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# BARD

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**FINAL REPORT**

**PROJECT NO. IS-1234-87**

הספריה המרכזית  
לפנימי החקלאות  
בית-17

**Growth Stimulation and Improved Feed  
Efficiency by Feed Restriction in Chickens and  
Turkeys**

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P.O. Box 6

Bet Dagan, ISRAEL

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*Title*

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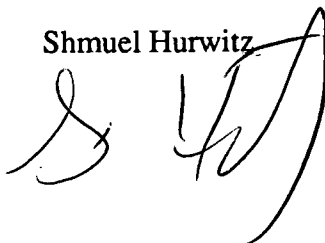
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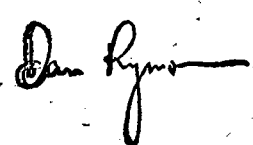
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### c. ABSTRACT

The results of the present project, inclusive of a large field experiment, substantiate the findings of the previous BARD project (I-735-84) which suggested a practical benefit due to improved feed efficiency and carcass quality in chickens and turkeys as a result of early-age feed restriction. The existence of compensatory growth of variable magnitude, following a large range of feed restriction conditions in terms of severity, duration, and some nutritional deficiencies such as protein or sodium, was validated. Furthermore, compensatory growth and improved feed efficiency were observed in several broiler strains characterized by a different growth potential. In effect, the large range of responses could be predicted fairly accurately by a single mathematical model. Thus, for practical implementation, economic consideration remains the main determinant of a suitable restriction regime, within the general context of nutrition and environment. The restriction-compensation model has been now incorporated into a general computerized optimization algorithm previously constructed as part of BARD project US-777-86.

In the search for a physiological mechanism, we found no specific involvement of the epiphyseal growth plate of the long bones, and no change in mineral gain in the compensatory growth response. In turkeys, muscle gain was specifically promoted by feed restriction. In chicks, this is also suggested by the increase in gain of carcass water. During feed restriction, lipogenesis decreases, whereas during refeeding, the normal temporal pattern of *in vivo* fat deposition changes so that for the first six weeks, fat deposition is considerably reduced.

Circulating levels of growth hormone and IGF-I were reduced by feed restriction. In another study, however, growth hormone level was increased during restriction. A severe energy restriction resulted in a reduction in triiodothyronine and an increase in the ratio of thyroxine to triiodothyronine. During refeeding, the normal decline in growth hormone was attenuated while IGF-I concentration remained similar to that the non-restricted birds. The level of testosterone was higher in restricted-refed than in *ad libitum*-fed turkeys and may thus play an important role in promoting muscle growth.

### **c. OBJECTIVES**

- a. To elucidate the mechanism of growth stimulation by early restriction.
- b. To determine breed differences in the response to feed restriction.
- c. to evaluate the responses in terms of growth, feed efficiency and carcass composition
- d. To examine the feed restriction technique under semi-practical conditions.

### **e. RESERACH REPORTS**



# Performance of Broiler Chickens and Turkey Poults Subjected to Feed Restriction or to Feeding of Low-Protein or Low-Sodium Diets at an Early Age<sup>1</sup>

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**ABSTRACT** The use of low-sodium (LoNa) and low-protein (LoPr) diets was examined in chickens and LoNa diets in turkey poults as a means of restricting growth at an early age. In the first trial, the LoNa, LoPr, or control diet were fed to 8-day-old male broiler chickens for *ad libitum* consumption for 6 days. An additional treatment group was pair-fed the LoNa diet (feed restricted). During the restriction period, the birds fed the LoPr diet gained the least BW. Also, these birds did not recover the lost weight as measured at 56 days of age; but feed efficiency was improved significantly. At 56 days of age, abdominal fat was reduced. Meat yield was not affected by any of the early-growth-restriction treatments terminated at 14 days of age.

In another experiment, 6-day-old female chicks received a LoNa diet for 6 or 9 days or were severely feed-restricted for 6 days. At 56 days of age, BW losses had been recovered completely only in the birds fed the LoNa diet for 6 days. Feed efficiency was improved and carcass fat was reduced in all restricted groups.

For 10 days, 7-day-old turkey poults were given 1) LoNa diet 2) pair-feeding regimen for the LoNa birds, or 3) a more severe regimen of feed restriction. At 147 days, the BW of the restricted birds, especially of the group pair-fed the LoNa treatment, exceeded the weight of the controls. A large part of this difference between feed-restricted birds and the control birds could be accounted for by a corresponding difference in the yield of breast muscle. Overall feed efficiency was improved for the birds in all of the regimens involving early growth restrictions. The results suggested that feeding a LoNa diet may be beneficial in terms of early growth restriction for chickens and turkeys. However, the recovery of BW appears to be more complete when a controlled, early feed restriction is employed.

(Key words: feed restriction, chickens, turkey poults, body weight, sodium deficiency)

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## INTRODUCTION

Feed efficiency for both broiler chickens (Plavnik and Hurwitz, 1985, 1988a; McMurtry *et al.*, 1988) and growing turkeys (Plavnik and Hurwitz, 1988b) was improved by early feed restriction. With broilers, feed restriction also resulted in a reduced carcass fat content (Plavnik and Hurwitz, 1985, 1988a).

Since feed restriction may require extra labor or mechanization, alternate methods of restricting growth have been examined. Ferket and Moran (1985) fed low-energy diets to breeder toms. Diets deficient (Singsen *et al.*, 1965) or imbalanced (Smith, 1968) with respect to various amino acids or diets containing amino-acid analogs (Fancher and Jensen, 1988) were fed to heavy-breed, re-

placement pullets. For growing turkeys up to 6 wk of age, Auckland and Morris (1971) obtained a 25% growth retardation by feeding a low-protein diet. By 14 wk of age, the lost weight was mostly regained; also, feed conversion was improved. With 21-wk-old, male turkeys, Plavnik and Hurwitz (1988b) found a complete recovery of BW after the birds (at 7 to 17 days of age) were fed on a diet with a maintenance protein content.

In the present study, the use of low-protein (LoPr) and low-sodium (LoNa) diets to restrict the growth of young broiler chickens was examined. The use of a LoNa diet with turkeys was also evaluated in comparison to feed restriction.

## MATERIALS AND METHODS

### The Birds

Day-old Cobb chicks or British United Turkey poults were obtained from commercial hatcheries and were raised for 4 wk in battery

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TABLE 1. Composition of the experimental diets for chickens<sup>1</sup>

Ingredient	Diet number				
	1	2	3	4	5
	(g per kg)				
Ground yellow corn	350	400	405	500	500
Ground sorghum	213	550	100	103	170
Soybean meal <sup>2</sup>	322	...	417	265	221
Fish meal	50	10	...	50	40
Soybean soapstock	34	...	45	50	40
Dicalcium phosphate	16	22	21.5	20	18.5
Ground limestone	7.1	9	5.7	5	5
Sodium chloride	2.5	2.5	...	2.5	2.5
DL-methionine	1.8	2.0	1.8	.5	...
Lysine-HCl	...	1.0	...	...	...
Vitamin mix <sup>3</sup>	2.5	2.5	2.5	2.5	2.5
Mineral mix <sup>3</sup>	1	1	1	1	1
Calculated composition					
ME, kcal per kg	3,060	3,190	3,010	3,200	3,200
Protein, %	22.6	9.4	22.8	20.1	18.1
Calcium, %	.93	.90	.83	.93	.84
Phosphorus, %	.77	.67	.78	.82	.75

<sup>1</sup>Diets 1 to 3 were for 0 to 3-wk-old chicks. Diets 4 and 5 were given to birds that were 3 to 6 wk old and 6 to 9 wk old, respectively. Diet 2 = LoPr; Diet 3 = LoNa.

<sup>2</sup>Containing 44.5% protein.

<sup>3</sup>The vitamin mixture supplied (per kilogram of feed): retinyl propionate, 8,500 IU; vitamin D<sub>3</sub>, 1,700 ICU; DL- $\alpha$ -tocopheryl acetate, 10 mg; menadione sodium bisulfite, 2 mg; riboflavin, 5 mg; Ca-pantothenate, 10 mg; niacin, 20 mg; vitamin B<sub>12</sub>, .01 mg; folic acid, .5 mg; pyridoxine, 1.5 mg; choline chloride, 200 mg; ethoxyquin, 125 mg; bacitracin, 10 mg. The mineral mix supplied (in milligrams per kilogram of feed): Mn, 80; Zn, 50; Fe, 25; Cu, 2; I, 1.2; Co, .2; Se, .1.

brooders in rooms at a constant temperature of 24 C. Subsequently, the birds were transferred to individual cages (with community feeders) situated in temperature-controlled rooms (20 C).

The experiments were started at 6, 7, or 8 days of age, the birds with extreme weights were eliminated then. The remaining birds were assigned to the experimental groups, equalizing both the mean and variance of BW within the groups. Each treatment was applied to four replicate groups of 10 birds in the chick experiment or to six replicate groups of 8 turkey poults.

During the growth-restriction period of 6 days for chickens and 10 days for turkeys, the birds were given either the deficient diets or a daily allowance of energy calculated to maintain BW according to the formula modified from Hurwitz *et al.* (1980):

$$\begin{aligned} \text{Energy intake (kcal ME per day)} \\ = M \cdot BW^{2/3}; \end{aligned} \quad [1]$$

where BW (in grams) is the weight at the beginning of the restriction period; and where M = 1.5 or 1.9, respectively, in the equations used

for chicks or turkeys. Following the restriction period, birds had *ad libitum* access to diets appropriate for their species and age (Tables 1 and 2).

The birds were weighed individually at the end of the restriction period and at weekly intervals thereafter. Feed-intake records, on a group basis, were also obtained at those times. At the termination of the experiments, 8 birds from each treatment group with BW around the mean treatment weight were killed. Estimates were made for the yields of carcass meat and fat.

#### The Diets

For both chickens and turkeys, the diets were designed to satisfy the recommendations of the National Research Council (1984) and were composed primarily of corn, sorghum grain, and soybean-oil meal. The MINOS (Murtagh and Saunders, 1983) program was used in the computer calculations of the diets. The LoPr diet (Diet 2, Table 1) did not contain soybean meal, but was supplemented with 1% fish meal in order to maintain a favorable balance of amino acids. The calculated protein content was confirmed by Kjeldahl analysis. The LoNa diet

## EARLY GROWTH RESTRICTION IN BIRDS

TABLE 2. Composition of the experimental diets for turkeys, Trial 3<sup>1</sup>

Ingredient	Diet number <sup>1</sup>						
	1	2	3	4	5	6	7
	(g per kg)						
Ground yellow corn	200	250	250	250	250	250	250
Ground sorghum	178	152	292	368	418	492	165
Soybean meal <sup>2</sup>	523	510	370	294	244	166	553
Fish meal	20	...	...	...	...	...	...
Soybean soapstock	34	50	50	50	50	55	40
Dicalcium phosphate	23	20	20	20	20	20	23
Ground limestone	13	10	10	10	10	10	13
Sodium chloride	3.5	3.5	3.5	3.5	3.5	3.5	...
DL-methionine	2	1.3	1.2	1	.5	.2	2
Vitamin mix <sup>3</sup>	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Mineral mix <sup>3</sup>	1	1	1	1	1	1	1
Calculated composition							
ME, kcal per kg	2,800	2,900	3,000	3,100	3,200	3,250	2,800
Protein, %	28.0	26.0	22.0	18.4	16.5	13.8	28.0
Calcium, %	1.26	.99	.95	.93	.93	.90	1.20
Phosphorus, %	.90	.79	.74	.71	.69	.66	.87

<sup>1</sup>Diets 1 to 6 are for wk of age, respectively: 0 to 4, 4 to 8, 8 to 12, 12 to 16, 16 to 20, and over 20. Diet number 7 is the LoNa diet.

<sup>2</sup>Containing 44.5% protein.

<sup>3</sup>The vitamin mixture supplied (per kilogram of feed): retinyl propionate, 10,000 IU; cholecalciferol, 2,000 ICU; DL- $\alpha$ -tocopheryl acetate, 10 mg; menadione sodium bisulfite, 2 mg; riboflavin, 7 mg; Ca-pantothenate, 15 mg; niacin, 60 mg; vitamin B<sub>12</sub>, .02 mg; folic acid, .5 mg; pyridoxine, 1.5 mg; choline chloride, 900 mg; ethoxyquin, 125 mg; bacitracin, 25 mg. The mineral mix supplied (in milligrams per kilogram of feed): Mn, 90; Zn, 75; Fe, 25; Cu, 5.2; I, 1.5; Co, .2; Se, .2.

(.03% Na, determined by flame photometry) contained no fish meal and no supplementary sodium chloride. The sodium content of the drinking water was not monitored.

experiment was terminated when the birds were 56 days of age.

### Trial 2

Here, the purpose was to compare the efficacy of using LoNa diets versus feed restriction in female broiler chicks. The experiment was started with 6-day-old, female chicks and included: 1) an *ad libitum*-fed control group (Treatment 1); 2) a 6-day regimen of feed restriction, according to Equation 1; 3) a 6-day feeding period using a LoNa diet (Treatment 3); and 4) a 9-day feeding period with a LoNa diet (Treatment 4). The experiment was terminated when the birds were 56 days of age.

### Trial 3

The purpose of this trial was to compare the efficacy of using LoNa diets with total feed restriction in turkey poults (Table 2). The experiment was started with 7-day-old male poults and included: 1) a control group fed on an *ad libitum* basis (Treatment 1); 2) another group on feed restriction for 10 days, according to Equation 1; 3) a 10-day feeding period using a

### Statistical Analyses

The results were subjected to a one-way analysis of variance (Snedecor and Cochran, 1967). The means were separated by multiple comparison tests (Duncan, 1955).

