FS FAMILY SELN	
	R	R%P	R	R%P	R	R%P
LWT	130.1	8	116.3	7	155.7	10
CT	-0.20	-6	-2.41	-71	-1.36	-40
B	-0.07	-5	-1.00	-67	-0.53	-36
S	-0.27	-7	-2.82	-74	-1.68	-44
TW	-0.02	-17	-0.09	-89	-0.07	-68
TD	-0.01	-3	-0.11	-54	-0.05	-24
KK	-0.09	-5	-1.13	-67	-0.61	-36
TK	0.00	0	-0.27	-54	-0.12	-24

Line 2

	MASS SELN		HS FAMILY SELN		FS FAMILY SELN	
	R	R%P	R	R%P	R	R%P
LWT	93.0	6	94.0	6	124.3	8
CT	-0.34	-7	-3.60	-75	-2.33	-48
B	-0.03	-2	-0.58	-45	-0.26	-20
S	-0.24	-8	-2.33	-78	-1.56	-52
TW	-0.01	-5	-0.07	-68	-0.04	-40
TD	-0.01	-2	-0.22	-45	-0.10	-20
KK	-0.07	-3	-1.38	-55	-0.69	-28
TK	0.01	-1	-0.20	-29	-0.08	-11

1. LWT expressed in g, defects as %I
2. Family selection assumes that all families with affected individuals are selected.

Table 7.7 Correlated response to selection on LWT and defect traits using parameters presented in Table 7.4 expressed as a % of the mean

(a) Line 0

Mass selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.06	-0.06	-0.05	0.00	-0.01	0.00	0.01
CT	13.43	-	-0.96	-0.68	0.04	0.07	0.04	0.45
B	22.34	-2.25	-	1.17	-0.25	-0.62	-0.19	0.59
S	23.57	-1.52	1.12	-	-0.56	-0.26	-0.21	-0.02
TW	9.16	1.47	-3.74	-8.74	-	-0.17	-2.64	0.83
TD	28.94	1.29	-4.92	-2.21	-0.09	-	1.26	0.04
KK	1.53	0.12	-0.26	-0.29	-0.24	0.21	-	-0.42
TK	-30.78	5.69	3.25	-0.12	0.31	0.03	-1.69	-

HS family selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.42	-0.80	-0.55	-0.01	-0.12	-0.03	0.16
CT	11.81	-	-12.74	-7.75	0.27	1.18	0.57	6.48
B	28.44	-15.33	-	13.35	-1.60	-10.43	-2.80	8.60
S	20.73	-10.35	14.29	-	-3.57	-4.46	-2.99	-0.30
TW	8.06	10.06	-47.59	-99.06	-	-2.89	-38.33	12.10
TD	25.45	8.83	-62.65	-25.04	-0.58	-	18.35	0.61
KK	1.34	0.84	-3.31	-3.30	-1.53	3.61	-	-6.05
TK	-27.07	38.87	41.31	-1.33	1.96	0.49	-24.59	-

FS family selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.31	-0.46	-0.33	-0.01	-0.06	-0.02	0.09
CT	15.92	-	-7.07	-4.70	0.20	0.59	0.31	3.50
B	38.34	-11.23	-	8.10	-1.19	-5.22	-1.51	4.65
S	27.94	-7.58	8.23	-	-2.67	-2.23	-1.62	-0.16
TW	10.86	7.37	-27.42	-60.12	-	-1.45	-20.74	6.55
TD	34.31	6.47	-36.10	-15.19	-0.44	-	9.93	0.33
KK	1.81	0.61	-1.90	-2.00	-1.14	1.81	-	-3.27
TK	-36.49	28.47	23.80	-0.81	1.46	0.24	-13.31	-

S.TR = selected trait, C.TR = correlated trait
Where selection is on a defect, family responses are calculated under the assumption that all families with affected individuals are selected.

Table 7.7 (b) Line 1

Mass selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.10	-0.07	-0.05	0.01	-0.01	0.02	0.00
CT	32.59	-	-1.79	-1.99	-0.09	-0.10	0.57	-0.10
B	49.87	-4.03	-	-0.28	-0.92	-0.15	1.10	0.02
S	12.78	-1.77	-0.11	-	0.00	-0.06	-0.78	0.08
TW	-47.56	-1.82	-8.30	0.00	-	0.00	0.87	0.12
TD	87.41	-2.19	-1.45	-1.59	0.00	-	-2.91	-0.32
KK	-9.86	1.18	1.00	-1.80	0.09	-0.27	-	-0.23
TK	0.00	-0.67	0.07	0.64	0.04	-0.10	-0.78	-

HS family selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-1.22	-0.94	-0.48	0.04	-0.23	0.20	0.00
CT	29.17	-	-23.81	-21.07	-0.47	-1.79	7.63	-1.74
B	44.63	-47.60	-	-2.99	-4.80	-2.68	14.65	0.40
S	11.44	-20.94	-1.49	-	0.00	-1.16	-10.37	1.48
TW	-42.56	-21.49	-110.6	0.00	-	0.00	11.57	2.20
TD	78.23	-25.86	-19.36	-16.89	0.00	-	-38.83	-5.79
KK	-8.83	13.93	13.38	-19.05	0.46	-4.91	-	-4.19
TK	0.00	-7.90	0.90	6.75	0.22	-1.82	-10.40	-

FS family selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.69	-0.50	-0.29	0.03	-0.10	0.11	0.00
CT	39.00	-	-12.71	-12.55	-0.36	-0.81	4.07	-0.79
B	59.67	-26.97	-	-1.78	-3.70	-1.21	7.82	0.18
S	15.30	-11.87	-0.79	-	0.00	-0.53	-5.54	0.67
TW	-56.91	-12.18	-59.07	0.00	-	0.00	6.18	1.00
TD	104.59	-14.66	-10.34	-10.06	0.00	-	-20.73	-2.62
KK	11.80	7.89	7.14	-11.35	0.35	-2.23	-	-1.90
TK	0.00	-4.48	0.48	4.02	0.17	-0.82	-5.55	-

Table 7.7 (c) Line 2

Mass selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.14	-0.03	0.00	0.01	-0.01	-0.02	-0.01
CT	30.78	-	-0.72	-1.41	-0.12	0.08	-0.59	-0.02
B	25.27	-2.82	-	0.50	0.00	-0.23	-0.14	-0.22
S	-1.35	-2.28	0.20	-	-0.45	-0.16	0.18	-0.15
TW	-140.0	-5.76	0.00	-13.49	-	-0.27	1.53	-0.97
TD	13.71	0.85	-0.62	-1.02	-0.06	-	-1.35	-0.57
KK	7.32	-1.16	-0.07	0.22	0.06	-0.26	-	-0.32
TK	11.54	-0.14	-0.37	-0.63	-0.13	-0.36	-1.07	-

HS family selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-1.46	-0.64	0.04	0.17	-0.13	-0.30	-0.22
CT	31.11	-	-16.05	-13.70	-1.61	1.82	-10.76	-0.59
B	25.54	-30.21	-	4.84	0.00	-5.23	-2.54	-6.27
S	-1.36	-24.44	4.59	-	-6.10	-3.53	3.25	-4.42
TW	-141.5	-61.66	0.00	-130.8	-	-6.09	28.11	-27.80
TD	13.86	9.06	-13.85	-9.85	-0.80	-	-24.78	-16.34
KK	7.40	-12.44	-1.56	2.11	0.85	-5.74	-	-9.27
TK	11.67	-1.45	-8.18	-6.08	-1.78	-8.04	-19.70	-

FS family selection

C.TR/S.TR	LWT	CT	B	S	TW	TD	KK	TK
LWT	-	-0.95	-0.29	0.02	0.10	-0.06	-0.15	-0.09
CT	41.13	-	-7.28	-9.20	-0.95	0.82	-5.44	-0.23
B	33.77	-19.54	-	3.25	0.00	-2.37	-1.28	-2.45
S	-1.80	-15.81	2.08	-	-3.58	-1.60	1.64	-1.73
TW	-187.1	-39.88	0.00	-87.85	-	-2.77	14.21	-10.88
TD	18.33	5.86	-6.29	-6.62	-0.47	-	-12.53	-6.39
KK	9.79	-8.05	-0.71	1.41	0.50	-2.61	-	-3.63
TK	15.43	-0.94	-3.71	-4.09	-1.05	-3.65	-9.96	-

Family selection here is done under the assumption that all families with affected birds are selected against. This is unlikely to be the case, nor is it likely that breeders will often wish to use half-sib family selection against defects, which would lead to the disposal of a lot of unaffected individuals, and also inbreeding.

