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Feathering in commercial poultry II. Factors influencing feather growth and feather loss

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In commercial production, there is often concern about the quantity and/or quality of feathering in both broilers and layers. For broilers, the concern is adequacy of protective feather cover, while in layers it is usually the necessary degree of feathering needed to optimise feed efficiency. Feather development is under the control of hormones such as thyroxine and oestrogen and indirectly by testosterone. Environmental or nutritional status that influences such hormonal output will indirectly affect feathering. In broilers, rate of feathering is influenced by genetics, since some 20 years ago there was a conscious decision to introduce slow (K) *vs.* fast (k) feathering as a means of sexing day-old chicks. With the relative "immaturity" of modern broilers, these genes influence feather cover well into the production cycle. In White Leghorn crosses, initial problems with apparent Leukosis susceptibility of the progeny of slow feathering dams had to be overcome by eradication of Leukosis before feather sexing could be generally introduced. Nutrition can influence rate of feathering as well as feather structure, colour and moulting. Amino acid balance and especially deficiencies of TSAA and branched chain amino acids will influence feathering in young birds. Deficiency of vitamins and certain trace minerals also induce characteristic feather abnormalities, as does the presence of dietary mycotoxins. A number of viruses, bacteria and mycoplasma can infect the feather follicle and so influence feather development. Feather pecking and feather licking are behavioural abnormalities, although these conditions can be induced by changes in environmental conditions or nutritional adequacy of the diet.

Keywords: feathers; broilers; layers; genetics; nutrition; environment

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Hormonal balance and feather growth

Thyroid hormones and the gonadotrophins have a major influence on feather growth and development. Follicle cell growth is under the direct action of thyroid hormones, although the primary wing feathers are little affected by this hormone. Down feathers in the young chick also develop without the influence of thyroid metabolism, but all subsequent feather generations are affected. Thyroidectomy, or the incorporation of thyroid antagonists in the diet, causes cessation of feather growth, although with the latter, the effect is reversible if normal nutritional status is resumed. In situations of an atrophied thyroid, young birds can be almost naked with the notable exception of some development of wing feathers (Schwarz, 1930 as cited by Spearman, 1971).

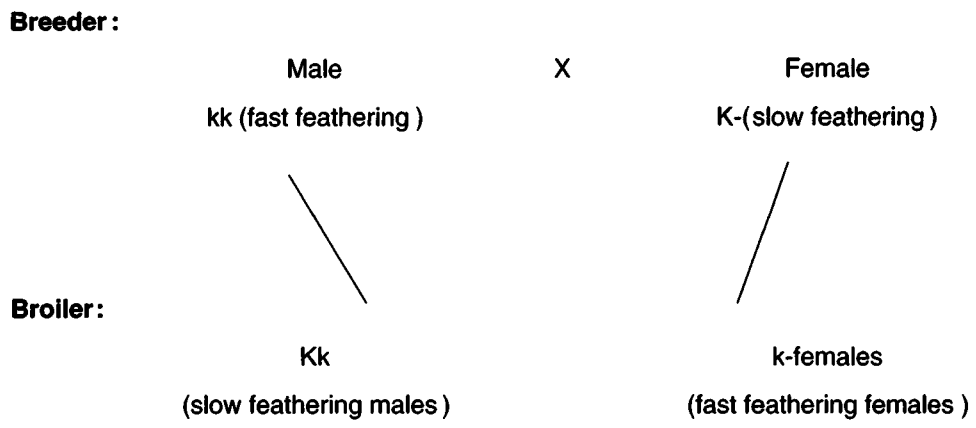
Thyroid imbalance is not likely to occur or to be diagnosed in commercial poultry, although the role of thyroid antagonists such as glucosinolates in rapeseed meal has not been studied. The condition is seen periodically in pet birds where diffuse feather loss, rough feathering and loss of feather pigments is seen in psittacines. In these situations, thyroid replacement therapy usually consists of 40 μg/kg Bwt/day or 1.7 mg thyroxine per litre drinking water (Olgesbee, 1992). Castration or ovariectomy of any bird results in continuous growth and renewal of the entire plumage, rather than an annual moult.

