Chapter 2

MATERIALS AND METHODS

2.1 Growth Trials

2.1.1 Diets and management

One day old chickens were placed in an electrically-heated brooder and fed a commercial broiler starter crumble diet (Fielders Stock Feeds, Tamworth, New South Wales) until the experiment started. Then the birds were allocated to cages (see Appendix A) according to the experimental design. A completely randomized experimental design was used for all trials. The room temperature was kept at about 22°C during the experimental period.

A commercial broiler finisher mash (Fielders Stock Feeds, Tamworth, New South Wales) was used in all the nine trials. The composition, ingredients and nitrogen and energy contents is shown in Table 2.1.

Trials 1 and 2 started at day 25 and 28 respectively and finished at day 56, the other seven trials commenced when the birds were 28 days of age and were finished at day 49. Feed intake and weight gain were measured weekly and when the trial was terminated, the birds, with an overnight feed withdrawal, were slaughtered by cervical dislocation and weighed individually. The abdominal fat pad (AFP) was then removed and weighed. In Trials 1, 2, 6 and 9 a number of birds were selected according to their body weight, frozen (minus AFP) and later finely minced twice. Subsamples of mince were taken for carcass fat

analysis.

Table 2.1: Specifications of the broiler finisher mash as supplied by manufacturer

Metabolizable energy	12.14 MJ	Lysine (total)	10.05 g/kg
Crude protein	19.0%	Methionine (total)	4.34 g/kg
Crude fat	3.6%	Methionine + Cystine	7.41 g/kg
Salt	0.5%	Threonine (total)	6.51 g/kg
Crude fibre max	3.5%	Tryptophan (total)	2.00 g/kg
Urea max	nil	Phosphorus (total)	6.95 g/kg
Fluorine max	0.02%	Calcium	7.55 g/kg

2.1.2 Birds and chemicals

Two experimental lines of chickens selected for high (F) and low (L) carcass fat (Pym, 1985b) were used in Trials 1 and 2. Birds were hatched from the eggs that were obtained from Dr. R.A.E. Pym of the University of Queensland. Tegel chickens used in Trial 3 were from A.A. Tegel Pty. Ltd. Hyline birds were from Table Talk, Poultry Farms Ltd., Luddenham, New South Wales. Steggles birds were obtained from Steggles Pty. Ltd., Beresfield, New South Wales.

The beta-agonist, cimaterol, was from American Cyanamid Company, Agricultural Research Division, Princeton, NJ 08540, USA. Theophylline and Caffeine were from Sigma Chemical Company, P.O. Box 14508, St. Louis, MO, 63178, USA. Iodinated casein was prepared by Dr. T.M. Sutherland using the direct iodination of skim milk powder as described by Reineke *et al.* (1942) with the modification that the iodinated proteir was freeze dried instead of being dried at room temperature so that it could be finely ground. Chemical agents were stored at 4°C and mixed into the diet as premix.

2.1.3 Experimental Design

2.1.3.1 Trial 1

The effects of cimaterol were tested in a genetically fat and lean line of chicken. Cimaterol was mixed into the finisher mash at four levels, 0, 0.2, 0.4 and 0.6 ppm. The four treatments, with three replicates each of six birds of mixed sex, commenced on day 25 and continued to day 56. After AFP was removed, six pairs of birds, three of each sex, were taken for carcass fat analysis.

2.1.3.2 Trial 2

On the basis of the results of Trial 1 and those published, 0.4 ppm cimaterol was chosen and evaluated in the same two lines of birds. Thirty six birds from each of the fat and lean lines were allocated to two treatments with three replicates each of six birds of mixed sex. The experiment was started at day 28 and finished at day 56. After AFP measurement, carcass fat analysis was determined on each individual bird.

2.1.3.3 Trial 3

Sixty Tegel female birds were used in this trial to test the effects of theophylline and/or cimaterol. There were four treatments, control, 0.1% theophylline (1g theophylline per kg diet), 0.5 ppm cimaterol and 0.1% theophylline plus 0.5 ppm cimaterol, with three replicates each of five birds.

2.1.3.4 Trial 4

The same treatments as those in Trial 3 were adopted with three replicates each of seven Hyline male birds.

2.1.3.5 Trial 5

Sixty three female Hyline chickens were allocated to three diets with different levels of theophylline: 0, 0.05% (0.5g theophylline per kg diet) and 0.1%, with three replicates each

of seven birds.

2.1.3.6 Trial 6

One hundred and fifty female Hyline birds were allocated to five treatments with five replicates each of six birds to test the effects of different levels of theophylline. Treatment 1 was a control, treatment 2 was 0.05% theophylline and treatment 3 was 0.1% theophylline. The birds on treatment 4 were on a control diet in the first half experimental period (from 28 to 38 days) and on 0.1% theophylline diet during the second half period (from 38 to 48 days). They were assumed to have the same amount of theophylline intake as those on 0.05% theophylline diet on treatment 2 during the whole period. Birds on treatment 5 were pair-fed the control diet to the same intake as birds on 0.1% theophylline diet on treatment 3 in order to exclude effects of feed intake on observations. After AFP was removed, two birds of average body weight from each replicate, thus 10 from each treatment, were selected for carcass fat analysis.

2.1.3.7 Trial 7

The effects of caffeine were tested in Steggles male chickens. There were four treatments with four replicates each of six birds: controls, 0.05% and 0.1% caffeine and a group on the control diet pair-fed to birds receiving 0.1% caffeine to exclude effects of differences in feed intake.

2.1.3.8 Trial 8

Steggles male chickens (144) were allocated to six treatments with four replicates each of six birds to explore the effects of iodinated casein and iodinated casein plus theophylline. Three levels of iodinated casein, 0, 50 ppm (50 mg/kg diet) and 100 ppm (100 mg/kg diet), were used. Each level of iodinated casein was combined with 0 or 0.05% theophylline. Thus the six treatments were: control, 0.05% theophylline, 50 ppm iodinated casein, 50 ppm iodinated casein plus 0.05% theophylline, 100 ppm iodinated casein and 100 ppm iodinated casein plus 0.05% theophylline.

2.1.3.9 Trial 9

This trial was designed to confirm the effects of caffeine at the same two levels as those in Trial 7 and effects of 50 ppm iodinated casein. A combination of 50 ppm iodinated casein and 0.05% caffeine was also included. Steggles male birds (140) were allocated to five treatments with four replicates each of seven chickens: controls, 50 ppm iodinated casein, 50 ppm iodinated casein plus 0.05% caffeine, 0.05% caffeine and 0.1% caffeine. After AFP measurement, four birds of average body weight from each replicate were combined into one sample for carcass fat determination giving four samples per treatment.

2.1.4 Chemical analysis

Carcass fat was determined in duplicate by increasing the density of a fat : tetrachloroethylene extract in a magnetic float cell as described by Usher *et al.* (1973) on a Foss-Let analyser (Appendix B).

2.1.5 Calculations

Abdominal fat pad (AFP) was expressed as a percentage of body weight after slaughter. Feed conversion ratio (FCR) was expressed as feed consumption (kg) per kg live weight gain. Fat-free body weight (FFBW) was calculated by subtraction of carcass fat and AFP from body weight after slaughter.

2.1.6 Statistical analysis

Data were subjected to an analysis of variance (AOV) and differences between treatment means were compared and determined using the Least Significant Difference (LSD) test (Steel and Torrie, 1960) using a Neva computer package. Standard error of means (SEM) was calculated using group means and not individual observations. Values given in all tables with the same superscript within a row are not significantly different ($P>0.05$).

2.2 Respiration Calorimetry Experiments

Three experiments were conducted to measure heat production of the birds subjected to different treatments and of different lines by using the closed-circuit respiration chambers described by Farrell and Swain (1977) and Farrell (1972). The relative humidity was held around 70% and the same broiler finisher mash as that in the growth trials was used in all the three experiments. The method of routine operation of the chambers, the calculation of heat production from gaseous exchange and method of gas analysis were as described by Farrell (1972).

2.2.1 Experiment 1

2.2.1.1 Respiration chambers

The respiration chambers and ancilliary equipment used in this experiment were described by Farrell and Swain (1977) and Swain (1980) and are shown in Appendix C. Basically, the equipment consisted of an external cabinet enclosing a moulded fibreglass reservoir filled with water, which held the two water-proof copper chambers ($46 \times 35 \times 51 cm^3$). The chickens were placed in cages inside the chambers which were sealed with a perspex lid fitted with a moulded silicone rubber seal and firmly champed to the perimeter of the chamber rim using 10 small 'G' clamps. The lids of the external cabinet (85cm long, 38cm wide and 2 cm thick) opened from the centre. This area was filled with polyurethane foam except for a small area within each lid where a small fluorescent light (26cm long, 8watts) was attached. The tank (120cm long, 76cm wide and 60cm deep) was completed enclosed by a 15mm thick expanded polyurethane for insulation purpose.

The two chambers were bolted centrally to the floor of the tank. The water was added regularly to a set level. The temperature of the water in the tank was regulated by heating and cooling coils connected to a thermostat and could be held at any selected temperature in the range of 0–40°C. Temperature within the chambers was recorded with three dry-bulb and one wet-bulb thermometers located in the lid.

Within each chamber was a rectangular, wire-mash cages fitted with a feeder and a

glass drinker. This cage was placed in a galvanised iron tray 5cm deep with two galvanised handles. As a result, the bird could be easily lowered into the chambers.

Air circulation through the enclosed system was drawn from the outlet of the chamber into a small pump and then blown through a potassium hydroxide (KOH) solution to absorb the exhaled CO_2, calcium chloride ($CaCl_2$) train and then returned to the chamber. This circulation system contained a length of polythene hose, 1.5cm in diameter. The drying cylinders containing silica gel were attached to the air circulating pump (240 Volts and 1.2 amps) which had a by-pass tube with a tap to vary air flow.

Oxygen to the chambers was controlled by a manometer and solenoid. The glass manometer (U-tube), connected to the chambers by a plastic tube, contained a solution of sodium hydrogencarbonate ($NaHCO_3$) and had one electrode in each arm and one at the base. When pressure inside the chambers dropped, the solution was gradually drawn up to the electrode and this caused the solenoid valve attached to the oxygen cylinder to open via a relay, allowing oxygen to flow into the chamber until the solution in the manometer contacted the second electrode switching off the solenoid.

2.2.1.2 Principles of operation

Immediately prior to the start of a measurement run, a plastic sheet was placed beneath the cage to collect excreta. Feeder, drinker and birds were weighed and then placed in the cages. The two cages were lowered into chambers and sealed with lids. The four thermometers were inserted through rubber stoppers in each perspex lid. Also tubing to the manometer and to the solenoid valve was attached to perspex nipples in the lid and the circulating pump was turned on . The oxygen cylinders were previously and accurately weighed and firmly connected to the copper tubing attached to the solenoid valves. When the temperature of both chambers had equilibrated, usually after about 30 minutes, the circulating pumps were stopped and the temperatures in each chamber were recorded. Gas samples for air composition were taken in metal syringes. The drying trains and KOH flasks were connected immediately giving a sealed system. The system was kept airtight throughout each period of measurement. The circulation pumps were again switched on. At this stage the time and

atmospheric pressure were recorded. When the $NaHCO_3$ solution in the manometer had moved up the arm to the electrode probe and activated the solenoid valve, the control tap on the oxygen was set to provide an even flow of O_2 into the chambers. At the end of the measurement period, the chambers were stopped, the time was recorded and gas samples were withdrawn with an airtight metal syringe for analysis. Feed and water containers were weighed and refilled. Excreta on the plastic were collected and a new plastic sheet was placed in the tray. New adequately concentrated KOH flasks were put on. The drying trains were removed, and silica gel palced in an oven set at 105°C overnight and replaced by new trains inserts containing silica gel.

The circulating pumps were checked every week for leaks and the diaphragm was changed. The chambers were tested regularly for leaks by altering the atmospheric pressure within the chamber and then observing changes in solution level in the manometer. Illumination was continuous.

2.2.1.3 Birds and management

The same fat and lean lines of chickens, aged 25 to 38 days, as those used in growth Trial 1 and 2 were used in this experiment. They were reared in the electrically-heated brooders and cages (Appendix A) before moving into the chambers. The birds were fed *ad libitum* and heat production was measured continuously for three days except for about 2 hours each day in order to service the chambers. Chamber temperature was kept at 25-27°C.

2.2.1.4 Treatments

This experiment was designed to test the difference between fat and lean lines of birds, males and females on a control and a 0.4 ppm cimaterol diet in heat production and energy and nitrogen metabolism.

Three birds of same sex and similar body weight were selected and paired as follows:

- control fat female birds (F_{-f}) and control lean female birds (L_{-f})
- control fat male birds (F_{-m}) and control lean male birds (L_{-m})

- control fat female birds ($F-_f$) and treated fat female birds ($F+_f$)
- control fat male birds ($F-_m$) and treated fat male birds ($F+_m$)
- control lean female birds ($L-_f$) and treated lean female birds ($L+_f$)
- control lean male birds ($L-_m$) and treated lean male birds ($L+_m$)

2.2.2 Experiment 2

The same chambers and birds as in Experiment 1 were used to compare the difference between fat and lean lines in energy and nitrogen metabolism. After one day for acclimatization on *ad libitum* feeding, three female birds of similar body weight in each chamber were given about 90% of their *ad libitum* intake according to the results from Experiment 1 and the previous day for five days to determine daily heat production. Chamber temperature was about 26°C.

2.2.3 Experiment 3

This experiment was conducted to study the effects of 0.5 ppm cimaterol, 0.05% theophylline and 50 ppm iodinated casein on heat production and energy and nitrogen metabolism.

2.2.3.1 Respiration chambers

Three different respiration chambers used in this experiment (Appendix D) were similar to those described by Farrell (1972) with some differences. They were constructed of 0.5cm thick glass and were measured 62cm long × 34cm wide × 63cm high. They sat in a 5cm deep metal trough containing water to seal the chambers. Chamber temperature was controlled by changing room temperature with a fan and an air-conditioning unit. Temperature and relative humidity were measured with one wet-bulb and three dry-bulb thermometers attached to the front, back, and side of the chamber. Chamber temperature was kept at about 22–24°C. Illumination was continuous. The birds were housed in a wire-mesh cage which sat in a galvanised tray 20cm deep.

2.2.3.2 Birds and treatments

Three Steggles female birds aged 21 to 35 days and of similar body weight were selected and placed in the chambers for one week. After one day for acclimatization, heat production was measured for six days.

There were four diets, control, 0.05% theophylline, 50 ppm iodinated casein and 0.5 ppm cimaterol. For the first three days birds were on the control diet and were pair-fed to about 90% of *ad libitum* intake level. For the second three days they were on one of the four treated diets and were pair-fed to the level of intake of those on theophylline diet since they showed the lowest feed intake in the growth trials. There were four repeated runs for each diet. Excreta were collected every day for the three days and combined for energy and nitrogen determinations.

2.2.4 Chemical analysis

2.2.4.1 Gas samples

CO_2 composition of chamber air was determined using a Haldane gas analyser. O_2 content was measured in a Beckman Oxygen Analyser (Model 755).

2.2.4.2 Feed and excreta

Excreta were dried at 70°C to constant weight in an oven and finely ground through a 1 mm^2 sift. Subsamples were then taken. Gross energy (GE) of representative subsamples of feed and excreta was determined in an adiabatic bomb calorimeter. Nitrogen content was measured using a micro-Kjeldahl procedure using a selenium catalyst and distillation by the methods of Ivan *et al.* (1974).