The above responses are offset by the largely unfavourable correlated responses to selection for live-weight. The type of selection does not appear to have a proportionately large effect on response, probably because the selection intensity assumed here is quite high (1 in 10 birds being selected). Full-sib family selection has the greater effect. Where the genetic correlations are positive, selection for body weight leads to increases in the incidence of the defects. This is most consistent amongst the three relatively high incidence leg abnormalities: crooked toe, bow and splay (except for the latter in Line 2), which all show an increase in the proportion affected in the order of 10-50%. Where traits are negatively correlated with live-weight, selection has a beneficial effect of reducing the incidence, as for twisted keel in Line 0.

Correlated responses in one defect trait to selection on another depend on the true genetic correlation between the two traits. It is hard to generalise, but selection against leg defects with a reasonably high incidence appears to produce a favourable correlated response in similar traits, in the order of 10-20%. Correlated responses to selection against twisted leg and tibial dyschondroplasia appear to be variable and largely higher than the rest. This latter point may be because of low incidence and

uncertainty in the data, respectively. Correlated responses to selection against the keel defects are small, as are responses in all traits to mass selection.

On consideration of the application of independent culling levels, it can be seen that use of mass selection on each trait is not sufficient to counteract the effect of the positive correlations between defect traits and live-weight. Unfavourable gains in incidence still occur, these being as much as 41% in bow in Line 1. Full-sib family selection on all traits, however, is capable of reducing the incidence in all the defect traits. This is also the case if full-sib family selection is used against the defects, and mass selection on live-weight; the decreases in incidence of the defect traits being even greater. In almost all of the latter cases, the incidences of defect traits are predicted to change by at least one percentage point. The responses in live-weight in all three lines, however, are worse than the corresponding responses to direct selection on weight, either with mass or full-sib family selection. This is particularly noticeable in selection option 9 of Table 7.5, and may lead to an overall loss to the producer despite the improvement amongst the defects. Use of independent culling levels may lead to successful reduction of the incidence of undesirable traits, but this can only be achieved by sacrificing gains in live-weight.

The latter point raises the question of value to the breeder and grower. It is little use ridding a population of leg defects if the improvement in body weight is only such that that it would make more

Table 7.8 Cumulative response to sequential restriction of traits in an index using FS family and individual selection (Line 0).

RESTRICTION	ECONOMIC GAIN (U)	R %LWT	% CHANGE IN R LWT [1]
NONE	11.82	12.14	-
-CT	11.77	12.04	-0.43
-CT,B	9.51	9.73	-19.19
-CT,B,S	8.36	8.52	-12.05
-CT,B,S,TW	7.47	7.29	-10.66
-CT,B,S,TW,TD	7.74	7.64	+3.57
-CT,B,S,TW,TD,KK	7.73	7.81	-0.11
-CT,B,S,TW,TD,KK,TK	6.86	6.86	-11.24

1. difference in response in LWT from previous restriction

economic sense to ignore those traits. One way of examining this is to use selection indices. Predicted results from the three indices proposed are shown in parts 10,11,12 of Table 7.5. Use of an index appears to give a higher response in live-weight than that which would be expected from direct selection on this trait. Responses in defects are quite strongly unfavourable, however, except in traits which are known to have a negative genetic correlation with body weight, such as splay in Line 2., or a very low incidence, as has twisted leg in all lines. The incidence in any one defect trait is likely to be worse after selection on an index, as a result of a correlated response to selection on live-weight. In the case of a restricted index, as has been calculated for Line 0 (see Table 7.8), exclusion of each successive trait from the index leads to a corresponding decrease in the response to live-weight. The implications of selection indices calculated using the economic weights suggested here, are that relative to live-weight, defects are of very little economic importance. This is so much so that the indices indicate that it is beneficial to select for defect traits, as this will cause further gain in weight!

To conclude, the outcome of the results discussed above is that defect traits can be selected against, but only at the expense of gains in live-weight. Use of an index suggests that it may be more worthwhile to ignore defect traits in the field. Use of independent culling levels is justified if strong selection is required to act against defects while small gains in body weight are maintained.

This means that the method of selection is really a matter of priority in breeding objectives, and various successful outcomes are available to the breeder. It should be noted that the responses achieved in this chapter refer to a particular set of data. It is felt that the equations used are robust, however. Direct responses to selection are proportional to the incidence of the trait involved, and the magnitude of correlated responses is determined by the incidences of the two traits and the genetic correlation between these. Given the fact that it appears that the methods of predicting selection responses proposed in this chapter are valid, it can be seen that, if desired, these methods may be used to predict changes in incidences of skeletal defects.

CHAPTER 8. GENERAL DISCUSSION AND CONCLUSIONS

Avian dyschondroplasia is typified by the presence of cartilage plugs at the end of long bones. This condition is widespread in domestic poultry. The importance of dyschondroplasia has been underlined by the findings of the present studies, field incidences in the order of 10-25% having been observed. This chapter attempts to draw together results and conclusions presented in this thesis, and to consider these in relation to previous knowledge of the problem. For the purpose of easy comparison, the chapter is therefore subdivided into sections corresponding to those used in the introductory chapter. Thus fresh insights into particular areas of study can be readily assessed.

Morphology and development

Although this thesis was not concerned directly with anatomical aspects of leg conditions, much incidental information has been gathered. Various points have been noted which differ from previous findings. The most important of these is that dyschondroplasia has been induced in chickens of egg-laying strains (Chapter 3, Chapter 5). These birds are therefore not genetically free of any susceptibility to the condition, and so all chickens may potentially develop growth plate abnormalities. A general feature noted in the numerous dissections which have been carried out is that the development of the growth plate in broilers is irregular. On a gross level, the transition between the cellular zones is indistinct, and it is hard to point to broilers with a so-called 'normal' growth

plate. This supports the results from comparisons made by Reiland et al (1978) of broilers and Leghorns.

If heavier birds such as broilers have a generalised disruption of growth processes, as indicated above, then it should be possible to find evidence of abnormal development elsewhere in the chicken. A very small study was performed in which eye lens tissue was examined for cartilage irregularities, from 24 chickens which had been killed and assessed for dyschondroplasia (R.M. Clayton and Mercer, unpublished). Only three birds were seen to be affected by dyschondroplasia. Of these, two had lenses which showed signs of a moderate disruption in cellular development, and the other had very mild symptoms of irregularity. In contrast, the remaining birds all had normal eye lenses. This evidence, although very slight, does suggest that disruption of growth processes is not merely a localised phenomenon. It may be possible by studying other cellular and biochemical processes in the chicken to achieve a better understanding of dyschondroplasia.

In all the studies carried out during the course of this thesis a greater incidence of growth plate abnormalities was found in cocks than pullets. Sex differences were not indicated in some of the previous studies (Riddell, 1977; Sheridan et al, 1978). It is possible that such differences are a consequence of faster growth rates and heavier weights at a given age in males, as suggested by Leach and Nesheim (1972).

Unmistakable abnormal cartilage plugs were noted at 10 days in

broilers. This is slightly earlier than has previously been recorded, and was seen in birds reared on both purified and practical diets. The initial investigatory survey (Chapter 2) indicated that more birds, on average, were likely to have cartilage plugs at an age of 3-4 weeks. This supports the conclusions of other authors (Poulos et al, 1978; Randall and Mills, 1981; Leach and Nesheim, 1972). In the various rearing trials discussed in Chapter 3, however, age was seen to have a significant effect on incidence in some cases, but not in others. The presence of lesions at 6 weeks in birds thought to be free of the condition at 4 weeks, in the selection experiment described in Chapter 4, may indicate that the time of onset is variable. It may be accepted that dyschondroplasia can develop at different times. It is reasonable to suggest, however, that the best time to assess birds by dissection is at the 4 week mark. Further comment is made about a suggested critical period of development in the section concerning growth rate.

One of the main criticisms which can be directed against the literature on dyschondroplasia in general, is that authors give very scant descriptions of methods which are used to assess the condition. For work to be repeatable, or comparable, it is vital that there should be consistency in the way in which growth plates are examined and scored for abnormalities. Much attention has been given to this problem throughout the present studies. Attempts have been made to find methods of assessment which are precise and simple, and which can be used for practical selection as well as post-mortem examination. A 1-5 grading system involving the dissection of growth plates has been proposed (Chapter 2), which can

be limited to the proximal tibiotarsus and proximal tarsometatarsus of one leg only. Any further information is superfluous if the intention is merely to establish the incidence of the condition.