### Trial 1

The purpose of this trial was to evaluate the use of LoNa or LoPr diets for restricting growth in male broilers. The experiment was started with 8-day-old, male chicks and included: 1) a control group given feed for *ad libitum* consumption (Treatment 1); 2) a 6-day period of feeding using a LoPr diet designed to supply amino acids for maintenance only, using a previous model (Hurwitz *et al.*, 1980) and containing 9.2% CP (Treatment 2); 3) a 6-day feeding period with the LoNa diet (Treatment 3); and 4) feed restriction by pair-feeding to Treatment 3 (Treatment 4). The

LoNa diet (Treatment 3); and 4) a diet-restriction group, pair-fed to Treatment 3 (Treatment 4). The experiment was terminated when the poults were 147 days old.

## RESULTS

### Trial 1

During the restriction period, the birds receiving the LoPr diet gained only 22% as much weight as the controls (Table 3). This reduction in growth was associated with reduced feed intake and very low feed efficiency. A considerably higher growth rate than that of the LoPr birds was exhibited by the birds that consumed the LoNa diet (Treatment 3), but their weight gain, feed intake, and feed efficiency from 8 to 14 days of age were lower than the controls. In Treatment 4, where the birds received the control diet pair-fed to the LoNa birds, feed efficiency was only about 22% less than for the controls; and growth was restricted considerably less than with the LoNa birds.

Following growth retardation, compensatory growth by the pair-fed birds resulted in a minimization of the difference in BW between the restricted (pair-fed) birds and the controls at 56 days of age (Table 3). During the period after growth retardation, feed efficiency was superior to that of the controls. Compared with the controls, overall feed efficiency was significantly improved only for the LoNa birds. Abdominal fat was significantly lower in the growth-restricted birds from either treatment, versus the controls. The percentage of thigh and breast meat was not affected significantly by the treatments. Mortality was 3, 5, 2, 1 (out of 40) birds in Treatments 1 to 4, respectively.

### Trial 2

The feed-restriction regimen (Treatment 2), as applied in this experiment with female chicks, retarded the BW gain from 6 to 12 days of age to about 19% of that achieved by the controls (Table 4). A considerably less growth reduction was obtained by feeding the LoNa diet. Following restriction, the growth of birds given the feed-restriction treatment failed to overcome the retardation of BW. The BW gains of the birds from 12 to 56 days of age were 2,094, 2,062, 2,099, and 2,042 g for Treatments 1, 2, 3, and 4, respectively. However, during the refeeding period, the feed efficiency of all the growth-

restricted birds was superior to that of the controls. However, overall feed efficiency was improved only by feed restriction or by feeding the LoNa diet for 9 days. Abdominal fat, expressed as a percentage of BW, was lower for all treatment groups, versus the controls. Only one bird (in Treatment 3) died during the experiment.

### Trial 3

In this trial, the response of growing turkeys to a LoNa diet was compared with the response to a feed restriction (Table 5, Figures 1 and 2). During the restriction period (Table 5), growth retardation was most severe (at approximately 31% of the control birds) in Treatment 2, in which the energy allowance was calculated only for BW maintenance. However, this allowance permits some growth due to a decrease in the energy needs for maintenance.

Due to extremely poor feed efficiency, the LoNa birds in Treatment 3 gained less weight during the restriction period than their pair-fed

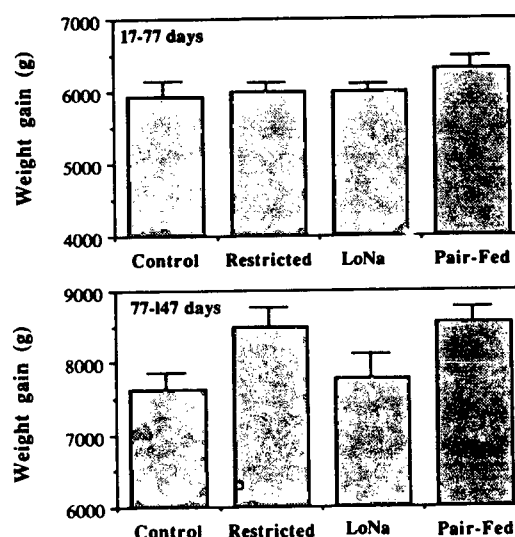


FIGURE 1. The effect of feed restriction (restricted), intake of a diet low in sodium chloride (LoNa), and feed restriction by pair-feeding the LoNa diet, at 7 to 17 days of age, on subsequent weight gain in turkey poults. Upper graph, between 17 and 77 days of age; no significant differences ( $P > .05$ ). Lower graph, between 77 and 147 days of age; significant differences between the controls compared with the restricted and pair-fed birds. The SE is given by the vertical bars (Trial 3).

## EARLY GROWTH RESTRICTION IN BIRDS

TABLE 3. The effect of protein, sodium, and quantitative feed restriction on male broiler performance, Trial 1<sup>1</sup>

Variable	Treatment <sup>2</sup>				SE <sup>3</sup>
	Control group	LoPr diet	LoNa diet	Pair-fed birds	
Body weight, g					
8 days	171	171	171	171	.1
14 days	359 <sup>a</sup>	212 <sup>d</sup>	257 <sup>c</sup>	285 <sup>b</sup>	2.7
56 days	2,766 <sup>ab</sup>	2,627 <sup>b</sup>	2,725 <sup>ab</sup>	2,796 <sup>a</sup>	45.0
Feed intake, g					
8 to 14 days	280 <sup>a</sup>	177 <sup>c</sup>	222 <sup>b</sup>	216 <sup>b</sup>	4.2
14 to 56 days	5,284 <sup>ab</sup>	4,980 <sup>b</sup>	5,091 <sup>ab</sup>	5,348 <sup>a</sup>	96.1
8 to 56 days	5,564 <sup>a</sup>	5,157 <sup>b</sup>	5,313 <sup>ab</sup>	5,563 <sup>a</sup>	96.3
Feed efficiency, g gain per g of feed					
8 to 14 days	.672 <sup>a</sup>	.230 <sup>d</sup>	.392 <sup>c</sup>	.525 <sup>b</sup>	.014
14 to 56 days	.456 <sup>c</sup>	.485 <sup>a</sup>	.485 <sup>a</sup>	.470 <sup>b</sup>	.004
8 to 56 days	.466 <sup>b</sup>	.476 <sup>ab</sup>	.481 <sup>a</sup>	.472 <sup>ab</sup>	.004
Abdominal fat, g	81.8 <sup>a</sup>	59.6 <sup>b</sup>	57.0 <sup>b</sup>	58.3 <sup>b</sup>	3.9
BW, % <sup>4</sup>	2.93 <sup>a</sup>	2.16 <sup>b</sup>	2.18 <sup>b</sup>	2.10 <sup>b</sup>	.13
Thigh meat, g	442 <sup>a</sup>	447 <sup>ab</sup>	423 <sup>b</sup>	456 <sup>ab</sup>	10.0
BW, % <sup>4</sup>	15.8	16.1	16.3	16.3	.39
Breast meat, g	392	379	369	383	11.6
BW, % <sup>4</sup>	13.9	14.0	13.8	13.8	.40

<sup>a-d</sup>Within rows, values with no common superscript differ significantly ( $P < .05$ ).<sup>1</sup>Each treatment included four groups with 10 birds per group.<sup>2</sup>Control birds fed on an *ad libitum* basis. LoPr = low protein; LoNa = low NaCl; Pair-fed = intake of control diet was restricted to intake of birds given the LoNa CL diet.<sup>3</sup>Calculated from the error term of the analysis of variance.<sup>4</sup>Percentage of live weight,  $n = 8$  individual birds.TABLE 4. The effect of sodium restriction on female broiler performance, Trial 2<sup>1</sup>

Variable	Treatment				SE <sup>2</sup>
	Control group <sup>3</sup>	Birds on feed restriction	Low sodium chloride		
			6 days	9 days	
Body weight, g					
6 days	116	116	116	116	.3
12 days	269 <sup>a</sup>	145 <sup>c</sup>	195 <sup>b</sup>	195 <sup>b</sup>	2.6
56 days	2,363 <sup>a</sup>	2,207 <sup>c</sup>	2,294 <sup>ab</sup>	2,237 <sup>bc</sup>	26.6
Feed intake, g					
6 to 12 days	211 <sup>a</sup>	74 <sup>c</sup>	162 <sup>b</sup>	166 <sup>b</sup>	2.7
12 to 56 days	4,534 <sup>a</sup>	4,233 <sup>bc</sup>	4,347 <sup>b</sup>	4,159 <sup>c</sup>	35.0
6 to 56 days	4,745 <sup>a</sup>	4,306 <sup>c</sup>	4,509 <sup>b</sup>	4,325 <sup>c</sup>	37.4
Feed efficiency, g gain per g of feed					
6 to 12 days	.730 <sup>a</sup>	.383 <sup>c</sup>	.486 <sup>b</sup>	.476 <sup>b</sup>	.010
12 to 56 days	.462 <sup>b</sup>	.487 <sup>a</sup>	.483 <sup>a</sup>	.491 <sup>a</sup>	.003
6 to 56 days	.474 <sup>b</sup>	.485 <sup>a</sup>	.483 <sup>ab</sup>	.491 <sup>a</sup>	.003
Abdominal fat, g	63.5 <sup>a</sup>	52.4 <sup>b</sup>	47.5 <sup>b</sup>	61.4 <sup>ab</sup>	4.8
BW, % <sup>4</sup>	3.26 <sup>a</sup>	2.52 <sup>b</sup>	2.65 <sup>b</sup>	2.79 <sup>b</sup>	.09

<sup>a-c</sup>Within rows, values with no common superscript differ significantly ( $P < .05$ ).<sup>1</sup>Each treatment included four groups, 10 birds per group.<sup>2</sup>Calculated from the error term of the analysis of variance.<sup>3</sup>Fed for *ad libitum* consumption.<sup>4</sup>Percentage of live weight,  $n = 8$  individual birds.

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TABLE 5. The effect of a low-sodium diet and quantitative feed restriction on the performance of male turkey poults, Trial 3<sup>1</sup>

Variable	Treatment				SE <sup>2</sup>
	Control group <sup>3</sup>	Birds in feed restriction	Low sodium chloride	Pair-fed birds <sup>4</sup>	
Body weight, g					
7 days	150	150	150	150	0
17 days	460 <sup>a</sup>	246 <sup>d</sup>	320 <sup>c</sup>	377 <sup>b</sup>	3
147 days	13,991 <sup>b</sup>	14,728 <sup>ab</sup>	14,111 <sup>b</sup>	15,291 <sup>a</sup>	273
Feed intake, g					
7 to 17 days	427 <sup>a</sup>	193 <sup>d</sup>	390 <sup>b</sup>	347 <sup>c</sup>	4
7 to 147 days	41,715 <sup>ab</sup>	41,982 <sup>ab</sup>	40,540 <sup>b</sup>	42,685 <sup>a</sup>	647
Feed efficiency, g gain per g of feed					
7 to 17 days	.726 <sup>a</sup>	.502 <sup>b</sup>	.438 <sup>c</sup>	.713 <sup>a</sup>	.014
7 to 147 days	.332 <sup>b</sup>	.347 <sup>a</sup>	.345 <sup>a</sup>	.359 <sup>a</sup>	.005
Thigh meat, g	2,746	2,894	2,912	3,038	111
BW, % <sup>5</sup>	19.0	19.0	19.9	19.5	.57
Breast meat, g	2,882 <sup>c</sup>	3,346 <sup>ab</sup>	3,132 <sup>bc</sup>	3,693 <sup>a</sup>	124
BW, % <sup>5</sup>	20.0 <sup>b</sup>	21.9 <sup>ab</sup>	21.4 <sup>b</sup>	23.8 <sup>a</sup>	.77

<sup>a-d</sup>Within rows, values with no common superscript differ significantly ( $P < .05$ ).

<sup>1</sup>Each treatment included six groups, with 6 birds each.

<sup>2</sup>Calculated from the error term of the analysis of variance.

<sup>3</sup>Birds fed on an *ad libitum* basis.

<sup>4</sup>The intake of the control diet was restricted to the intake of the birds given the diet low in sodium chloride.

<sup>5</sup>Percentage of live weight,  $n = 8$  individual birds.

counterparts (Treatment 4), although they consumed even slightly more feed. During the first half of the repletion period of 130 days, weight gains did not differ significantly among any of the treatments (Figure 1), but were significantly greater for birds in the feed-restriction treatments (Treatments 2 and 4) than in the control group (Treatment 1) during the second half of the repletion period. Such improvement was not observed in the chickens previously fed the LoNa diet (Treatment 3).

Under all growth-restriction treatments, feed efficiency was higher for those birds than for the controls during the first half of the refeeding period (Figure 2). No significant differences in feed efficiency were observed during the second half of the refeeding period.

Accelerated growth during refeeding resulted in a complete recovery of BW (Table 5). In Treatment 4, the BW at 147 days even exceeded that of the controls and that of turkeys fed the LoNa diet. Under all growth-restricted treatments, feed efficiency was greater than for the control birds. The yield of thigh meat was not significantly affected by the treatment. However, the absolute yield (grams of breast meat) from the feed-restricted birds (Treatments 2 and

4) significantly exceeded that of the control birds. Mortality during the entire experiment was 6, 4, 7, and 7 poults (out of 48), for Treatments 1 to 4, respectively.