2.2.4.3 Carbon dioxide recovery

The basis of the quantitative determination of CO_2 is described by the following chemical equations:

- CO_2 (exhaled) + 2KOH —> K_2CO_3 + H_2O

- $K_2CO_3 + BaCl_2 \rightarrow BaCO_3$ (precipitate) + 2KCl

The KOH solution was washed with distilled water into a 2-litre flask and made up to volume. The amount of CO_2 absorbed in KOH solution was determined gravimetrically. To a 10 ml aliquot of the KOH, K_2CO_3 was added 6–10 ml of ammonium chloride (NH_4Cl) (200 g/l) to neutralize it, and 20–25 ml of barium chloride ($BaCl_2$) (300 g/l) was added to precipitate the barium carbonate ($BaCO_3$). The precipitate was centrifuged at 3500 rpm for 15 minutes and rechecked for the white precipitate by adding drops of $BaCl_2$. The clear supernatant was decanted, the precipitate was washed with distilled water and centrifuged again at 4000 rpm for 30 minutes. After decanting the supernatant, the precipitate was dried at 110°C overnight and allowed to cool in a dessicator. The weight of CO_2 produced was calculated from the weight of $BaCO_3$ precipitated from 2 litres.

2.2.5 Calculations

O_2 uptake was calculated by difference between the weight of the cylinder measured each day at the start and finish of a measurement period. CO_2 output was calculated from the amount of dry precipitate collected from the KOH flask as described previously. Differences in gas concentrations of chamber air at the start and finish of a daily measurement period were used to adjust final O_2 and CO_2 values.

The heat production was calculated, without correction for urinary nitrogen loss, using the equation of Romijn and Lokhorst (1961):

Heat production (kJ) = 16.20 O_2 (l) + 5.00 CO_2 (l).

Metabolizable energy (ME) was calculated by the difference between feed energy intake and excreta energy output. Maintenance energy was calculated when energy retention was zero.

$$\text{Metabolizability (\%)} = \frac{\text{Feed intake (g)} \times \text{GE in feed (kJ)} - \text{Excreta (g)} \times \text{GE in excreta (kJ)}}{\text{Feed intake} \times \text{GE in feed}} \times 100$$

Feed ME (kJ/g) = Metabolizability × GE in feed (kJ)

Energy balance (E balance) = ME intake - Heat production

$$\text{Energy retention efficiency (E efficiency, \%)} = \frac{\text{Energy balance}}{\text{ME intake}} \times 100$$

Nitrogen balance (N balance) = Nitrogen intake – Nitrogen excretion

Nitrogen retention efficiency (N efficiency, %) = $\frac{\text{Nitrogen retention}}{\text{Nitrogen intake}} \times 100$

The live weight (W) of the three birds in each chamber was taken as the means of weights recorded at the start and finish of the period of measurement. Data were expressed on a daily (d) and a body weight (W) or a metabolic body size ($W^{.75}$) basis.

2.2.6 Statistical analysis

The comparison of means was made for body weight, weight gain, feed intake metabolizability of dietary energy, feed ME, heat production, energy balance, nitrogen balance and energy and nitrogen retention efficiency using AOV (Steel and Torrie, 1960) on a Neva computer package. Energy balance and nitrogen balance were plotted and regressed against ME intake and nitrogen intake respectively and differences between these regression equations were compared using Lines and BMDP computer packages.

Chapter 3

RESULTS AND DISCUSSION: GROWTH TRIALS

3.1 Results

3.1.1 Trial 1

The results from this trial with four levels (0, 0.2, 0.4 and 0.6 ppm) of cimaterol on lean and fat line birds are shown in Table 3.1 and 3.2 respectively.

In the lean line (Table 3.1), no significant differences were observed in the size of abdominal fat pad between levels of cimaterol treatment although a 35% reduction in females and data for the combined sex at 0.2 ppm cimaterol approached significance ($0.05<P<0.10$). At the 0.2 and 0.4 ppm levels of cimaterol, carcass fat was significantly decreased ($P<0.05$) in the female group (20%) and for the combined sex (19%) but differences were not significant in males. Although final body weight and body weight gain were depressed on all the cimaterol-treated birds and significantly so at 0.2 and 0.4 ppm by 6% and 5% in final body weight, and by 8% and 7% in body weight gain ($P<0.05$), respectively, there was no difference in fat-free body weight. Feed intake was significantly depressed by 5% at 0.4 ppm cimaterol ($P<0.05$). No significant difference was observed between treatments in feed conversion ratio.

Table 3.1: The effects of cimaterol on the growth performance and fat content of lean line birds grown from 25 to 56 days of age in growth trial 1

Cimaterol levels (ppm)	0	0.2	0.4	0.6	SEM
Body weight (g)					
Start (25 days)	434 ^{a}	432 ^{a}	414 ^{a}	438 ^{a}	5.3
Finish (55 days)	1387 ^{a}	1310 ^{b}	1320 ^{b}	1347 ^{ab}	9.9
Weight gain (g)	953 ^{a}	878 ^{b}	886 ^{b}	909 ^{ab}	7.5
Feed intake (g)	2499 ^{a}	2444 ^{a}	2381 ^{b}	2441 ^{a}	16.9
FCR[1]	2.63 ^{a}	2.79 ^{a}	2.69 ^{a}	2.69 ^{a}	0.024
AFP(%)[2]					
Sex combined	1.30 ^{a}	0.84 ^{a}	0.93 ^{a}	1.25 ^{a}	0.047
Male	0.87 ^{a}	0.54 ^{a}	0.60 ^{a}	0.86 ^{a}	0.095
Female	1.72 ^{a}	1.13 ^{a}	1.25 ^{a}	1.63 ^{a}	0.101
Carcass fat (%)					
Sex combined	9.5 ^{a}	7.7 ^{b}	7.8 ^{b}	8.7 ^{ab}	0.13
Male	8.3 ^{a}	6.8 ^{a}	7.0 ^{a}	7.4 ^{a}	0.39
Female	10.8 ^{a}	8.7 ^{b}	8.5 ^{b}	10.0 ^{ab}	0.37
FFBW (g)[3]					
Sex combined	1245 ^{a}	1222 ^{a}	1212 ^{a}	1182 ^{a}	14.7
Male	1316 ^{a}	1311 ^{a}	1231 ^{a}	1217 ^{a}	42.5
Female	1175 ^{a}	1133 ^{a}	1193 ^{a}	1146 ^{a}	13.8

[1] *Feed conversion ratio = feed intake/weight gain;*

[2] *Abdominal fat pad;*

[3] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

Table 3.2: The effects of cimaterol on the growth performance and fat content of fat line birds grown from 25 to 56 days of age in growth trial 1

Cimaterol levels (ppm)	0	0.2	0.4	0.6	SEM
Body weight (g)					
Start (25 days)	421 ^{a}	419 ^{a}	411 ^{a}	430 ^{a}	5.3
Finish (55 days)	1442 ^{a}	1444 ^{a}	1423 ^{a}	1387 ^{a}	18.1
Weight gain (g)	1020 ^{a}	1025 ^{a}	1013 ^{a}	956 ^{a}	18.4
Feed intake (g)	2704 ^{a}	2701 ^{a}	2699 ^{a}	2586 ^{a}	35.3
FCR[1]	2.65 ^{a}	2.64 ^{a}	2.67 ^{a}	2.70 ^{a}	0.183
AFP(%)[2]					
Sex combined	3.28 ^{a}	3.20 ^{a}	2.78 ^{a}	3.06 ^{a}	0.073
Male	3.37 ^{a}	2.50 ^{ab}	3.15 ^{ab}	2.22 ^{b}	0.134
Female	3.19 ^{ab}	3.89 ^{a}	2.41 ^{b}	3.90 ^{a}	0.133
Carcass fat (%)					
Sex combined	13.3 ^{a}	13.1 ^{a}	12.6 ^{a}	13.1 ^{a}	0.33
Male	13.2 ^{a}	12.2 ^{a}	12.8 ^{a}	11.3 ^{a}	0.56
Female	13.4 ^{a}	14.0 ^{a}	12.4 ^{a}	15.0 ^{a}	0.65
FFBW (g)[3]					
Sex combined	1178 ^{a}	1220 ^{a}	1152 ^{a}	1174 ^{a}	10.2
Male	1310 ^{a}	1270 ^{a}	1265 ^{a}	1246 ^{a}	24.2
Female	1045 ^{b}	1170 ^{a}	1040 ^{b}	1103 ^{ab}	12.4

[1] *Feed conversion ratio = feed intake/weight gain;*

[2] *Abdominal fat pad;*

[3] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

In the fat line birds (Table 3.2), the size of abdominal fat pad was reduced by 34% ($P<0.05$) at 0.6 ppm cimaterol and by 26% at 0.2 ppm ($0.05<P<0.10$) in males. In females, a 24% decrease in abdominal fat ($P<0.05$) at 0.4 ppm level was observed. No significant differences were seen in carcass fat content and growth performance. There was a significant 12% ($P<0.05$) increase in fat-free body weight in females on diets with 0.2 ppm cimaterol.

Table 3.3 shows the effects of cimaterol on combined fat and lean line birds in this trial. Except for a 6% depression in growth rate at 0.6 ppm level ($P<0.05$), cimaterol showed no effect on growth performance and fat-free body weight. Abdominal fat was decreased significantly by 19% ($P<0.05$) at 0.4 ppm cimaterol for sex combined data. In males, abdominal fat was reduced by 28% at 0.2 ppm ($P<0.05$) and by 27% at 0.6 ppm ($0.05<P<0.10$); in females abdominal fat declined by 27% at 0.4 ppm of cimaterol ($P<0.05$). Carcass fat was decreased by 14% in females ($P<0.05$) and by 11% when sex of bird was combined ($0.05<P<0.10$).

3.1.2 Trial 2

The effects of 0.4 ppm cimaterol were tested in this trial and the results are shown in Table 3.4. Cimaterol showed no effect on the fat content of lean line birds while a significant reduction ($P<0.05$) was observed in both abdominal (18%) and carcass fat (12%) in fat line birds. Cimaterol-treated lean line birds ate 10% more feed ($P<0.05$) and both fat and lean lines of birds had a poorer feed conversion ratio ($P<0.05$).

A comparison of the growth performance and fat content between the genetically fat and lean line birds used in Trials 1 and 2 is shown in Table 3.5. The most striking differences between these two lines of birds were seen in their fat content. Fat line birds had 65% more abdominal fat and 35% more carcass fat in Trial 1 and 63% more abdominal fat and 39% more carcass fat in Trial 2 (all $P<0.001$). Fat line birds also had a higher feed intake in Trial 1 (9%, $P<0.001$) and a higher liveweight gain (10% in Trial 1, $P<0.001$ and 9% in Trial 2, $P<0.05$) which led to a better feed conversion in Trial 2 ($P<0.01$). There was no difference in fat-free body weight although fat line birds were about 6% heavier than lean

Table 3.3: The effects of cimaterol on the growth performance and fat cor tent of fat and lean line birds grown from 25 to 56 days of age in growth trial 1

Cimaterol levels (ppm)	0	0.2	0.4	0.6	SEM
Body weight (g)					
Start (25 days)	428 ^{ab}	426 ^{ab}	412 ^{b}	434 ^{a}	2.8
Finish (56 days)	1418 ^{a}	1377 ^{a}	1367 ^{a}	1367 ^{a}	7.2
Weight gain (g)	991 ^{a}	951 ^{ab}	947 ^{ab}	933 ^{b}	7.1
Feed intake (g)	2614 ^{a}	2573 ^{a}	2532 ^{a}	2514 ^{a}	15.0
FCR[1]	2.64 ^{a}	2.71 ^{a}	2.68 ^{a}	2.70 ^{a}	0.011
AFP(%)[2]					
Sex combined	2.29 ^{a}	2.02 ^{ab}	1.85 ^{b}	2.15 ^{ab}	0.030
Male	2.12 ^{a}	1.52 ^{b}	1.87 ^{ab}	1.54 ^{ab}	0.057
Female	2.46 ^{a}	2.51 ^{a}	1.83 ^{b}	2.77 ^{a}	0.062
Carcass fat (%)					
Sex combined	11.4 ^{a}	10.4 ^{a}	10.2 ^{a}	10.9 ^{a}	0.13
Male	10.7 ^{a}	9.5 ^{a}	9.9 ^{a}	9.4 ^{a}	0.23
Female	12.1 ^{ab}	11.4 ^{ab}	10.4 ^{b}	12.5 ^{a}	0.3
FFBW (g)[3]					
Sex combined	1212 ^{a}	1221 ^{a}	1182 ^{a}	1178 ^{a}	5.7
Male	1313 ^{a}	1290 ^{a}	1247 ^{a}	1231 ^{a}	15.4
Female	1110 ^{a}	1151 ^{a}	1116 ^{a}	1125 ^{a}	6.8

[1] *Feed conversion ratio = feed intake/weight gain;*

[2] *Abdominal fat pad;*

[3] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

Table 3.4: The effects of cimaterol on the growth performance and fat content of fat and lean line birds grown from 28 to 56 days of age in growth trial 2

	Lean line		Fat line		
Cimaterol levels (ppm)	0	0.4	0	0.4	SEM
Body weight (g)					
Start (28 days)	555 ^{b}	563 ^{ab}	568 ^{ab}	573 ^{a}	1.3
Finish (55 days)	1381 ^{b}	1429 ^{ab}	1514 ^{a}	1484 ^{ab}	10.8
Weight gain (g)	826 ^{a}	866 ^{a}	946 ^{a}	912 ^{a}	9.5
Feed intake (g)	2100 ^{b}	2313 ^{a}	2288 ^{ab}	2318 ^{a}	20.2
FCR[1]	2.54 ^{b}	2.68 ^{a}	2.42 ^{c}	2.54 ^{b}	0.007
AFP (%)[2]	0.97 ^{c}	1.30 ^{c}	3.37 ^{a}	2.75 ^{b}	0.041
Carcass fat (%)	7.8 ^{c}	8.9 ^{c}	14.5 ^{a}	12.8 ^{b}	0.12
FFBW (g)[3]	1185 ^{a}	1224 ^{a}	1191 ^{a}	1215 ^{a}	7.2

[1] *Feed conversion ratio = feed intake/weight gain;*

[2] *Abdominal fat pad;*

[3] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

Table 3.5: The influence of strain and sex on the growth performance and fat content in growth trials 1 and 2

	Trial 1				Trial 2			
	Lean	Fat		SEM	Lean	Fat		SEM
Body weight (g)								
Start	430	420	NS	1.3	554	570	*	0.7
Finish	1341	1424	***	3.7	1400	1499	*	4.8
Weight gain (g)	907	1004	***	3.6	846	929	*	5.2
Feed intake (g)	2444	2673	***	7.5	2207	2303	NS	10.1
FCR[1]	2.70	2.67	NS	0.005	2.61	2.48	**	0.003
AFP (%)[2]								
Sex combimed	1.08	3.08	***	0.015	1.14	3.06	***	0.019
Male	0.72	2.81	***	0.028				
Female	1.44	3.35	***	0.031				
Carcass fat (%)								
Sex combined	8.4	13.0	***	0.07	8.4	13.7	***	0.06
Male	7.4	12.4	***	0.12				
Female	9.5	13.7	***	0.15				
FFBW (g)[3]								
Sex combimed	1215	1181	NS	3.1	1205	1203	NS	3.6
Male	1269	1273	NS	7.7				
Female	1162	1089	NS	3.4				

NS: not significant;
** P<0.05;*
*** P<0.01;*
**** P<0.001;*
[1] *Feed conversion ratio = feed intake/weight gain;*
[2] *Abdominal fat pad;*
[3] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

Table 3.6: The effects of cimaterol and/or theophylline on the growth performance and fat content of female Hyline broilers grown from 28 to 49 days of age in growth trial 3

Treatment	1	2	3	4	SEM
Body weight (g)					
Start (28 days)	817 [a]	817 [a]	816 [a]	816 [a]	1.2
Finish (48 days)	1822 [a]	1612 [b]	1813 [a]	1474 [c]	20.8
Weight gain (g)	1005 [a]	789 [b]	997 [a]	658 [c]	20.0
Feed intake (g)	2366 [a]	1909 [b]	2358 [a]	1851 [b]	33.9
FCR[5]	2.36 [b]	2.42 [b]	2.37 [b]	2.82 [a]	0.039
AFP (%)[6]	2.05 [a]	1.20 [b]	2.17 [a]	1.79 [a]	0.037

1: control;
2: 0.1% theophylline;
3: 0.5 ppm cimaterol;
4: 0.1% theophylline plus 0.5 ppm cimaterol;
[5] *Feed conversion ratio = feed intake/weight gain;*
[6] *Abdominal fat pad.*

line birds at slaughter ($P<0.001$ in Trial 1 and $P<0.05$ in Trial 2).

