

STUDY ON APPETITE REGULATION AND NUTRITIONAL PHYSIOLOGY IN CHICKENS
SELECTED FOR FEED EFFICIENCY OR ITS COMPONENTS

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SUMMARY

Effects of variation in dietary nutrient density upon growth, food intake, body composition and digestive physiology and anatomy were studied in two experiments in lines of chickens selected for 12 generations for either increased liveweight gain (line **W**), increased food consumption (line **F**), improved food utilisation efficiency (line **E**), or at random (line **C**).

Unlike the other lines, the F line in both experiments ate less of the low than the high nutrient density diet indicating that these birds were eating to gastrointestinal capacity. However, these birds had the shortest food retention time, the smallest gizzard and proventriculus and the shortest intestine of all the lines. The E line had the longest food retention time and the heaviest proventriculus, gizzard and intestine but only about a quarter as much abdominal fat as the F line.

These results shed further light on physiological parameters underlying observed differences between these lines in production performance and reinforce the argument for direct selection for food conversion efficiency in broilers.

INTRODUCTION

The increasing cost of feed and the fact that feed accounts for some 70 percent of total costs in broiler production, has led commercial breeders to reassess selection for growth rate alone and to consider the merits of direct selection for feed conversion efficiency.

However, it has been recognised (Pym, 1985), that there is a lack of information on the relative response of birds selected for growth or feed efficiency to variation in dietary nutrient composition. The definition of factors that control voluntary feed intake in these differently selected lines of birds is of importance in determining appropriate feeding strategies for similarly selected commercial lines of broilers.

We have therefore investigated the relative response to diets varying in nutrient density of four lines of chickens selected either for increased 5 to 9 week weight gain (line **W**), increase 5 to 9 week food consumption (line **F**), decreased 5 to 9 feed conversion ratio (line **E**), or at random control (line **C**), (Pym and Nicholls, 1979).

MATERIALS AND METHODS

Management of Birds

Fertile eggs obtained from the four lines after three generations of relaxation following selection for twelve generations (Pym, 1985), were

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incubated and hatched at the University of Queensland Veterinary School Farm for two experiments in May and September 1985. The chicks were reared in electrically heated brooder cages to four weeks of age and fed a commercial starter diet in crumble form (12.5 MJ ME/kg, 230g protein/kg). At four (experiment 2) or five (experiment 1) weeks of age, the chicks were randomly allocated to individual cages provided with individual feed troughs and automatic drinkers (Pym and Nicholls, 1979).

Experiment 1

This experiment commenced at 5 weeks of age and terminated at 9 weeks of age. The two dietary treatments shown in Table 1 were formulated to vary in nutrient density but to contain all other nutrients in the same **proportion** to ME. The low density diet was obtained by diluting the high density diet (diet 1), which contained 230g protein and 13.0 MJ ME/kg, with **350g** finely ground rice hulls per kg. The other nutrients in diet 1 were formulated close to the values suggested by NRC (1977). The experiment was designed such that there were 2 diets x 4 lines x 2 sexes x 4 birds per sub-cell.

Individual weights and food intakes were recorded. A subsample of excreta was collected once a week from each bird and stored frozen. The four excreta collections from each bird were pooled and dried in a force draft oven at **60-70°** for about 48 hours. Feed and excreta were analysed for gross energy, nitrogen and acid insoluble ash, for determination of ME and nitrogen retention.

Analysis of variance was carried out using the balanced factorial (BALF) computer programme of **Beattie** (1982). Treatment means were compared using the least significant difference (LSD) test.

Experiment 2

A second experiment identical in design to experiment 1 was undertaken to study effects upon body composition and digestive organ size. The experiment commenced at 4 weeks of age.

At 8 weeks of age, food retention time was measured in the birds using ferric oxide (**Fe₂O₃**) as an indigestible indicator (**Tuckey et al**, 1958; **Golian** and **Polin**, 1984). Food retention time was **determined as** the duration from the time of placing a gelatine capsule of **Fe₂O₃** (200 mg per kg body weight) in the oesophagus to **the time** of the first appearance of a distinct red colour in the excreta.