## DISCUSSION

The present results confirm previous observations by Plavnik and Hurwitz (1985, 1988a,b), by Plavnik *et al.* (1986), and by McMurtry *et al.* (1988), which suggested that there was an advantage for early feed restriction in terms of improving feed efficiency with broiler chickens and turkey poults and reducing carcass fat with broilers. Similar improvements in feed efficiency at market age for both species, and the reduced carcass fat for broilers, can be achieved by using diets fed at an early age that are severely deficient in protein or sodium. However, the practical use of such deficient diets requires additional standardization.

The poor feed efficiencies on the protein-deficient or sodium-deficient diets during growth restriction indicated that a reduced appetite accounts for only part of the induced depression in weight gain, as suggested by the

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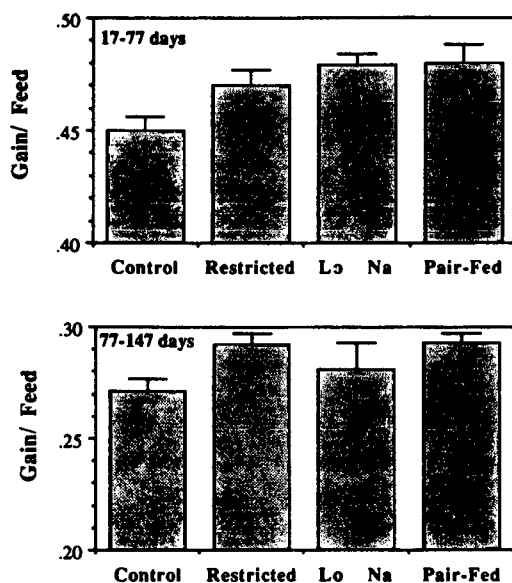


FIGURE 2. The effect of feed restriction (restricted), intake of a diet low in sodium chloride (LoNa), and feed restriction by pair-feeding the LoNa diet, at 7 to 17 days of age, on subsequent feed efficiency in turkey poults. Upper graph, between 17 and 77 days of age; significant differences occurred between the controls and the growth-restricted birds. Lower graph, between 77 and 147 days of age; no significant differences ( $P > .05$ ). The SE is given by the vertical bars (Trial 3).

comparison between the deficient groups and their pair-fed counterparts (Tables 3 and 5). During the growth-restriction period both growth rate and feed efficiency were always greater for the pair-fed birds than for the ones on a sodium-deficient diet. Furthermore, weight gain in turkeys during the second half of the refeeding period was significantly lower for the LoNa birds than for those subjected to feed restriction (Figure 1). As a result, the BW of the turkeys on the LoNa treatment was lower than for the birds in the pair-fed treatment at 147 days of age. No explanation can be offered at present for this outcome.

The results of a previous study (Plavnik and Hurwitz, 1988a) as well as those from the present study suggest that compensatory growth (Wilson and Osborn, 1960) in female chicks is not as marked as in males. The present results, however, suggest that after a mild growth restriction as imposed by feed restriction or by feeding a LoNa diet, feed

efficiency can be improved and that carcass fat as a percentage of BW, can be reduced also in females.

Although BW at market age was not suppressed, and feed efficiency was improved in male broilers and turkeys by feeding a LoNa diet, superior performance was obtained by a feed-restriction regimen using a complete feed (Table 3, Treatment 3 versus Treatment 4; Table 5, Treatment 3 as opposed to Treatments 2 and 4), suggesting that feed restriction is preferable to feeding a deficient diet.

Plavnik and Hurwitz (1985; 1988a,b) suggested previously that accelerated growth in chickens occurred about 4 to 5 wk (for turkeys, 10 wk) after the end of a severe feed restriction. This finding is supported by the results of the present turkey experiment in which the weight gain between 77 and 147 days of age was greater for the restricted groups than for the controls receiving feed *ad libitum*. As shown previously (Frisch, Y., and S. Hurwitz, unpublished results), skeletal growth during this period is minimal, so most of the weight gain is derived from muscle growth. Therefore, it is not surprising that the weight and proportion of pectoral muscle were increased by the feed-restriction treatments (Table 5). This result, however, has not been observed in broiler chicks.

The previous observation by Plavnik and Hurwitz (1988b) that the BW of turkeys restricted at 7 days of age sometimes exceeded that of the birds (controls) on an *ad libitum* feeding routine—was also made in the present study (Treatment 4, Table 5 and Figure 1). The feed restriction applied in Treatment 4 was milder than that used in Treatment 2, suggesting that growth could be promoted by using a mild feed restriction at an early age.

## ACKNOWLEDGMENTS

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## RESPONSE OF BROILER CHICKENS AND TURKEY POULTS TO FOOD RESTRICTION OF VARIED SEVERITY DURING EARLY LIFE

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**Abstract** 1. Cobb male (7-d-old) and female (6-d-old) chicks, and BUT male and female turkeys (7-d-old), were subjected to food restriction of varying severity for 7, 5, 10 and 7 d, respectively.  
2. The energy allowance was calculated to support from 0% to 75% of the growth rate typical of species, strain and sex.  
3. In male and female broilers kept up to 56 and 50 d, respectively, a high rate of weight gain after the restriction period (refeeding period) overcame the growth retardation attributable to restriction, except in the birds which had been subjected to the most severe regimen. In the mildest regimens, body weights reached slightly higher values than those of the *ad libitum*-fed controls. Overall food conversion efficiency was significantly improved in males and abdominal fat content was reduced in both sexes, regardless of the severity of restriction.  
4. Body weights of male and female turkeys, aged 147 and 112 d respectively, of the groups subjected to the milder (75% growth) restriction, significantly exceeded those of the controls. Food conversion efficiency and meat yield were improved by early-age food restriction but carcass fat was not affected.  
5. The results for both species of either sex suggest that a mild food restriction applied at an age of about 1 week and allowing for 60 to 75% of normal growth, may offer an economic advantage over a continuous *ad libitum* feeding regimen.

### INTRODUCTION

Compensatory growth (Wilson and Osborn, 1960; Auckland and Morris, 1971) following food restriction can permit an early reduction in rate of weight gain to be recouped by marketing age (Plavnik and Hurwitz, 1985). Body weights of chicks following restriction can even exceed those of the *ad libitum*-fed (Plavnik and Hurwitz, 1990), with restriction regimens less severe than those previously evaluated (Plavnik and Hurwitz, 1985). Early age food restriction resulted in improved food conversion efficiency in broiler chickens (Plavnik and Hurwitz, 1985; 1988a; McMurtry *et al.*, 1988) and growing turkeys

(Plavnik and Hurwitz, 1988b; 1990). Furthermore, the severe early food restriction practiced in previous studies with broilers always resulted in a decrease in the fat content of the abdominal fat pad (Plavnik and Hurwitz, 1985; 1988a; McMurtry *et al.*, 1988). The data of Cartwright *et al.* (1986) suggested that the reduction in abdominal fat resulted from a corresponding reduction in the number of adipocytes, induced by the early food restriction. Because only severe food restriction regimens were applied in these studies, a possible relationship between carcass fat and the severity of restriction, or the possible existence of a minimum restriction threshold for the effect on carcass fat, have not been investigated. In turkeys, early food restriction did not affect carcass fat at marketing age, but meat yield was increased (Plavnik and Hurwitz, 1988b; 1990).

In the present study, a wide range of food restriction regimens has been investigated in male and female broiler chickens and turkeys in an effort to achieve greater flexibility in the use of this technique.

#### MATERIALS AND METHODS

One-d-old Cobb chicks and British United Turkey poults were obtained from commercial hatcheries and reared for 4 weeks in battery brooders in constant-temperature rooms at 24°C. Subsequently, they were transferred to individual cages situated in temperature-controlled (20°C) rooms with continuous artificial illumination.

The experiments started at 6 or 7 d of age. At that time, birds with extreme weights were eliminated, and the remaining birds were assigned to the experimental groups, equalising both mean and variance of body weight within the groups. Each treatment was applied to 4 replicate groups of 10 chicks or to 6 replicate groups of 6 turkey poults.

During the growth restriction period, the birds were given a daily allowance of energy (EI, in kJ/d), calculated to maintain body weight and support a rate of growth varied according to treatment, using an equation modified from Hurwitz *et al.* (1980):

$$EI = M \cdot W^{2/3} + D \cdot G$$

where  $W$  (g) is the body weight at the beginning of the restriction period. Forsum *et al.* (1981) and Plavnik and Hurwitz (1985; 1988a) showed that maintenance energy requirement decreased considerably during food restriction. The results of Plavnik and Hurwitz (1985) were then used to calculate an approximate dependence of maintenance requirement ( $M$ ) on degree of restriction. In this study,  $M$  for broilers was varied between 8.37 and 4.18 kJ/g  $W^{2/3}$ , proportional to the degree of restriction. For turkeys,  $M = 5.44$  kJ/g  $W^{2/3}$  was used. The value for  $D$ , the coefficient for weight gain ( $G$ , in g/d) was the same as that of  $M$ ; it varied with the degree of restriction because of the expected graded responses of carcass fat deposition during the restriction period. Daily energy allowances were calculated for the period of restriction. However, an average allowance was fed daily throughout the period. Following

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the restriction period, birds were fed *ad libitum* diets appropriate for their species and age (Tables 1 and 2).

TABLE 1

*Composition of the experimental diets fed to broiler chickens (Trials 1 and 2)<sup>1</sup>*

Diet No.	1	2	3
Ingredients (g/kg)			
Maize	350	500	500
Sorghum	203	103	170
Soyabean meal <sup>2</sup>	322	265	221
Fish meal	50	50	40
Soya soapstock	34	50	40
Dicalcium phosphate	16.0	21.2	18.5
Ground limestone	7.1	5.0	5.0
Sodium chloride	2.5	2.5	2.5
DL-methionine	1.8	0.5	...
Vitamin mix <sup>3</sup>	2.5	2.5	2.5
Mineral mix <sup>3</sup>	1.0	1.0	1.0
Calculated composition			
Metabolisable energy (MJ/kg)	12.8	13.4	13.4
Protein (g/kg)	220	201	181
Sulphur amino acids (g/kg)	9.3	7.4	6.3
Lysine (g/kg)	13.2	11.6	10.0

<sup>1</sup> Diets 1, 2 and 3 were fed to 0–3, 3–6 and 6–8-week-old chicks, respectively.

<sup>2</sup> Containing 445 g/kg protein.

<sup>3</sup> Vitamin mix (per kg food): retinyl acetate 2.7 mg, cholecalciferol 42.5 µg, DL-α-tocopheryl acetate 10 mg, menadione sodium bisulphite 2 mg, riboflavin 5 mg, Ca pantothenate 10 mg, niacin 20 mg, cyanocobalamin 0.01 mg, folacin 0.5 mg, pyridoxine 1.5 mg, biotin 0.1 mg, choline chloride 200 mg, ethoxyquin 125 mg, bacitracin 10 mg. Mineral mix (mg/kg food): manganese 80, zinc 50, iron 25, copper 2, iodine 1.2, cobalt 0.2, selenium 0.1.

The birds were weighed individually at the end of the restriction period and at weekly intervals thereafter. Food intake records, on a group basis, were also obtained at those times. At the end of the experiments, two birds of each replicate group in the broiler experiments, and one bird per replicate of the turkey experiments, with body weights near the mean treatment weight, were killed and dissected for estimation of yields of carcass meat and fat.

The diets for both chickens and turkeys (Tables 1 and 2) were designed to satisfy the recommendations of the National Research Council (1984) and were composed primarily of maize, sorghum grain and soyabean meal. The MINOS (Murtagh and Saunders, 1983) program was used in the computer calculations of the diets. Calculated protein content was confirmed by Kjeldahl analysis.

### Trial 1

The purpose of this trial was to evaluate graded food restriction in male broilers. The experiment was started with 7-d-old male chicks and included an *ad libitum*-fed control and 4 food-restriction treatments, A, B, C, and D, designed to support 22, 15, 6 and 0 g/d of weight gain, respectively during

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TABLE 2  
Composition of the experimental diets fed to turkeys (Trials 3 and 4)<sup>1</sup>

Diet No.	1	2	3	4	5	6
Ingredients (g/kg)						
Maize	200	250	250	250	250	250
Sorghum	178	152	292	368	418	492
Soyabean meal <sup>2</sup>	523	510	370	294	244	166
Fish meal	20	...	...	...	...	...
Soyabean soapstock	34	50	50	50	50	55
Dicalcium phosphate	23	20	20	20	20	20
Ground limestone	13	10	10	10	10	10
Sodium chloride	3.5	3.5	3.5	3.5	3.5	3.5
DL-methionine	2.0	1.3	1.2	1.0	0.5	0.2
Vitamin mix <sup>3</sup>	2.5	2.5	2.5	2.5	2.5	2.5
Mineral mix <sup>3</sup>	1.0	1.0	1.0	1.0	1.0	1.0
Calculated composition						
Metabolisable energy (MJ/kg)	11.7	12.1	12.6	13.0	13.4	13.6
Protein (g/kg)	280	260	220	184	165	138
Calcium (g/kg)	12.6	9.9	9.5	9.3	9.3	9.0
Phosphorus (g/kg)	9.0	7.9	7.4	7.1	6.9	6.6

<sup>1</sup> Diets 1-6 were fed to 0-4, 4-8, 8-12, 12-16, 16-20 and over 20-week-old turkeys, respectively.

<sup>2</sup> Containing 445 g/kg protein.