Of the gonadotrophins, oestrogen (presence or not) has the greatest effect on feather appearance. Feather formation will be delayed by high levels of oestrogen. In adult birds, the initial stages of feather growth are influenced by thyroxine levels as previously described, although development of the emerging feather is greatly influenced by oestrogen. In most birds, the presence of oestrogen results in the typical female feather shape while absence of oestrogen causes typical male feathering. Testosterone *per se* usually has little effect on feathering and so it is the absence of oestrogen, rather than the presence of testosterone that causes typical male feathering characteristics. Exceptions to this situation occur in the Campine and Sebright strains of chicken, where the feather follicle is equally responsive to testosterone and oestrogen (Spearman, 1971). In these strains, both sexes have female type feathering underlying an obvious genetic basis for feathering characteristics in poultry.

Moulting is also associated with elevated levels of plasma progesterone, although this is not likely a causal situation. Progesterone levels do decline as the new feathers emerge. Thyroxine levels are also elevated during a moult and then they decline during feather regeneration. Conversely, the levels of tri-iodothyronine are low during initiation of a moult and then gradually increase as feathers start to grow (Pethes *et al.*, 1982). Cortisone administration has also been shown to suppress feather growth (Siegel *et al.*, 1989) with the effect being more pronounced in broiler than in layer strains.

Genetics of feather growth

Choice of broiler strain can have a profound effect on feathering because most breeding companies offer a choice of feather-sexable birds. It has been recognized for many years that a single recessive sex-linked gene controls the rate of feather growth, especially in the very young chick. After introducing this gene into primary breeding stock, the two sexes have different rates of feathering that allow for the determination of sex in the newly hatched chick. Slow feathering (K) is dominant to fast feathering (k). Males carry two sets of sex chromosomes, and females only one. Males can therefore be KK or Kk (slow feathering), or kk (fast feathering). Females, on the other hand, can only be K- (slow feathering) or k- (fast feathering). In the breeding program, a male that is homozygous for fast feathering (kk) is crossed with a slow feathering (K-) female:



At the breeder level, therefore, it is the female chicks that are expected to be slower feathering than are the males, while, for the broiler offspring, the females will be fast feathering and can be differentiated from the males simply by the extra length of their primary feathers. This differential rate of feathering is less pronounced even at 3-4 days of age, although slow feathering male chicks will always be retarded in feathering compared to non feather-sexable, fast-feathering male broilers. Because about 90% of broiler chicks today are feather sexable, producers must accept a male broiler that is somewhat slower to feather. Chambers *et al.* (1994) provide an excellent review of sex-linked feathering.

The genes involved essentially delay the emergence of the feather more so than the subsequent growth of the feathers. As adults, therefore, or even as 7-8 week old broilers, the fast *vs.* slow feathering broilers are virtually indistinguishable. If there are differences, it will invariably be seen in the length of the tail feathers, because late feathering males do not normally show extensive tail feather development until 17-20 days of age. There has been considerable discussion about the effect of sex-linked feathering on nutrient requirements. Because feathers contain a high level of cystein it is suggested that fast-feathering female chicks have a greater need for sulphur amino acids. A slight complication to this concept arises from the observation of Zelenka *et al.* (1992) showing that methionine digestibility is higher in fast feathering females compared to slow-feathering males.

The same genes have been introduced into commercial egg layers, although a number of problems have been identified with this breeding programme. It has been suggested that slow-feathering genotypes have reduced egg production (Harris *et al.*, 1984) that may be associated with leucosis. Even the fast-feathering female progeny from these slow feathering dams are sometimes affected, suggesting problems of maternal egg transmission. It has since been determined that the slow-feathering gene is intimately associated with the ev-21 gene, which is an endogenous gene that can be responsible for producing infectious virus particles. Such endogenous genes can compromise the bird's immune response to field strains of avian leucosis virus—effectively the bird recognizes the field strain similar to the ev-21 gene and therefore, does not react to this. Because of such immune tolerance problems, many breeding companies have removed the slow-feathering gene from their primary egg type breeders. Dunnington and Siegel (1986) suggested that genetically fast-feathering chicks were more resistant to *E. Coli* infection. There have been studies on developing sex-linked feathering traits in other poultry species such as guinea fowl (Pal and Singh, 1997) and turkeys (Zakrzewska and Savage, 1997) although these have yet to gain commercial acceptance.