At 9 weeks of age, individual weights and food **consumptions** were recorded. Birds were then starved overnight and on the following day were killed by neck dislocation. The digestive organs were dissected and removed from the body, cleaned from mesenteric fat and the remaining **digesta** were removed mechanically before certain components were weighed and measured. After the measurements were recorded, the organs were replaced and the bodies were stored frozen at **-20°C**. Body chemical analysis as described by Pym and Solvyns (1979) was subsequently carried out .

Table 1. Composition of diets (g/kg) used in experiments 1 and 2

Ingredient	Diet	
	H	L
Wheat	308	200.2
Sorghum	306	198.9
Soybean meal	230	149.5
Meat meal	80	52
Fish meal	30	19.5
Vegetable oil	30	19.5
Rice hull	-	350
Salt	2.5	1.62
Vitamin premix ^a	1.5	0.97
Mineral premix ^b	1.5	0.97
Methionine	2	1.3
Diatomaceous earth	7.5	4.9
Coccidiostat	1	0.64
Total	1000	1000
<u>Calculated:</u>		
ME, MJ/kg	13.0	8.5
Protein, g/kg	230	150
Ca, g/kg	9.1	5.9
P, g/kg	7.6	4.9
Lysine, g/kg	9.2	6.0
Methionine, g/kg	4.6	3.0
Protein ME ratio	17.9	17.6

^a Each gram of vitamin premix contained: Vit. A 8000 i.u., Vit. D3 2000 i.u., Vit. B2 6.5 mg; F, 0.1 mg, Vit. B6, Vit. B12; E, K3, Biotin, folic acid, Ca phantotenate, niacin and antioxidant.

^b Each gram of mineral premix contained: Co, 0.4 mg; I, 0.6 mg; Mo, 0.6 mg; Fe²⁺, 30 mg; Mn, 60 gm; Zn, 50 mg; Cu, 4 mg; Se, 0.05 mg; and F, 0.1 mg.

RESULTS

Effects of dietary nutrient density upon performance of the four lines are shown in Table 2. Results in the performance traits were similar in both experiments, although the liveweight gain and feed intake values were higher in experiment 2 which was due to the longer test period in that experiment. Liveweight gain was, as expected, highest in line W and lowest in line C. Food intake was highest in line F and lowest in lines C and E and feed efficiency was, as expected, highest in line E and lowest in line F. Birds given the high density diet performed better than those on the low density diet whilst males performed better than females.

Table 2. Performance of chickens from the four lines given diets varying in nutrient density

	Liveweight gain (g/bird)		Food intake (g/bird)		Food conversion ratio (feed/gain)	
	Exp.1	Exp.2	Exp.1	Exp.2	Exp.1	Exp.2
Line (L)						
W ⁺	807 ^{a++}	1160 ^a	2793 ^b	3591 ^a	3.67 ^b	3.30 ^a
F	730 ^b	997 ^b	3053 ^a	3643 ^a	4.45 ^a	3.95 ^b
E	633 ^c	861 ^c	2028 ^c	2730 ^b	3.38 ^b	3.44 ^a
C	520 ^d	759 ^d	1955 ^c	2803 ^b	3.91 ^a	3.91 ^b
LSD (5%)	69	59	183	250	0.30	0.31
Diet (D)						
H ⁺⁺⁺	803 ^a	1152 ^a	2337 ^b	3048 ^b	2.92 ^b	2.71 ^b
L	541 ^b	737 ^b	2578 ^a	3335 ^a	4.79 ^a	4.59 ^a
LSD (5%)	49	42	129	176	0.21	0.22
Sex (S)						
Male	738 ^a	1057 ^a	2656 ^a	3356 ^a	3.38 ^b	3.47 ^b
Female	606 ^b	831 ^b	2258 ^b	3027 ^b	3.87 ^a	3.83 ^a
LSD (5%)	49	42	129	176	0.21	0.22
Interaction						
L x D	**	**	**	*	NS	NS

⁺ W, line selected for increased 5-9 week high liveweight gain.

F, line selected for increased 5-9 week high feed consumption.

E, line selected for decreased 5-9 week feed conversion ratio.

C, random control line.

⁺⁺ Values with the same superscript are not significantly ($P < 0.05$) different. *, $P < 0.05$; ** $P < 0.01$; NS, Not significant ($P > 0.05$).