<sup>3</sup> Vitamin mix (per kg food): retinyl acetate 3.2 mg, cholecalciferol 50 µg, DL-α-tocopheryl acetate 10 mg, menadione sodium bisulphite 2 mg, riboflavin 7 mg, Ca-pantothenate 15 mg, niacin 60 mg, cyanocobalamin 0.02 mg, folacin 0.5 mg, pyridoxine 1.5 mg, biotin 0.1 mg, choline chloride 900 mg, ethoxyquin 125 mg, bacitracin 25 mg. Mineral mix (mg/kg food): manganese 90, zinc 75, iron 25, copper 5.2, iodine 1.5, cobalt 0.2, selenium 0.1.

restriction. Food restriction was applied for 7 d and the experiment was terminated at the age of 56 d.

### Trial 2

The purpose of this trial was to evaluate graded food restriction in female broiler chicks. The experiment was started with 6-d-old female chicks and included an *ad libitum*-fed control and three food-restriction treatments, A, B and C, designed to support 18, 12 and 6 g/d weight gain, respectively during restriction. Food restriction was applied for 5 d and the experiment was terminated at the age of 50 d.

### Trial 3

The purpose of this trial was to evaluate graded food restriction in male turkeys. The experiment included an *ad libitum*-fed control and three food-restriction treatments, A, B and C, designed to support a weight gain of 23, 15 and 10 g/d, respectively during restriction. The experiment was started at the age of 7 d and terminated at 147 d. Food restriction was applied for 10 d (7 to 17 d of age).

#### *Trial 4*

The purpose of this trial was to evaluate graded food restriction in female turkeys. The experiment included an *ad libitum*-fed control and three food-restriction treatments, A, B and C, designed to support a weight gain of 18, 12 and 6 g/d, respectively during restriction. The experiment was started at the age of 7 d and terminated at 112 d. Food restriction was applied for 7 d (7–14 d of age).

To determine carcass fat birds were killed by neck dislocation, their carcasses were defeathered, autoclaved for 3 h and homogenised. Fat was determined gravimetrically in an aliquot after extraction with a 2:1 mixture of chloroform:methanol (Folch *et al.*, 1957).

The results were subjected to a one-way analysis of variance (Snedecor and Cochran, 1967) and multiple comparison tests (Duncan, 1955). Results of carcass composition, calculated as fractions of body weight, were subjected to arcsine-square root transformation prior to analysis.

### RESULTS

#### *Trial 1*

During the restriction period, daily weight gains of the restricted birds differed slightly from those planned (Methods section). These differences may have resulted from inaccuracies in the calculation of energy needs or may have been attributable to the daily feeding of the average rather than the actual calculated daily energy allowance.

Compensatory growth following growth retardation resulted in minimisation of the difference in body weight between the restricted and the control birds at 56 d of age (Table 3). Body weights were reduced by the most severe restriction regimens (C and D) but tended to be higher than controls in treatment A and B; the latter difference, however, did not reach significance. Overall food intake was lower in the restricted birds, but significantly ( $P < 0.05$ ) so only in treatments C and D. Food conversion efficiency, both overall and during the period subsequent to growth retardation, was superior in all restricted treatments to that of the controls. Abdominal fat was less in all restricted treatments than in the controls, with inconsistent differences among the restricted groups. The proportion of breast meat was significantly reduced by treatments C and D. Mortality was 1, 0, 1, 1 and 0 birds (out of 40) in the control, A, B, C and D treatments, respectively.

#### *Trial 2*

Weight gains of the female chicks during the restriction period were slightly lower than planned (Methods section). Compensatory growth during the refeeding period resulted in a narrowing of the gap between the control and the restricted birds (Table 4). However, body weights on restriction treatments B and C remained significantly lower than those of the controls.

TABLE 3  
*The effect of food restriction of varied severity on male broiler performance (trial 1)<sup>1</sup>*

	Restriction					SE <sup>2</sup>
	Control	A	B	C	D	
Body weight (g)						
7 d	156	156	156	156	156	0.1
14 d	385 <sup>a</sup>	285 <sup>b</sup>	246 <sup>c</sup>	203 <sup>d</sup>	169 <sup>c</sup>	3.1
56 d	2974 <sup>a</sup>	3046 <sup>a</sup>	2983 <sup>a</sup>	2879 <sup>b</sup>	2798 <sup>b</sup>	27.7
Food intake (g)						
7-14 d	310 <sup>a</sup>	221 <sup>b</sup>	169 <sup>c</sup>	110 <sup>d</sup>	66 <sup>c</sup>	3.4
14-56 d	5478 <sup>ab</sup>	5526 <sup>a</sup>	5531 <sup>a</sup>	5342 <sup>bc</sup>	5239 <sup>c</sup>	41.8
8-56 d	5787 <sup>a</sup>	5747 <sup>a</sup>	5690 <sup>a</sup>	5452 <sup>b</sup>	5305 <sup>c</sup>	44.1
Food conversion efficiency (g gain/g food intake)						
7-14 d	0.742 <sup>a</sup>	0.587 <sup>b</sup>	0.538 <sup>c</sup>	0.433 <sup>d</sup>	0.208 <sup>c</sup>	0.012
14-56 d	0.472 <sup>b</sup>	0.500 <sup>a</sup>	0.494 <sup>a</sup>	0.501 <sup>a</sup>	0.502 <sup>a</sup>	0.004
7-56 d	0.486 <sup>b</sup>	0.503 <sup>a</sup>	0.497 <sup>ab</sup>	0.499 <sup>a</sup>	0.498 <sup>a</sup>	0.004
Abdominal fat (g/kg) <sup>3</sup>	25.6 <sup>a</sup>	19.8 <sup>b</sup>	20.6 <sup>b</sup>	19.3 <sup>bc</sup>	16.2 <sup>c</sup>	...
Breast meat (g/kg) <sup>3</sup>	148 <sup>ab</sup>	152 <sup>a</sup>	144 <sup>ab</sup>	139 <sup>bc</sup>	130 <sup>c</sup>	...

<sup>1</sup> Each treatment included 4 groups of 10 birds each. Values within rows with no common superscript are significantly ( $P < 0.05$ ) different.

<sup>2</sup> Calculated from the error term of the analysis of variance.

<sup>3</sup> Average of 8 birds per treatment. Statistical analysis was on the arcsine-square-root transformed results.

Food intake was lower than the control in restriction treatments B and C. Food conversion efficiency was improved by early food restriction during the refeeding period, but there were no significant differences among treatments in overall conversion efficiency. Abdominal fat was reduced by early feed restriction with no differences among the restricted treatments. Mortality was 5, 1, 1 and 6 birds (out of 40) in the control, A, B, and C treatments, respectively.

### Trial 3

As in the chick experiments, weight gains during the restriction period of 10 d, were lower than planned. Because of accelerated growth during the refeeding period, body weights at 147 d (Table 5) were higher in all restriction treatments than in the controls but the difference reached significance ( $P < 0.05$ ) only in restriction treatment A. Food intake tended to be lower in all restriction treatments than in the controls but the difference reached significance only for restriction treatment B. Overall food efficiency during the refeeding period was higher in all restriction treatments than in the controls. The proportion of breast meat was significantly increased but the percentage of thigh meat was not significantly affected by the restriction regimens. Mortality was 11, 6, 12 and 10 birds (out of 36) in the control, and A, B and C treatments, respectively.

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TABLE 4

*The effect of food restriction of varied severity on female broiler performance (trial 2)<sup>1</sup>*

	Restriction				SE <sup>2</sup>
	Control	A	B	C	
Body weight (g)					
6 d	126	126	126	126	0.1
11 d	247 <sup>a</sup>	199 <sup>b</sup>	174 <sup>c</sup>	150 <sup>d</sup>	3.1
50 d	2025 <sup>a</sup>	2044 <sup>a</sup>	1945 <sup>b</sup>	1956 <sup>b</sup>	21.0
Food intake (g)					
6-11 d	180 <sup>a</sup>	128 <sup>b</sup>	98 <sup>c</sup>	69 <sup>d</sup>	1.2
11-50 d	3844 <sup>ab</sup>	3896 <sup>a</sup>	3716 <sup>b</sup>	3773 <sup>ab</sup>	41.4
6-50 d	4023 <sup>a</sup>	4024 <sup>a</sup>	3814 <sup>b</sup>	3846 <sup>b</sup>	41.2
Food conversion efficiency (g gain/g food intake)					
6-11 d	0.677 <sup>a</sup>	0.568 <sup>b</sup>	0.495 <sup>c</sup>	0.354 <sup>d</sup>	0.012
11-50 d	0.462 <sup>b</sup>	0.474 <sup>a</sup>	0.477 <sup>a</sup>	0.479 <sup>a</sup>	0.003
6-50 d	0.472	0.477	0.481	0.477	0.003
Abdominal fat (g/kg) <sup>3</sup>	32.3 <sup>a</sup>	26.2 <sup>b</sup>	26.3 <sup>b</sup>	27.2 <sup>b</sup>	1.2

<sup>1</sup> Each treatment included 4 groups of 10 birds each. Values within rows with no common superscript are significantly ( $P < 0.05$ ) different.<sup>2</sup> Calculated from the error term of the analysis of variance.<sup>3</sup> Average of 8 birds per treatment. Statistical analysis was on the arcsine-square-root transformed results.

TABLE 5

*The effect of food restriction of varied severity on male turkey performance (trial 3)<sup>1</sup>*

Variable	Restriction				SE <sup>2</sup>
	Control	A	B	C	
Body weight (g)					
7 d	147	147	147	147	0
17 d	383 <sup>a</sup>	286 <sup>b</sup>	251 <sup>c</sup>	227 <sup>d</sup>	4
147 d	14201 <sup>b</sup>	15170 <sup>a</sup>	14853 <sup>ab</sup>	14401 <sup>ab</sup>	288
Food intake (g)					
7-17 d	343 <sup>a</sup>	270 <sup>b</sup>	221 <sup>c</sup>	193 <sup>d</sup>	3
17-147 d	41637	39958	39217	39762	861
7-147 d	41980 <sup>a</sup>	40228 <sup>ab</sup>	39438 <sup>b</sup>	39955 <sup>ab</sup>	862
Food conversion efficiency (g gain/g food intake)					
7-17 d	0.690 <sup>a</sup>	0.514 <sup>b</sup>	0.468 <sup>b</sup>	0.414 <sup>c</sup>	0.015
17-147 d	0.330 <sup>b</sup>	0.372 <sup>a</sup>	0.373 <sup>a</sup>	0.356 <sup>a</sup>	0.007
7-147 d	0.333 <sup>b</sup>	0.373 <sup>a</sup>	0.373 <sup>a</sup>	0.356 <sup>a</sup>	0.007
Thigh meat (g/kg) <sup>3</sup>	196	195	192	202	...
Breast meat (g/kg) <sup>3</sup>	200 <sup>b</sup>	224 <sup>a</sup>	220 <sup>a</sup>	217 <sup>a</sup>	...

<sup>1</sup> Each treatment included 6 groups of 6 birds each. Values within rows with no common superscript are significantly ( $P < 0.05$ ) different.<sup>2</sup> Calculated from the error term of the analysis of variance.<sup>3</sup> Average of 6 birds per treatment. Statistical analysis was on the arcsine-square-root transformed results.

...al 4

Again, weight gain during the restriction period was somewhat lower than planned. Compensation for the weight gain lost during the restriction period had been completed by 112 d, even exceeding that of the controls (Table 6). Food intake was not significantly affected by treatment but food conversion efficiency during the refeeding period and for the entire experiment was significantly improved by food restriction. As in male turkeys, the proportion of breast meat was increased without any significant change in the percentage of thigh meat. Carcase fat was not affected by treatment. Mortality during the entire experiment was 3, 3, 4, and 3 (out of 36) birds of the control, A, B and C treatments, respectively.

TABLE 6

*The effect of food restriction of varied severity on female turkey performance (trial 4)<sup>1</sup>*

Variable	Control	Restriction			SE <sup>2</sup>
		A	B	C	
Body weight (g)					
7 d	153	153	153	153	0
14 d	314 <sup>a</sup>	240 <sup>b</sup>	219 <sup>c</sup>	199 <sup>d</sup>	3
112 d	8 045 <sup>b</sup>	8 389 <sup>ab</sup>	8 444 <sup>b</sup>	8 200 <sup>ab</sup>	118
Food intake (g)					
7-14 d	205 <sup>a</sup>	163 <sup>b</sup>	140 <sup>c</sup>	118 <sup>d</sup>	2
14-112 d	22 065	21 830	22 132	21 247	383
7-112 d	22 271	21 993	22 272	21 366	383
Food conversion efficiency (g gain/g food intake)					
7-14 d	0.788 <sup>a</sup>	0.538 <sup>a</sup>	0.470 <sup>b</sup>	0.391 <sup>c</sup>	0.011
14-112 d	0.348 <sup>b</sup>	0.368 <sup>a</sup>	0.371 <sup>a</sup>	0.376 <sup>a</sup>	0.005
7-112 d	0.352 <sup>b</sup>	0.370 <sup>a</sup>	0.372 <sup>a</sup>	0.376 <sup>a</sup>	0.004
Thigh meat (g/kg) <sup>3</sup>	192	195	192	202	...
Breast meat (g/kg) <sup>3</sup>	198 <sup>b</sup>	211 <sup>a</sup>	209 <sup>a</sup>	200 <sup>a</sup>	...
Carcase fat (g/kg) <sup>3</sup>	149	165	159	152	...

<sup>1</sup> Each treatment included 6 groups of 6 birds each. Values within rows with no common superscript are significantly ( $P < 0.05$ ) different.