Feather colour is also affected by sex-linked genes and so this trait offers potential for chick sexing. Starting in the 1920's, the era of "autosexing" came into being, where various crosses were established to differentiate the sex of chicks by the colour or pattern of their feathering. Such characteristics as barring, gold/silver, spangling and lacing were commonly used for autosexing (Smyth, 1990). Today few commercial colour-sexed strains of bird remain.

The only other gene that has been intentionally introduced into breeding stock that relates to feathering is the naked-neck (Na) gene. The Na gene is a single autosomal dominant gene that causes almost complete lack of feathers in the neck region. The main characteristic of such birds is greater resistance to heat stress (Cahaner *et al.*, 1994), presumably due to enhanced heat dissipation through the exposed skin. These same workers showed that the frizzle gene (F), which causes feathers to turn away from the body and expose more skin, was additive to the Na gene in promoting resistance to heat stress.

Most other genetically related feathering characteristics are unintentional and generally cause problems with feather growth or development. McGibbon (1977) described a sex-linked mutation in chickens that affected rate of feathering. A barebacked cockerel was observed at 6 weeks of age and subsequent mating with various types of hens showed a new allele. The "slow" feathering gene is given the symbol Ks and is dominant to the more common K (slow feathering) allele described earlier. Somes (1969) also described a mutant, termed Kn, where birds were almost naked. The dominance seems to be $K^n \rightarrow K^S \rightarrow K \rightarrow k$. The occurrence of some naked back or naked broiler chickens seen in commercial flocks resembles the photographs given by Somes (1969) and McGibbon (1977).

Bitgood *et al.* (1987) describes the location of the tardy feathering gene (t), which also causes slower than normal rate of feathering in young birds. Genetic abnormalities can also influence feather appearance. Urrutia *et al.* (1983) described a severe abnormality in the development of primary feathers in growing birds. Although chicks appear normal at hatch, by 28 d they have unusual feather follicle enlargement leading to necrosis and eventual feather loss. As adults, the birds lack a variable number of primaries while other feathers may be smaller than usual or show bent or knotted vanes. The birds appear very rough and a nutritional deficiency may be suspected. However, Urrutia *et al.* (1983) showed the condition to be due to a single autosomal recessive gene mutation, producing the name dysplastic remiges with the symbol *dr*.

Nutrition and feather growth

Diet nutrient profile, feed intake, antinutrients and various feed additives can all influence feather development to some extent. Because of the high content of protein and amino acids in feathers, these nutrients have received considerable attention. Using a simple feather litter score system, Twining *et al.* (1976) showed that crude protein content of the diet had a direct effect on feather regeneration. More feathers were found on the litter when a high protein diet was fed, being indicative of greater feather growth and development and subsequent moult. Deficiency of crude protein is known to have the opposite effect, where poorly feathered chicks will invariably be seen if birds younger than 10-15 d of age are given diets with much less than 16% crude protein. This latter situation sometimes occurs with broiler breeder chicks and seems to be a protein effect *per se* since use of relatively high levels of supplemental amino acids does not correct the problem. This situation suggests that either there is a requirement for protein *per se* for feathers, or that we have not correctly established the optimum balance of amino acids needed for feather growth independent of other traits such as growth and feed efficiency.