3.1.3 Trial 3

The effects of 0.1% theophylline, 0.5 ppm cimaterol and the combination of these two on female Hyline birds are shown in Table 3.6. There was no difference between cimaterol-treated and control birds in either growth performance or fat content. Theophylline significantly decreased the size of the abdominal fat pad by 41% ($P<0.01$) and this was accompanied by about a 20% ($P<0.05$) depression in both feed intake and growth rate. Feed conversion was not influenced. Cimaterol plus theophylline showed no significant effect on the size of the abdominal fat pad but depressed ($P<0.05$) growth rate (35%) and feed intake (22%) which led to a poorer feed conversion ($P<0.05$) than both control birds and birds treated with theophylline alone.

Table 3.7: The effects of cimaterol and/or theophylline on the growth performance and fat content of male Hyline birds grown from 28 to 49 days of age in growth trial 4

Treatment	1	2	3	4	SEM
Body weight (g)					
Start (28 days)	858 [a]	859 [a]	859 [a]	860 [a]	0.7
Finish (48 days)	2006 [a]	1639 [b]	2012 [a]	1511 [b]	24.8
Weight gain (g)	1147 [a]	780 [b]	1153 [a]	651 [b]	25.4
Feed intake (g)	2691 [a]	1999 [b]	2687 [a]	1886 [b]	48.5
FCR[5]	2.34 [c]	2.56 [b]	2.34 [c]	2.91 [a]	0.030
AFP (%)[6]	2.42 [a]	1.40 [c]	2.37 [a]	1.87 [b]	0.028

1: control;
2: 0.1% theophylline;
3: 0.5 ppm cimaterol;
4: 0.1% theophylline and 0.5 ppm cimaterol;
[5] *Feed conversion ratio = feed intake/weight gain;*
[6] *Abdominal fat pad.*

3.1.4 Trial 4

The effects of 0.1% theophylline, 0.5 ppm cimaterol and cimaterol plus theophylline were re-examined in male Hyline birds and the results are shown in Table 3.7. As observed with the female Hyline birds, no effect of cimaterol was seen on either growth performance or fat content and theophylline significantly reduced the size of abdominal fat pad (42%, $P<0.01$), feed intake (26%, $P<0.05$), growth rate (32%, $P<0.05$) and feed conversion ratio ($P<0.05$). In birds treated with cimaterol plus theophylline, a significant 23% ($P<0.01$) reduction in the size of abdominal fat pad and a depressed ($P<0.05$) growth rate (43%), feed intake (30%) and feed conversion ratio were found.

3.1.5 Trial 5

Two levels of theophylline, 0.05% and 0.1%, were tested on female Hyline birds grown from 28 to 49 days of age in this trial and the results are shown in Table 3.8. As seen in Trials 3 and 4, theophylline at 0.1% inclusion significantly decreased the size of the abdominal fat

Table 3.8: The effects of theophylline on the growth performance and fat content of Hyline male birds grown from 28 to 49 days of age in growth trial 5

Theophylline (%)	0	0.05	0.1	SEM
Body weight (g)				
Start (28 days)	758 ^{a}	760 ^{a}	758 ^{a}	0.5
Finish (48 days)	1717 ^{a}	1587 ^{b}	1379 ^{c}	9.3
Weight gain (g)	958 ^{a}	826 ^{b}	621 ^{c}	9.0
Feed intake (g)	2405 ^{a}	2076 ^{b}	1717 ^{c}	20.9
FCR[1]	2.51 ^{b}	2.51 ^{b}	2.76 ^{a}	0.016
AFP (%)[2]	2.81 ^{a}	2.45 ^{a}	1.76 ^{b}	0.034

[1] *Feed conversion ratio = feed intake/weight gain;*
[2] *Abdominal fat pad.*

pad (37%, P<0.01) and depressed growth rate (35%, P<0.01), feed intake (29%, P<0.01) and feed conversion ratio (P<0.05). The effects of 0.05% level of theophylline were similar to those of 0.1% but less dramatic. A significant (P<0.05) depression in growth rate by 8% and feed intake by 14% was found. There was no difference in feed conversion ratio and the size of the abdominal fat pad at this theophylline level compared with controls.

3.1.6 Trial 6

The effects of the two levels of theophylline were re-examined on female Hyline birds in this trial and the results are presented in Table 3.9. Again, the inclusion of 0.1% theophylline decreased the size of the abdominal fat pad (42%, P<0.01), feed intake (27%, P<0.01), growth rate (36%, P<0.01) and feed conversion (P<0.05). Carcass fat was also measured in this trial and it was reduced by 22% (P<0.05). As observed in Trial 5, theophylline at 0.05% showed similar but less dramatic effects than those at 0.1% inclusion with a depression in growth rate by 16% and in feed intake by 16%. But a significant reduction in abdominal fat pad by 26% (P<0.05) was also seen here. There were no differences in feed conversion ratio and carcass fat content at this theophylline level compared with control birds. Theophylline included at the rate of 0.1% in the diet and administered during 39–48 days showed a 17%

Table 3.9: The effects of theophylline on the growth performance and fat content of female Hyline broilers grown from 28 to 49 days of age in growth trial 6

Theophylline (%)	0	0.1	0.05	0.1 (39-48 days)	Pair-fed[1]	SEM
Body weight (g)						
Start (28 days)	792 ^{a}	792 ^{a}	792 ^{a}	792 ^{a}	792 ^{a}	0.2
Finish (48 days)	1696 ^{a}	1374 ^{c}	1548 ^{b}	1441 ^{c}	1393 ^{c}	11.5
Weight gain (g)	904 ^{a}	583 ^{c}	756 ^{b}	650 ^{c}	601 ^{c}	11.6
Feed intake (g)	2360 ^{a}	1723 ^{c}	1973 ^{b}	1896 ^{b}	1747 ^{c}	18.9
FCR[2]	2.61 ^{b}	2.98 ^{a}	2.61 ^{b}	2.92 ^{a}	2.91 ^{a}	0.033
AFP (%)[3]	2.62 ^{a}	1.51 ^{c}	1.95 ^{b}	2.18 ^{b}	2.06 ^{b}	0.019
Carcass fat (%)	13.4 ^{a}	10.5 ^{b}	12.2 ^{a}	12.4 ^{a}	12.6 ^{a}	0.17
FFBW (g)[4]	1371 ^{a}	1159 ^{c}	1255 ^{b}	1174 ^{c}	1123 ^{c}	7.6

[1] *on control diet with restricted feed intake to that of 0.1% theophylline;*
[2] *Feed conversion ratio = feed intake/weight gain;*
[3] *Abdominal fat pad;*
[4] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

reduction in abdominal fat ($P<0.05$) and a poorer feed conversion ratio ($P<0.05$). Feed intake and growth rate ($P<0.01$) were reduced. The only difference between 0.1% level of theophylline treated birds and those on the control diet but pair-fed to the 0.1% theophylline treated group was in fat content, i.e. the birds treated with 0.1% theophylline had 27% less abdominal fat ($P<0.05$) and 17% less carcass fat ($P<0.05$) than the pair-fed birds. The pair-fed birds had 21% less abdominal fat ($P<0.05$) than *ad libitum* fed control birds. Fat-free body weight was similarly influenced as was final body weight; this was 15% lower on the diet with 0.1% theophylline ($P<0.01$), 8% with 0.05% ($P<0.05$), 14% with 0.1% for the final 10 (39–48) days, ($P<0.01$) and 14% in the pair-fed group ($P<0.01$).

3.1.7 Trial 7

The effects of two levels of caffeine, 0.05 and 0.1%, on growth rate and fat content of Steggles male broilers are shown in Table 3.10. A significant 66% ($P<0.05$) reduction in abdominal fat was seen in birds treated with 0.1% caffeine. There was no difference in feed conversion ratio between caffeine-treated and control birds while a similar depression was

Table 3.10: The effects of caffeine on the growth performance and fat content of Steggles male chickens grown from 28 to 49 days of age in growth trial 7

Caffeine (%)	0	0.05	0.1	Pair-fed[1]	SEM
Body weight (g)					
Start (28 days)	718 ^{a}	719 ^{a}	719 ^{a}	721 ^{a}	4.9
Finish (48 days)	1876 ^{a}	1771 ^{a}	1622 ^{b}	1421 ^{c}	17.8
Weight gain (g)	1163 ^{a}	1052 ^{b}	903 ^{c}	700 ^{d}	17.1
Feed intake (g)	2414 ^{a}	2189 ^{b}	1867 ^{c}	1845 ^{c}	29
FCR[2]	2.10 ^{b}	2.09 ^{b}	2.07 ^{b}	2.65 ^{a}	0.034
AFP (%)[3]	0.89 ^{a}	0.79 ^{a}	0.30 ^{b}	0.87 ^{a}	0.013

[1] *on control diet with feed intake restricted to 0.1% caffeine group;*
[2] *Feed conversion ratio = feed intake/weight gain;*
[3] *Abdominal fat pad.*

observed both in feed intake (23% at 0.1% inclusion, $P<0.01$ and 9% at 0.05%, $P<0.05$) and in growth rate (22% at 0.1% inclusion, $P<0.01$ and 10% at 0.05% inclusion, $P<0.05$). Compared with controls, the pair-fed birds showed no difference in abdominal fat, a 24% ($P<0.01$) lower feed intake, 40% ($P<0.001$) lower growth rate and a poorer feed conversion ratio ($P<0.05$). Compared with 0.1% caffeine-treated birds to which they were pair-fed, the pair-fed birds had a 22% lower growth rate ($P<0.05$), a poorer feed conversion ratio ($P<0.05$) and 66% higher abdominal fat ($P<0.05$).

3.1.8 Trial 8

The effects of iodinated casein and/or theophylline on the growth performance and fat content of Steggles male broilers from 4 to 7 weeks of age are shown in Table 3.10. Theophylline at 0.05% inclusion gave similar results to those obtained in Trial 6, i.e. a significant ($P<0.05$) reduction in the size of the abdominal fat pad (31%), in feed intake (15%) and in growth rate (12%). Both levels of iodinated casein, 50 and 100 ppm, significantly ($P<0.05$) decreased abdominal fat by 39% and 32% respectively. The depression in feed intake at these two levels (9 and 10 % respectively, $P<0.05$) combined with the maintained growth

Table 3.11: The effects of iodinated casein and/or theophylline on the growth performance and fat content of Steggles male chickens grown from 28 to 49 days of age in growth trial 8

Treatment	1	2	3	4	5	6	SEM
Body weight (g)							
Start (28 days)	723 ^{d}	724 ^{cd}	725 ^{c}	727 ^{bc}	728 ^{ab}	729 ^{a}	0.3
Finish (48 days)	1855 ^{a}	1750 ^{b}	1908 ^{a}	1657 ^{c}	1862 ^{a}	1679 ^{c}	13.7
Weight gain (g)	1133 ^{a}	1001 ^{b}	1183 ^{a}	931 ^{b}	1134 ^{a}	950 ^{b}	13.1
Feed intake (g)	2594 ^{a}	2194 ^{cd}	2362 ^{b}	2000 ^{e}	2338 ^{bc}	2070 ^{de}	24.3
FCR[7]	2.29 ^{a}	2.19 ^{ab}	2.00 ^{d}	2.15 ^{bc}	2.06 ^{cd}	2.19 ^{ab}	0.021
AFP (%)[8]	1.16 ^{a}	0.80 ^{b}	0.71 ^{bc}	0.54 ^{c}	0.79 ^{b}	0.53 ^{c}	0.013

1: control;
2: 0.05% theophylline;
3: 50 ppm iodinated casein;
4: 0.05% theophylline plus 50 ppm iodinated casein;
5: 100 ppm iodinated casein;
6: 0.05% theophylline plus 100 ppm iodinated casein;
[7] *Feed conversion ratio = feed intake/weight gain;*
[8] *Abdominal fat pad.*

rate was reflected in an improved feed conversion ($P< 0.01$). The inclusion of 0.05% theophylline plus 50 ppm or 100 ppm iodinated casein depressed growth rate by 17% on average ($P<0.05$) and feed intake by 22% ($P<0.05$). The abdominal fat pad was reduced by 54% ($P<0.01$) and this reduction was more dramatic than that seen with either iodinated casein or theophylline alone. This further decrease in abdominal fat was about 34% ($P<0.05$) over that caused by theophylline, 25% ($0.05<P<0.10$) over that caused by 50 ppm iodinated casein and 33% ($P<0.05$) over that caused by 100 ppm iodinated casein. No further response was found when the inclusion rate of iodinated casein was doubled (100 ppm).

3.1.9 Trial 9

The effects of iodinated casein and/or caffeine on the growth performance and fat content of male birds are shown in Table 3.12. In this trial, 50 ppm iodinated casein showed no effect on feed intake and feed conversion as it did in Trial 8; abdominal fat was decreased by 18% ($P<0.05$). The effects of the two levels of caffeine, 0.05% and 0.1%, obtained in

Table 3.12: The effects of iodinated casein and/or caffeine on the growth performance and fat content of male Steggles broilers grown from 28 to 49 days of age in growth trial 9

Treatment	1	2	3	4	5	SEM
Body weight (g)						
Start (28 days)	827 ^{a}	829 ^{a}	832 ^{a}	829 ^{a}	828 ^{a}	0.9
Finish (48 days)	2152 ^{a}	2164 ^{a}	2003 ^{b}	2018 ^{b}	1838 ^{c}	14.0
Weight gain (g)	1323 ^{a}	1337 ^{a}	1175 ^{b}	1158 ^{b}	1008 ^{c}	12.5
Feed intake (g)	2628 ^{a}	2641 ^{a}	2426 ^{b}	2380 ^{b}	2077 ^{c}	20.4
FCR[6]	1.99 ^{a}	1.98 ^{a}	2.07 ^{a}	2.06 ^{a}	2.06 ^{a}	0.016
AFP (%)[7]	1.30 ^{a}	1.06 ^{b}	1.27 ^{a}	1.05 ^{b}	0.70 ^{c}	0.014
Carcass fat (%)	10.2 ^{a}	9.6 ^{a}	9.7 ^{a}	9.7 ^{a}	8.1 ^{b}	0.18
FFBW (g)[8]	1841 ^{ab}	1888 ^{a}	1736 ^{c}	1755 ^{bc}	1626 ^{d}	14.9

1: control;
2: 50 ppm iodinated casein;
3: 50 ppm iodinated casein plus 0.05% caffeine;
4: 0.05% caffeine;
5: 0.1% caffeine;
[6] *Feed conversion ratio = feed intake/weight gain;*
[7] *Abdominal fat pad;*
[8] *Fat free body weight = slaughter body weight - (AFP + carcass fat).*

Table 3.13: The regression analysis for body weight (W, g), abdominal fat pad (AFP, %) and carcass fat (FAT, %)

Trials	Regression equations	R^2	RSD
1	AFP=-1.93 + 0.0029 W	0.09	1.15
2	AFP=-1.28 + 0.0025 W	0.11	1.22
1	FAT= 2.36 + 0.0061 W	0.06	2.88
2	FAT= 1.67 + 0.0068 W	0.12	3.27
1	FAT= 5.90 + 2.33 AFP	0.89	1.01
2	FAT= 6.02 + 2.42 AFP	0.81	1.52
6	FAT= 8.30 + 1.94 AFP	0.57	1.20
9	FAT= 6.32 + 2.75 AFP	0.71	0.51

Trial 7 were confirmed in this trial except for a significant 19% reduction in abdominal fat at the inclusion rate of 0.05% ($P<0.05$). The size of the abdominal fat pad declined at 0.1% inclusion of caffeine by 46% ($P<0.01$) and carcass fat by 21% ($P<0.05$). Iodinated casein plus caffeine showed no effect on feed conversion as feed intake and growth rate were similarly depressed ($P<0.05$) by 8% and 11% respectively. The effect of this combination on abdominal fat in this trial was different from that obtained with theophylline plus iodinated casein in Trial 8 in that no reduction in abdominal fat was seen here. Except for a 12% ($P<0.05$) lower fat-free body weight in 0.1% caffeine-treated birds than the controls, there was no difference in this measurement between controls and other treatments.