<sup>2</sup> Calculated from the error term of the analysis of variance.

<sup>3</sup> Average of 6 birds per treatment. Statistical analysis was on the arcsine-square-root transformed results.

## DISCUSSION

The present results confirm previous observations (Plavnik and Hurwitz, 1985, 1988a; b; Plavnik *et al.*, 1986; McMurtry *et al.*, 1988) which suggested that early food restriction had advantages in improving food conversion efficiency in male broiler chickens and turkey poults and in reducing carcase fat in broilers. In male turkeys, accelerated growth following food restriction resulted in greater body weights, an effect which reached significance in the



## EARLY GROWTH RESTRICTION IN BIRDS

case of the milder restriction (treatment A, Table 5), confirming earlier suggestions (Plavnik and Hurwitz, 1990). Similar tendencies were observed in broilers, but did not reach significance.

In broiler chickens the growth and food conversion efficiency responses of females were not as pronounced as those of males, so that overall conversion efficiency hardly changed in response to food restriction, although there was complete compensation for the weight gain lost during restriction. In contrast, a significant improvement in food conversion efficiency attributable to food restriction was observed in female turkeys. It is possible that accelerated growth may be better expressed during the longer growing period of turkeys.

Broiler chickens responded to food restriction differently from turkeys, with regard to body composition. In chickens, regardless of sex, early food restrictions of varying severity resulted in a reduction of carcass fat at the marketing age of 7 to 8 weeks (Tables 3 and 4). The reduction in carcass fat was associated with a reduction in adipocyte number in a previous study (Cartwright *et al.*, 1986). In turkeys of either sex, carcass fat was not affected by food restriction. On the other hand, meat yield as a fraction of body weight was increased greatly in turkeys of either sex, but was not affected by food restriction in chickens, substantiating previous observations (Plavnik and Hurwitz, 1990). The differences in the response of either species to food restriction could be related to the difference in length of the growing period applied in the present studies; these were chosen to correspond with commercial practices. Alternatively, the differences may be related to differences in the growth patterns of the various tissues between species. Whereas carcass fat content was found to increase continuously with age in broiler chicks (Plavnik and Hurwitz, 1983), it remained rather uniformly low in turkeys up to 9 weeks of age, when it started to increase rapidly, although remaining lower than in broiler chickens (Hurwitz *et al.*, 1983). In turkeys, muscle growth relative to skeletal growth occurs mostly during the second half of the growing period. As shown previously (Plavnik and Hurwitz, 1990), this component of growth was stimulated by food restriction.

Abdominal fat, weighed at 56 and 50 d in male and female broilers, respectively, was markedly reduced by food restriction applied several weeks previously. The maximal response appears to have been reached with the mildest food restriction, which had been aimed at supporting 60% to 70% of normal growth rate. Thus, within the range tested, no quantitative relationship existed between the degree of early food restriction and deposition of carcass fat during refeeding. A threshold for this effect has not been detected in this study but may exist under milder restriction regimens.

In accord with the main purpose of this study, a consistent response was obtained to a wide range of restriction severity. The results show that the severe food restriction conditions applied previously (Plavnik and Hurwitz, 1985) were not essential for eliciting the desired responses of food conversion efficiency in both broiler chickens and turkeys or of carcass fat in broilers. A choice of the most economic regimen can be made using an algorithm (Talpoz *et al.*, 1988) based on the variables estimated in this study.

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## EDUCATION AND PRODUCTION

### Characterization of Growth and Development of Male British United Turkeys<sup>1</sup>

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**ABSTRACT** Body weight and the size of various organs (tibia, pectoral muscle, leg muscle, liver, spleen, and testes) were monitored in growing male turkeys (British United Turkeys) in an effort to characterize their growth pattern. The results were fitted with either a single- or a double-component Gompertz equation, describing single and diphasic growth patterns, respectively, using an iterative nonlinear estimation algorithm. The diphasic model with an early and a late growth component provided a better description of the body weight function than the single-phase model. The start of sexual maturation, evidenced by testicular development, marked the transition age between the early and the late phases of growth. A single-component Gompertz equation was sufficient to describe growth of various individual organs. Growth of bone and liver appeared to follow the early growth component, whereas the path of muscle development appeared to be closer to the later growth component. The results suggest that the diphasic growth behavior of male turkeys is caused by a differential growth rate of various organs, rather than by a periodicity in the overall growth rate. (Key words: turkey, growth models, organ weights, Gompertz equation, parameter estimation)

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#### INTRODUCTION

The process of growth involves a continuous accretion of body mass that is conveniently described by growth equations. Of the various growth models (Zeger and Harlow, 1987), the Gompertz (1825) equation was found appropriate to describe the growth path of chickens (Tzeng and Becker, 1981; Talpaz *et al.*, 1987, 1991). The classical Gompertz equation describes the growth curve by one double exponential term and thus assumes a single-phase behavior of growth along the entire path. As reviewed by von Bertalanffy (1957), the simple sigmoidal shape is most common in fish and in some mammals. However, in other mammals, such as the rat, the growth pattern is best described by two cycles, the second beginning at the time of sexual maturation. Moreover, Koops *et al.* (1987) found that growth in mice can be described by a triphasic function, each phase

given by a term that is a single logistic function. Preliminary examination of detailed growth curves of male turkeys suggested the possible presence of more than a single growth component, because attempts at fitting the single-phase Gompertz equation to turkey growth failed to satisfy the statistical requirements.

The multiphasic behavior of a growth path could be caused either by oscillatory behavior of growth of all organs, or by a combination of differential single-phase growth rates of organs of quantitative importance. These possibilities can be tested if growth of selected organs is monitored along with body weight during the growth period. In the present study, the growth pattern of male turkeys, including selected organs, has been characterized.

#### MATERIALS AND METHODS

##### Birds

Day-old male British United Turkey poults were obtained from a commercial hatchery and raised for 4 wk in battery brooders at a constant temperature of 24 C and then transferred to individual cages at 20 C. The birds were provided *ad libitum* access to diets designed to

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satisfy the recommendations of the National Research Council (1984), which were composed primarily of corn, sorghum grain, and soybean oil meal according to the schedule: 0 to 4 wk, 2,800 kcal ME, 28.2% protein; 4 to 8 wk, 2,945 kcal ME, 26.4% protein; 8 to 12 wk, 3,070 kcal ME, 22.2% protein; 12 to 16 wk, 3,160 kcal ME, 19.1% protein; 16 to 20 wk, 3,250 kcal ME, 16.9% protein; and 20 to 24 wk, 3,330 kcal ME, 14.2% protein.

One group of 24 birds was weighed individually at variable intervals up to 147 days of age. Another group of 90 birds was distributed at random into 15 replicates of 6 birds each. All birds in a replicate were killed by cervical dislocation, at the ages of 0, 7, 17, 21, 42, 60, 66, 73, 80, 87, 94, 101, 108, 115, and 136 days, respectively. The weights of tibia, liver, testes, spleen, and pectoral and leg muscles were determined immediately. Tibia length was measured at these times.

#### Numerical Analysis

**Model 1.** The body weight time path,  $W_t$ , under *ad libitum* feeding was found to be governed in chickens (Talpez *et al.*, 1988, 1991) by a single-phase Gompertz curve. This curve is given by

$$W_t = \omega t^{\beta e^{-\gamma t}} \quad [1]$$

where  $\omega$  is the maximal (asymptotic) body weight;  $\beta$  and  $\gamma$  are Gompertz rate constants describing the sigmoid growth curve;  $t$  is the age in days; and  $e$  is the base of the natural logarithm, i.e., 2.71828....

**Model 2.** This model describes the body weight time path,  $W_t$ , under *ad libitum* feeding as governed by a diphasic function of the double Gompertz curve. The function is

$$W_t = \omega_1 t^{\beta_1 e^{-\gamma_1 t}} + \omega_2 t^{\beta_2 e^{-\gamma_2 t}} \quad [2]$$

where,  $\omega_1$  and  $\omega_2$  are the maximal (asymptotic) weights of the two components of body weight (Component 1 and Component 2) as defined mathematically and later identified with specific organs;  $\beta_1$  and  $\beta_2$ , and  $\gamma_1$  and  $\gamma_2$  are the growth rate constants for the respective components; and  $t$  is the age in days.

**The Inflection Point.** Gompertz equations, single or double, were characterized by the age

at the inflection point  $t^*$  when growth rate is maximal. To do so, consider the equivalent form of the Gompertz Equation [1]:

$$W_t = \omega t^{\beta e^{-\gamma(t-t^*)}} \quad [3]$$

The identity of Equation [3] to Equation [1] can be established by substituting

$$\beta = e^{-\gamma t^*} \quad [4]$$

into Equation [1] and rearranging the terms.

The interpretation of  $t^*$  is of physiological importance, as this age is signified by maximal growth. The derivative of [3] is the daily body weight gain:

$$\dot{W} = \gamma e^{\gamma t^*} W_t e^{-\gamma t} \quad [5]$$

By definition, body weight gain is maximal at the inflection point. Setting the derivative of Equation [5] to equal 0 and solving for  $W_t$ , it is possible to obtain,  $W_{t^*} = \omega/e$ , which is the body weight at which the maximum weight gain is achieved. The maximum weight gain at that point is given by

$$\dot{W}_{\max} = \frac{\gamma \omega}{e}; \text{ at } t = t^* \quad [6]$$

From the Gompertz parameters (Table 1) the inflection points ( $t^*$ ) were calculated for each individual curve by substituting the estimated values of  $\beta$  and  $\gamma$  into Equation [5] and rearranging,

$$t^* = \frac{\text{LN}(\beta)}{\gamma} \quad [7]$$

where LN is the natural logarithm operator.

#### Parameter Estimation

A nonlinear least squares procedure was employed to estimate the parameters. The average values of the variables for each time point were used as "observations". The minimization algorithm utilizes the Murtagh and Saunders (1987) MINOS<sup>®</sup> code as described in Talpez *et al.* (1991). To overcome problems of heteroscedasticity, the squared errors to be minimized have been inversely weighted by the respective observations raised to the .75th

## GROWTH IN TURKEYS

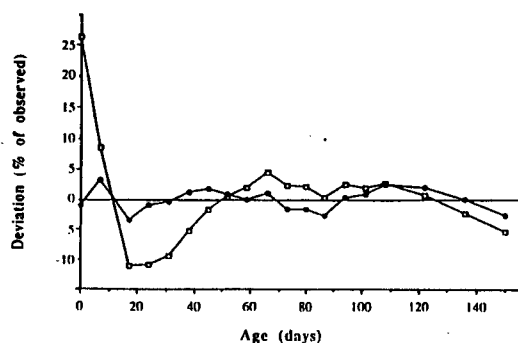


FIGURE 1. Deviations of observed from predicted values of a single (Model 1,  $\square$ ) or double (Model 2,  $\circ$ ) Gompertz model fitted to body weight of male British United turkeys.

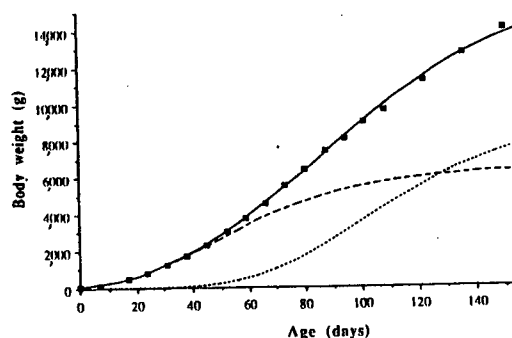


FIGURE 2. The fit of the double Gompertz model (Model 2, —) to body weights of British United turkeys ( $\square$ ), and the breakdown of the body weight path to its two Gompertz components: Component I --- and Component II ----.

power, yielding a weighted objective function (WSSE):

$$\text{Minimize WSSE} = \sum_{t=0}^{t_f} \left[ \frac{\hat{w}_t - w_t}{w_t^{.75}} \right]^2 \quad [8]$$

for all sampling times from 0 to  $t_f$  (final age). The estimation of the parameters' standard errors resulting from the deviations of the observed values from the respective function, was carried out using the nonlinear procedure presented previously by Talpaz *et al.* (1987). For each estimation, the measures of serial correlations (Kennedy and Gentle, 1980) were computed to establish the goodness of model specifications.

## RESULTS

Results of body weight were fitted by the single (Model 1) or the double (Model 2) Gompertz equation, according to Equation [1] and Equation [2], respectively. The deviations between the predicted and actual values are given in Figure 1. In most cases the deviations were far greater for Model 1 than for Model 2, especially up to 49 days of age. On the other hand, deviations resulting from Model 2 were not localized to any time point, and were in most cases lower than 2% of the observed values. Furthermore, the sum of squared error and the serial correlations (in absolute values) were considerably lower for Model 2 than for Model 1 (.0063 versus 2.4954 for the sum of squared error, and -.0236 versus -.217 for the serial correlation, respectively), suggesting that

TABLE 1. Estimated Gompertz parameters for male turkey growth<sup>1</sup>

Variable	Parameter			
	$\omega$	$\beta$	$\gamma$	$t^*$
Body weight, g, I	6,409 $\pm$ 2,698	4.70 $\pm$ .24	.0332 $\pm$ .0067	46.6
Body weight, g, II	9,609 $\pm$ 2,925	15.17 $\pm$ 3.01	.0271 $\pm$ .0018	100.3
Tibia weight, g	128.6 $\pm$ 1.7	5.36 $\pm$ .22	.0309 $\pm$ .0009	54.4
Tibia length, cm	25.2 $\pm$ .2	1.90 $\pm$ .02	.0267 $\pm$ .0004	24.0
Liver weight, g	122.7 $\pm$ 1.8	4.27 $\pm$ .22	.0344 $\pm$ .0013	42.2
Pectoral muscle, g	3,796 $\pm$ 58	6.53 $\pm$ .15	.0228 $\pm$ .0005	82.3
Leg muscle, g	3,704 $\pm$ 79	6.30 $\pm$ .19	.0220 $\pm$ .0006	83.7
Spleen, g	13.2 $\pm$ .6	5.00 $\pm$ .44	.0252 $\pm$ .0019	64.1

<sup>1</sup>Gompertz parameters  $\pm$  standard errors were estimated for the growth data given in Figures 1 to 5.  $\omega$  is the maximal size whereas  $\beta$  and  $\gamma$  are Gompertz exponents.  $t^*$  is the inflection point of the growth curve, representing the age (in days) of maximal rate of weight gain of the respective organ.