The major amino acids involved in the synthesis of feather keratin are the sulphur containing amino acids, cystine and methionine (Wheeler and Latshaw, 1981). Cystine is the major component of keratin while methionine is involved through its conversion to cystine (Champe and Maurice, 1984). This conversion is believed to occur in both the feather follicle and in the liver (Moran, 1984). In addition to their role in feather synthesis, methionine and cystine are also required for general maintenance and growth (Wheeler and Latshaw, 1981), and in most instances the latter needs are greater than that of feather development. The relative proportion of the sulphur containing amino acids is much greater in the integument than in muscle tissue and so, marginal dietary deficiencies of these amino acids will often be initially manifested as abnormal feathering (Moran, 1984). Although abnormal feathering can be used as an aid in diagnosing certain nutritional deficiencies, our experience suggests this to be non-specific, such that antagonisms, deficiencies and/or imbalances of various amino acids often result in similar gross abnormalities of the plumage. For example, imbalance of leucine (Penz *et al.*, 1984), valine (Farran and Thomas, 1992) or even glycine, phenylalanine and tyrosine (Robel, 1977) all cause similar types of feather abnormalities in young chicks. In most situations, the appearance of affected chicks on such deficient or imbalanced diets is best described as "rough feathering" and upon closer examination the body feathers are often sticking out from the body while primary wing feathers are invariably unsheathed closer to the body.

Because methionine and cystine are frequently the first limiting dietary amino acids, accurate determination of requirements for sulphur-containing amino acids during various stages of growth are necessary to ensure proper feather growth. This situation becomes most critical with diets formulated specifically for feather-sexed broilers (Moran, 1984). Wheeler and Latshaw (1981) conducted a study to determine the bird's total sulphur containing amino acid (TSAA) requirements and the replacement value of cystine for methionine. During the period of rapid feather development, maximum feed efficiency was obtained with cystine representing 54% of TSAA. Once feather development was complete, the need for cystine declined. With feather-sexable broilers, therefore, there is the potential for differential feeding programmes for male and female birds related to rate of feathering. However, under commercial conditions, one must also take into account other variables that can influence feather growth, such as environmental temperature and anticoccidial use, and so absolute guidelines are often misleading.

Tsiagbe *et al.* (1987) showed that methionine and cystine levels could influence the texture of feathers. Adding 0.20% cystine to a basal diet containing 0.37% cystine with 0.35% methionine caused feathers to be stronger. Adding a large dose of extra methionine (+1.45%) resulted in much softer feathers, a condition which was correlated with increased content of bound sulphide. Source of methionine is often questioned in evaluation of feathering in commercial broilers, yet there is surprisingly little information available on this topic. There is a wealth of information published on the efficacy of DL-methionine *vs.* various methionine hydroxy analogues. Lack of comparable information on methionine source for feathering suggests it is not an issue.

Although the sulphur containing amino acids are quantitatively the most important for feather growth, numerous bio-assays have demonstrated the importance of others (Hegsted *et al.*, 1941; Wilkening *et al.*, 1947; Anderson and Warnick, 1967; Robel, 1977; Penz *et al.*, 1984). Characteristic deficiency symptoms include a spoon-like appearance of the chick's primary and secondary feathers caused by the retention of an abnormally long sheath that covers the proximal end of the feather shaft (Sanders *et al.*, 1950). Furthermore, the birds lose their characteristic smooth appearance since barbules on adjacent barbs no longer interlock normally (Anderson and Warnick, 1967). Another characteristic of amino acid deficiencies is the abnormal curling of feathers away from the body (Robel, 1977). These signs are noted in chicks fed diets deficient in arginine, valine,

leucine, isoleucine, tryptophan or phenylalanine and tyrosine. However the degree to which the abnormality progresses varies with the individual amino acid and the severity of deficiency (Anderson and Warnick, 1967). It is interesting to note that some of these feather abnormalities and in particular the abnormally long sheathing, are also characteristic of a range of vitamin deficiencies (Summers *et al.*, 1978).