Present methods of live assessment for tibial dyschondroplasia (TD) have been shown to be totally inadequate for the prediction of underlying growth plate lesions. This is because TD bears little, if any, relation to avian dyschondroplasia as scored by dissection, and therefore perfectly normal birds are being unnecessarily culled. Indeed, the important criterion in diagnosing TD appears to be the size of the tibiotarsal head, as felt by palpation, which may be subject to natural variation. It is recommended that this information should be ignored in the field. Live assessment may be of some value in aiding diagnosis of birds with the use of radiography. It may also be used to obtain a general indication of prevalence of leg problems related to dyschondroplasia, although this is of limited application. The condition has been seen to be correlated with shorter legs and wider growth plates (Chapter 2), but measurement of these criteria is not useful in aiding diagnosis.

A quick and harmless method of radiographing tibiotarsi of chickens, similar to that of Burton and Howlett (1979), has been devised (Chapter 2). Positioning of the bird in order to achieve a good radiograph is thought to be critical to success. Initial studies suggested that the method was extremely accurate in detecting lesions, and even for assessing their severity. Practical application of the technique, however, produced slightly

disappointing results, in that some affected birds were probably missed, and diagnosis was uncertain in others. despite these reservations, radiography seems to be the most reliable technique available. It remains the best way of assessing birds for dyschondroplasia in a mass selection programme.

Effect of nutrition on dyschondroplasia in the chicken

A feature of the various nutritional experiments carried out in this study, and indeed of both previous experiments reported in the literature and at the PRC, is that birds grown on purified diets had consistently higher incidences of dyschondroplasia than birds reared simultaneously on practical diets. It is possible that birds may be able to pick up vital trace elements or obtain more of an active ingredient for the prevention of the condition by feeding on practical diets. Problems also arise in the mixing of a purified diet in ensuring that trace elements and vitamins are evenly distributed throughout the feed. As no single dietary factor has been successfully identified as being responsible for dyschondroplasia, it is probable that feeding of a purified diet may offer a variety of ways in which the birds' requirements are not fully met. Their overall condition is therefore lowered, and their susceptibility to dyschondroplasia increased.

An advantage of using the same experimental facilities for a series of tests, as was the case in this instance at both the PRC and breeding company Y, is that rearing conditions are repeatable (Chapter 3). Results from different experiments may be compared with

more confidence than is the case between laboratories. The fact that the literature on nutritional effects is full of discrepancies may be partly attributed to this, in addition to the general instability of birds in relation to the condition.

No specific practical-type diets were seen to have an effect on incidence in any of the rearing trials performed. Purified diets, however, were formulated which consistently produced high incidences of dyschondroplasia when birds were reared in wire cages (Chapter 3). These contained various soy-protein isolates, one of which, FP950, is readily available for use in commercial mixes. Use of a diet known to produce a high proportion of affected birds under a particular set of environmental conditions could be beneficial in selection experiments. Indeed, even commercially, one way of imposing greater selection pressure on a population would be to lower the threshold above which birds would exhibit dyschondroplasia. Used in conjunction with radiography, it should be possible to make rapid progress towards eliminating the condition.

Effect of growth rate

The relationship of dyschondroplasia with body weight has been examined critically throughout this thesis. Incidence of the condition does not appear to be related to mean group weights at 6-7 weeks. This was seen amongst the different strains discussed in Chapter 3, and in the ML and MT strains used in Chapter 5. An exception to this was noted in the trial comparing birds reared on the floor and in cages (Chapter 5). Birds grown on wire were seen to

be heavier, and had higher incidences than birds kept in litter pens.

Where indivdual weights were recorded, however, dyschondroplasia was found to have a negative phenotypic correlation with 4 week weight (-0.10, Chapter 4; -0.23, -0.14, Chapter 5). This implies that birds affected by the condition are likely to be smaller than their contemporaries. Affected birds are probably reluctant to move about, are less able to compete for feed, and so have poorer growth rates. This is supported by the negative phenotypic correlation between growth plate abnormalities and length of tibiotarsus in the initial survey (chapter 2).

In contrast to the above, the nature of genetic correlations between dyschondroplasia and live-weight is as yet uncertain. In selected G2 birds (Chapter 4) the condition was seen to have a somewhat surprising correlation with 4 week weight of -0.41. In the rearing trial discussed in Chapter 5, however, the genetic correlations between growth plate abnormalities and weight were positive at 1, 2, 3 and 4 weeks old. In neither of these studies were the numbers of sires and progeny considered sufficient to give reliable estimates of genetic parameters. A somewhat marginal line of evidence for the support of positive correlations between the two are the largely positive genetic correlations found between TD and 6 week live-weight presented in Chapter 6 (rg=0.12, 0.15 and 0.04 overall in Lines 0, 1 and 2 respectively). Reservations must be made about using this information because, as discussed, TD is thought not to be representative of true dyschondroplasia. A more important

indicator is that the other leg traits considered in Chapter 6 seem also to have positive correlations with live-weight. If leg problems are thought to be related, as indicated in this study, then it is likely that they all share the same relationship with growth rate. The above results suggest that dyschondroplasia is in fact a problem in faster growing birds.

More direct evidence for the involvement of weight with the formation of cartilage abnormalities may be found if the period of rapid development in the skeleton of the chicken is considered. In the restriction trial reported in Chapter 3, birds were seen to develop fewer abnormalities if restricted at 1-2 or 1-3 weeks, the time at which cartilage remodelling is most rapid. Results from the rearing trial show that the genetic correlations between weight and incidence are greater at 1 and 2 weeks (r_g=0.16, 0.15) than at 3 and 4 weeks (r_g=0.01, 0.04), suggesting that the earlier period of development is more important. These figures must be treated with caution because of the large standard errors involved. However, this supports the theory of a critical period of growth presented in Chapter 1. It may be possible to prevent a certain number of cases of dyschondroplasia by restricting feed at this time under field conditions. No evidence was found to support this, however, in one of the rearing trials described in Chapter 3, although the number of birds considered was very small.

If, as is thought, dyschondroplasia and other leg problems are positively correlated with body weight, this presents an obvious problem to the breeder and grower. As shown from predictions

presented in Chapter 7, selection for live-weight at 6 weeks will increase the incidence of leg problems. By use of a selection programme involving independent culling levels, the incidence of defects may be lowered while the weight is increased, but gains in weight are considerably less than would be the case if defects were ignored. Computation of selection indices designed to predict maximum economic gain shows that it is financially beneficial to actually award a positive weighting to defects in order to maximise responses in increased weight. Whilst this is hardly likely to be adopted by commercial breeders, serious questions must be raised as to what should be done. How much importance should be attached to the eradication of leg problems in order to produce a more aesthetically pleasing flock, despite the fact that this may not be as profitable overall? Alternatively, should leg problems be ignored in selection programmes in the hope of optimising financial gain per bird placed?

Genetic influences

The evidence which arose from these studies concerning the inheritance of dyschondroplasia was insufficient to make any positive conclusions. Definite strain differences were recorded from rearing trials, but no evidence of a genotype x environment interaction was found (Chapter 3). A selection line was successfully founded (Chapter 5), in which the incidence of dyschondroplasia was increased by use of family information obtained by dissection. Responses were similar to those noted by other authors, particularly Sheridan et al (1978). Responses in this line varied somewhat from

those predicted, however. This may be partly explained by the facts that a highly selected line of birds was used, and that, inevitably, environmental conditions varied from one hatch of birds to the next.

Heritability estimates obtained for the G1 and G2 generations of this selection line were large (1.00 and 0.36), as was the estimate calculated for birds in the rearing trial described in Chapter 5 (h^2 =0.63). All the above estimates were made using analysis of variance techniques, with the estimates being corrected for continuity. These estimates may be compared with those of Sheridan et al (1978), of 0.57 and 0.52 in the F2 and F3 generations respectively, although the numbers of birds involved in the present study were low. Sire component estimates were seen to be smaller than dam component estimates of heritability, suggesting that maternal or dominance effects may influence the incidence of dyschondroplasia in progeny. A comparison with the somewhat variable estimates obtained for TD in Chapter 6 is not valid, because of the doubts surrounding the nature of this trait. It appears conclusive, though, that dyschondroplasia can be inherited.

Little evidence was found to support the theory proposed by Sheridan et al (1978) that dyschondroplasia is controlled by a single recessive sex-linked gene. Sire family distributions of affected progeny were examined in two hatches of both G1 and G2 selected birds (Chapter 4 and 5), together with unselected ML birds in the rearing trial desribed in Chapter 5. Indications of bimodality were seen only in G1 birds grown in the rearing trial, and the numbers examined in this instance were small. In all other

cases the sire family incidences were continuously distributed. Although scant, results from test crosses made refute the existence of a single gene which exclusively controls the presence of dyschondroplasia. The ratio of affected to unaffected progeny in any of the matings could not be fitted to Mendelian theory. Indeed, affected progeny arose from the mating of unaffected individuals, and the mating of an unaffected cock to two affected hens produced different results in each case. The matings performed were insufficient, however, to make firm conclusions concerning single gene models involving variable penetrance. Dyschondroplasia may not therefore be categorically defined as a polygenic trait.