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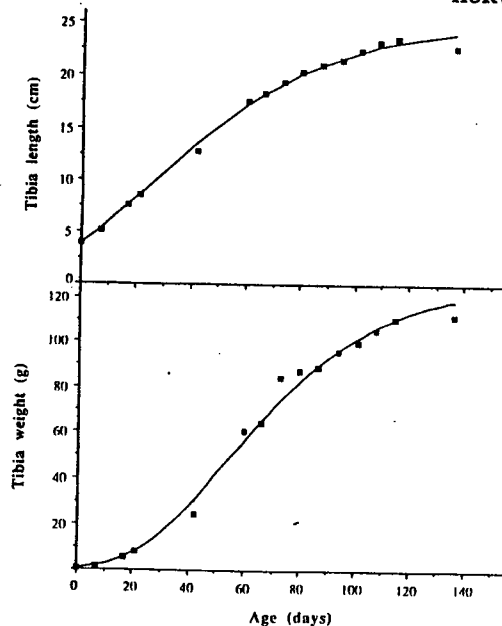


FIGURE 3. Tibia length and weight in male British United turkeys. Observed values (□). The solid lines represent the respective fitted single Gompertz Model 1.

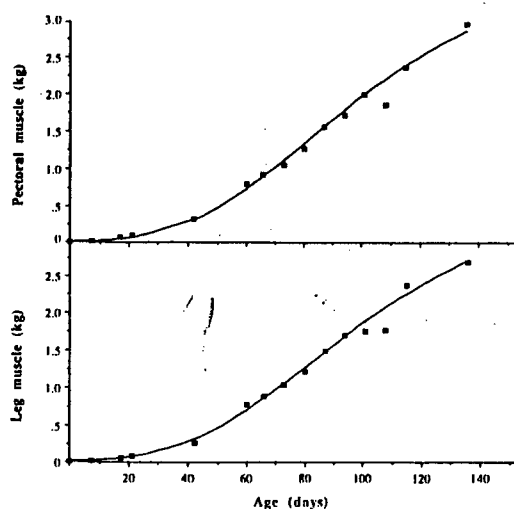


FIGURE 4. Development of pectoral and leg muscles in male British United turkeys. Observed values (□). The solid lines represent the respective fitted single Gompertz Model 1.

Model 2 fits the growth path much better than Model 1.

The results of body weight together with the computed double Gompertz growth functions are shown in Figure 2. The equation parameters are shown in Table 1. The decomposition of the growth curve into its two Gompertz components shows that Component 1 rises rapidly and approaches its maximal value between 80 and 100 days. Maximum gain occurred at approximately 47 days for the first, and 100 days for the second, slower growth component. The first Gompertz term can account for nearly the entire body weight during the growth period of up to 40 days of age. The second component becomes apparent only after 40 days of age, and does not approach any maximal value during an observation period of 147 days. Obviously, a longer growing period would be necessary to obtain a more accurate assessment of this maximal value. Although the maximal weight of the first component appeared lower than that of the second, the large standard errors suggest that this difference is not significant.

Model 2 was found inappropriate to describe the growth functions of individual organs. During estimation, some of the deter-

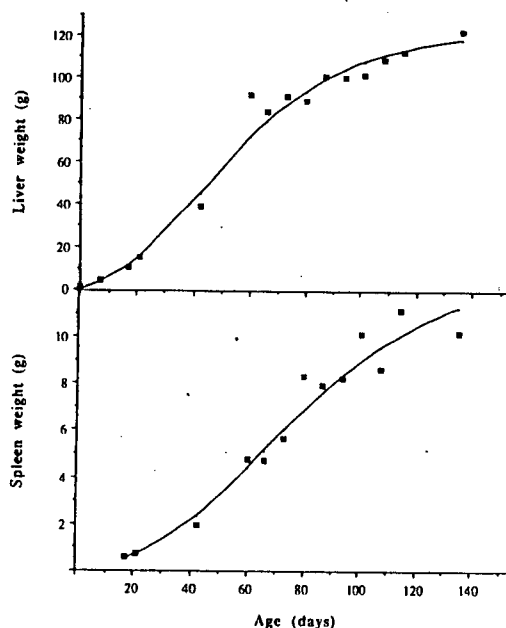


FIGURE 5. Liver and spleen growth in male British United turkeys. Observed values, (□). The solid lines represent the respective fitted single Gompertz Model 1.

## GROWTH IN TURKEYS

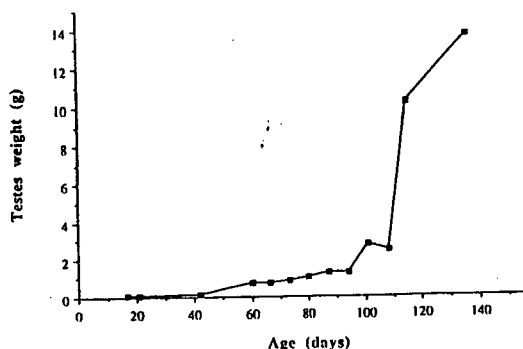


FIGURE 6. Testes growth in male British United turkeys. The solid line connects the experimental points given by the solid squares.

mined parameters were not statistically significant, and the high correlations between parameters prevented the arrival at a unique solution. On the other hand, a fit of Model 1 with its single Gompertz component to weights of various organs (excluding the testes), and to tibial length provided a good description of their temporal behavior (Figures 3 to 5). In most cases the standard errors of the estimated Gompertz parameters (Table 1) were lower than 3% of the estimated values. The Gompertz function fitted poorly to testes weight. With different weighting factors, an estimate of at least one of the three parameters of the Gompertz equation ( $W_t$ ,  $\beta$ ,  $\gamma$ ) was not significant by *t* test. Hence, testes weight is given in Figure 6 without any fitted function.

The growth pattern of bone length, liver weight, spleen weight, and pectoral and leg muscle weights is given in Figures 3 to 5. The shape of the functions suggests two main classes of organs. One, represented by tibia weight, tibia length, and liver weight, exhibits rapid initial growth, with a very short lower sigmoid part, and a leveling off at the age of 80 to 100 days. The Gompertz function describing their growth is characterized by a short inflection age  $t^*$ . The other class, represented by muscle tissue taken from two sites, exhibits slow initial development, a striking acceleration in development rate at the age of 40 to 60 days, with only a slight decrease in the rate towards the end of the observation period, and a long  $t^*$ . Spleen weight appears to fall between the two classes, as suggested by the intermediate  $t^*$ . Although results of testes development could not be

fitted with a Gompertz function, the shape of its path resembled that of Component 2, because significant development started only at 40 days of age, with a rapid acceleration at approximately 100 days of age (Figure 6).

## DISCUSSION

The partitioning of the growth curve of male turkeys into two Gompertz components improved the formal description of growth over the use of a single component, as indicated by several statistical criteria. The suggested diphasic behavior of turkey growth is similar to the pattern described for the rat (von Bertalanffy, 1957), but different than that for the broiler chicken, in which a Gompertz model with a single component provided a near perfect fit to body weights recorded during 64 days of life (Talpez *et al.*, 1991). Although it is possible that the 64-day period is somewhat short, in terms of maturity, the age of 70 to 84 days in the broiler appears to be equivalent to 140 to 147 days in the turkey, on the basis of testes development, plasma testosterone levels, and approach to maximal weights (Hurwitz, unpublished results). The difference in growth pattern between the two species appears to result from a relatively earlier muscle deposition in the broiler chicken than in the turkey. The growth behavior in the turkey is also different than that reported for the mouse, which was best described by a triphasic growth path (Koops *et al.*, 1987). The present model, however, uses Gompertz terms rather than the logistic equations employed by Koops *et al.* (1987) to describe the phasic growth behavior.

The large standard errors for the estimated parameters representing maximum size [ $\omega_1$  and  $\omega_2$  (Table 1)] limit the exact quantitative assignment of anatomical entities into each of the estimated formal components. Nevertheless, when the shape of the growth path, and especially the inflection point  $t^*$ , are examined, the growth path of various organs can be classified as following the kinetics of the first or the second component of the body weight path. Tibia weight, representing body frame, appears to follow the kinetics of the more rapid Component 1. Tibia length develops even more rapidly with an inflection age  $t^*$  of only 24 days. Muscle weight, the main contributor to overall body weight, acts more in accord with the slower Component 2, although the initial development appears to be somewhat more rapid than that predicted by Component 2.

The path of the slower body weight Component 2 appears to coincide with the acceleration in the development of the testes, in agreement with von Bertalanffy (1957), who found that the transition from one to the other growth cycle in the rat was associated with sexual maturation. Sexual maturation is accompanied by the closure of the epiphyseal growth plate and cessation of longitudinal bone growth, explaining the kinetics of tibia weight and especially tibia length. The rapid increase in muscle growth also occurs during the acceleration of testicular development, and the resulting increase in concentration of circulating testosterone (S. Hurwitz and M. Pines, unpublished observations). Thus, it may be suggested that the late increase in muscle growth results from the increase in circulating androgens. A large acceleration in carcass fat deposition starts at between 60 and 70 days of age (Hurwitz *et al.*, 1983), also following the kinetics of the late growth phase and providing additional evidence of the profound changes in development of the turkey at that age.

Liver development appears to follow the kinetic behavior of growth Component 2. This behavior could result from processes other than sexual maturation. It is tempting to speculate that because one of the main functions of the liver is to process metabolic fuel, its size could be proportional to energy intake, which is, to a large extent, proportional to body surface. The present results of organ development suggest that the observed diphasic behavior of growth in the male turkeys is caused by a differential growth rate of the various organs, rather than by periodicity in overall growth rate.

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## **Modeling of the Dynamics of Accelerated Growth Following Feed Restriction in Chicks\***

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### **ABSTRACT**

*A model for normal growth and accelerated growth resulting from an early-age feed restriction in chickens, has been constructed. The model describes normal growth by a Gompertz equation, whereas compensatory growth is the product of three terms: a time derivative of the Gompertz equation; a compensation factor which is a function of the severity of feed restriction and a time-dependent exponential term. The model was fitted to experimental results obtained in male and female broiler chicks, with the aid of an iterative algorithm, based on numerical integration of the time-function, in two steps.*

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*In Step I, the Gompertz parameters were estimated both analytically and numerically, using the performance of ad libitum-fed birds. In Step II, compensation parameters were obtained by analyzing data of the performance of chicks subjected to early-age feed restriction of variable severity. The standard errors for the estimated parameters were less than 3% of the values. The deviations of the predicted from the observed values did not exceed 4% and were, in most cases, less than 2% of the observed values. The usefulness of the presented model in simulation and economic optimization algorithms is discussed.*

## INTRODUCTION

The phenomenon of accelerated growth following an early period of restricted feeding was first reported by Osborne & Mendel (1915) and more recently reviewed by Wilson & Osborn (1960). In chicks, early-age feed restriction resulted in a reduced carcass fat and an improved feed efficiency (Plavnik & Hurwitz, 1985, 1988a, 1989; McMurtry *et al.*, 1988). Improved growth and feed efficiency, and increased muscle yield were also induced in turkeys by early-age feed restriction (Plavnik & Hurwitz, 1988b).

The importance of the description of growth in mathematical terms has long been recognized. Several types of models have been suggested: (a) the logistic (autocatalytic), (b) the Gompertz and (c) the Brody two-stage model (Zeger & Harlow, 1987). Of these, the Gompertz (1985) equation was found to be most suitable for describing growth in broiler chickens (Tzeng & Becker, 1981). This model has been tested previously (Talpaz *et al.*, 1986) and found useful in an algorithm aimed at an economic optimization of the growth trajectory (Talpaz *et al.*, 1988). In that model, which was based on experimental data obtained by Plavnik & Hurwitz (1985), it was assumed that accelerated growth following feed restriction at an early age, could not completely overcome the initial growth retardation. The model included a Compensation Factor which was based on the gap in body weight between the *ad libitum* and the early-age feed-restricted (restricted) chicks. However, a recent study by Plavnik & Hurwitz (1989) showed that in restricted chicks, body weights at the age of 7–8 weeks may, under certain conditions, exceed those of the *ad libitum*-fed ones. Therefore, the gap between the *ad libitum*-fed and the restricted birds cannot be taken as the driving force behind the phenomenon of growth acceleration; an improved model which is capable of accounting for an overshoot in body weight due to early feed restriction, was needed.

The objective of this study has been to remodel the accelerated growth in order to facilitate prediction scenarios of growth rates under different feeding regimes subject to the induced accelerated growth.