Apart from studies with sulphur amino acids, metabolism of the branch chain amino acids is known to have a major effect on feather development. Farran and Thomas (1992) showed that chicks fed a diet marginal in valine, but with adequate levels of leucine and isoleucine, developed characteristic feather abnormalities as described previously. These authors concluded that a deficiency of valine alone is much more detrimental to feathering than is a concurrent deficiency of all branch chain amino acids. Penz *et al.* (1984) showed a similar feathering abnormality by feeding high levels of leucine. Feathers were found deficient in valine, isoleucine and cystine, and the problem could be corrected by adding extra valine or isoleucine.

Feather pigmentation is also influenced by nutrition. Because certain diets resulted in normal feather structure, but decidedly abnormal pigmentation, separate factors were implicated in feather structure and feather pigmentation (Fuller and Wilke, 1942). Lysine has since been identified as the major amino acid involved in pigment formation (Vohra and Kratzer, 1957, 1959a; Owings and Balloun, 1959). Anderson and Warnick (1967) also observed reduced pigmentation when chicks were fed diets deficient in phenylalanine and tyrosine, which are precursors of melanin. Lysine deficiency may also cause depigmentation by inhibiting activity of the enzyme tyrosinase so inhibiting the formation of melanin (Owings and Balloun, 1959). Grau *et al.* (1989) provides a comprehensive review of the relationship between nutrition and feather pigmentation.

Vitamins and minerals also influence feather development. Supplee (1966) observed an unusual feather development when chicks received diets deficient in vitamin E and selenium. Similar feather conditions have been described in chicks fed inadequate pyridoxine (Daghir and Balloun, 1963) panthothenic acid, folic acid, biotin or nicotinic acid (Taylor, 1967). Deficiency of riboflavin causes perhaps the most characteristic signs of deficiency in embryos and chicks. Affected embryos exhibit clubbed down, which, as its name implies, involves a swollen tip for each down feather (Leeson and Summers, 1997). In the last few years, there has been increased incidence of clubbed down or defective down as it is sometimes called. Whitehead *et al.* (1993) studied this problem in breeders and concluded that the defective down syndrome now seen in commercial chicks is not due to a deficiency of riboflavin at the breeder level. These authors consider the feather abnormality to be more likely the result of some infectious agent. Payne (1977) also described a clubbed down condition in chicks that also had characteristic long ginger hairs in the breast area. The condition was not responsive to riboflavin, but chicks developed normal feathers if given an oral dose of molybdenum.

Summers *et al.* (1978) studied the broiler's response to a number of individual vitamin deficiencies. In these situations, birds were fed corn-soybean meal diets and thus were not totally devoid of vitamins, the deficiency being created by lack of supplemental synthetic vitamins. As previously described for amino acids, the effect on feathering was dramatic, but not characteristic for any single vitamin. In all cases, feathering was delayed and primaries had barbs and barbules only on the distal part of the feather.

Inadequate levels of trace minerals will also impede feather development. Deficiencies of zinc, tin, vanadium, chromium and nickel have all been reported to affect feathering (Scott *et al.*, 1959; Baker and Molitoris, 1975). Supplee *et al.* (1958) were among the first to show that zinc is needed for feather development of growing pullets while Scott *et al.* (1959) indicated that cereal diets for pheasants needed to be supplemented with zinc in order to prevent poor feathering. A zinc deficiency causes frayed feathers especially in the

rapidly growing primaries and secondaries (Sunde, 1972). In other studies, Cook *et al.* (1984) described a zinc deficiency in pheasants, expressed as poor feathering, although the diet contained normal levels of this mineral. These authors concluded that some unidentified dietary factor was responsible for inducing zinc deficiency and associated abnormal feathering. Sunde (1972) also described a feathering problem in Leghorn birds that was responsive to zinc supplementation. Characteristic blisters appeared on the shaft of the feathers about 3 cm from the follicle and the feather often broke off at this point. Only about 5-10% of birds were affected and the condition was corrected by a supplement of 100 ppm zinc as zinc carbonate even though the basal diet contained 50 ppm zinc. Birds deficient in sodium will also exhibit poor feathering, and this is often associated with cannibalism and feather pecking.