⁺⁺⁺ H diet contained 230g protein with 13.0 MJ ME/kg.

L diet contained 150g protein with 8.5 MJ ME/kg.

There were significant ($P < 0.05$) interactions between line and diet for liveweight gain and food intake in both experiments as shown in Figure 1.

There was 3 change in ranking of the lines in liveweight gain from the high to the low nutrient density diet. On the high diet, gain was greatest in the W line followed by the F, E then C lines. On the low diet in experiment 1, the ranking of the E and F lines was reversed and in experiment 2, gains in the E and C lines were similar. Lines E and C showed substantial increases in food intake on the low nutrient density diets in both experiments, whereas the F line showed a marked decrease in intake on the low diet in experiment 1 and a marginal decrease in intake in experiment 2. Food intake in the W line increased on the low density diet in experiment 1, but showed little change in experiment 2.

Experiment 1

Experiment 2

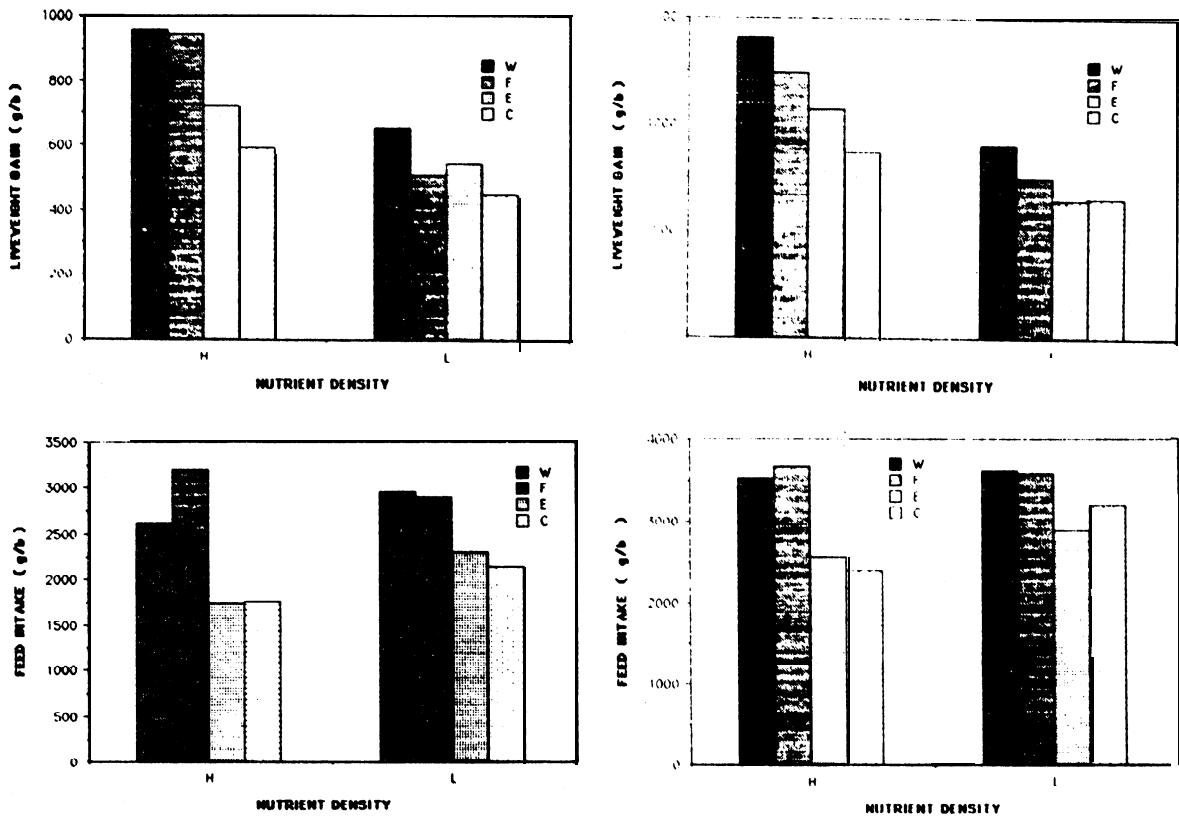


Figure 1. Histograms showing the effect of line and diet upon liveweight gain and food intake in the two experiments