More work has been presented here on theoretical aspects of the analysis of discontinuous data, as typified by information collected on leg problems. A comparison of different methods of estimating heritabilities and genetic correlations is made in Chapter 6. Simulation studies show that heritability estimates obtained by analysis of variance methods, and corrected for continuity using the transformation of Robertson and Lerner (1949), are consistently greater than estimates made using proband methods (Falconer, 1965; Reich, James and Morris, 1972), and generally show an upward bias. Estimates are less reliable if the defect involved has a very low incidence ($p<0.01$). In contrast, estimates of genetic correlations made using analysis of variance methods are lower than those using proband methods. Estimates obtained using either method are quite robust unless the incidences involved are very small, or the true genetic correlations small. It is recommended that analysis of variance methods be applied for estimation of genetic correlations,

as these are more flexible than proband methods, which should be used for calculation of heritability estimates, if possible.

Prediction equations are presented for calculation of direct and correlated reponses to both individual and family selection in defect (0-1) traits (Chapter 7). Simulation studies show that these formulae may be used with some confidence for the prediction of selection responses, at least on theoretical populations. The direct response to selection on a defect is directly proportional to the heritability of that trait, and is scaled by its incidence. Greater responses are achieved using half-sib as opposed to full-sib family selection, but family size is not very important in determining the magnitude of this response. Correlated responses to selection on a 0-1 trait in other traits are dependent on the incidence and heritability of the 0-1 trait, and are scaled by the genetic correlation between the two traits.

Application of the above theory, together with the high heritability of dyschondroplasia, will make it possible for breeders to eliminate the condition from their birds.

Miscellaneous influences

It has been demonstrated several times in this thesis that the environmental conditions in which chickens are reared can moderate the expression of growth plate abnormalities. For this reason, consistency in experimental conditions is vital if results are to be compared. Perhaps the most encouraging finding in this respect is

the lack of any genotype x environment interaction in birds reared over a series of separate trials described in Chapter 3. This would have complicated the whole picture concerning dyschondroplasia still further.

It is hard to separate the various different factors which may be important in regulating the incidence of dyschondroplasia. In the rearing trial described in Chapter 5, the incidence of the condition in birds reared on litter was seen to be lower than would be expected from the literature (Ferguson et al, 1977; Veltmann and Jensen, 1980; Leach, 1982). A subsequent experiment revealed the presence of a floor type x diet interaction. Birds in wire cages attained heavier weights, however, may have been restricted in the amount they could move around, and were also subjected to a slightly different lighting regime. Any of these factors, or a combination of all three, may have been critical in promoting a higher incidence, in addition to the indicated variable 'floor type'.

In a rearing trial (Chapter 3) designed specifically to test the effect of stocking density in litter pens, no apparant differences in incidence of dyschondroplasia were noted between treatments. Birds from a 'heavy' male-line thought to be badly affected by leg problems were seen to have few cases of dyschondroplasia, however, when reared at an obviously low stocking density. Other variables which may have had an effect in the selection experiment (Chapter 4) are hatch effects. As birds from consecutive hatches and reared on the same farm had widely varying incidences ($p=0.18$ and 0.51), hatch effects deserve further consideration. The possibility of unknown

changes in husbandry cannot be ruled out, however.

In all probability, a whole variety of single or combinations of environmental variables may alter the susceptibility of birds to the development of cartilage abnormalities. These, including nutritional effects, will act upon the penetrance of genes controlling dyschondroplasia, and so vary the incidence.

Relation to general leg weaknesses

Information on various other leg problems has been collected and analysed in several of the studies presented here. It appears from the extensive analyses presented in Chapter 6 that crooked toe is the commonest leg problem encountered, although this is of little economic significance. Of the more important traits, splay has a field incidence of 2-4% in pure-line stock, with that of bow being slightly less. True twisted leg, accompanied by slipped tendon, is relatively scarce ($p<0.001$). This ranking of incidences was repeated in birds examined during the course of the selection experiment described in Chapter 4. Incidences here were approximately double those found previously (more so in the case of twisted leg). Few overt leg abnormalities were recorded in the rearing trial reported in Chapter 5, but most birds examined in this test had at least a very mild splay. Splay may be considered as a form of twisted leg, and is described as such by other authors (including Simons, 1977; Randall and Mills, 1981; Riddell, 1976).

The relationship between various leg problems and live-weight has

been considered in some detail in the data analyses presented in Chapter 6. Negative phenotypic correlations were reported between splay and 6 week live-weight, and also between twisted leg and live-weight, but the latter was seen to be positively correlated with bow and crooked toe. All these correlations were small ($r_p \leq \pm 0.07$). In birds examined in the selection experiment (Chapter 4), crooked toe, bow, splay and twisted leg were all seen to have negative phenotypic correlations with 4 week weight (results not presented). All leg abnormalities, with the exception of twisted leg, were seen to have positive genetic correlations with 6 week live-weight, those for bow and crooked toe being quite substantial, and ranging from $r_g=0.08$ to $r_g=0.32$, overall. These results show that leg problems, with the possible exception of true twisted leg, are more likely to occur in faster growing birds. The negative phenotypic correlations found indicate that birds which exhibit any severe forms of twisted leg develop poorly as a consequence. Bow and crooked toe may be exacerbated by heavy weight in individual birds, as suggested by Poulos et al (1978). The incidence of true twisted leg was very low in all studies presented, and therefore no definite conclusions can be made regarding this condition.

All of the leg abnormalities considered in Chapter 6 appeared to have high components of heritable variance. Heritability estimates calculated using the modified proband method averaged approximately 0.18 for bow, 0.24 for splay, 0.27 for crooked toe and 0.46 for twisted leg. An estimate of $h^2=0.28$ for splay was similarly obtained in the rearing trial (Chapter 5), which is comparable to the previous estimate. Hartmann (1979) also found that twisted leg had a

high heritability. It was seen in the present study that leg abnormalities could be pooled, and heritability estimates obtained for 'leg defects' considered as a single trait. These estimates were again quite substantial, and in the order of 0.20. Not surprisingly, application of selection theory to the above parameters showed that it was possible to change the incidence of the various leg defects.

The fact that leg problems can be considered together suggests in itself that they are interrelated. Phenotypic correlations between leg abnormalities presented in Chapter 6 were small, and all negative. Slight problems may have been caused by underscoring, in that only one defect may have been recorded where two were actually present. These correlations indicate that a bird with one leg defect is unlikely to develop another, and may possibly be interpreted as meaning that the various conditions are merely different manifestations of an underlying physiological syndrome. Genetic correlations between the various different conditions were generally positive, with the possible exception of that between bow and splay. This is further evidence that leg defects are related. Other skeletal defects were also considered, keel problems having negative phenotypic correlations with leg abnormalities, but variable genetic correlations. Tibial dyschondroplasia, as scored in the field, has not been commented on above, as it was not thought to be an actual leg problem.

Studies investigating true dyschondroplasia were not adequate for the determination of the relationship between this and other leg problems. They all indicate, however, that the two are closely

associated. It is notable that the incidences of various other defects were high in birds selected for dyschondroplasia (Chapter 4). In the initial survey on mortalities (Chapter 2), a significant association between all leg problems and growth plate abnormalities was found, this being similar to that described by Randall and Mills (1981). In the present study bow in particular seemed to be closely associated with the presence of dyschondroplasia, and slight evidence was also found for a relationship between dyschondroplasia of the hock joint and true leg twist. Phenotypic correlations between bow and dyschondroplasia were also seen to be positive in the selection experiment discussed in Chapter 4. It is likely that bow is often a consequence of growth plate abnormalities. Dyschondroplasia was found to have negative phenotypic correlations with crooked toes in this study. The phenotypic correlations between the former, and both splay and twisted leg were inconsistent from generation to generation, as were all genetic correlations calculated.

If positive genetic correlations exist between various leg problems, and between these and live-weight, it is likely that dyschondroplasia is also related genetically to these other defects. Evidence, although largely circumstantial, points to the conclusion that leg defects have arisen as a consequence of rapid growth in broilers, and that these are interrelated. Although the abnormalities, including dyschondroplasia, can be treated separately, selection against one is likely to reduce the incidence in others.

Much time has been spent in the past on searching for factors which determine the presence of dyschondroplasia. It has now been established that the condition is almost certainly not attributable to any single factor, but that its expression is susceptible to a large range of environmental variables. It would seem rather pointless therefore to continue trying to isolate specific diets, for example, which will reduce the incidence of the condition. Changes thus achieved will probably not be the same under all environmental circumstances.