Another mineral of current concern is boron, which is sometimes used as a litter treatment to control darkling beetle. Dufour *et al.* (1992) give details of a feathering abnormality in young chicks raised on boric acid-treated litter (7.2 kg/9.3 m^2). The primary wing feathers were sheathed with feathers appearing curled and rough in appearance. It is not clear if the boric acid causes problems due to contact with the skin, or if birds are affected due to eating the contaminated litter. Over the years there has also been concern expressed regarding the relationship between feathering and use of certain anticoccidials (Charles and Kiker, 1974; Kiker and Sherwood, 1974). It was initially thought that ionophore anticoccidials induced abnormal feathering by increasing the bird's methionine requirement, although many reports fail to support this contention (Damron *et al.*, 1977; Leeson and Summers, 1983). Patel *et al.* (1980) in fact, reported a methionine-sparing effect of lasalocid. Certain ionophore anticoccidials will cause poorer feathering if chick starter diets are low in protein (<16%) and associated amino acids.

Dietary mycotoxins are also known to cause feather abnormalities. Birds fed T-2 toxin develop only a sparse covering of feathers, which tend to protrude from the bird at odd angles. One possibility is that T-2 toxin alters the metabolism of certain nutrients, which are required for proper feather development (Wyatt *et al.*, 1975). With T-2 toxicosis most of the body feathers are influenced, unlike the localized situation seen with vitamin or amino acid imbalance. Feather problems occur at 4-16 ppm T-2 toxin and severity is dose related, although the effect is quite variable from bird to bird. Parkhurst *et al.* (1992) implicate T-2 toxin in "helicopter wing", which is characterized by one or two primary wing feathers sticking out from the body. Using up to 8 ppm T2 mycotoxins, these researchers showed frayed and missing barbs and barbules on the medial side of the bottom half of the feather. The feather shafts tended to have an accentuated curve away from the body and in some cases there was a distinct dark bar across the feather about one-quarter the length down from the tip. Of the mycotoxins, 15-monoacetoxyscirpenol was problematic even at 0.5 ppm whereas diacetoxyscirpenol toxicity occurred at 2 ppm. Niemiec *et al.* (1991) suggested 1.5 ppm ochratoxin A to cause delayed feathering in broilers.

Infectious agents and feather growth

A number of viruses and mycoplasma have been implicated in poor feathering of chicks, poultry and especially pet birds. Kisary (1985) artificially infected chicken embryos with parvovirus and resultant chicks had enteritis and poor feathering. A goose parvovirus was also suspected of causing abnormal feathering in Muscovy ducks (Takehara *et al.*, 1994).

Stunting syndrome has been quite common in broiler chicks and poultry for a number of years. A characteristic of the disease is poor feathering and in particular helicopter wing. A reovirus is suspected as being causative (Rosenberger, 1983). The situation with reovirus poses the question of whether or not feather abnormalities are due to the

infectious agent *per se*, or due to poor nutrient uptake caused by associated enteritis (Neressian *et al.*, 1985). Stunting and associated poor feathering is also caused by reticuloendothelial viruses that are retroviruses comparable to the ev-21 gene described earlier. Fowl pox vaccine contaminated with reticuloendothelial virus has been implicated in situations of poor feathering and associated proventriculitis (Diallo *et al.*, 1998).

Ritchie *et al.* (1989) characterized a new virus responsible for Psittacine Beak and Feather Disease in captive cockatoos. Affected birds quickly lose their feathers. The condition seems quite common in Asia. *Mycoplasma iowae* is perhaps best known for its effect on hatchability of turkeys, although Bradbury and Kelly (1991) attributed a feathering problem in young broiler breeders to this infectious agent. Feathering was delayed, with retention of feather down in the head region, while primaries remained sheathed. Again it is unclear if the mycoplasma influences the feather follicle directly or if the feather abnormality is due to concurrent reduction in feed intake.