The effects of nutrient density upon metabolisable energy (ME), nitrogen retention (NR) and body composition in the four lines are shown in Table 3. The ME value was highest in the E line and lowest in the F line. Nitrogen retention also followed the same pattern being greatest in the E line and least in the F line. Birds on the high density diet retained more nitrogen than those on the low diet. Body fat was, as expected, highest in the F line and lowest in the E line ($P < 0.05$). Birds on the high density diet were fatter ($P < 0.05$) than those on the low density diet and females were also fatter than males.

There was a significant interaction between line and diet for body fat, caused by a differential reduction in fatness in the four lines given the low density diet. The relative decrease in fatness from the high to the low density diet in the W, F, E and C lines was 6, 10, 3 and 3% respectively.

Table 3. Metabolisable energy (ME), nitrogen retention (NR) and body composition in the four lines of chickens given diets varying in nutrient density in the two experiments

	ME	NR	Body fat		Body protein	
	(MJ/kg) Exp.1	(g/kg) Exp.1	Exp.1#	Exp.2	Exp.1	Exp.2
Line (L)						
W ⁺	10.90 ^{a++}	17.51 ^b	11.80 ^b	11.98 ^b	19.25 ^a	20.18 ^a
F	10.78 ^a	14.93 ^c	12.45 ^a	14.86 ^a	20.17 ^a	19.98 ^a
E	11.08 ^a	19.13 ^a	7.71 ^d	7.39 ^d	19.93 ^a	20.55 ^a
C	10.97 ^a	17.70 ^b	9.25 ^c	10.24 ^c	20.12 ^a	20.66 ^a
LSD (5%)	0.32	1.82	2.22	1.47	0.95	0.80
Diet (D)						
H ⁺⁺⁺	12.84 ^a	20.37 ^a	10.88 ^a	13.93 ^a	19.61 ^a	20.06 ^a
L	10.62 ^b	14.27 ^b	8.74 ^b	8.30 ^b	20.13 ^a	20.62 ^a
LSD (5%)	0.22	1.29	1.57	1.04	0.67	0.58
Sex (S)						
Male	10.93 ^a	17.68 ^a		10.00 ^b		20.65 ^a
Female	10.93 ^a	16.96 ^a		12.23 ^a		20.03 ^a
LSD (5%)	0.22	1.29		1.04		0.57
Interaction						
L x D	NS	NS	NS	**	NS	NS

⁺, ⁺⁺, ⁺⁺⁺ See footnotes in Table 2.

Values taken from male birds only.

The effects of line, dietary nutrient density and sex upon feed retention time and digestive organ size is shown in Table 4: There was a significant difference in mean retention time between the lines. Retention time in the F line was only 80% that in the E line. The E line also had the relatively heaviest proventriculus, gizzard and intestine whilst the F line had the smallest proventriculus and gizzard but the largest liver and about four times as much abdominal fat as the E line. Birds given the high density diet had a smaller crop, gizzard and intestine but a longer intestine and more abdominal fat than those given the low density diet. Males had a relatively smaller proventriculus and gizzard and less fat than females.

DISCUSSION

The response in food intake in the E and C lines in both experiments to variation in dietary nutrient density is in keeping with the now well accepted proposition that food intake is regulated to meet energy requirements. The F line, however, responded quite differently by actually decreasing food intake on the low nutrient density diet, significantly in the first experiment and marginally in the second. Food intake in the W line responded similarly to the E and C lines in experiment 1 but showed little response to dietary nutrient density in experiment 2.