Practically, it would seem sensible to select against leg conditions if these were felt to be sufficiently important. If the various different problems are all related then it might be profitable to perform an extensive study, using test crosses, to determine categorically whether or not these traits are quantitative. Little is yet known about the physiological changes which are responsible for leg problems. If developmental stages can be seen to be influenced by certain biochemical properties, and these related back to the occurence of dyschondroplasia, then our understanding of the condition would be greatly enhanced. Ultimately it might be possible to involve other genes in the regulation of leg problems. This could be achieved by either selection or gene manipulation. Before this can be considered, however, the biology of leg conditions must be investigated.

It is possible to summarise some of the results presented above in an attempt to clarify the ways in which they advance the current knowledge on dyschondroplasia:

1. Avian dyschondroplasia has been found to be prevalent in several strains of British broilers.

2. The condition is not restricted to broilers, and has been induced in egg-laying strains of chicken.

3. Evidence is presented to suggest that dyschondroplasia is only one phenomenon of a generalised syndrome of physiological defects resulting from rapid growth.

4. A simple and consistent method of assessing birds for dyschondroplasia by dissection is suggested.

5. Radiography seems to be the only reliable method of in vivo screening for growth plate abnormalities, and may be used in selection against these.

6. A purified diet containing a soy-protein isolate has been formulated which increases the incidence of dyschondroplasia in broilers reared in wire cages. It is suggested that use of this might be made to increase selection pressure against the condition.

7. Susceptibility of birds to dyschondroplasia is sensitive to nutrient balance.

8. Dyschondroplasia has a negative phenotypic correlation with live-weight. Evidence suggests that the genetic correlation between the two is positive.

9. It is proposed that growth rate during the period of cartilage remodelling at 1-3 weeks is critical to the development of growth plate abnormalities in the chicken.

10. Accurate methods of predicting selection responses involving leg defects have been described. These suggest that breeders face a dilemma of choosing between selection programmes designed to increase weight or eradicate leg problems.

11. Dyschondroplasia is highly heritable, and the incidence may be changed by selection. No evidence for the exclusive involvement of a single gene has been found, however.

12. Definite strain differences exist regarding incidence of dyschondroplasia, but no sign of a genotype x environment interaction was found.

13. A comparison of methods of estimating genetic parameters for 0-1 traits shows that robust estimates of heritability may be obtained using a modified proband technique, and genetic correlations using analysis of variance techniques.

14. Floor type, stocking density and hatch effects are all thought to influence the incidence of dyschondroplasia. It is suggested that a wide range of environmental variables may alter the susceptibility of birds to this condition.

15. Other leg problems have also been found to be inherited, and may be selected against. These appear to have positive genetic correlations with each other and with live-weight.

16. Dyschondroplasia is thought to be closely related to other leg problems, and it is suggested that all of these conditions have arisen as a consequence of the rapid growth rates found in the modern broiler.

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REFERENCES

BAKER, R.J. and NELDER, J.A. 1978 GLIM manual Release 3. Royal Statistical Society.

BEIRNE, M.J. and JENSEN, L.S. 1979 Influence of broiler breeder hen diet on incidence of leg disorders in offspring. Poultry Science 58, 1009 Abstract.

BERGMANN, von V. and SCHEER, J. 1976 Beitrage zur Differentialdiagnose der Bewegungsstorungen beim Junghun. 3 Mitt: Dyscondroplasie als Ursache von Schlactverlusten und Lahmheiten bei Broilern. Monatschafte fur Vetrinarmedizin 31, 576-581.

BURTON, R.W. and HOWLETT, C.R. 1979 A method for rapid radiographic assessment of tibial dyschondroplasia in broiler chickens. Journal of the American Veterinary Radiological Society. 20, 80.

BURTON, R.W., SHERIDAN, A.K., and HOWLETT, C.R. 1981 The incidence and importance of tibial dyschondroplasia to the commercial broiler industry in Australia. British Poultry Science 22, 153-160.

COCHRAN, W.G. 1940 The analysis of variance when experimental errors follow the Poisson or Binomial Laws. Annals of Mathematical Statistics 11, 335-347.

CRAIG, F.R. 1978 Leg problems and their economic implications. Proceedings of the National Meeting on Poultry Health and Condemnations 13, 18-23.

CUE, R.I.E. 1981 A genetic analysis of lamb mortality in hill sheep. Ph.D. Thesis, University of Edinburgh.

CUNNINGHAM, E.P. 1972 Theory and application of statistical selection methods. Proceedings of the 15th British Poultry

Breeders Roundtable, Birmingham. Nov. 14-16th.

EDWARDS, H.M. Jr. and VELTMANN, J.R. Jr. 1981 Influence of calcium and phosphorous levels of practical type rations on the incidence of tibial dyschondroplasia (TD) in broilers. Poultry Science 60, 1378 Abstract.

ELSTON, R.G. 1977 Response to query on estimating 'heritability' of a dichotomous trait. Biometrics 33, 232-233.

FALCONER, D.S. 1965 The inheritance of liability to certain diseases, estimated from the incidence amongst relatives. Annals of Human Genetics 29, 51-76.

FALCONER, D.S. 1967 The inheritance of liability to diseases with variable age of onset, with particular reference to diabetes mellitus. Annals of Human Genetics 31, 1-20.

FALCONER, D.S. 1981 Inroduction to quantitative genetics. 2nd Edition. Longmans, London.

FERGUSON, A.E., SUMMERS, J.D., LESLIE, A.J. and CARLSON, H.C. 1974 Leg deformities in chicken broilers. The Canadian Veterinary Journal 15, 185-190.

FERGUSON, A.E., LEESON, S., JULIAN, R.J. and SUMMERS, J.D. 1978 Leg bone abnormalities and histopathology of caged and floor reared broilers fed diets devoid of selected vitamins and minerals. Poultry Science 57, 1559-1562.

GRISTWOOD, A. 1980 Report on heritability of leg problems. - unpublished.

HARTMANN, W. 1979 Line and family effects on the incidence of 'twisted legs' in meat chickens. Proceedings of the British Poultry Breeders Roundtable, Glasgow Nov. 20-22nd.

HARVEY, W.R. 1977 User's guide for LSML76. Ohio State University.

HAYE, U. and SIMONS, P.C.M. 1975 Tibiale dyschondroplasie bij slachtkuikens in Nederland. Tijdschriff voor diergenskunde 100, 1048-1051.

HEMSLEY, L.A. 1970 A cartilage abnormality of broiler chickens. The Veterinary Record 86, 385.

HILL, W.G. and SMITH C. 1977 Alternative response to query on estimating 'heritability' of a dichotomous trait. Biometrics 33, 234-236.

HOWLETT, C.R. 1974 Morphological studies of tibial dyschondroplasia in the broiler chicken. Proceedings of the Australian Poultry Science Convention, Hobart, p87.

HOWLETT, C.R. 1979 The fine structure of the proximal growth plate of the avian tibia. Journal of Anatomy 128, 377-399.

HUFF, W.E. 1980 Evaluation of tibial dyschondroplasia during aflatoxicosis and feed restriction in young broiler chickens. Poultry Science 59, 991-995.

HULAN, H.W., PROUDFOOT, F.G., RAMEY, D. and McRAE, K.B. 1980 Influence of genotype and diet on general performance and incidence of leg abnormalities of commercial broilers reared to roaster weights. Poultry Science 59, 748-757.

ITAKURA, C., GOTO, M. and FUJIWARA, Y. 1973 Tibial dyschondroplasia in broiler chickens in Japan. Japanese Journal of Veterinary Science 35, 289-297.

LAURSEN-JONES, A.P. 1970 A cartilage abnormality of broiler chickens. The Veterinary Record 86, 445.

LEACH, R.M. Jr. 1982 Nutrition and tibial dyschondroplasia. Proceedings of the 31st. Western Poultry Disease Conference and 16th. Poultry Health Symposium, University of California,

Davis Feb 24th-Mar 3rd p59-61.

LEACH, R.M. Jr. and LILBURN, M.S. 1980 Effect of diet and environment on the incidence of tibial dyschondroplasia. Poultry Science 59, 1630 Abstract.

LEACH, R.M. Jr. and NESHEIM, M.C. 1965 Nutritional, genetic and morphological studies of an abnormal cartilage formation in young chicks. Journal of Nutrition 86, 236-244.

LEACH, R.M. Jr. and NESHEIM, M.C. 1972 Further studies on tibial dyschondroplasia (cartilage abnormality) in young chicks. Journal of Nutrition 102, 1673-1680.

LILBURN, M.S. and LEACH. R.M. Jr. 1980 Metabolism of abnormal cartilage cells associated with tibial dyschondroplasia. Poultry Science 59, 1892-1896.