The environment and feather growth

Feathers play a major role in insulating the body of the bird. Therefore, except in situations of very high temperature, good feather coverage will optimise energy metabolism and feed efficiency. Partially defeathered birds will loose more heat, especially when exposed to cold temperatures where they are forced to use various thermoregulatory responses in order to maintain body temperature at 41°C. Richards (1977) showed birds at 15°C to have 60% higher metabolic rate than birds kept at 25°C. Below about 25°C, well-feathered birds increase heat production by around 2% per °C, whereas for poorly feathered birds, the rate increases by about 5% per °C. These data are confirmed by Tullet *et al.* (1980). Lee *et al.* (1983) showed that insulative jackets reduced the fasting heat production of poorly feathered birds at 20°C from 148% to 126% of that of normally feathered birds. Leeson and Morrison (1978) indicated that feed efficiency in laying hens was significantly correlated with feather cover, while O'Neill *et al.* (1971) showed 85% greater feed intake for naked birds at 22°C. In addition, to a direct effect on maintenance energy needs, there will also be increased energy demand for rejuvenation of feathers.

Prolonged exposure of young birds to high environmental temperatures (> 32°C) leads to retardation of feathering (Geraert *et al.*, 1996), although short-term exposure (<4 d) has little detrimental effect. What is generally referred to as "poor feathering" is, in fact, beneficial when birds are subjected to high temperatures because they can simply dissipate heat more quickly. For example, Pech-Waffenschmidt (1995) showed that birds with extensive feather loss had a higher body surface temperature when subjected to high environmental temperatures, but that body core temperature was reduced. Such poor feather coverage is the likely reason for the better heat-resistant characteristics of naked-neck strains of bird.

There has been relatively little work conducted on the effect of lighting on feather development. Working with pheasants, Slaugh *et al.* (1990) suggested that intermittent photoperiods were beneficial in promoting feathering in the back region, compared to continuous light. Although back feathering was almost 50% greater with such lighting, the most striking feature was an increased length of tail feathers (10 *vs.* 20 cm).

Feather pecking, cannibalism and behaviour

Feather pecking is often regarded as a starting point for the onset of more serious cannibalism. The cause of feather pecking, or feather "licking" as occurs in broiler

breeders, is rarely identified, and in many situations, there are no serious consequences to the bird. Deficiencies of protein and sulphfur amino acids are most often suspected, and such diet adjustment seems to be the standard approach to the problems, usually without any beneficial effect.

Kjaer and Sorensen (1997) showed that feather pecking was a heritable trait (h^2 0.38 at 69 weeks) and that selection for layers with no or little tendency to feather peck should be feasible. Loss of feathers in layers is sometimes caused by feather pecking, especially in multi-bird cages. However, Hagger (1993) recently suggested that there is a negative correlation between feather cover of older birds and egg production. Adult birds, however, continuously experience feather loss as an unavoidable consequence of their housing conditions. A certain amount of feather loss arises due to abrasion on the metal surfaces of cages, feed troughs etc., however, these losses are generally insignificant (Hughes, 1980). Feather pecking among hens represents the main avenue of feather loss in caged birds (Hunton, 1985). Although both nutritional and environmental factors have been associated with the behavioural patterns of feather pecking, it is most frequently attributed to stress of intensive management (Hughes and Duncan, 1972). Various management techniques, such as minimal light intensity and the elimination of unnecessary stresses, will reduce feather pecking and consequently limit feather losses (Hunton, 1985). A certain amount of feather loss is, however, unavoidable and most layers are partially defeathered before the cycle is complete.

Apart from genetics and age, the most common reasons for feather pecking and possible associated cannibalism are boredom, high light intensity, low humidity, restricted feeding were practiced and perhaps nutritional deficiencies. If the latter is suspected then birds often eat feathers, and this is diagnosed by the existence of feathers in the gizzard and also lack of feathers appearing on the litter or underneath the cages. Because of the potential for feather pecking to lead to cannibalism, beak trimming is frequently considered as a treatment. Lee and Craig (1991) observed no difference in feather pecking activity of regular *vs.* beak trimmed White Leghorns. However beak-trimmed birds appeared less nervous and so their better feather cover was most likely a consequence of less wear and tear within the cage environment.

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