## THE MODEL

The body weight time path,  $W_t$ , under *ad libitum* feeding is governed by the Gompertz curve,

$$W_t = \omega e^{-\beta e^{-\gamma t}} \quad (1)$$

where  $\omega$  is the maximal body weight,  $\beta$  and  $\gamma$  are parameters;  $t$  is the age in days. These parameters can characterize the chicken's genotype (Talpaz *et al.*, 1988). The daily gain is given by the time derivative of eqn (1),

$$\dot{W}_t = \beta\gamma W_t e^{-\gamma t} \quad (2)$$

The interpretation of the parameters  $\beta$  and  $\gamma$  may be facilitated by the examination of eqn (2). For newborn chicks ( $t=0$ ) the product  $\beta\gamma$  is the fractional initial weight gain,  $\dot{W}_0 = \beta\gamma W_0$ .

Preliminary examination of the experimental results showed that:

- (a) The compensatory growth rates were not uniform among the groups which had been subjected to different degrees of feed restriction.
- (b) The accelerated growth rates appear to be driven by the degree of restrictions in a non-linear fashion. Up to a certain restriction level, the rate of accelerated growth increased, and beyond that point it decreased.
- (c) The magnitude of growth acceleration diminished with time.
- (d) The fed-restricted birds exhibited higher peaks of growth compared with the control *ad libitum*-fed birds.

Such a behavior may be represented by a general daily gain function,  $G_t$ ,

$$G_t = C(R_t)H(R_t, t)\dot{W}_T \quad (3)$$

where  $\dot{W}_T$  is the *normal* weight gain (the gain without any compensation) calculated by eqn (2) using  $T$ , the *physiological age*, substituting for  $t$ . The physiological age of the restricted chick is defined as the age of an *ad libitum*-fed chick when reaching the body weight identical to that of the restricted chick.  $T$  can be calculated from eqn (1). Let  $W_T$  be the weight of a previously feed-restricted chick of an age  $t$ , then,

$$T = -\log_e [-\log_e (W_T/\omega)/\beta]/\gamma \quad (4)$$

where  $\log_e$  is the log operator with base  $e$ —the *natural number*.  $\dot{W}_T$  is computed in eqn (2) by substituting  $T$  for  $t$ .

The compensation function,  $C(R_t)$ , is given by a variant of the Gamma density function,

$$C(R_t) = 1 + \frac{\sqrt{1-R_t}}{\lambda^x} \frac{e^{-R_t/\lambda}}{\alpha^2} R_t^{x-1} \quad (5)$$

where  $R_\tau$  is the ratio of the actual  $W_\tau$  and the potential weight (*ad libitum*)  $W_{p\tau}$ ,  $W_\tau/W_{p\tau}$ , at the end of the feed restriction period,  $\tau$ .  $\lambda$  and  $\alpha$  are the function parameters estimated as detailed below.

$H(R_\tau, t)$  is a time-dependent function given by,

$$H(R_\tau, t) = 1 + \eta \sqrt{1 - R_\tau} e^{-\zeta(t - \tau)} \quad (6)$$

with the amplitude  $\eta$  and dumping  $\zeta$  parameters to be estimated as detailed below.

### Parameter estimation

In order to calculate the expected body weight at time  $t$  for the *ad libitum*-fed chicks, one can either evaluate eqn (1) analytically, providing an exact solution, or by integrating eqn (2) numerically, yielding an approximate evaluation. An analytical solution is unattainable for the fed-restricted chicks. Moreover, the numerical solution is dependent on the method of numerical integration while the level of restriction is continuous, including the neighborhood of no restriction. Therefore, one should use numerical integration for either case (normal and restricted birds) in order to remain consistent. Since  $T$  is evaluated from eqn (1), the use of the parameters  $\beta$  and  $\gamma$  estimated by *numerical* integration is bound to bias the value of  $T$ . Therefore,  $\omega$ ,  $\beta$  and  $\gamma$  are estimated in two steps: first, eqn (1) is used directly for the purpose of obtaining an unbiased  $T$ , providing the estimates  $\omega_1$ ,  $\beta_1$  and  $\gamma_1$ ; second, eqns (2) through (6) are used via numerical integration *given* the estimation of eqn (1) in the first step, providing the estimates  $\omega_2$ ,  $\beta_2$  and  $\gamma_2$ .

A non-linear least square procedure is employed to estimate the parameters. The minimization algorithm utilizes Murtagh & Saunders' MINOS (1987) code. Equation (2) is repeatedly evaluated using a numerical rectangular integration process. To overcome problems of heteroscedacity, the squared errors to be minimized have been inversely weighted by the squared root of the respective observed values, yielding a weighted objective function (WSSE):

$$\text{Minimize WSSE} = \sum_{j=A}^D \sum_{t=\tau}^{t_f} \left[ \frac{\hat{W}_{jt} - W_{jt}}{\sqrt{W_{jt}}} \right]^2 \quad (7)$$

for all restricted feeding groups  $j = A, B, C, D$  and at all sampling times from  $\tau$  to  $t_f$ . The estimation of the parameters' standard error was carried out using the non-linear procedure presented previously by Talpaz *et al.* (1987).

## EXPERIMENTAL

The detailed experimental procedure as well as a more comprehensive report of the results is given elsewhere (Plavnik & Hurwitz, in press). Briefly, 7-day-old male or 5-day-old female Cobb broiler chicks were assigned to the experimental groups, equalizing both mean and variance of body weight within the groups. Each treatment was applied to four replicate groups of 10 birds each. The male and female chicks were subjected to feed restriction for 7 and 5 days, respectively. During the restriction period, the birds were given a daily allowance of energy ( $EI$ , in  $\text{kcal d}^{-1}$ ), calculated to maintain body weight and support a selected rate of growth, according to the equation modified from Hurwitz *et al.* (1980):

$$EI = MW_i^{2/3} + DG \quad (8)$$

where  $W_i$  (in g) is the body weight at the beginning of the period;  $G$  (in  $\text{g d}^{-1}$ ) is the daily body weight gain;  $M$  (in  $\text{kcal g}^{-2/3}$ ) is the coefficient of maintenance in terms of energy requirements;  $D$  (in  $\text{kcal g}^{-1}$ ) is the energy requirement for growth. On the basis of previous studies (Plavnik & Hurwitz, 1985, 1988a)  $M$  was varied between 2.0 and 1.0, proportional to the degree of restriction. Following the restriction period, birds were fed *ad libitum* diets appropriate for their species and age. Body weights were recorded individually at the end of the restriction period and at weekly intervals thereafter.

## RESULTS

The body weights of male and female broilers, following the early-age feed restriction, and at the end of the trials are given in Table 1. A full account of the experiments is given elsewhere (Plavnik & Hurwitz, in press). A full compensation for the weight retardation at the end of 14 days was accomplished in the restriction treatments A and B in males where in effect body weight slightly exceeded that of the non-restricted controls. Fifty-six-day-old body weight remained lower than that of the controls in the more severe restriction treatments C and D.

In females, only the mildest restriction (treatment A) was followed by a full compensation. Body weight of the more severe restriction groups remained lower than in the controls.

Body weights of the *ad libitum*-fed chicks together with the fitted Gompertz function are shown in Fig. 1. The Gompertz equation provides a near perfect description of the growth behavior which is also evident from the relatively low values of the parameter's standard errors given in Table 2. As expected, male chickens gained more weight than females.

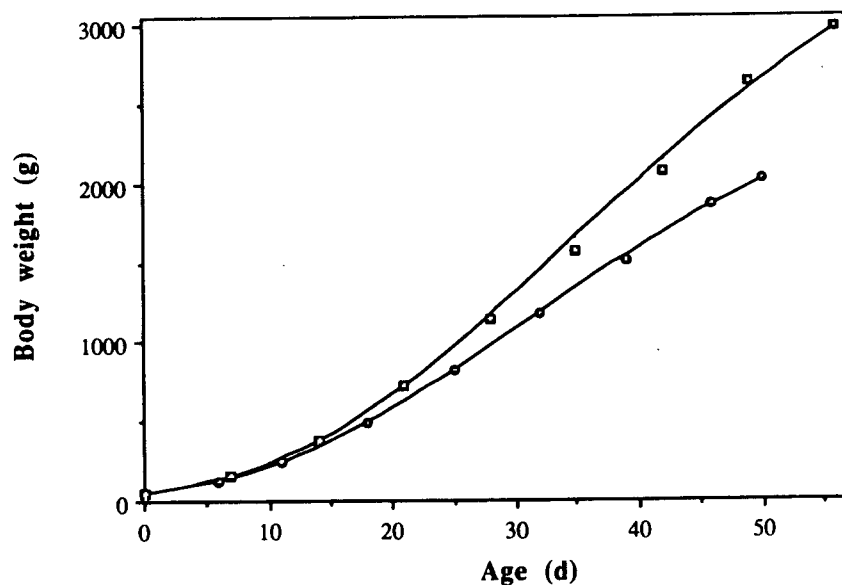
**TABLE 1**  
Average Body Weights (g) of Broiler Chickens After Feed Restriction  
(14 d) and at Marketing Age<sup>a</sup>

	Ad libitum	Restriction treatment			
		A	B	C	D
Males					
14 d <sup>b</sup>	385	285	246	203	169
56 d	2974	3046	2983	2879	2798
Females					
11 d <sup>b</sup>	247	199	174	150	—
50 d	2025	2044	1945	1956	—

<sup>a</sup> Detailed results are given elsewhere (Plavnik & Hurwitz, in press).

<sup>b</sup> Age at the end of feed restriction applied for 7 and 5 days in male and female broiler chicks, respectively.

The time trajectory of body weights of the restricted male and female chickens after the end of the restriction, together with their respective predicted functions, are given in Figs 2 and 3, respectively. An excellent fit of the growth pattern predicted by the Gompertz parameters (of the *ad libitum*-fed birds) and modified by the compensation coefficients, is apparent. The



**Fig. 1.** Body weight (observed) of male (□) and female (○) broiler chickens. The solid lines represent the Gompertz functions fitted by the numerical integration procedure.

## Dynamics of growth in chicks following feed restriction

TABLE 2

Estimated Parameters  $\pm$  Their Corresponding Standard Errors of the Growth and Compensation Equations for Broiler Chickens by Sex Under *Ad libitum* Feeding and After Early-Life Feed Restriction

Parameter	Equation	Sex	
		Males	Females
$\omega_1$	Gompertz, analytical	$4\,563 \pm 1\,011$	$3\,003 \pm 1\,0$
$\beta_1$	Gompertz, analytical	$4\,533 \pm 0\,000\,8$	$4\,305 \pm 0\,000\,8$
$\gamma_1$	Gompertz, analytical	$0\,042\,2 \pm 0\,6 \cdot 10^{-5}$	$0\,049\,0 \pm 0\,7 \cdot 10^{-9}$
$\omega_2$	Gompertz, numerical	$4\,843 \pm 821$	$3\,278 \pm 26$
$\beta_2$	Gompertz, numerical	$4\,656 \pm 0\,095$	$4\,223 \pm 0\,032$
$\gamma_2$	Gompertz, numerical	$0\,045\,9 \pm 0\,009\,5$	$0\,047\,1 \pm 0\,000\,6$
$\alpha$	Compensation	$3\,065 \pm 0\,109$	$5\,0 \pm 0\,860$
$\lambda$	Compensation	$0\,435 \pm 0\,015$	$0\,645 \pm 0\,032$
$\eta$	Compensation	$1\,287 \pm 0\,189$	$0\,989 \pm 0\,475$
$\zeta$	Compensation	1.0	$0\,367 \pm 0\,164$

deviations of the predicted from the expected values were between 0.5% and 4.4%, and 0.1% and 2.2%, for the males and females, respectively.