LOWTHER, D.A. 1971 Cited by Sheridan et al (1974).

LOWTHER, D.A., ROBINSON, H.C., DOLMAN, J.W. and THOMAS, K.W. 1974 Cartilage matrix components in chickens with tibial dyschondroplasia. Journal of Nutrition 104, 922-929.

LUTFI, A.H-M. 1967 Growth and development of the upper end of the tibia of the domestic fowl (Gallus domesticus). Ph.D. Thesis, University of Belfast.

McCAPES, R.H. 1967 Lameness in turkeys due to faulty bone formation. Animal Nutrition and Health 22, 17.

MERKELEY, J.W. 1979 Tibial dyschondroplasia in broilers. Poultry Science 58, 1021 Abstract.

MOEN, R. 1968 Selection index for performance tested boars - unpublished report cited by Cunningham (1972).

MONGIN, P. and SAUVEUR, B. 1977 Interrelationships between mineral nutrition, acid-base balance, growth and cartilage abnormalities.

In Growth and Poultry Meat Production 235-247 ed. Boorman K.N. and Wilson B.J., British Poultry Science Ltd., Edinburgh.

NAIRN, M.E. 1969 Osteomyelitis-synovitis complex of turkeys. Proceedings of the California Conference on Leg Weakness in Turkeys, University of California p19.

OLAUSSON, A. and RÖNNINGEN, K. 1975 Estimation of genetic parameters for threshold characters. Acta Agriculturae Scandinavica 25, 200-208.

POULOS, P.W. Jr. 1978 Tibial Dyschondroplasia (osteochondrosis) in the turkey. Acta Radiologica Supplement 358, 197-227.

POULOS, P.W. Jr., REILAND, S., ELWINGER, K. and OLSSON, S-E. 1978 skeletal lesions in the broiler, with special reference to dyschondroplasia (osteochondrosis). Acta Radiologica Supplement 358, 229-375.

PRASAD, S., HAIRR, W.T. and DALLAS, J.T. 1972 Observations of abnormal cartilage formation associated with leg weakness in commercial broilers. Avian Diseases 16, 457-461.

RANDALL, C.J. and MILLS, C.P.J. 1981 Observations on leg deformity in broilers with particular reference to the intertarsal joint. Avian Pathology 10, 407-431.

REICH, T., JAMES, J.W. and MORRIS C.A. 1972 The use of multiple thresholds in determining the mode of transmission of semi-continuous traits. Annals of Human Genetics 36, 163-184.

REILAND, S., POULOS, P.W. Jr., HANSEN, H-J,. LINDGREN, N-O. and OLSSON S-E. 1977 Tibial dyschondroplasi hos broiler - ett delfenomen i en generaliserad sjukdom - osteochondros. Svensk Veterinartidning 29, 123-127.

REILAND, S., OLSSON, S-E., POULOS, P.W. Jr. and ELWINGER, K. 1978

Normal and pathological skeletal development in broiler and Leghorn chickens. Acta Radiologica Supplement 358, 277-298.

RICHET, G., ARDAILLOU, R. and AMIEL, C. 1966 Cited in Sauveur and Mongin (1978).

RIDDELL, C. 1975 The development of tibial dyschondroplasia in broiler chickens. Avian Diseases 19, 443-462.

RIDDELL, C. 1975 Studies on the pathogenesis of tibial dyschondroplasia in chickens. 1. Production of a similar defect by surgical interference. 2. Growth rate of long bones. 3. Effect of body weight. Avian Diseases 19, 483-505.

RIDDELL, C. 1976 Selection of broiler chickens for a high and low incidence of tibial dyschondroplasia with observations on spondylolisthesis and twisted legs (perosis). Poultry Science 55, 145-151.

RIDDELL, C. 1977 Studies on the pathogenesis of tibial dyschondroplasia in chickens. 4. Some features of the vascular supply to the growth plate of the tibiotarsus. Avian Diseases 21, 9-15.

RIDDELL, C., HOWELL, J. and KAYE, M.M. 1971 Tibial dyschondroplasia in broiler chickens in Western Canada. Avian Diseases 15, 557-565.

ROBERTSON, A. 1977 The effect of selection on the estimation of genetic parameters. Zeitschrift fur Tierzuchtung und Zuchtungsbiologie 94, 131-135.

ROBERTSON, A. and LERNER, I.M. 1949 The heritability of all-or-none traits: viability of poultry. Genetics 34, 395-411.

SAUVEUR, B. 1969 Cited in Sauveur and Mongin (1978)

SAUVEUR, B. and MONGIN, P. 1978 Tibial dyschondroplasia, a cartilage

abnormality in poultry. Annales Biologie, Animale Biochemie et Biophysique 18, 87-98.

SHERIDAN, A.K. 1974 Genetic studies of tibial dyschondroplasia in the broiler chicken. Proceedings of the Australian Poultry Science Convention, Hobart p86.

SHERIDAN, A.K. 1979 Summary of leg weakness experimental results - unpublished report.

SHERIDAN, A.K., HOWLETT, C.R., and BRUYN, J.A. 1974 Genetical factors influencing tibial dyschondroplasia in Australian meat chickens. Proceedings of the 15th World Poultry Congress, New Orleans p34-35.

SHERIDAN, A.K., HOWLETT, C.R, and DETTMANN, E.B. 1976 Selection for tibial dyschondroplasia in Australian meat chickens. Proceedings of the 1st Australian Poultry and Stock Food Convention, Melbourne Oct. 10-15th. 2.

SHERIDAN, A.K., HOWLETT, C.R. and BURTON, R.W. 1978 The inheritance of tibial dyschondroplasia in broilers. British Poultry Science 19, 491-499.

SILLER, W.G. 1970 Tibial dyscondroplasia in the fowl. Journal of Pathology 101, 39-46.

SILLER, W.G. and DUFF, R.H. 1970 Cartilage abnormality in broiler fowls. The Veterinary Record 86, 757.

SIMONS, P.C.M. 1979 Twisted legs in broilers. Proceedings of the British Poultry Breeders Roundtable, Glasgow Nov. 20-22nd.

SMITH, C. 1980 The prediction of frequency of discrete traits in the progeny of extreme individuals. - unpublished.

SNEDECOR, G.W. and COCHRAN, W.G. 1967 Statistical Methods 6th Edition. Iowa State University Press.

SOMES, R.G. Jr. 1969 Genetic perosis in the domestic fowl. Journal of Heredity 60, 163-166.

STEINKE, F.H. 1971 Osteodystrophy - nutritional and related factors Proceedings of the Symposium of Leg Weakness in Turkeys, Iowa State University p30-36.

SUMMERS, J.D, LEESON, S. and FERGUSON, A.E. 1978 Performance and leg conditions of caged and floor reared broilers fed diets deficient in selected vitamins and minerals. Poultry Science 57, 506-512.

TALBOT, H. 1981 An introductory guide to GENSTAT. Edinburgh Regional Computing Centre, Edinburgh University.

TURNER, H.N. and YOUNG, S.S.Y. 1969 Quantitative genetics in sheep breeding. Macmillan of Australia.

VELTMANN, J.R. Jr. and JENSEN, L.S. 1980 Variations in the incidence of tibial dyschondroplasia associated with different environmental conditions. Avian Diseases 24, 625-630.

VELTMANN, J.R. Jr. and JENSEN, L.S. 1981 Tibial dyschondroplasia in broilers: comparison of dietary additives and strains. Poultry Science 60, 1473-1478.

WALSER, M.M., ALLEN, N.K., MIROCHA, C.J., HANLON, G.F. and NEWMAN, J.A. 1982 Fusarium-induced osteochondrosis (tibial dyschondroplasia) in chickens. Veterinary Pathology 19, 544-550.

WISE, D.R. 1975 Skeletal abnormalities in table poultry - a review. Avian Pathology 4, 1-10.

WISE, D.R. and JENNINGS, A.R. 1972 Dyschondroplasia in domestic poultry. The Veterinary Record 91, 285-286.

WISE, D.R. and JENNINGS, A.R. 1973 The development and morphology of the growth plates of two long bones of the turkey. Research in Veterinary Science 14, 161-166.

WISE, D.R. and NOTT, H. 1975 Studies on tibial dyschondroplasia in ducks. Research in Veterinary Science 18, 193-197.

APPENDIX

DEEBLE, F.K., MERCER, J.T. and HILL, W.G. 1982 A comparison of the conformation assessment systems used for Friesian and Holstein heifers in Britain. Proceedings of the British Cattle Breeders' Club Winter Conference. Digest No. 37 p1-8.

