Nutritional influences on carcass composition in the broiler chicken

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During the period 1974–1984 the annual consumption of poultry meat has doubled to approximately 15 kg per person (Simpson, 1985). The determinant factor for this increased consumption is the low price of chicken compared with other meat. This has been a direct result of genetic selection for efficiency of production in terms of increased growth rate and improved feed conversion efficiency combined with a further understanding of a bird's environmental and nutritional needs. Twenty years ago it took 68 d for a broiler chicken to reach a slaughter weight of 2 kg whereas in 1985 it takes approximately 45 d (K. Laughlin, personal communication).

As a broiler chicken grows its carcass composition changes. The most obvious change is the increase in carcass fat. Since a broiler chicken does not reach maturity, in terms of lean body mass, until about 30 weeks of age, broilers are slaughtered while relatively immature and, to date, the producer has given little or no attention to the composition of the end-product. However, the consumer is becoming far more health conscious and a recent report on diet and cardiovascular disease by the Committee on Medical Aspects of Food Policy (1984) recommended that 'The consumption of saturated fatty acids and fat in the United Kingdom should be decreased'. Since meat and meat products account for about 25% of the saturated fat ingested, the report went on to state that 'Consumption of saturated fatty acids could be decreased now by substituting, for example, some non-fatty fish or poultry, e.g. chicken'. When compared with other meat, chicken has a slightly higher protein content but the total fat content is no more than 20% of that of other meat. The saturated fatty acid content is 50% that of other meat and the level of polyunsaturated fats slightly higher, thus placing chicken very favourably with respect to human nutrition (Paul & Southgate, 1978).

Although the carcass composition of the broiler chicken is to a certain extent genetically predetermined, it can be influenced by nutritional factors, of which the most widely investigated is the dietary energy:protein (E:P) value.

The dietary E:P value

Frapps (1943) demonstrated that chickens with widely differing amounts of body fat could be produced by manipulation of the dietary E:P value. Subsequently many workers (Spring & Wilkinson, 1957; Summers *et al.* 1965; Bartov *et al.* 1974*a,b*; Griffiths *et al.* 1977*a,b*; Summers & Leeson, 1979) have investigated the effect of the dietary E:P value on carcass composition and have established that the E:P value is directly correlated with carcass fat. Narrowing

the E:P value generally prevents excessive fat deposition. The dietary E:P value can be manipulated by altering the energy level, protein level or both.

In the work of Jackson *et al.* (1982*a*,*b*), dietary treatments comprising six levels of apparent metabolizable energy (AME) and six levels of crude protein (nitrogen × 6·25, CP) were offered *ad lib.* to birds from 1-d-old until 49 d of age. The AME content of the diet was increased from 10·9 to 15 MJ/kg while maintaining the protein content at 260 g/kg. As the dietary energy level increased the body-weight at 49 d increased although it approached an asymptote between 14·2 and 15 MJ AME/kg. Similar results were reported by Brown & McCartney (1982) who found that with diets containing 230 g CP/kg the optimum level for live-weight gain was 14·2 MJ AME/kg. At higher levels, food intake and body-weight were depressed.

In accordance with Spring & Wilkinson (1957) and Summers *et al.* (1965), Jackson *et al.* (1982*b*) reported that increasing the dietary energy level caused an increase in the proportion of total carcass fat and abdominal fat and a reduction in carcass protein. However, the whole carcass is not normally eaten and Hakannson *et al.* (1978) investigated the effect of dietary energy level on the edible portion (meat plus skin) of the carcass. The male broilers were given diets containing 10·08, 11·61 or 13·14 MJ AME/kg and were slaughtered at 2 kg live-weight. On the highest energy diet, the birds achieved this weight in 52 d whereas it took 65 d on the lowest energy diet. The yield of the edible portion was similar for all dietary energy levels but the composition was considerably affected. Feeding the highest-energy diet as compared with the lowest one resulted in a threefold increase in the fat content of the edible portion (13 *v.* 4·7%).

Work with other species (Baratunde *et al.* 1967; Knittle & Hirsch, 1968) has shown that restricting energy intake during the early stage of growth can cause a permanent reduction in the size of the fat depots. This suggests that early energy restriction can be used to decrease broiler fat deposition at market age. The work of Deaton *et al.* (1973) and Kubena *et al.* (1974) had indicated that the energy level of the finisher diet influenced the effect of the starter diet. However, Jones & Wiseman (1985) conducted a trial in which three isonitrogenous starter diets (230 g CP/kg) and three finisher diets (200 g CP/kg) were formulated to contain 10·78, 12·78 and 14·78 MJ AME/kg. The three starter diets were given to broiler chickens from 0 to 24 d of age and the three finisher diets from 24 to 49 d of age such that there were nine dietary combinations. Birds fed on the low-energy starter diet had significantly (*P*<0·05) lower body-weights and their carcass contained proportionally less abdominal and total carcass fat at 24 d of age. This effect remained until 49 d of age irrespective of the finisher diet given. The energy level of the finisher diet had no significant effect on body-weight but the proportion of abdominal fat and total carcass fat deposited was lower for birds fed on the low-energy finisher diet. Feeding a low-energy starter diet in order to reduce carcass fat is unlikely to prove economic since it is accompanied by a reduction in body-weight. Feeding a low-energy finisher diet would seem to be a more viable alternative. However, the birds fed on the low-energy finisher diet had a higher feed intake than those fed on the high-energy one and consequently had a similar

energy intake but a higher protein intake. Thus, it would appear that the reduction in carcass fat was actually a response to the increased protein intake.