3.1.10 The correlations between body weight, abdominal and carcass fat

The correlations between body weight, abdominal and carcass fat are shown in Table 3.13. Neither abdominal nor carcass fat correlated well with body weight as seen from the R^2 values. A close relationship was obtained between abdominal and carcass fat content, this was particularly so in the two genetically fat and lean lines of birds in Trials 1 and 2. The regression lines for abdominal fat pad and carcass fat in Trials 1 and 2 are shown in Figure 3.1.

3.2 Discussion

The striking difference in both abdominal and carcass fat contents between the two genetically fat and lean lines of birds in Trials 1 and 2 (Table 3.5) demonstrates the effectiveness of selection against abdominal and body fat content using fat pad calipers (Pym and Thompson, 1980). This agrees with other studies showing marked difference in fat content of broilers by using direct measurement of abdominal fat content in slaughter experiments (LeClercq, 1984; Cahaner *et al.*, 1986) or by measuring plasma VLDL levels in live birds (Whitehead and Griffin, 1984). The correlated response of feed conversion with selection for low body fat observed here is inconstant with the study of Whitehead *et al.* (1984) who found that plasma VLDL concentration was significantly correlated with feed conversion

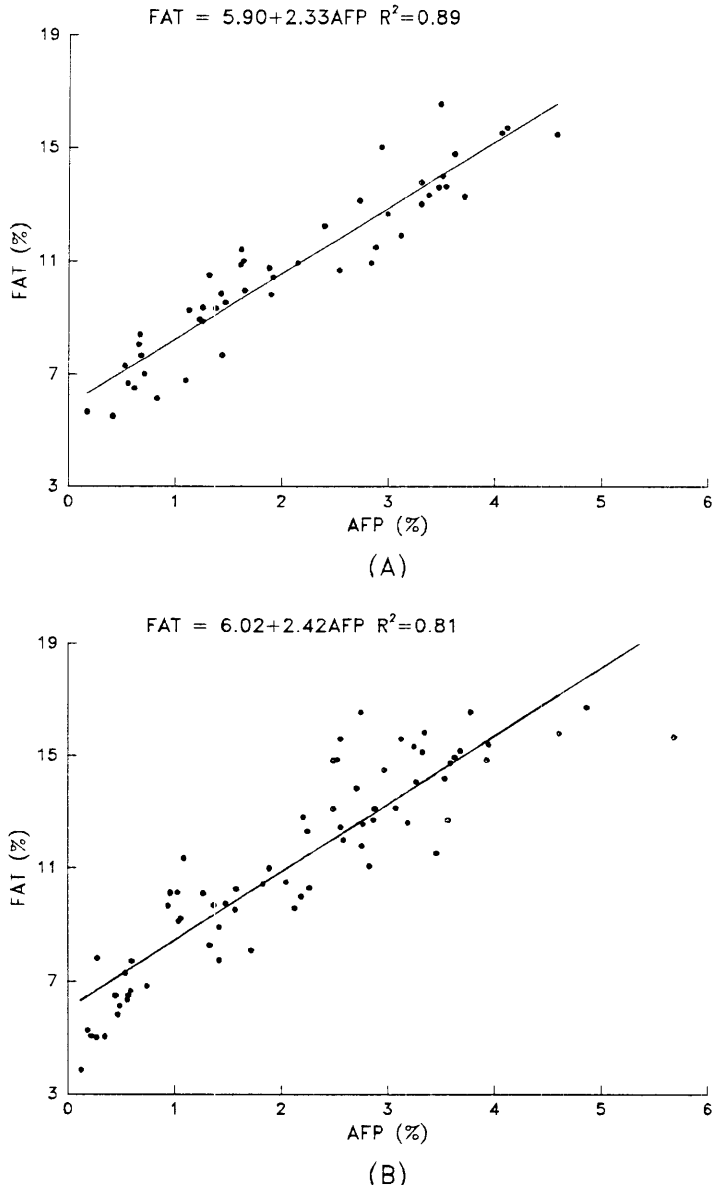

Figure 3.1: Relationship between abdominal fat pad (AFP) and carcass fat content (FAT) in Trial 1 (A) and Trial 2 (B)

as well as with body fat content. It, however, agrees with the result of the study of Pym (1985a) in which he showed that a line of broilers selected for lean tissue growth was 33% leaner than the line selected for growth rate alone and that despite the difference in fatness, there was no difference in feed conversion ratio from 4–8 weeks of age. This was unexpected since the decreased fat deposition with its higher energetic cost should lead to an improvement in feed conversion as proposed by Brody (1935) and selection in chickens for improved feed conversion has generally resulted in a decrease in fat content (Thomas *et al.*, 1958; Washburn *et al.*, 1975; Pym and Solvyns, 1979; Chambers *et al.*, 1983). The similar fat-free body weights observed in sex combined data between fat and lean lines of birds in Trials 1 and 2 confirm the suggestion that selection for abdominal fat should reduce fat in other locations without a change in fat-free body weight (Pym, 1979; Becker *et al.*, 1979).

The reduced correlation between body weight and abdominal or carcass fat content observed in this study (Table 3.13) is in keeping with previous findings (Nir, 1984; Cahaner, 1986). This may be evidence for the view of Pym and Solvyns (1979) that selection for growth rate is not necessarily accompanied by an increase in fat content. The reasonable agreement between abdominal and total fat content observed by other researchers (Hood, 1982; Whitehead and Griffin, 1984) is confirmed in the present study (Table 3.13, Figure 3.1) and the correlation coefficient is close to the range of values of 0.40 and 0.80 as summarized by Pym (1979) from the published results. Thus, abdominal fat is representative of total body lipid.

Trials 1 and 2 were the first to compare the effects of cimaterol on chickens genetically disposed towards high and low fat contents. It was expected that a lipolytic agent would show its fat mobilizing effect more readily in animals with a high fat content. This proved to be the case in Trial 2 (Table 3.4) where a significant reduction was seen in both abdominal and carcass fat in a fat line of birds but this was not so in Trial 1 (Table 3.1 and 3.2) where less consistent results were obtained. An apparent sex effect on the magnitude of response to cimaterol or clenbuterol was noted in the experiments conducted by Dalrymple *et al.* (1984b) and female birds were shown to respond more consistently than males to both growth promoting and repartitioning effects of these two beta-agonists under the conditions

used in those studies. Results from laboratory rats also suggested that beta-agonists were more effective if body fat content was high and if the capacity for protein retention was low (Greife *et al.*, 1987; Berschauer *et al.*, 1987), i.e., females were more responsive than males in fat mobilization and effects on body protein increased as animals approached maturity. Such a sex effect was observed in lean line birds treated with 0.2 and 0.4 ppm levels of cimaterol (Table 3.1) but not in fat line birds (Table 3.2). This may be explained partly by the much larger difference in fat content between males and females in the lean line (50% for abdominal fat and 22% for carcass fat, $P<0.01$, Table 3.5) and relatively smaller difference between males and females in the fat line (16% for abdominal fat and 9% in carcass fat, $P<0.05$). No marked sex effect was found in cimaterol-treated birds in studies in West Germany (Scholtyssek, 1987).

While cimaterol was shown to be effective in decreasing fat content in Trials 1 and 2 in the genetically fat and lean lines of birds, in Trials 3 (Table 3.6) and 4 (Table 3.7) with commercial birds, neither abdominal fat pad nor the growth performance was influenced by the inclusion of cimaterol. There are no ready and easy reasons to explain why the commercial birds, whose proportion of abdominal fat was between fat and lean lines of birds used in Trials 1 and 2, did not respond to cimaterol at all. Carcass fat was not analysed in these two trials.

Except for the significant improvement in fat-free body weight in the high-fat line birds treated with 0.2 ppm cimaterol in Trial 1 (Table 3.2), no effect of cimaterol on improving growth performance was observed in this study. This does no seem to be in agreement with the results obtained by Dalrymple *et al.* (1984b) and Dalrymple and Ingle (198") in the US with broiler chickens where cimaterol or clenbuterol showed small but significant effects on both improving the growth performance and on decreasing carcass fat content (see Table 1.3). The depression in growth rate in Trial 1 in lean line birds treated with cimaterol may be seen partly as the consequence of the significant decrease in carcass fat and marked reduction in abdominal fat content since there was no difference in fat-free body weight (Table 3.1).

In the investigations conducted in the US with broiler chickens (Dalrymple *et al.*, 1984b;

Dalrymple and Ingle, 1987), little effect of cimaterol or clenbuterol on abdominal fat pads was found. This does not agree with the results from Trials 1 and 2 in the present study where cimaterol showed lipolytic effects on both abdominal fat and carcass fat and with those of the six trials carried out in West Germany where abdominal fat was reduced significantly by about 10% on average (Scholtyssek, 1987). Dalrymple *et al.* (1984b) attempted to explain this unresponsiveness of abdominal fat to clenbuterol or cimaterol in their trials by citing the study of Carlson *et al.* (1964) which was the first published paper showing the unresponsiveness of chicken adipose tissue to catecholamines which are structurally similar to clenbuterol and cimaterol (see Figure 1.7). It should be noted, however, that in the study of Carlson *et al.* (1964), the unresponsiveness to catecholamines was found both *in vivo* from the anesthetized fowl and *in vitro* from adipose tissue pieces and there was no indication of the origin of the fat tissue.

The exact mode of action of the β-adrenergic agonists is still unknown but it has been shown that these agonists act in muscle and adipose tissue and exert their effects on both protein and lipid metabolism (Buttery and Dawson, 1987). In chickens, data from the *in vitro* study suggested that cimaterol inhibits protein breakdown primarily via a nonlysosomal proteolytic mechanism (Rogers and Fagan, 1988) and data from an *in vivo* study demonstrated that differences in muscle weight observed in cimaterol treatment are due to the change in protein fractional degradation rate and not fractional synthesis rate (Morgan *et al.*, 1988b). These observations with chickens agree with those found in rats (Reeds *et al.*, 1986) and calves (Williams *et al.*, 1987) and may partly explain the improved fat-free body weight in female fat line birds treated with 0.2 ppm cimaterol in Trial 1 (Table 3.2).

There has been so far no fundamental study using clenbuterol or cimaterol to investigate their effects on avian lipid metabolism. The results from a study with other β-agonists (isoproterenol, epinephrine and norepinephrine) in chicken hepatocytes and adipose tissue explants *in vitro* tended to suggest that the effects of clenbuterol or cimaterol on lipid metabolism is due to inhibition of lipogenesis rather than stimulation of lipolysis (Campbell and Scanes, 1985). This is supported by the findings with sheep which showed a marked suppression of *in vitro* acetate incorporation (Thornton *et al.*, 1985) and a reduced total

number of fat cells (Coleman *et al.*, 1985) induced by clenbuterol treatment. A study with rats also showed that clenbuterol appeared to be 5–10 times more potent as an anti-lipogenic than as a lipolytic agent (Duquette and Muir, 1985). The less pronounced effects of clenbuterol or cimaterol on chickens than on mammals tested may be due to the chicken liver being the major site of fatty acid synthesis as found by Goodridge (1968c) and O'Hea and Leveille (1969) while adipose tissue in most mammals fulfills this function. It was shown that most of the lipolytic hormones exert their effects on adipose tissue but there is a striking species variation in the response of adipose tissue to these hormones (Butcher *et al.*, 1972). Cimaterol and clenbuterol tend to bind selectively to the β_2-receptor which exhibits a greater affinity for adrenalin in rats (Buttery and Dawson, 1987). It is noteworthy then that the adrenal medullary output of chickens is about 80% norepinephrine and in pigs 49% while in ruminants only 30% (West, 1955). This may explain to some degree the smaller response of chickens to clenbuterol and cimaterol.

There has been so far no work published to explore the effects of theophylline or caffeine on fat content and growth performance in farm animals. These two alkaloids have shown very consistent and similar effects in decreasing both the size of the abdominal fat pad (37–66%) and carcass fat content (21–22%) at 0.1% level of inclusion. The clear difference in fat content between the birds treated with 0.1% theophylline (Table 3.9) or caffeine (Table 3.10) and those pair-fed to them on the control diet demonstrates that theophylline and caffeine *per se* had an effect in reducing fat content over that caused by depression of feed intake. This marked decrease in fat content observed in the present study with commercial broiler chickens is in accord with the results from both *in vivo* (Freeman and Manning, 1971) and *in vitro* findings showing the effects of theophylline or caffeine on increasing the release of glycerol and NEFA (Langslow and Hales, 1969; Langslow, 1972). The mechanism by which theophylline and caffeine exert their lipolytic effects has been proposed as they inhibit the enzyme cAMP phosphodiesterase which hydrolyses cAMP to AMP within the cell of adipose tissue (Butcher and Sutherland, 1962), therefore, the concentration of cAMP required for activation of hormone-sensitive lipase is maintained and the process of lipolysis is enhanced (see Figure 1.1).

Although there have been some findings showing the synergistic lipolytic effects of catecholamines and theophylline or caffeine *in vitro* on adipose tissue of birds (Langslow and Hales, 1969; Langslow, 1972), Trials 3 and 4 were the first to explore their effects in live commercial birds. In both trials (Table 3.6 and 3.7) the effect of cimaterol plus theophylline on reducing the size of abdominal fat was smaller than that of theophylline alone. This was not expected from the *in vitro* studies with isolated chicken fat cells which showed that theophylline was able to potentiate the lipolytic effects of adrenalin (Table 1.1, Langslow and Hales, 1969) and noradrenalin (Langslow, 1972), which are structurally similar to cimaterol (Figure 1.7). Cimaterol itself did not show any effects on either abdominal fat or growth performance in these two trials. Thus, the observed difference between control birds and those treated with the combined cimaterol and theophylline should be attributed to the effects of theophylline. But cimaterol must have exerted some effects in some way since there was a discrepancy in both abdominal fat and growth performance between the birds treated with theophylline alone and those with the combination. The reason for this discrepancy is unknown.

The marked decrease in the size of abdominal fat pad in Trial 8 (Table 3.11) and Trial 9 (Table 3.12) and the improvement in the growth performance in Trial 8 observed at the two levels of iodinated casein used in the present study are in agreement with previous findings at lower levels (Irwin *et al.*, 1943; Turner *et al.*, 1944; Wheeler *et al.*, 1948; Boone *et al.*, 1950; Herbert and Brunson, 1957; Singh *et al.*, 1968; Ringer, 1976; Adams and Stadelman, 1978; Wilson *et al.*, 1983). The reduction in the size of abdominal fat pad obtained here also agrees with the fundamental work in rats by Malbon *et al.* (1978) who reported the weight of adipose tissue from hypothyroid rats was 57% greater and that from hyperthyroid rats was 46% less than that of controls. The improved feed conversion in birds treated with iodinated casein during the finisher period (4–7 weeks of age) in Trial 8 confirms the conclusion of Koger *et al.* (1943) that the thyroactive iodocasein-fed chicks utilized their feed more efficiently than the controls only during the periods when growth was most rapid.