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A COMPARISON OF THE CONFORMATION ASSESSMENT SYSTEMS USED FOR FRIESIAN AND HOLSTEIN HEIFERS IN BRITAIN

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INTRODUCTION

Early in 1978 the Government completed a review of the standards used for the selection of bulls for use in artifical insemination (AI) in Great Britain. In July, Agriculture Ministers announced that the Committee on the Artificial Insemination of Cattle would be set up to bring together representatives of organisations in Great Britain interested in artificial insemination of cattle, independent scientific experts and officials of the three Agricultural Departments in Great Britain. In December 1978 the appointment of Sir Richard Trehane as Chairman was announced together with the Committee's terms of reference. One of these was to develop a comprehensive standardised system of information on both production and conformation features of the progeny of dairy breed bulls. The Committee first met on 26 April 1979 and decided that devising a standardised information system for production traits should be given a higher priority than that for the assessment of conformation. The Committee considered that a standard information system implied that the information, from different sources, made available to users could be readily compared. This presupposed that the information had been:

(a) collected in the same way to the same standards of accuracy,
(b) subjected to the same analytical or aggregating procedures or, alternatively, that information which did not meet criterion(a) had been subjected to valid scaling procedures so that the information from one source could be compared with that from another source.

CONFORMATION ASSESSMENT OF DAIRY CATTLE

The Committee studied the two broad groups into which dairy conformation assessment systems in Britain fell: those carried out by Breed Societies and those made by members of the Artificial Insemination Organisation. It noted that the differences between the systems, both in principle and detail, arose mainly because the breed society schemes were developed to meet the needs of pedigree breeders for information on females, which was aggregated subsequently for the evaluation of their sires, whereas members of the AI Organisation were concerned only with sire evaluation primarily to supply information to their customers. The Committee noted also that breed societies and members of the AI Organisation usually assess different progeny of the same bull. The former operated in the pedigree herds and the latter, primarily, in the non-pedigree sector: only some 15% of the black and white first lactation females assessed by Milk Marketing Board (MMB) staff, for the purpose of the Dairy Progeny Testing Scheme (DPTS) sire evaluation were registered with breed societies. Furthermore, assessments by AI Organisation staff were of first lactation females only whereas the breed societies assessed both first and later lactation females. The systems also differed, but in varying degrees, over the characteristics (traits) or parts of the animal's body assessed, the score card format used, the proportions of the total score allocated to the same part or

characteristic of the animal assessed and the standard used as the basis of the assessment.

THE POSSIBILITIES FOR STANDARDISED INFORMATION ON CONFORMATION

The Committee concluded that it would not be easy to obtain agreement to a standardised information system to accord with the principles given above. A degree of uniformity was, however, developing between some breed societies and members of the AI Organisation by the use of the same score card layout, the same range of scoring and holding joint workshops of assessors from breed societies and the AI Organisation. It was decided that the problem was more acute in respect of black and white cattle than other dairy breeds since the numbers involved were larger and the differences in the systems were greater. The British Friesian Cattle Society (BFCS) and members of the AI Organisation assessed virtually the same traits but the British Holstein Society (BHS) assessed different traits in the main and used different methods of assessment. Thus it was possible for one assessment of the progeny of a Holstein bull to be published by BHS, another by members of the AI Organisation and a third by BFCS and for each not to be directly comparable with the others. The Committee agreed that whilst it would be desirable to achieve a common system of type assessment and score card for all breeds it was not practicable to make such a recommendation. It agreed also that it would not be practicable to recommend establishing a new organisation to be responsible for all type assessments for sire evaluation purposes. It could not accept the retention of the status quo however and decided that it would explore the possibility of using a mathematical technique either to bring together the information from the present schemes into a single set of figures for each bull or to provide a basis for comparison of the separate results for each bull under the different schemes.

The Committee had first, therefore to establish whether or not the organisations ranked cattle in the same order so as to provide the basis for the comparability. It noted that the basis for a continuing comparability assumed that assessors would continue to assess cattle in the future in the same way as they did in establishing the basis of the comparability.

A classification trial was therefore set up to:

(1) ascertain whether assessors from the four organisations, Associated Artificial Insemination Centres (AAIC's), British Holstein Society (BHS), British Friesian Cattle Society (BFCS) and the Milk Marketing Board (MMB), generally rank animals in the same order and with the same range of scores;

2. Find a mathematical way of combining the information from the diffferent sources into one assessment or, alternatively, a way of scaling the information so that the assessments are made comparable. Either method would be expected to provide reliable information for semen users.

The Committee was advised that to meet its objectives it should obtain conformation assessment records on some 200 first lactation animals by several inspectors from each organisation. It would then be possible to compare classifications by inspectors from different organisations with those by inspectors from the same organisation.

DESIGN AND DATA PREPARATION

The type-conformation assessment trial was carried out from 5th-7th May, 1981 on farms in Wiltshire and Dorset, with the valued help and co-operation of six breeders. These were chosen so that there were two farms of each of the following herd types represented: Pedigree Friesian, Pedigree Holsteins and D.P.T.S. herds

(those taking part in the progeny testing of young sires). Approximately the same number of animals were classified on each farm. These were mostly heifers, but included a small number of second calvers (Table I) which have been ignored in the subsequent analyses, because the benefits could not justify the complications. Four classifiers from each of the four organisations inspected each animal at the same time, and used the form and procedure normally adopted by his own organisation.

The classifications by BFCS, AAIC and MMB were on a numerical scale up to 10 for the same nine traits, with breed character also assessed by BFCS (Table II). Classifications by BHS were, except for a numerical final score out of 100, on a letter grade for a somewhat different set of traits. For the purpose of the analyses these were converted into numbers using a formula suggested by BHS (Table III). An alternative scale was also used for the statistical analyses but as this did not affect any of the conclusions, the results are not reported. For the purpose of the combined analyses some manipulation of the scores had to be carried out. Those for feet and legs (FL) and mammary systems (MS) awarded by BHS were repeated to represent individual scores for feet (F) and legs (L), and the two teat characteristics respectively. The converse was also applied, and the mean scores of F and L together, and teat size, shape and quality (T.S.Q.) and teat position (T.P.) together with BFCS, AAIC and MMB, were taken to represent FL and MS respectively. In addition the rump score (R) given by BHS was made equivalent to the top line and rump score (TLR) given by BFCS, AAIC and MMB.

ANALYSES BY ORGANISATIONS SEPARATELY

(i) Means and Variability of Scores

The mean scores for each organisation are shown in Table IV. There are large and obvious differences among those using the same numerical scoring system; the most extreme are for feet. Differences between mean scores of different classifiers from the same organisation were found, but were generally smaller. In combining data for sire evaluation purposes, these differences in mean scores between organisations or classifiers are not important **provided** that comparisons are made amongst contemporaries in the same herd inspected by the same person - "within herd/visit comparisons." As an indication of the degrees of variability in the scores awarded by the different organisations, the standard deviations of the scores have been presented together with the means. Analyses not presented here have shown that there is reasonable agreement in the level of variability shown by separate classifiers within each organisation, so data from these have been considered together subsequently. It is evident, however, that variability differed from organisation to organisation. In combining data for sire evaluation these differences in the spread of scores cause problems, which cannot be resolved by applying the normal best linear unbiased predictions (BLUP) procedure. To overcome these it would be necessary to use a transformation which makes the scoring systems more similar in any "combining" procedure.

(ii) Correlation of Scores by Different Classifiers

The degree to which individual classifiers from the same organisation agree in their assessments can be estimated from the correlation (formally the intra-class correlation) of their scores on each cow. A correlation of 100% would imply complete agreement, 0% no agreement. Results are presented for each trait in Table V. They reflect the subjective nature of all type classifications, because typical correlations of 50% imply there is as much variation in scores attributable to differences in opinion among classifiers as there are differences between cows, even after correcting for the average scores. Generally classifications carried out on both head, neck and shoulder and legs showed the poorest level of agreement, whilst those on udder traits showed better agreement. There are some differences in

the degrees of correlation displayed by the various organisations. The results in Table V are directly applicable only to the classifiers used in the trial and another "sample" from each organisation may have agreed better or worse with each other. Further, without reclassification of the same animal by each inspector it was not possible to tell how repeatable were individual classifications. Nevertheless the results imply that there may well be a need for more classification workshops in each organisation.

Similar analyses were conducted within each herd type, and from these it was concluded that there was no evidence to suggest that:

(a) BFCS classifiers agree best in Friesian herds,
(b) BHS classifiers best in Holstein herds,
(c) MMB and AAIC best in DPTS herds.

(iii) Effects of Age and Stage of Lactation

Some attempt was made in the above analyses to correct for the effects of age of cow and time since calving, factors which the classifiers are supposed to take into account in their allocation of scores. The results obtained suggest that for at least some traits, and particularly for body capacity in relation to age, adjustment should be made statistically for the purposes of sire evaluation, rather than being left to the individual classifier. No attempt was made to correct for these factors in any of the combined analyses.