Jackson *et al.* (1982*a,b*) maintained the dietary energy at 13 MJ AME/kg and increased the dietary protein content from 160 to 360 g/kg. As the protein level increased to 240 g/kg the body-weight at 49 d increased, but further increases beyond 240 g/kg had no effect on body-weight. Brown & McCartney (1982) reported a depression in body-weight when birds were fed on a diet containing 310 g CP/kg. Jackson *et al.* (1982*a,b*) found no significant increases in protein deposition at dietary protein levels higher than 240 g/kg or any significant reduction in carcass fat at protein levels beyond 280 g/kg. These results are in agreement with Velu *et al.* (1971) who reported that the level of protein needed to maximize body-weight gain was 40% less than that needed to minimize fat deposition.

There is no information available, at present, on the effect of dietary protein level on the composition of the edible portion of the carcass. However, Summers & Leeson (1984) showed that increasing the dietary protein level resulted in an increase in yield of edible meat and it would be reasonable to assume that increasing the dietary protein level would cause a reduction in the fat content of the edible portion.

Elvery (1983) reported that giving a low-protein starter diet to broiler chickens from 0 to 24 d resulted in a reduction in the body-weight at 29 d but that this effect had disappeared by 49 d of age. The protein level of the diet given during the finisher phase (24–49 d) had no significant effect on body-weight but there was a tendency for body-weight to increase with each increment of dietary protein. Neither the protein level of the starter nor the finisher diet had a significant effect on fat deposition at market age, but there was a general trend for fat deposition to decrease as dietary protein increased. Thus it appears that increasing the dietary protein level can be used as a means to reduce carcass fat levels without a concomitant decrease in body-weight.

Bartov & Bornstein (1979) found that the degree of saturation of the carcass fat was partially dependent on the dietary E:P value. A narrow E:P value such as would be obtained by increasing the dietary protein level, results in a less saturated carcass. The degree of saturation of carcass fat is of practical importance since unsaturated fatty acids are readily oxidized (Bartov & Bornstein, 1977). This gives rise to problems with rancidity of the fat, thus reducing the storage quality of the carcass, although the stability of the fat can be improved by supplementation of the diet with vitamin E (Bartov & Bornstein, 1977).

Protein balance

The growing bird should receive sufficient essential amino acids and non-essential nitrogen for optimum synthesis and deposition of tissue proteins. Velu *et al.* (1972) showed that when chicks were given *ad lib.* a diet containing a mixture of amino acids devoid of isoleucine, but otherwise balanced, each increment of the limiting amino acid resulted in an increase in food intake,

body-weight gain and body fat. However, when equalized feeding was employed in order to minimize the influence of variation in energy intake, the concentration of body fat decreased as the level of isoleucine was increased up to a plateau of o·6%. As the level of isoleucine was increased the level of effective protein in the diet was also increased, thus enabling the birds to fulfil their potential for muscle growth.

In contrast to the depression in food intake observed with a severe amino acid imbalance, mild imbalances tend to stimulate food intake and growth rate. Experiments of Tobin & Boorman (cited by Boorman, 1980) showed that when a mixture of amino acids lacking histidine was added to a 100 g CP/kg diet, food intake increased over a 10 d period with up to 2% addition. However, continued feeding of a mildly imbalanced diet eventually leads to a reduction in food intake, in a similar manner to that caused by a severe imbalance. Thus most effects of dietary amino acid imbalances on growth and carcass composition are due to the differences in food intake that these conditions promote.

Nutrient density

Theoretically, birds adapt their feed intake so as to maintain a constant nutrient intake over a range of nutrient density. However, if the nutrient density of the diet is too low the birds are physically unable to consume sufficient feed and their growth is retarded. Since such birds are physiologically younger they also tend to be leaner. At the other extreme, if the nutrient density of the diet is too high the birds will not reduce their feed intake sufficiently since such a reduction leaves them physically hungry. The excess nutrient intake is deposited as fat. Even in the middle range, birds do not adapt their feed intake exactly and Fisher & Wilson (1974) found that at a constant nutrient:energy value, a 1 MJ/kg increase in the diet caused the fat content of the carcass to increase by 6·7 g/kg.

Dietary fat

Bartov et al. (1974b), Bartov & Bornstein (1977), Bartov (1979) and Fuller & Rendon (1977) reported that increases in the fat content of the diet, without alteration of the dietary E:P value, had no effect on the growth or carcass composition of broilers. However, Edwards et al. (1973) and Fuller & Rendon (1977) found that the level and type of fat given (i.e. degree of saturation) influenced the fatty acid composition of the carcass fat.

In birds given a fat-free diet all the carcass fat is of endogenous origin and the predominant fatty acids are palmitic and oleic acids. In birds given a fat-supplemented diet the carcass fat is composed of a mixture of dietary fatty acids, which are being deposited without being metabolized (Salmon & O'Neil, 1973; Bartov et al. 1974b), and fatty acids synthesized from carbohydrates. Feeding a fat-supplemented diet depresses hepatic lipogenesis (Yeh et al. 1970; Pearce, 1974). Thus, as the level of fat given is increased, the carcass fat deposited more closely resembles that of the dietary fat in composition (Salmon & O'Neil, 1973). Feeding vegetable oils with high levels of unsaturated fatty acids will result in a less-saturated carcass fat than feeding, for example, beef tallow. Since a large

proportion of the carcass fat present in the broiler chicken at market age is deposited during the 2 weeks before slaughter, the influence of dietary fat on the composition of the carcass fat is only of importance during the finisher phase.

Conclusion

The composition of the edible portion of the chicken carcass can be manipulated nutritionally. The fat content can be reduced by either decreasing the dietary energy level or increasing the dietary protein level, the latter being the more viable alternative. The degree of saturation of the carcass fat can be reduced by feeding high-protein–low-energy diets or by the inclusion of unsaturated fats in the diet. However, at present the producer is paid entirely on a live-weight basis and only consumer pressures will alter the nature of the end-product.

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