Some of the researchers measured the change in the weight of thyroid in birds fed thyroxine and found that treated birds generally had lighter thyroids than those of untreated

controls (Irwin *et al.*, 1943; Turner *et al.*, 1944; Roberson and Trujillo, 1975; Wilson *et al.*, 1983). This was explained as thyroactive protein supply was replacing the normal thyroid production and Turner *et al.* (1944) found a tendency for the thyroid to return towards the normal weight and normal histological picture upon the elimination of iodinated casein from the diet. This was, in fact, in agreement with the quite well accepted conclusions that thyroid hormones are required for normal growth and growth rate is independent of the thyroid over a fairly wide range of thyroid activities; but growth retardation may occur under conditions of hypothyroidism or hyperthyroidism of genetic or nutritional origin (Ringer, 1976; Falconer, 1984; Scanes *et al.*, 1984).

The regulation of adipose tissue metabolism by thyroid hormones has been clearly demonstrated in both *in vivo* and *in vitro* studies in both chickens and rats (see section 1.2.4.4 and for review see Fain, 1980). However, the data concerning the mechanisms involved are scanty in chickens and conflicting in rats. It has also been difficult to obtain consistent *in vitro* effects of triiodothyronine at low concentrations on rat fat cell metabolism. The hyperthyroid state is the opposite of the hypothyroid state and it results in increased synthesis of lipid, enhanced breakdown and mobilization of lipid, and increased fatty acid oxidation in rats (Diamant *et al.*, 1972). Hypothyroidism in the chicken has been shown to induce liver hypertrophy and increased glycogen deposition (Snedecor and King, 1964; Snedecor, 1968). However, these early studies paid little attention to possible changes in lipid metabolism due to the change in liver which is the main site for lipogenesis (Goodridge, 1968c; O'Hea and Leveille, 1969). Data from a study with rats indicated that it is changes in the size rather than the number of fat cells which are responsible for the alteration in the weight of adipose tissue (Malbon *et al.*, 1978). The activation of adenylate cyclase by catecholamines is reduced by hypothyroidism and it was proposed that thyroid hormones may exert their influence on fat cells by regulating the coupling of the hormone-receptor complexes to adenylate cyclase (Malbon *et al.*, 1978). The observations from other studies with rats demonstrated that the sensitivity of fat cells to all lipolytic agents and the ability of lipolytic agents to elevate cAMP are influenced by thyroid hormones, and hypothyroid reduced the sensitivity of fat cell lipolysis to all agents such as catecholamines, ACTH and

glucagon (Goodman and Bray, 1966). This can be taken to explain the synergistic effect of iodinated casein plus theophylline on decreasing the size of abdominal fat pad in Trial 8 (Table 3.11). Vaughan (1967), however, found an immediate effect of triiodothyronine on the lipolytic response of rat adipose tissue to ACTH, thyroid-stimulating hormone (TSH) and catecholamines, but not that to theophylline or dibutyryl cAMP and concluded that the 'sensitivity' of the rat adipose to theophylline was not altered by thyroid status. TSH was once shown to increase cAMP levels in the presence of 1 mM caffeine in rat fat cells (Butcher *et al.*, 1968). As far as the author is aware, no such work has been reported with chicken fat cells. If this is also the case in birds, it is hard then to explain why fat content was synergistically decreased by the combination of theophylline and iodinated casein and not by iodinated casein plus caffeine since caffeine is an analogue of theophylline and has shown very similar effect on decreasing fat content to that of theophylline as discussed previously. Again, a species difference between chickens and rats in their response to various lipolytic agents may be involved.

Chapter 4

RESULTS AND DISCUSSION: RESPIRATION CALORIMETRY EXPERIMENTS

4.1 Results With Tables and Figures

4.1.1 Experiment 1

Daily measurements of body weight, feed intake, R.Q. and heat production for the two genetically fat and lean lines on either the control or the 0.4 ppm cimaterol diet are presented in Appendix E. Comparisons between the two lines for heat production and energy and nitrogen metabolism are shown in Table 4.1.

4.1.1.1 Fat and lean lines

Heat production was significantly higher in the lean line by 8% on both a body weight (W) and a metabolic body size basis ($W^{.75}$) ($P<0.05$) and this difference in heat production between these two lines in males was close to significance ($0.05<P<0.10$). The regression equations and regression lines for ME intake and energy balance, maintenance energy (E_m) calculated from these linear regressions, and the results of the analysis of these regression

Table 4.1: Comparisons of heat production and energy (E) and nitrogen (N) balance for 3 days of fat and lean line birds aged from 25 to 38 days in calorimetry experiment 1

	Sex combined (N=7)			Males (N=4)			Female (N=3)		
	Fat	Lean	SEM	Fat	Lean	SEM	Fat	Lean	SEM
Body weight (kg)	2.33	2.33	0.018	2.01	2.00	0.008	2.76	2.77	0.061
Weight gain (g)	202	194	14.1	268	185	34.2	114	206	17.0
Feed intake (g)	542	526	8.0	551	496	15.1	529	566	7.4
Metabolizability (%)	74.2	74.8	0.69	74.4	74.1	0.24	73.9	75.8	0.58
ME[1] intake									
kJ/d/W	1051	1047	22.2	1232	1170	53.9	808	884	7.8
kJ/d/$W^{.75}$	1257	1248	23.2	1419	1329	55.8	1042	1140 *	4.0
Heat production									
kJ/d/W	714	774 *	6.3	771	846	11.9	638	677	9.5
kJ/d/$W^{.75}$	855	926 *	6.1	886	967	11.7	814	872	11.6
Energy balance									
kJ/d/W	337	273	22.2	462	324	48.0	170	207	8.2
kJ/d/$W^{.75}$	402	322	24.6	533	362	51.9	228	269	10.3
E efficiency (%)[2]	30.0	25.3	1.31	36.6	26.7	2.37	21.0	23.4	0.88
N intake									
g/d/W	2.51	2.48	0.050	2.94	2.79	0.117	1.94	2.07	0.019
g/d/$W^{.75}$	3.02	2.96	0.054	3.41	3.19	0.122	2.50	2.67 *	0.009
N excretion									
g/d/W	1.23	1.20	0.018	1.39	1.36	0.047	1.02	0.98	0.023
g/d/$W^{.75}$	1.49	1.43	0.021	1.61	1.54	0.050	1.31	1.27	0.032
N balance									
g/d/W	1.28	1.29	0.035	1.55	1.44	0.076	0.92	1.08	0.028
g/d/$W^{.75}$	1.53	1.53	0.041	1.79	1.64	0.078	1.18	1.40	0.032
N efficiency (%)[3]	50.1	51.9	0.52	52.1	51.6	0.64	47.4	52.3	1.17

* *$P<0.05$;*

[1] *Metabolizable energy;*

[2] *Energetic efficiency = (Energy balance×100)/ME intake;*

[3] *Nitrogen retention efficiency = (N balance×100)/N intake.*

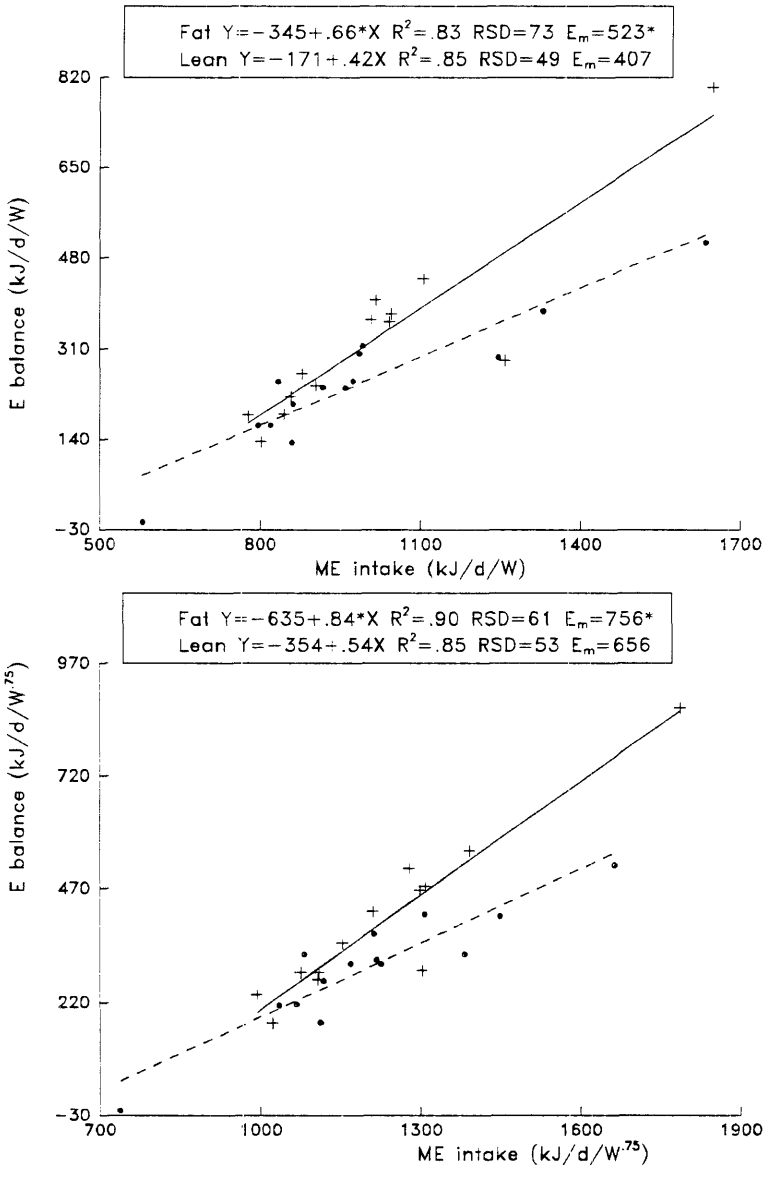

Figure 4.1: Relationship between ME intake and energy balance of fat (+ —) and lean (... - - -) lines of birds in Experiment 1 (* $P<0.05$ compared with lean line.)

equations are shown in Figure 4.1. The significant difference in the slope of the regression equations indicated that fat line birds had higher E_m and retained more energy per unit of ME intake ($P<0.05$ on both a W and a $W^{.75}$ basis).

4.1.1.2 Control and cimaterol-treated birds

Comparisons between controls and 0.4 ppm cimaterol treated fat line birds are shown in Table 4.2. The regression equations, the regression lines for ME intake and energy balance and E_m are presented in Figure 4.2. No difference was found in heat production and energy or nitrogen metabolism between control and cimaterol-treated fat line birds.

Table 4.3 presents comparisons between control and cimaterol-treated lean line birds. Control female birds had a higher heat production on a $W^{.75}$ basis ($P<0.05$). A 10% higher nitrogen intake was seen in control female birds ($P<0.05$) and since there was no difference in nitrogen excretion, they retained 17% more nitrogen on a W basis ($P<0.05$) and 20% on a $W^{.75}$ basis ($0.05<P<0.10$). There was no difference in nitrogen retention efficiency between control and cimaterol-treated birds ($P>0.05$). Maintenance energy, the regression equations and regression lines for ME intake and energy balance are presented in Figure 4.3. The regression analysis revealed no difference between control and treated lean line birds.

4.1.2 Experiment 2

The aim of this experiment was to repeat part of Experiment 1. ME intake was closely regulated to examine differences between the same fat and lean lines of female birds in heat production and energy and nitrogen metabolism that were independent of feed intake during eight measurement periods. Daily measurements of body weight, feed intake, R.Q. and heat production for each five-day period are presented in Appendix F. Comparisons between these two lines in heat production and energy and nitrogen metabolism are shown in Table 4.4.

Table 4.2: The effects of cimaterol on heat production and energy (E) and nitrogen (N) balance for 3 days of fat line birds aged from 25 to 38 days in calorimetry experiment 1

	Sex combined (N=6)			Male (N=3)			Female (N=3)		
	–[1]	+[2]	SEM	–	+	SEM	–	+	SEM
Body weight (kg)	2.41	2.39	0.049	2.30	2.23	0.016	2.53	2.55	0.097
Weight gain (g)	237	248	17.2	241	326	13.5	233	169	28.0
Feed intake (g)	549	544	16.4	563	580	20.0	535	507	38.2
Metabolizability (%)	74.4	72.8	0.25	73.6	72.8	0.61	75.3	72.9	0.39
ME[3] intake									
kJ/d/W	972	961	31.9	1018	1089	42.2	926	834	35.8
kJ/d/$W^{.75}$	1206	1189	38.4	1255	1328	52.6	1157	1049	57.1
Heat production									
kJ/d/W	655	665	5.0	666	672	10.1	644	658	10.3
kJ/d/$W^{.75}$	812	826	3.4	820	821	12.6	805	830	8.1
Energy balance									
kJ/d/W	317	297	29.6	352	417	34.8	282	176	41.5
kJ/d/$W^{.75}$	394	363	36.8	436	507	43.3	352	219	56.9
E efficiency (%)[4]	32.2	27.5	2.44	34.1	38.2	2.01	30.2	16.7	4.14
N intake									
g/d/W	2.35	2.38	0.076	2.51	2.71	0.083	2.19	2.06	0.075
g/d/$W^{.75}$	2.91	2.94	0.091	3.10	3.30	0.103	2.73	2.58	0.119
N excretion									
g/d/W	1.17	1.20	0.028	1.22	1.31	0.025	1.11	1.09	0.021
g/d/$W^{.75}$	1.45	1.49	0.036	1.51	1.61	0.032	1.39	1.37	0.038
N balance									
g/d/W	1.18	1.18	0.057	1.29	1.40	0.069	1.08	0.97	0.054
g/d/$W^{.75}$	1.46	1.45	0.067	1.59	1.70	0.086	1.34	1.21	0.081
N efficiency (%)[5]	50.1	48.6	1.05	51.4	51.3	1.14	48.9	45.8	0.88

[1] *Control birds;*

[2] *0.4 ppm cimaterol treated birds;*

[3] *Metabolizable energy;*

[4] *Energetic efficiency = (Energy balance ×100)/ME intake;*

[5] *Nitrogen retention efficiency = (N balance×100)/N intake.*

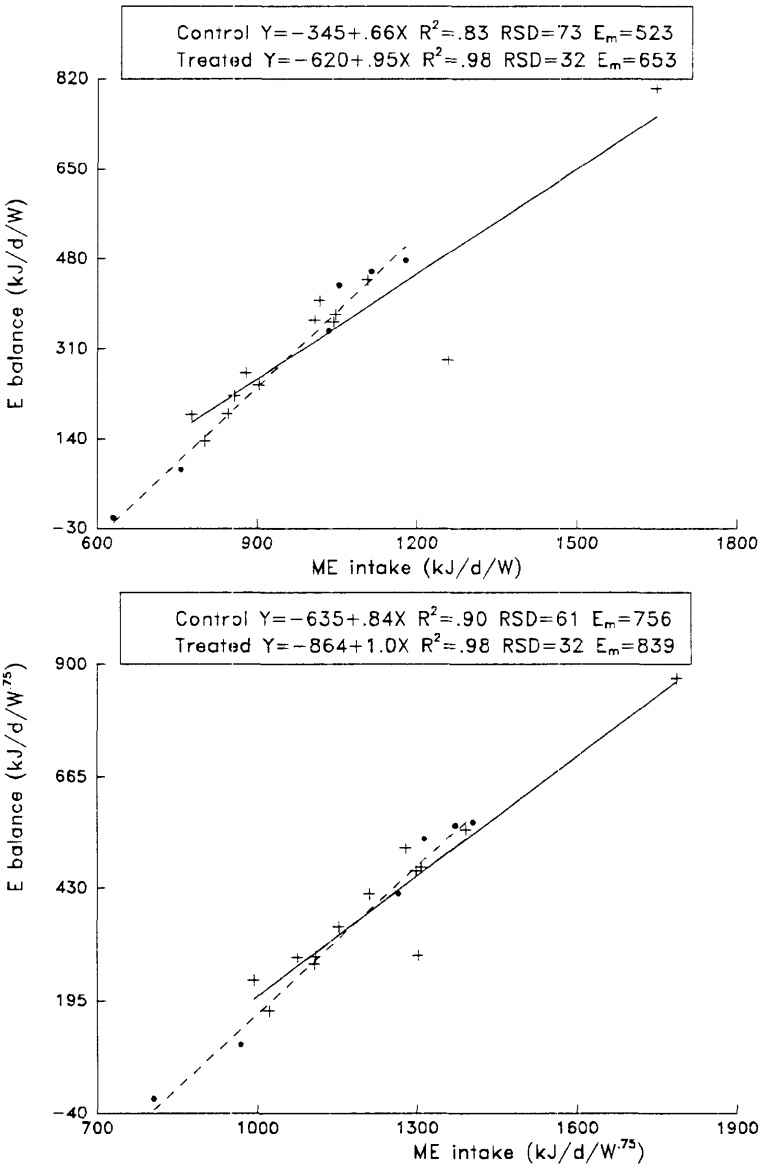

Figure 4.2: Relationship between ME intake and energy balance of controls (+ —) and cirnaterol-treated (... – – –) fat line birds in Experiment 1