ANALYSIS BY ORGANISATIONS COMBINED

(i) Cow x organisation interaction

The objective of this, the main analysis, was to test whether there were differences between organisations in the way individual cows were rated (technically to test whether there was a substantial cow x organisation interaction) after allowing for differences in scores of classifiers from the same organisation (a cow x classifier interaction). As shown previously (Table IV), the variability of scores differed between organisations, and if this were not corrected for in the analysis, a cow x organisation interaction would appear even if cows were ranked the same by each organisation. The data were therefore combined by dividing the scores given to each cow by the standard deviation for cows, calculated for each organisation for each trait, and estimated in constructing Table V (i.e. by the square root of the variance due to cows). The analyses were performed both including and excluding BHS in order to compare the numerically scoring organisation only. After the transformation a comparison of means becomes pointless. The variability between scores was partitioned into that due to individual cows, that due to cow x organisation interaction and that due to cow x classifier organisation (Table VI). The proportion due to cows was the correlation of scores by classifiers from different organisations and that due to cows plus cows x organisation was the correlation of scores by classifiers from the same organisation (comparable to Table V).

As can be seen, a proportion of the variation for all traits was due to organisation x cow interactions, these tending to be larger when the scores for BHS were included. Although small, all these cow x organisation interactions were statistically significant. This may be partly accounted for by the facts that a different grading scheme was used for BHS with fewer values, and that also the traits examined were not exactly the same. However, these cow x organisational effects are smaller than those due to cow x classifiers, i.e. those arising from the decisions on the day by individual classifiers within organisations.

(ii) Genotype x organisation interactions

There is some indication from the trial, from comparing mean scores on each herd type (Holstein, Friesian or DPTS) for each organisation, that genetic groups are

ranked differently by the four organisations, but this is not conclusive because different herds were involved, rather than progeny groups in the same herd. An analysis was also performed in which sire family effects were included on data from four herds which used several sires (Table I). This gave no evidence that sire families were assessed differently by the different organisations, but the low numbers of sires and daughters included limited the power of this analysis. (The trial was not designed to make tests at the sire level). Even if the small differences in ranking of individual cows by AAIC, BFCS and MMB do also apply to sire progeny groups, it still seems appropriate to pool their data. However, the inclusion of data from BHS would require, in the first place, agreement as to the exact traits to be classified. Unpublished comparisons of sire assessments using BLUP or "ICC type" by MMB and BFCS have established rather different base values. Adjustment for this would have to be made in any combination of data.

SUMMARY AND CONCLUSIONS FOR SIRE EVALUATION

1. (i) There are substantial differences between the organisations in the mean scores allocated to the various traits, but these can be eliminated in sire evaluations using contemporary comparison procedures.

   (ii) The variability of scores differs among the numerically classifying organisations and between them and BHS. For the former this can be overcome in pooling data for sire evaluation by appropriate scaling.

   (iii) Different classifiers from the same organisation showed a considerable lack of agreement given to traits, especially for legs and H.N.S. Further "workshop" sessions could help to improve the level of agreement.
2. Although there are significant organisation x cow interactions, these are small in comparison to classifier x cow interactions, especially when only BFCS, MMB and AAIC are considered together. Thus it would still be justifiable to combine data from these organisations.
3. Unless there was to be agreement on the actual traits to be assessed, it would not be possible or justifiable to combine data produced under the BHS assessment system with that produced under the systems operated by the other three organisations.
4. Combination of the data would need to cater for the possibility that the reference bases used by BFCS, MMB and AAIC are substantially different unless the data were analysed as one set.

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Table I. Numbers Classified and Numbers of Sires Represented.

Type of herd	Farm Number	No. of heifers (number of sires)	No. of second calvers	Total
PEDIGREE HOLSTEIN	1	33 (4)*	0 -	33
	4	27 (9)	7 (4)	34
D.P.T.S.	2	28 (4)*	7 (2)	35
	3	23 (10)	10 (5)	33
PEDIGREE FRIESIAN	5	35 (8)	0 -	35
	6	27 (9)	0 -	27
TOTAL		173	24	197

*One bull only largely used in the herd sample.

Table II. Traits examined with abbreviations used.

	AAIC, BFCS & MMB		BHS
HNS	- Head, Neck & Shoulders	GA	- General Appearance
B.Ca	- Body Capacity	DC	- Dairy Character
TLR	- Top line and Rump	B.Ca	- Body Capacity
L	- Legs	R	- Rump
F	- Feet	FL	- Feet and Legs
FU	- Fore Udder	MS	- Mammary system
RU	- Rear udder	FU	- Fore udder
TSQ	- Teat shape/size & quality	RU	- Rear udder
TP	- Teat position	FC	- Final class
FS	- Final Score (calculated from above)	FS	- Final Score (numerical)
BC	- Breed Character (BFCS only)		

Table III. Conversion of BHS Letter Grades to Scores

Grade	EX	VG	GP	G	F	P
Normal Range	100-90	89-85	84-80	79-75	74-65	Less than 65
Value suggested by BHS	95	87	82	77	70	55
Scaled for one trait	9.5	8.7	8.2	7.7	7.0	5.5

Table IV. Organisation Means and Standard Deviations.

	Means			Standard Deviations				Means	Standard Deviations
	B.F.C.S.	A.A.I.C.	M.M.B.	B.F.C.S	A.A.I.C.	M.M.B.		B.H.S.	B.H.S.
B.C.	6.96	-	-	0.81	-	-	F.C.	76.07	3.91
H.N.S.	7.61	6.83	7.31	0.65	1.18	0.64	F.S.	75.86	2.97
B.Ca.	8.01	7.08	7.49	0.78	1.16	0.89	G.A.	7.61	0.39
T.L.R.	6.98	6.74	7.41	0.94	1.35	0.83	D.C.	7.95	0.41
L.	6.69	5.99	6.67	0.81	1.15	0.84	B.Ca.	8.15	0.37
F.	6.94	5.29	6.37	0.93	1.43	1.13	R.	7.82	0.54
F.U.	6.90	5.75	6.33	1.04	1.59	1.15	F.L.	7.91	0.32
R.U.	7.03	6.08	6.37	1.11	1.40	1.13	M.S.	7.71	0.36
T.S.Q.	7.04	6.62	7.11	1.09	1.28	1.01	F.U.	7.72	0.42
T.P.	6.57	6.13	6.13	1.36	1.56	1.05	R.U.	7.77	0.34
F.S.	70.73	56.51	61.19	5.32	6.76	4.76			

Table V. % Correlations of Scores by different Classifiers.

	ORGANISATION				
	B.F.C.S.	A.A.I.C.	M.M.B.		B.H.S.
F.S.	71.6	65.6	79.9	F.S.	69.7
B.Ca	67.3	58.2	68.3	B.Ca.	50.3
F.U.	60.7	64.5	76.3	F.U.	58.2
R.U.	55.6	58.7	67.2	R.U.	39.9
B.C.	51.6	-	-	F.C.	55.1
H.N.S.	44.4	28.2	39.4	G.A.	53.9
T.L.R.	63.7	58.9	69.0	D.C.	51.6
L.	34.4	35.0	55.3	R.	35.3
F.	59.4	50.4	75.4	F.L.	34.3
T.S.Q.	63.0	48.5	72.5	M.S.	54.5
T.P.	71.2	66.7	72.3		

Table VI. Percentages of Variation in Scores attributable to (A) differences between cows alone, (B) organisational differences in scoring and (C) differences in opinion between individual classifiers.

	4 Organisations Combined				3 Organisations Combined		
	Cows	Organisations	Classifiers		Cows	Organisations	Classifiers
B.Ca.	58.4	5.9	35.7	B.Ca.	60.4	7.4	32.2
T.L.R.	47.2	8.6	44.2	T.L.R.	62.6	2.6	34.8
L.	33.9	6.5	59.6	L.	39.0	2.6	58.4
F.	43.9	10.0	46.1	F.	60.1	3.5	36.4
F.U.	54.2	12.1	33.7	F.U.	62.3	6.1	31.6
R.U.	46.6	9.7	43.7	R.U.	58.4	3.4	38.2
T.S.Q.	42.3	19.9	37.8	T.S.Q.	57.7	4.2	38.1
T.P.	53.7	13.8	32.5	T.P.	66.8	4.2	29.0
F.S.	63.3	11.2	25.5	F.S.	71.1	4.1	24.8
F.L.	43.5	7.6	48.9	H.N.S.	34.4	5.8	59.8
M.S.	51.9	16.1	32.0				