Table 4. Mean values for food retention time, digestive organ and fat pad size in the four lines of chickens given diets varying in nutrient density

	Food retention time, min	Crop g/kgW	Proventriculus g/kgW	Gizzard g/kgW	Intestine g/kgW	Intestine cm	Liver g/kgW	Fat pad g/kgW
Line (L)								
W ⁺	150 ^{ab++}	5.0 ^a	3.4 ^{ab}	22.6 ^{bc}	14.9 ^b	144 ^{ab}	18.7 ^b	22.7 ^b
F	132 ^b	5.1 ^a	3.1 ^b	21.6 ^c	15.9 ^{ab}	135 ^b	21.8 ^a	37.3 ^a
E	166 ^a	5.0 ^a	3.6 ^a	27.8 ^a	16.8 ^a	148 ^a	18.7 ^b	9.9 ^d
C	148 ^{ab}	5.1 ^a	3.6 ^a	24.5 ^b	15.6 ^{ab}	144 ^{ab}	20.8 ^a	15.3 ^c
LSD (5%)	24	0.5	0.4	2.5	1.2	12	1.9	4.4
Diet (D)								
H ⁺⁺⁺	157 ^a	4.4 ^b	3.2 ^b	18.2 ^b	14.6 ^b	149 ^a	20.2 ^a	30.1 ^a
L	140 ^b	5.6 ^a	3.7 ^a	30.1 ^a	17.1 ^a	137 ^b	19.8 ^a	12.5 ^b
LSD (5%)	17	0.4	0.3	1.8	0.9	8	1.3	3
Sex (S)								
Male	153 ^a	5.1 ^a	3.3 ^b	23.2 ^b	15.8 ^a	145 ^a	20.0 ^a	17.9 ^a
Female	145 ^a	4.9 ^a	3.6 ^a	25.1 ^a	16.0 ^a	141 ^a	20.0 ^a	24.7 ^b
LSD (5%)	17	0.4	0.3	1.8	0.9	8	1.3	3
Interaction								
L x D	NS	NS	NS	NS	NS	NS	NS	NS

+, ++, +++ See footnotes in Table 2.

It is probable that the response of the F line can be explained in terms of an impairment of the **hypothalamic satiety mechanism** in these birds as observed by Burkhardt *et al* (1983) in a line of chickens which had been selected for 22 **generations for increased juvenile growth rate**. These workers showed that electrolytic **lesioning** of the satiety centre in the ventromedial hypothalamus led to hyperphagy and obesity in the low weight selected line but had no effect on the high weight line. They suggested that long term selection for increased growth rate, through its correlated effect upon food intake, results in "genetic lesions" of the higher neural centres involved with satiety, and in such birds consumptive limits are set mainly by gastrointestinal **capacity**. As indicated in the present study, selection for increased food intake per se more rapidly approaches this situation than does selection for growth rate alone,

It is thus likely that selection for increased food intake should result in an alteration of gastrointestinal function to accommodate the greater quantities of food entering the digestive tract. It has been suggested by Renner (1965) and Hurwitz *et al* (1973) that the small intestine, being the major site for **digestion** and absorption, is likely to be altered by such **selection**. In the present study, however, the F line did not have a larger intestinal capacity than the other lines (in fact, it had a smaller proventriculus, gizzard and intestine than line E) and the

means by which the former line was able to accommodate the larger amounts of food **was** to increase the rate of passage of **ingesta**. The effect of **such increase** upon metabolisability of dietary energy was clearly shown in the study by Pym (1985) where **metabolisability** of dietary energy in the W, F, E and C lines was **73.0 + 0.3**, **62.7 + 1.8**, **76.0 + 0.3** and **72.7 + 0.5** respectively after 12 generations of **selection**. The very poor **metabolisability** in the F line in that study was due in all probability to the expression of a major gene which appears now to have been lost in that line after three generations of relaxed selection and high mortality in the affected individuals.

The improved feed efficiency in the E line may be partly due to the larger relative size of the proventriculus, intestine and gizzard and the slower rate of passage of the **ingesta** allowing for greater digestion and thus absorption of nutrients. This is reflected in the higher ME observed in this line in the present study and in the earlier study of Pym (1985).

A number of studies are presently underway or proposed, to elucidate the mechanisms for appetite control in the four lines. A study is now underway of the glucostatic control mechanism. Glucose solutions are infused into the hepatic portal vein to determine differential food intake responses in the four lines (Shurlock and Forbes, 1981; Forbes, 1982).

It is further proposed to shortly investigate the role of the satiety centre in the ventromedial hypothalamus in regulating appetite by electrolytically lesioning the centre using the method described by Burkhart'et al (1983) and determining the subsequent responses in food intake **in the four lines**.

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