Table 4.3: The effects of cimaterol on heat production and energy (E) and nitrogen (N) balance for 3 days of lean line birds aged from 25 to 38 days in calorimetry experiment 1

	Sex combined (N=6)			Male (N=3)			Female (N=3)		
	-[1]	+[2]	SEM	-	+	SEM	-	+	SEM
Body weight (kg)	2.43	2.40	0.029	2.40	2.25	0.064	2.45	2.55	0.038
Weight gain (g)	216	138	13.8	280	195	30.6	152	80	30.8
Feed intake (g)	527	458	14.7	569	460	28.4	485	455	4.1
Metabolizability (%)	74.3	73.8	0.43	74.5	73.0	1.27	74.2	74.5	0.37
ME[3] intake									
kJ/d/W	933	842	19.4	1019	916	61.1	846	768 *	2.6
kJ/d/$W^{.75}$	1154	1029	24.6	1253	1092	76.1	1056	967 *	6.9
Heat production									
kJ/d/W	714	689	5.3	761	735	16.4	668	643	2.6
kJ/d/$W^{.75}$	884	846	6.4	935	879	19.0	834	812 *	0.44
Energy balance									
kJ/d/W	219	153	14.3	258	181	45.3	179	125	5.1
kJ/d/$W^{.75}$	270	184	18.7	318	213	57.7	221	155	7.1
E efficiency (%)[4]	21.5	15.2	1.30	25.2	18.0	4.12	17.8	12.3	0.67
N intake									
g/d/W	2.25	2.05	0.038	2.45	2.24	0.120	2.04	1.86 *	0.013
g/d/$W^{.75}$	2.78	2.51	0.047	3.01	2.68	0.147	2.54	2.34 *	0.006
N excretion									
g/d/W	1.08	1.09	0.017	1.21	1.21	0.02	0.96	0.97	0.030
g/d/$W^{.75}$	1.34	1.34	0.012	1.48	1.46	0.020	1.20	1.22	0.032
N balance									
g/d/W	1.16	0.96	0.031	1.24	1.03	0.097	1.08	0.89 *	0.017
g/d/$W^{.75}$	1.44	1.17	0.041	1.54	1.23	0.127	1.35	1.12	0.026
N efficiency (%)[5]	51.6	45.8	0.87	51.3	45.0	2.53	51.9	46.5	1.06

* *$P<0.05$;*

[1] *Control birds;*

[2] *0.4 ppm cimaterol treated birds;*

[3] *Metabolizable energy;*

[4] *Energetic efficiency = (Energy balance× 100)/ME intake;*

[5] *Nitrogen retention efficiency = (N balance× 100)/N intake.*

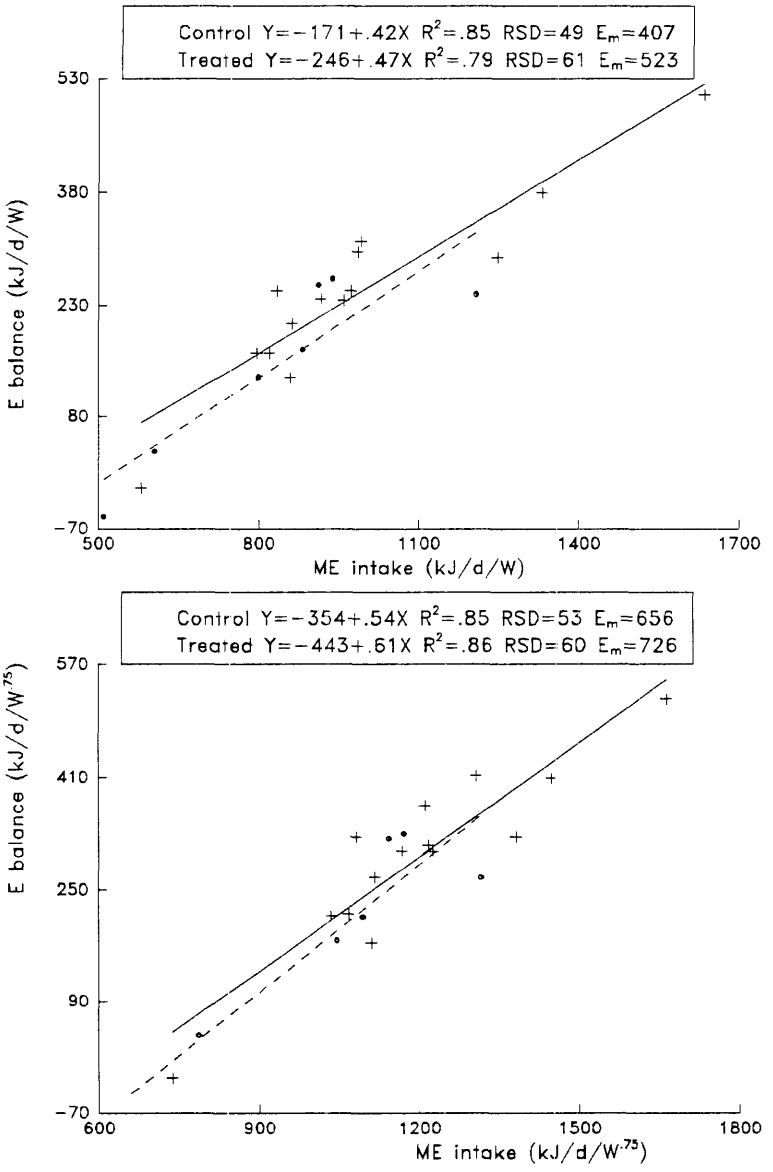

Figure 4.3: Relationship between ME intake and energy balance of control (+ —) and cimaterol-treated (... – – –) lean line birds in Experiment 1

Table 4.4: Comparisons of heat production and energy (E) and nitrogen (N) balance for 3 days of fat and lean line female birds aged from 28 to 42 days in calorimetry experiment 2

N=8	Fat line	Lean line	Statistical significance	SEM
Body weight (kg)	2.03	1.99		0.016
Weight gain (g)	379	380		5.2
Feed intake (g)	883	879		4.8
Metabolizability (%)	71.8	71.9		0.18
ME[1] intake				
kJ/d/W	1094	1124		2.5
kJ/d/$W^{.75}$	1300	1325		3.4
Heat production				
kJ/d/W	747	790	*	4.2
kJ/d/$W^{.75}$	887	934	*	3.3
Energy balance				
kJ/d/W	347	333		4.4
kJ/d/$W^{.75}$	413	391		5.6
E efficiency (%)[2]	31.0	28.8		0.41
N intake				
g/d/W	2.77	2.83		0.012
g/d/$W^{.75}$	3.28	3.34		0.013
N excretion				
g/d/W	1.40	1.43		0.016
g/d/$W^{.75}$	1.66	1.69		0.019
N balance				
g/d/W	1.37	1.40		0.010
g/d/$W^{.75}$	1.62	1.65		0.013
N efficiency (%)[3]	49.0	49.3		0.45

* *P<0.05;*

[1] *Metabolizable energy;*

[2] *Energetic efficiency = (Energy balance×100)/ME intake;*

[3] *Nitrogen retention efficiency = (N balance×100)/N intake.*

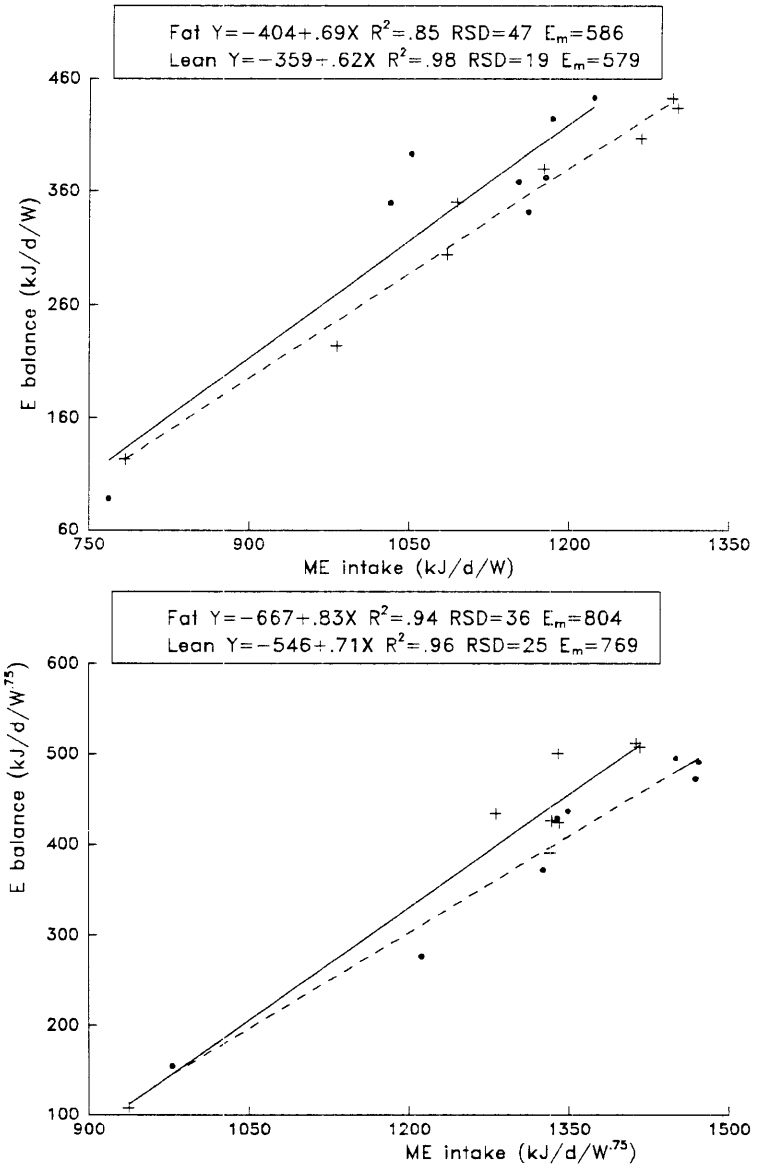

Figure 4.4: Relationship between ME intake and energy balance of fat (-- —) and lean (... – – –) lines of bird in Experiment 2

The regression equations and regression lines for ME intake and energy balance are shown in Figure 4.4. Except for a 5% ($P<0.05$) higher heat production in lean birds on a body weight and a $W^{.75}$ basis, no difference was observed between these two lines in other measurements of energy or nitrogen metabolism.

4.1.3 Experiment 3

The effects of 0.05% theophylline, 50 ppm iodinated casein and 0.5 ppm cimaterol on heat production and energy and nitrogen balance in female Steggles birds were tested in this experiment and the results are shown in Table 4.5. Daily measurements of body weight, feed intake, R.Q. and heat production are presented in Appendix G.

Cimaterol-treated birds had a 3% higher ($P<0.05$) heat production than controls in the treated period on a W but not on a $W^{.75}$ basis. The lower heat production observed in theophylline-treated birds was close to significance on a body weight basis ($0.05<P<0.10$). The result of the analysis of the six days daily heat production is shown in Table 4.6. The only significant change ($P<0.05$) as a result of the transfer from the control to treated diets on day 4 was seen in birds on the theophylline diet where compared with the previous day a 6% drop in heat production and a lower heat production compared with controls on both a W and a $W^{.75}$ basis were observed.

During the control period, the birds to be on the cimaterol diet deposited a significant 37% more energy on both a W and a $W^{.75}$ basis than controls ($P<0.05$), but this difference disappeared in the treated period (Table 4.5). The birds on the iodinated casein diet retained less energy on both a body weight (22%, $P<0.05$) and a $W^{.75}$ basis (18%, $0.05<P<0.10$) than controls in the treated period. The lower energy balance in theophylline-treated birds compared with controls approached significance on both a W and a $W^{.75}$ basis ($0.05<P<0.10$). There was no significant difference in energetic efficiency between the four treatments. The only difference in nitrogen metabolism during the treated period was found in theophylline-treated birds where a lower nitrogen retention efficiency ($P<0.05$) resulted from a lower nitrogen intake (5%, $P<0.05$).

Table 4.5: The effects of theophylline, iodinated casein and cimaterol on heat production and energy (E) and nitrogen (N) balance of female Steggles broilers for 3 days from 21 to 35 days of age in calorimetry experiment 3

	Day 1-3 (all on control diet)				Day 4-6 (on treated diets)				SEM
Treatment	CON[1]	THEO[2]	CAS[3]	CIM[4]	CON	THEO	CAS	CIM	(N=4)
Body weight (kg)	1.98 ^{f}	2.12 ^{c}	2.01 ^{e}	1.90 ^{g}	2.13 ^{c}	2.24 ^{a}	2.18 ^{b}	2.07 ^{d}	0.003
Feed intake (g)	464 ^{d}	478 ^{c}	477 ^{cd}	477 ^{cd}	518 ^{ab}	505 ^{b}	523 ^{a}	525 ^{a}	2.3
Metabolizability (%)	70.7 ^{d}	70.7 ^{d}	70.6 ^{d}	71.5 ^{bcd}	73.2 ^{a}	72.0 ^{abc}	72.4 ^{ab}	71.0 ^{cd}	0.21
Feed ME[5] (kJ/g)	12.1 ^{b}	12.1 ^{b}	12.1 ^{b}	12.4 ^{a}	12.5 ^{a}	12.3 ^{a}	12.4 ^{a}	12.3 ^{a}	0.03
ME intake									
(kJ/d/W)	952 ^{c}	918 ^{c}	959 ^{bc}	1049 ^{a}	1043 ^{a}	953 ^{c}	1009 ^{ab}	1060 ^{a}	8.6
(kJ/d/$W^{.75}$)	1123 ^{b}	1102 ^{b}	1138 ^{b}	1228 ^{a}	1248 ^{a}	1154 ^{b}	1218 ^{a}	1265 ^{a}	9.3
Heat production									
(kJ/d/W)	788 ^{b}	757 ^{de}	820 ^{a}	791 ^{b}	744 ^{e}	702 ^{f}	777 ^{bc}	764 ^{cd}	3.0
(kJ/d/$W^{.75}$)	929 ^{ab}	909 ^{ab}	946 ^{a}	925 ^{ab}	893 ^{bc}	855 ^{c}	928 ^{ab}	914 ^{ab}	7.0
E balance									
(kJ/d/W)	164 ^{c}	161 ^{c}	139 ^{c}	259 ^{ab}	298 ^{a}	250 ^{ab}	232 ^{b}	296 ^{a}	9.2
(kJ/d/$W^{.75}$)	194 ^{b}	193 ^{b}	192 ^{b}	303 ^{a}	355 ^{a}	299 ^{a}	290 ^{a}	352 ^{a}	11.0
E efficiency[6]									
(%)	15.1 ^{b}	17.1 ^{ab}	14.1 ^{b}	24.1 ^{ab}	27.7 ^{a}	25.1 ^{ab}	22.5 ^{ab}	27.3 ^{a}	1.9
N intake									
(g/d/W)	2.36 ^{d}	2.32 ^{d}	2.43 ^{cd}	2.60 ^{ab}	2.51 ^{bc}	2.36 ^{d}	2.50 ^{c}	2.65 ^{a}	0.016
(g/d/$W^{.75}$)	2.79 ^{c}	2.78 ^{c}	2.89 ^{c}	3.04 ^{b}	3.00 ^{b}	2.86 ^{c}	3.01 ^{b}	3.16 ^{a}	0.017
N excretion									
(g/d/W)	1.16 ^{d}	1.16 ^{d}	1.16 ^{d}	1.20 ^{bcd}	1.24 ^{abc}	1.27 ^{ab}	1.19 ^{cd}	1.28 ^{a}	0.011
(g/d/$W^{.75}$)	1.37 ^{c}	1.39 ^{c}	1.38 ^{c}	1.40 ^{c}	1.49 ^{ab}	1.54 ^{a}	1.44 ^{bc}	1.52 ^{ab}	0.014
N balance									
(g/d/W)	1.21 ^{bcd}	1.16 ^{cd}	1.27 ^{abc}	1.40 ^{a}	1.27 ^{abc}	1.09 ^{d}	1.31 ^{cb}	1.37 ^{a}	0.022
(g/d/$W^{.75}$)	1.43 ^{bcd}	1.39 ^{cd}	1.50 ^{abc}	1.64 ^{a}	1.51 ^{abc}	1.32 ^{d}	1.57 ^{cb}	1.64 ^{a}	0.025
N efficiency									
(%)[7]	50.6 ^{ab}	49.7 ^{bc}	52.1 ^{ab}	53.9 ^{a}	50.0 ^{ab}	46.0 ^{c}	52.0 ^{cb}	51.6 ^{ab}	0.61

[1] *Control diet;*
[2] *0.05% theophylline diet;*
[3] *50 ppm iodinated casein diet;*
[4] *0.5 ppm cimaterol diet;*
[5] *Metabolizable energy;*
[6] *Energetic efficiency = (Energy balance×100)/ME intake;*
[7] *Nitrogen retention efficiency = (N balance×100)/N intake.*

Table 4.6: The effect of theophylline, iodinated casein and cimaterol on daily heat production in female Steggles birds aged from 21 to 35 days in calorimetry experiment 3

	Control	Theophylline	Iodinated casein	Cimaterol	SEM
Day[1]	0	0.0%	50 ppm	0.5ppm	(N =4)
kJ/d/W					
1	818 $^{A\ 2\ a3}$	773 $^{A\ a}$	840 $^{A\ a}$	823 $^{A\ a}$	10.8
2	778 $^{AB\ ab}$	754 $^{A\ b}$	814 $^{AB\ a}$	792 $^{AB\ ab}$	7.2
3	767 $^{AB\ ab}$	744 $^{AB\ b}$	805 $^{AB\ a}$	771 $^{B\ ab}$	7.0
4	763 $^{AB\ a}$	699 $^{C\ b}$	798 $^{B\ a}$	785 $^{AB\ a}$	8.4
5	724 $^{A\ ab}$	698 $^{C\ b}$	775 $^{BC\ a}$	756 $^{B\ ab}$	11.2
6	745 $^{A\ a}$	711 $^{BC\ a}$	757 $^{C\ a}$	752 $^{B\ a}$	8.9
SEM	11.8	5.8	7.3	8.3	
kJ/d/W$^{.75}$					
1	957 $^{A\ ab}$	923 $^{A\ b}$	987 $^{A\ a}$	939 $^{A\ ab}$	9.8
2	919 $^{A\ b}$	906 $^{AB\ b}$	965 $^{AB\ a}$	926 $^{A\ ab}$	6.2
3	912 $^{A\ b}$	897 $^{AB\ b}$	961 $^{AB\ a}$	910 $^{A\ b}$	7.2
4	912 $^{A\ b}$	847 $^{C\ c}$	959 $^{AB\ a}$	932 $^{A\ ab}$	6.9
5	868 $^{A\ b}$	849 $^{C\ b}$	937 $^{AB\ a}$	904 $^{A\ ab}$	11.3
6	898 $^{A\ a}$	868 $^{BC\ a}$	921 $^{B\ a}$	905 $^{A\ a}$	8.8
SEM	14.9	7.7	9.1	10.0	

[1] *Day 1-3 on control diet, day 4-6 on treated diet;*

[2] *Values with the same upper case superscript within a column are not significantly different (P>0.05) between the six days in one treatment*

[3] *Values with the same lower case superscript within a row are not significantly different (P>0.05) between the four treatments in one day.*

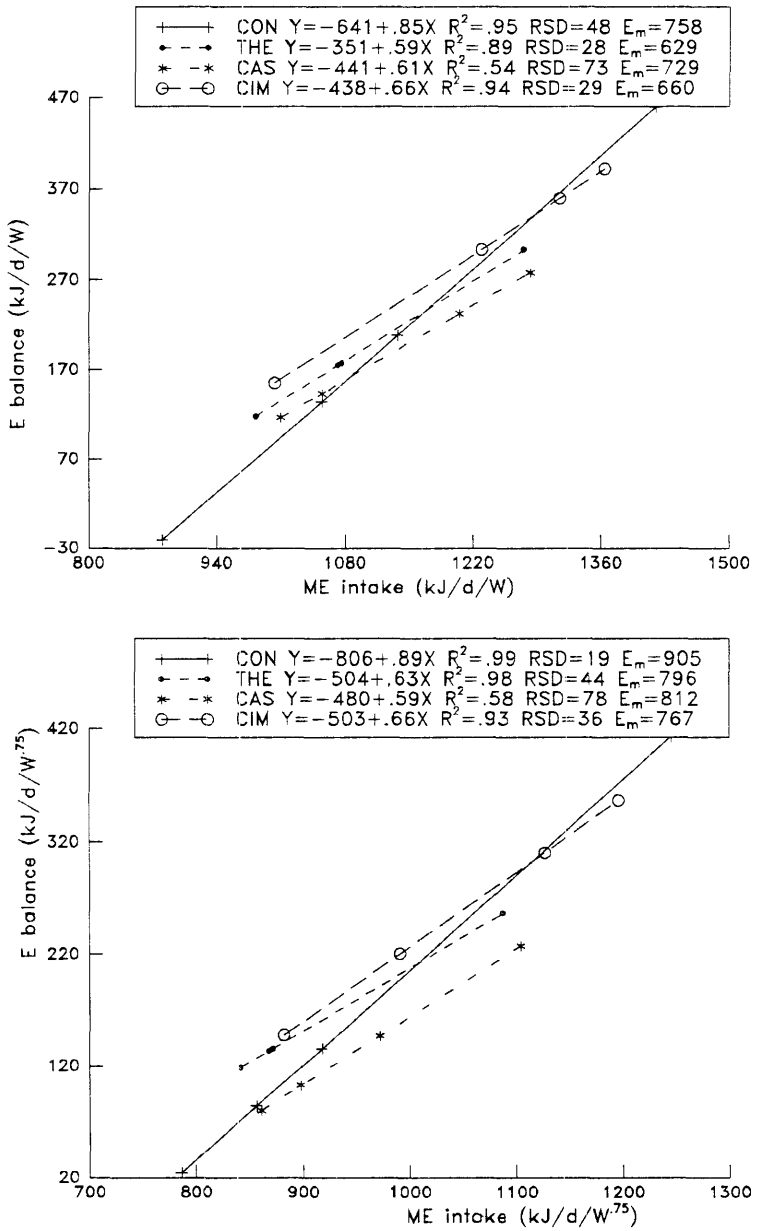

Figure 4.5: Relationship between ME intake and energy balance during the control period in Experiment 3 (CON: control diet, THE: 0.05% theophylline diet, CAS: 50 ppm iodinated casein diet, CIM: 0.5 ppm cimaterol diet).

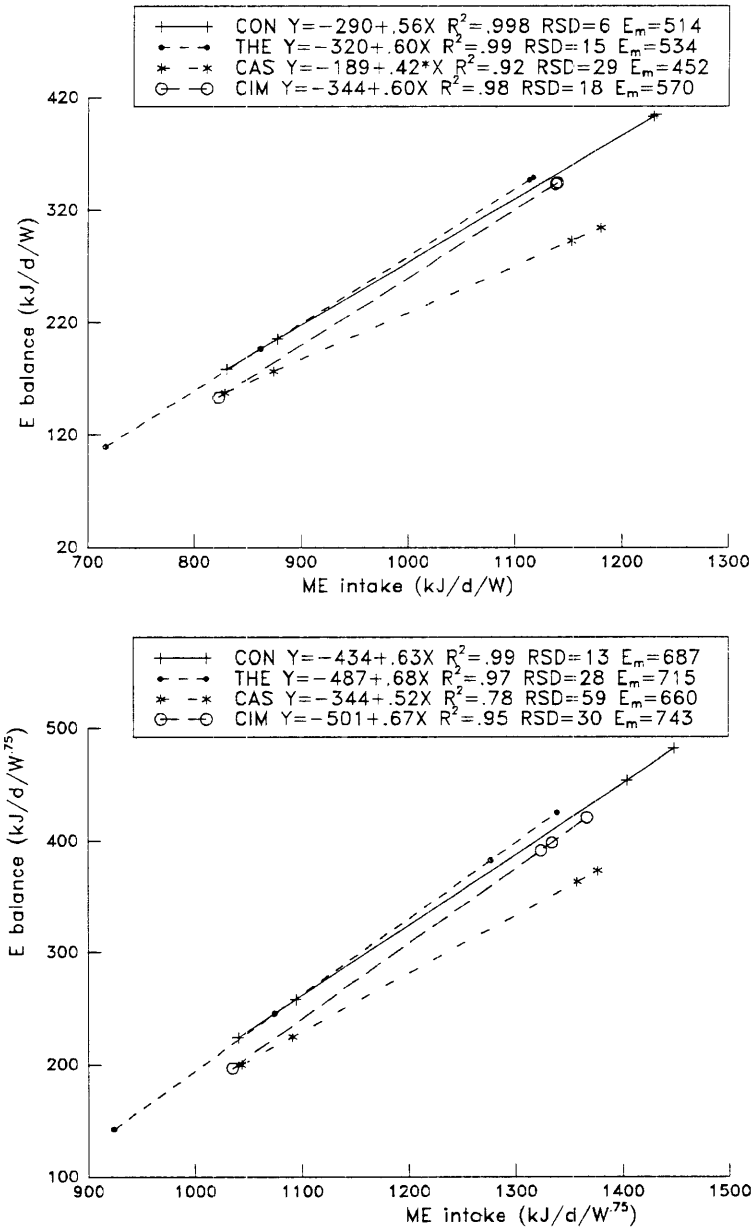

Figure 4.6: Relationship between ME intake and energy balance during the treatment period in Experiment 3 (CON: control diet, THE: 0.05% theophylline diet, CAS: 50 ppm iodinated casein diet, CIM: 0.5 ppm cimaterol diet; * $P<0.05$ compared with controls).

The regression equations and the regression lines for ME intake and energy balance during the control and the treated periods are shown in Figure 4.5 and 4.6 respectively. There was no significant difference in the relationship between energy retention and ME intake between the four treatments in the control period, but in the treated period the birds on the iodinated casein diet deposited less energy per ME intake than controls on a body weight basis ($P<0.05$).

4.2 Discussion

The similar metabolizability of dietary energy observed between the genetically fat and lean lines of birds in both Experiments 1 (Table 4.1) and 2 (Table 4.4) was in agreement with the results from other studied with different lines of birds selected for leanness and fatness (Pym and Farrell, 1977; LeClercq and Saadoun, 1982b; Sorensen *et al.*, 1983). Maintenance metabolizable energy requirement (E_m) calculated from linear regression between energy retention and metabolizable energy intake was significantly higher in fat line birds in Experiment 1 (Figure 4.1) which agrees with the results of Pym *et al.* (1984) but not with those of Experiment 2 (Figure 4.4) and of Geraert *et al.* (1987) and MacLeod *et al.* (1988) where similar E_m was found.

As expected, significantly higher heat production was found in lean line birds in both Experiments 1 and 2 on both a body weight and a $W^{.75}$ basis. This is in agreement with the results from rats (Pullar and Webster, 1974) that lean animals have a higher metabolic rate, but not with those from other genetically fat and lean lines of birds as summarized by MacLeod and Geraert (1987) where no difference in either fasting or fed heat production was found. The hypothesis, put forward in an attempt to explain the divergence in body composition, that a difference in energy expenditure would result in differing energy retentions was excluded by MacLeod and Geraert (1987). It would seem, however, that the higher heat production in lean birds is a consequence, rather than a cause, of the higher proportion of lean mass which is metabolically more active than fat tissue and the high energy cost of lean deposition is associated largely with amino acid turnover.

The observation that there was no difference in nitrogen retention efficiency between the genetically fat and lean lines of birds in Experiments 1 and 2 was consistent with the findings of Sorensen *et al.* (1983) and Pym *et al.* (1984) who reported no significant changes in protein retention efficiency of their lines except for a lower ratio of nitrogen retained to nitrogen intake in the line selected for increased growth rate. This was, however, inconsistent with the results of LeClercq (1983) and Whitehead and Griffin (1986) who observed an increased gross efficiency of protein retention by selection for leanness. This increased nitrogen retention efficiency has been attributed to the greater nitrogen losses of the fat lines as measured by an increase in uric acid excretion (Geraert *et al.*, 1987) and in the rate of N^T-methylhistidine excretion (Tomas *et al.*, 1988). It was concluded then (MacLeod and Geraert, 1987; MacLeod *et al.*, 1988) that the most important difference between the existing fat and lean lines of fowl in energy metabolism terms is in the partition of a given amount of retained energy between fat and protein deposition. Birds directly or indirectly selected for high abdominal fat content, or criteria other than feed intake, exhibit a higher rate of protein catabolism than their lean counterparts. The amino acid carbon chain is therefore being diverted from incorporation into protein to the pool of substrates for energy metabolism.

There have been no previous studies undertaken to investigate the effects of beta-adrenergic agonists on heat production and energy and nitrogen balance in chickens. There can be dangers in comparing the results observed in the chicken with those from laboratory rats or from ruminants; this is clear from different responses to beta-agonists due to species difference already demonstrated in the growth trials. With this caution in mind, however, it is still worthwhile to examine what has been obtained in other species when discussing the results of this study with birds.

The significant increase in heat production on a body weight basis on the inclusion of cimaterol and the reversal of energy balance in cimaterol-treated birds from significantly superior to controls in the control period to no difference from controls in the treated period in Experiment 3 (Table 4.5), and the the trend of reduced energetic efficiency seen in genetically lean birds in Experiment 1 (Table 4.3) were in agreement with the results

obtained with rats (Emery *et al.*, 1984; Stock and Rothwell, 1986; Rothwell and Stock, 1987a; Sainz and Wolff, 1988), calves (Williams *et al.*, 1987) and lambs (MacRae *et al.*, 1986; 1988). But this effect of cimaterol on heat production was not seen in the genetically fat and lean lines of birds in Experiment 1 (Table 4.2 and 4.3). A study to investigate the influence of clenbuterol on energy balance, thermogenesis and body composition in lean and genetically obese Zuker rats showed that the main difference in response to clenbuterol between these two strains of rats was in energy expenditure and energy metabolism and not in protein metabolism (Rothwell and Stock, 1987a). In clenbuterol-treated rats a significant increase in weight gain in lean rats was found mainly due to the elevated deposition of protein with water while in obese rats both the improvement in protein and water content and the decrease in fat content, increase in energy expenditure and depression in energetic efficiency resulted without changes in body weight gain. This different response of lean and obese rats to clenbuterol was attributed to the stimulated thermogenesis and brown adipose tissue activity in the genetically obese animals which may lead to reduced fat content. In keeping with these findings of Rothwell and Stock (1987a), Reeds *et al.* (1988) also reported the increased interscapular brown fat mass in clenbuterol-treated rats.

The differences in energy metabolism and the possible mechanisms causing these differences between genetically fat and lean birds and fat and lean mammals (rats) have recently been reviewed by MacLeod and Geraert (1987). Brown adipose tissue, with its numerous mitochondria and a very rich blood supply, has been shown to be the common effector of non-shivering, cold-induced and diet-induced thermogenesis in most mammals (Rothwell and Stock, 1986; Néchad, 1986). It is characterized by a high rate of substrate oxidation and consequently, with a large capacity to produce and distribute heat and thus specially capable for thermoregulation by non-shivering thermogenesis (Glick *et al.*, 1981). The occurrence of functional brown adipose tissue is not certain in birds. Johnston (1971) could not find any histological evidence for brown adipose tissue in eight species of birds known to be relatively unstable thermoregulators. Oliphant (1983) described brown-fat-like tissues in two North American species. Although these attempts to find brown adipose tissue in birds have led to contradictory reports and the question remains open, the characteristics of

the avian thermoregulation appear to differ from those of the mammal (Barre *et al.*, 1985) and birds are generally regarded as devoid of brown adipose tissue. It has been shown that if non-shivering thermogenesis does occur in birds, catecholamines are not involved (Hissa and Palokargao, 1970). Thus, if cimaterol or other beta-adrenergic agonists do have effects in birds, particularly in genetically fat birds, on increasing thermogenesis, which can be reflected in the changes in heat production, it would appear that such effects would not be as marked as those seen in mammals due to their different mechanisms of thermoregulation.

The studies conducted with theophylline have been mainly related to its effects on cAMP and its synergistic effect with catecholamines on elevating the release of non-esterified fatty acids and glycerol (see 3.2). It appears that no experiments have so far been carried out to investigate the effects of theophylline on heat production and energy and nitrogen metabolism in the chicken.

The immediate drop in heat production from the previous day in theophylline-treated birds and their lower heat production than controls only on the first day of the inclusion of theophylline in the diet (Table 4.6) could be attributed to their reduced feed intake and significantly lower ME intake during the 3-day treated period resulting from its anoretic effect (Table 4.5). The lower nitrogen retention efficiency in the theophylline-treated birds resulted from the lower ME intake and lower nitrogen intake than controls. A study with seven drugs, including theophylline, known to stimulate the sympathetic nervous system in different strains of rats (Dullo and Miller, 1984) led to variable results with theophylline shown to be effective in increasing thermogenesis in some strains but not in others for which no explanation was given. It was suggested (Dullo and Miller, 1984) then that drugs that are capable of increasing noradrenalin levels at the sympathetic neuro-effector junctions or of stimulating noradrenalin action on cell membrane beta-adrenoreceptors are effective in causing marked increase in thermogenesis while on the other hand, the inhibition of phosphodiesterase by theophylline is unlikely to cause drastic changes in cAMP levels in the absence of sufficient noradrenalin. No such studies have been conducted in chickens. The results observed in the present study demonstrated only the anoretic effect of theophylline but not its thermogenic effect as shown in some of the studies with rats (Dullo and Miller,

1984). The absence of brown adipose tissue and the different thermoregulation mechanisms in birds may explain this species difference in response to theophylline as discussed previously. The results from a recent study with obese rats indicated little effect of theophylline or caffeine alone on energy balance (Dullo and Miller, 1986). This was confirmed in Experiment 3 (Table 4.5) where no difference in energy metabolism between controls and theophylline-treated birds was found.

The changes in body composition, i.e., the decreased fat content, by the treatment of iodinated casein (Table 3.11 and 3.12) probably indicated that thyroid hormone levels had changed and this was reflected in the changed thyroid weight in iodinated casein-treated birds (Irwin *et al.*, 1943; Turner *et al.*, 1944; Roberson and Trujillo, 1975; Wilson *et al.*, 1983). Oxygen consumption, or metabolic rate, is closely correlated with hormone levels and the action of thyroprotein in elevating the metabolic rate seems to be well established (Singh and Shaffner, 1950). In Experiment 3 (Table 4.5), no difference was observed in heat production between controls and iodinated casein treated birds. This, however, would not be unexpected since the level of iodinated casein used in this experiment (50 ppm) was relatively low and would not be expected to cause much interruption of the normal growth. It seems that critical levels need to be reached before any effects are observed. Singh and Shaffner (1950) found that the metabolic response of chickens to graded doses of thyroprotein appeared to be proportional to dosage at levels only greater than 110 ppm. This level of thyroprotein was proposed to be about equivalent to the natural secretion rate and was very close to 90 ppm determined by Schultze and Turner (1945). Both of the reported levels are obviously higher than the 50 ppm used in Experiment 3 in the present study. The coefficient of correlation between oxygen consumption and thyroid hormone concentration in the plasma in different age groups of White Rock chickens was reported to range between 0.78 and 0.98 (Bobek *et al.*, 1977). Hyperthyroidism is an extreme condition in which excess thyroid hormones that can produce pronounced alteration in thyroid function giving an altered metabolic rate. This dose-response relationship in iodinated casein-treated birds was confirmed in growth trials where no sign of any depression in growth rate was seen at the two levels tested (see 3.2) and higher levels used in other

studies caused reduced growth rate (Wilson *et al.*, 1983). It has been found that in contrast to the mammal, in which prolonged metabolic stimulation follows thyroxine injection, the chicken exhibits only a small and a transitory rise in metabolic rate following administration of thyroid hormones which lasts only for 2–3 hours and the maximum effect occurred only at 2-hour after the administration (Singh *et al.*, 1968). The lower energy balance ($P<0.05$ on a body weight basis) in iodinated casein-treated birds during the 3-day treated period observed in Experiment 3 (Table 4.5; Figure 4.6) may account for the significant reduction in fat content in iodinated casein-treated birds as observed in growth trials (Table 3.11 and 3.12).

Chapter 5

GENERAL DISCUSSION AND CONCLUSIONS

Growth in poultry and other species is controlled by a multiplicity of hormones. It should be kept in mind that the final expression of growth is the result of interactions between nutritional, environmental, and genetic factors with the endocrine secretions. These interactions can be manipulated by management practices to maximize growth rate and feed efficiency and to optimize carcass composition. There is no doubt that there is considerable potential for further stimulating growth and improving leanness in the animal body. This area has been, and is likely to be, a focus of interest in the coming years.

Since animal growth is mediated to a large extent through the action of hormones, both directly or indirectly, many current growth promoting strategies involve some form of manipulation of hormonal status. The meaning of growth promoter can change with time and now a successful growth promoter is one which reduces the cost of meat without diminishing the confidence of the general public in meat as a wholesome food (Buttery and Dawson, 1988).

The use of closed-circuit respiration chambers developed by Farrell (1972) and Farrell (1974b) allows the metabolic changes following the administration of a growth promoter and genetic selection for leaner body mass in the chickens to be tested. The effectiveness of

genetic selection for leanness is demonstrated again in the present study as observed from the marked differences in the fat content between the fat and lean lines of birds (Table 3.5). With the simple and non-destructive techniques of measuring fatness in live birds such as the abdominal fat pad caliper (Pym and Thompson, 1980) and the measurement of plasma VLDL (Whitehead and Griffin, 1982), further improvement in carcass quality in terms of an increased proportion of lean body mass would be expected. A higher proportion of metabolically active lean body mass in the genetically lean birds resulted in a higher heat production. Although no difference was observed in nitrogen metabolism with the two lines selected in the present study for increased or decreased abdominal fat pad, some other studies with different genetically lean lines have shown an improved protein retention efficiency (see 4.2). Further experiments with the lines used in the present study to investigate in some depth amino acid metabolism may help to reveal differences in nitrogen metabolism. The correlated improvement in feed conversion with the decreased fat content, or the increased lean proportion, obtained under different feeding situations with birds of different genetic potential (see 3.1.1) was not observed here (Table 3.5). This disagreement suggests more growth trials and to pair-feed these two lines of birds so as to confirm the correlated response of feed conversion with the selection for body fat content.

Of the four compounds tested, theophylline and caffeine are for the first time explored in farm animals and were shown to markedly reduce fat content over that caused by the depression in feed intake. Both theophylline and caffeine are naturally-occurring alkoloids in tea or coffee. Their potential as a feed additive to produce leaner but smaller animals is worthy of further testing. Caffeine showed an advantage over theophylline in terms of feed conversion since at both 0.1% and 0.05% inclusion rate, caffeine-treated birds utilized their feed as efficiently as did controls while theophylline at 0.1% led to a poorer feed conversion ratio. There clearly exists a dose-effect relationship in the birds treated with theophylline or caffeine with 0.1% level showing a more dramatic effect than the 0.05% level. Considering the concomitant depression in growth rate with the marked reduction in both abdominal fat pad and carcass fat content at the 0.1% inclusion, it is worthwhile testing lower levels of theophylline or caffeine. By doing so, the depression in growth rate caused by the reduced

feed intake which due probably to their unpalatability would be expected to be alleviated but the effect on fat content may be also smaller. It should be kept in mind then that when smaller effects than those observed here are to be detected, the number of animals and replicates should be increased accordingly .

The β-adrenergic agonists, clenbuterol and cimaterol, have been of interest since 1983 to animal scientists who want to improve the efficiency and quality of animal products when their effects on improving animal growth and protein deposition while decreasing body fat content were first reported (Ricks *et al.*, 1983). Their effects on the performance and carcass composition were then tested in all the major farm animals as well as in laboratory rodents (see Section 1.4). Cimaterol was shown to be effective in decreasing fat content in both genetically fat (Table 3.2) and lean (Table 3.1) lines of birds and to have the potential to improve the lean body mass (Table 3.2). Although no effect of cimaterol on the growth performance and fat content was seen in commercial birds in Trials 3 and 4, this should not be taken as conclusive evidence that cimaterol has no effects on Australian broiler chickens. Braude (1976) once stressed that one should not be allowed to challenge established normal distribution of response of many biological variables to a growth-promoting compound without a very substantial replication because such a response is often affected by many interacting and sometimes antagonistic factors. The results obtained by Dalrymple (1984b) and Dalrymple and Ingle (1987) may lend support to this point since 8–10 replicates with a large number of birds were used in their studies so that relatively small but significant effects could be detected. Thus, further studies with more replicates than the three used in Trials 3 and 4 would help to clarify or to confirm the effects of cimaterol in chickens. The disagreement between the results of the present study and those of Scholtyssek (1987) and of Dalrymple and Ingle, (1987) in the response of the abdominal fat pad to cimaterol may only be explained by speculating that animals respond differently to the same dose of the same substance under different feeding situations. In addition, there may be an effect of strain of birds. Further extensive trials are therefore required under different husbandry conditions to substantiate the observed effects of cimaterol in the chicken. The effects of cimaterol on chickens fed on diets of different levels of protein are also worth testing. A

study with rats on a normal and a low protein diet has indicated that clenbuterol may help to conserve body protein at the expense of fat in protein deficient animals resulting in a smaller but leaner body mass (Rothwell and Stock, 1986).

It seems that there exists species difference in the response to growth-promoting compounds. This has been clearly demonstrated in experiments with ruminants and those with swine or poultry where the effects of β-agonists were studied (see Section 1.4.1.2). The magnitude of response of the farm animals to these agonists was biggest in ruminants, smaller in swine and least in poultry. Rudman (1963) reviewed studies on the adipokinetic action of polypeptide and amine hormones upon the adipose tissue of various animal species and summarised that pigs were unresponsive to either pituitary polypeptide or to catecholamines. Mersmann (1984) reported that the swine adipose tissue adrenoceptors were not readily classified as either β_1- or β_2-specific and most β_1- and β_2-adrenergic agonists were inactive although they were active with rat adipose tissue. It would be expected that there would be differences in response to lipolytic agents between birds and mammals since it has been noticed for some time that the regulation of lipolysis in chicken adipose tissue is markedly different from that in mammalian species (see Section 1.2.4). There is considerable species variation with respect to both the minimum dose and the actual hormones which elicit a lipolytic response from adipose tissue. The absence of functional brown adipose tissue and different thermoregulation mechanisms in birds seems to contribute to the lack of thermogenic effects of the β-adrenergic agonist, cimaterol, in Experiment 1 (Table 4.2 and 4.3) and of theophylline in Experiment 3 (Table 4.5) while adrenergic activation of non-shivering thermogenesis can be easily demonstrated in rats and other mammals possessing brown adipose tissue (Dullo and Miller, 1984; Stock and Rothwell, 1986).

Iodinated casein showed very consistent results in reducing body fat (Table 3.11 and 3.12) and in improving feed conversion efficiency (Table 3.11) at the two levels (50 and 100 ppm) tested. At the 50 ppm inclusion rate in respiration calorimetry Experiment 3 (Table 4.5), iodinated casein exhibited no influence on metabolic rate as measured by heat production. These positive effects make a potential for its use in the chicken meat industry although further trials on larger and commercial scales are required. Some lower

concentrations up to 50 ppm are worth testing to determine the lowest level with the same favourable effects.

There is a need to repeat the treatment of iodinated casein plus caffeine (Table 3.12) since this combination is believed to have a similar synergistic lipolytic effect as observed in birds treated with iodinated casein plus theophylline (Table 3.11). Iodinated casein plus theophylline reduced the size of the abdominal fat pad by over 50%. Although no carcass fat analysis was done, a reduction in whole body fat content would be expected with such a striking decrease in abdominal fat. Compared with the results from birds treated with theophylline or iodinated casein alone, a combination of the two gave a further reduction in fat content but at the cost of a decrease in growth rate. It would appear that whether such a further decrease in fat content with a depression in growth rate is more beneficial than a lesser reduction in fat content without reduced growth rate as seen in birds treated with iodinated casein alone, is related more to economic considerations.

As for the increasing public concern for the quality of animal products, Buttery and Dawson (1988) argued that the fear of hormones and the consequent ban on use of natural sex steroids within the European Economic Community were absolutely unfounded and irrational. They showed evidence that the hormone levels in animals treated with sex steroids are often less than can be found in untreated animals of a different sexual status. Any growth promoter even to be considered for use in meat industry will need long and extensive testing for its safety and the efficacy. It must be economically efficient as well as having public acceptance. All this testing has to be done in a climate when the application of new technology and new methods to improve the animal growth and carcass quality is viewed with increasing suspicion by the public.

In the present study, the results obtained from the growth trials demonstrated that feeding broiler chickens with diets mixed with compound(s) known to be lipolytic may be a means of combating the current excess fat problem in the chicken meat industry. The observations from the respiration calorimetry experiments suggested that these compounds could be included in the diet at low but still effective levels without causing significant interruption of normal physiological functions within the chicken. Cimaterol is effective in

reducing fat content in the chicken and shows the potential to improve lean body mass, but its effects in commercial broiler birds needs further trials. Both theophylline and caffeine have an effect *per se* on decreasing fat content over that caused by the depression in feed intake the dose-effect relationship in the birds treated with either of these alkoloids makes it worthwhile to test lower levels to overcome the depression in growth. Iodinated casein can reduce fat content while maintaining growth and improving feed conversion efficiency at the two levels tested. Iodinated casein plus theophylline has a synergistic effect on further reducing fat content and such a combination requires economic considerations.

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