The growth and compensation parameters, calculated for either male or female broiler chickens, are given in Table 2. The calculated  $\omega$  (maximal weight) was higher for males than females and the product of  $\beta$  and  $\gamma$ ,

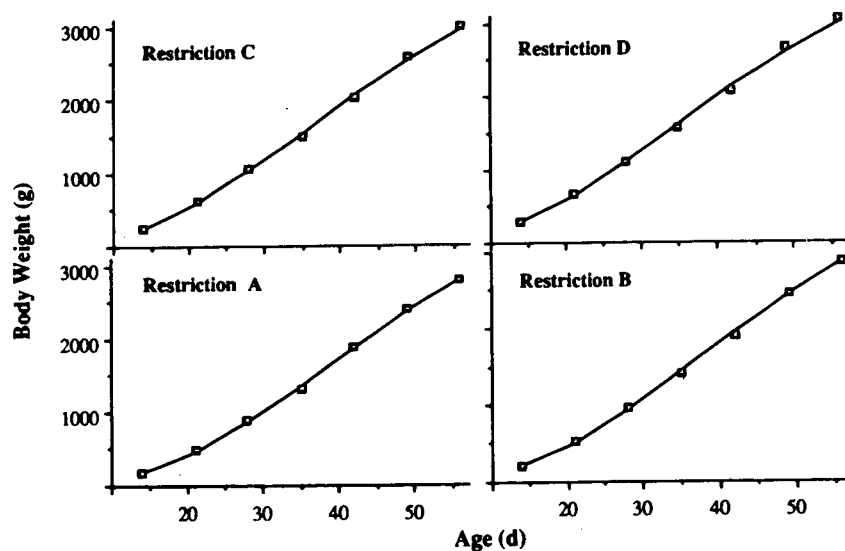
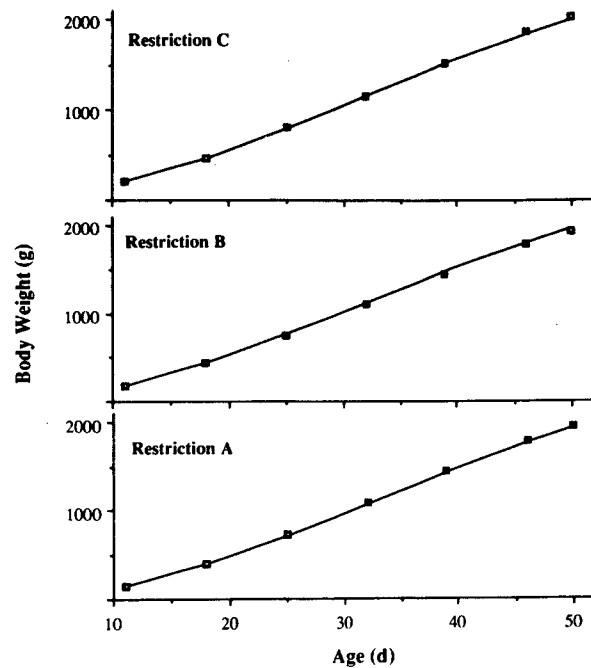
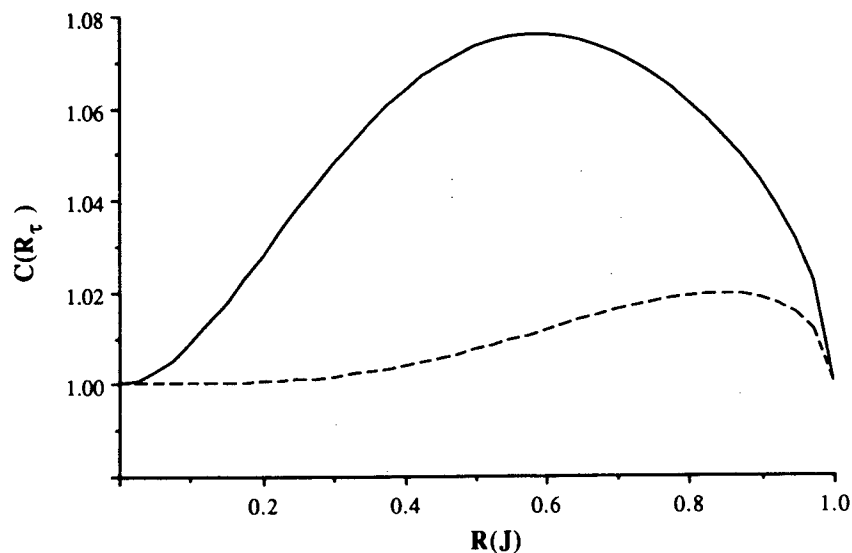


Fig. 2. Body weights of feed-restricted male chickens. A, B, C, and D represent restriction regimes of variable severity. For extent of restriction see Table 1. Symbols represent the actual observations and lines represent the model-calculated functions.



**Fig. 3.** Body weights of feed-restricted female chickens. A, B, and C represent restriction regimes of variable severity. For extent of restriction see Table 1. Symbols represent the actual observations and lines represent the model-calculated functions.



**Fig. 4.** The growth compensation factor as a function of the ratio of the restricted body weight to that of the *ad libitum*-fed male (solid line) and female (broken line) chickens, at the end of feed restriction.



representing the initial rate of weight gain, was also higher in males than in females. The compensation factors  $\alpha$  and  $\eta$  were lower in males than females whereas the reverse is true with regard to dumping parameter  $\zeta$ .

The computed compensation function [ $C(R_t)$ ] as a function of the ratio of body weight following restriction to normal body weight at that time ( $R_t$ ) is shown in Fig. 4. It exhibited a skewed distribution, with maximal compensation at an  $R_t$  of approximately 0.6 and 0.8 in males and females, respectively. Furthermore, the higher coefficient  $\eta$  in females than in males, is the result of the higher early compensation response and its faster rate of decay for this sex.

## DISCUSSION

Some techniques for estimation of the parameters of systems of non-linear equations have been described. For example, the procedure SYSNLIN by SAS (Sall, 1982) is, in general, capable of such estimations. However, that procedure failed to perform satisfactorily in our hands, since its minimization of the sum of squared error is based on the *analytical* derivation of the gradient with respect to the vector of parameters. Whereas the SAS procedure is efficient in general, it is handicapped by problems of rounding errors due to the application of the chain rule which is required for the analytical integration. In contrast, the present procedure avoids this problem by calculating the gradient via *numerical* finite difference approximation. In a way, it is a generalization of the estimation procedure employed by Talpaz *et al.* (1987) in the parameter estimation of their simulation model.

In agreement with Tzeng & Becker (1981), the Gompertz equation provides a good description of the growth pattern of male and female broilers, since the small deviations between the predicted curves and the actual body weight as functions of age, are symmetrically distributed. Furthermore, the maximal body weights ( $\omega$ ) predicted by the equations derived from results for ages of less than 8 weeks, are in close agreement with the adult size typical for the strain used in this study, which are usually reached at the age of about 20 weeks. As expected, male and female broiler chickens exhibited different growth paths (Fig. 1). Among the estimated Gompertz parameters, maximal body weight,  $W_F$ , was considerably different in males than females, whereas the two other parameters,  $\beta$  and  $\gamma$ , were similar.

Variable degrees of growth acceleration were exhibited by birds of either sex after being subjected to a wide range of feed restriction severity. The present model includes two parameters which describe the factor for growth

acceleration, and two which determine the time-dependent component of the acceleration. These were sufficient to describe the growth curves following feed restriction of variable severity. As suggested by the behavior of the compensation factor,  $C(R_t)$  (Fig. 4), males were more responsive than females to feed restriction. The time dependent component was also higher in males than in females soon after feed restriction but it declined faster with time, as indicated by the greater  $\zeta$ . The greater compensatory responsiveness of males than of females is in agreement with previous observations (Plavnik & Hurwitz, 1988a).

In contrast to the previous model (Talpaz *et al.*, 1988), the present one can accommodate an overshoot in body weight induced by feed restriction. The excellent fit of the model to the experimental results provides the basis for its use together with the determined parameters in an algorithm aimed at the economic optimization of the growth trajectory in broilers (Talpaz *et al.*, 1988), in which the accuracy of simulation of the biological response is crucial.

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*Dynamics of growth in chicks following feed restriction*

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**Prediction of the Response of Chicks to Early-Age Feed  
Restriction: Model Validation**

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Running head: VALIDATION OF RESTRICTION-COMPENSATION MODEL

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**Abstract** The simulation model (Talpaz *et al.*, 1991) which formalized accelerated growth following early-age feed restriction and included a formal description of feed intake and efficiency, was validated in four separate trials with broilers. All trials included *ad libitum* -fed and feed-restricted birds. In the latter, feed restriction was applied for 6 to 21 days starting at the age of 5 to 7 days. The first trial, conducted with Cobb males, confirmed a two-phase growth response to feed restriction: an early rapid phase, including only the first day of refeeding, and a slower phase occurring for several weeks after restriction. In three trials in which severity and duration of restriction were varied and three broiler strains were tested, growth acceleration resulted in narrowing or closure of the gap in body weight resulting from early-age feed restriction, at 7 to 8 weeks of age. The body weight values predicted by the model formulating this concept deviated by  $-.43 \pm 2.42\%$  from actual observations. Similarly, the predicted feed intake and feed efficiency values deviated by  $.95 \pm 3.07\%$  and  $-.55 \pm 2.88\%$ , respectively. In all trials, feed efficiency was improved by early age feed restriction. It is proposed that the simulation model be used to economically optimize the growth trajectory in broilers. (Keywords: growth, simulation, energy needs, feed efficiency, broiler strains)

## INTRODUCTION

In previous studies (Plavnik and Hurwitz, 1985, 1988a,b, 1990, 1991; Plavnik *et al.*, 1986) the accelerated growth response to feed restriction (Wilson and Osborn, 1960) and the resulting improvement in feed efficiency and reduced carcass fat were evaluated. Recent studies (Plavnik and Hurwitz, 1991) showed that the magnitude of response to feed restriction was a function of both length and severity of restriction. The selection of the most economic method of feed restriction is dependent on market conditions, as demonstrated by an algorithm developed to optimize the growth trajectory (Talpaz *et al.*, 1988). The optimization algorithm was refined by inclusion of a model which assumed a two-phase compensatory response to feed restriction (Talpaz *et al.*, 1991). The appropriate model-parameters were estimated using experimental results of a 6-day restriction experiment conducted with Cobb broiler chicks.

In the present study, the assumption of a two-phase response to feed restriction was tested. The model (Talpaz *et al.*, 1991) and parameters included were validated by comparing the calculated predictions with experimental results obtained for different broiler strains, and for restriction conditions different from those used in parameter estimation.

## MATERIALS AND METHODS

### *The simulation model*

For simulations applied in trials 2-4, body weights of the control groups were fitted with a Gompertz equation as described by Talpaz *et al.* (1991):

$$W_t = \omega e^{-\beta} e^{\gamma t} \quad [1]$$

where  $\omega$  is the maximal body weight,  $\beta$  and  $\gamma$  are Gompertz exponents,  $t$  is age

(days). The daily gain is given by the time derivative of [1],

$$\dot{W}_t = \beta \gamma W_t e^{-\gamma t} \quad [2]$$

To describe the change in body weight of the restricted birds during refeeding, the 'developmental age' (T) of the restricted birds is defined as the age of the *ad libitum*-fed birds when reaching body weight equal to that of the restricted group ( $W_T$ ):

$$T = -\log_e[-\log_e(W_T/\omega)/\beta]/\gamma \quad [3]$$

The daily weight gain during refeeding ( $G_t$ ) is calculated by multiplying the the equivalent daily weight gain of the *ad libitum*-fed bird ( $\dot{W}_T$ ) for  $t=T$  in [2] ( $\dot{W}_T$ ) by the compensation factors:

$$G_t = C(R_\tau) H(R_\tau, t) \dot{W}_T \quad [4]$$

where  $C(R_\tau)$  is the time-independent and  $H(R_\tau, t)$  is the time-dependent compensation factor, both negatively dependent on  $R_\tau$  - the ratio of body weight of the restricted to that of the *ad libitum* -fed birds at the end of restriction ( $t=\tau$ ):

$$H(R_\tau, t) = 1 + \eta \sqrt{1 - R_\tau} e^{-\xi(t-\tau)} \quad [5]$$

and,

$$C(R_\tau, t) = 1 + \frac{\sqrt{1 - R_\tau}}{\lambda^\alpha} \frac{e^{-R_\tau/\lambda}}{\alpha^2} R_\tau^{\alpha-1} \quad [6]$$

where  $\eta$ ,  $\xi$ ,  $\alpha$ , and  $\lambda$  are model parameters equal to 1.827, 1.0, 3.065, and 0.435, respectively. From [5] it is evident that the term  $H(R_\tau, t)$  is also a function of time, as shown previously (Talpez *et al.*, 1991).

The actual body weight of the restricted birds was obtained by numerical integration of the  $G_t$ 's. The mathematical representation of the compensation factors and the related parameters were detailed previously (Talpez *et al.*, 1991).

The energy intake,  $EI_t$  (in kcal/d), was calculated using an equation modified from Hurwitz *et al.* (1980):

$$EI_t = M_t \cdot W^{2/3} + D_t \cdot G_t \quad [7]$$

where  $W$  is the body weight (in g) and  $G_t$  is the body weight gain (in g/d). The maintenance requirement was given by Hurwitz *et al.* (1991):

$$M_t = M_{\max} (1 - e^{\delta t^{1.25}}) \quad [8]$$

where  $t$  is age (d); maximum coefficient,  $M_{\max}$  is 2.275 and the exponent  $\delta = .281$ .

The coefficient of growth requirement  $D_t$  is given by:

$$D_t = 0.5 + 9.2 [F_t] \quad [9]$$

where  $[F_t]$  is the fraction of fat in the weight gained. Feed intake ( $FI_t$ , g/d) is then calculated by:

$$FI_t = EI_t / [E] \quad [10]$$

where  $[E]$  is the energy concentration of the diet (kcal/g).

## Birds

Day-old male broiler chicks (Cobb, Trials 1-4; Anak and LaBelle, Trial 4) were obtained from commercial hatcheries and raised for 4 weeks in battery brooders in a constant-temperature room at 24 C. Subsequently, they were transferred to individual cages situated in a temperature-controlled (20 C) room with continuous artificial illumination.

At the start of the experiments, birds with extreme weights were eliminated, and the remaining ones were assigned to the experimental groups, equalizing both mean and variance of body weight within the groups. Each treatment was applied to four replicate groups of ten chicks, except for Trial 1, where only one group was subjected to each of the restriction treatments.

During the growth restriction period, the birds were given a daily allowance of energy, calculated (Eq. 6) to maintain body weight and support a rate of growth, which was varied according to treatment. Since energy needs decrease during restriction (Plavnik and Hurwitz, 1985),  $M$  was varied between 2 and 1 kcal/ g  $W^{2/3}$ , proportional to the degree of restriction. The value for  $D$ , the coefficient for



weight gain (G, in g/d), was varied with the degree of restriction, due to the expected graded response of carcass fat during the restriction period. Daily energy allowances were calculated for the period of restriction. However, an average allowance was fed daily throughout the period. Following the restriction period, birds were fed *ad libitum* diets appropriate for their age.

### *Diets*

The diets for the various age periods (Table 1) were designed to satisfy the recommendations of the National Research Council (1984). The MINOS® (Murtagh and Saunders, 1983) program was used in the computer calculations of the diets. Calculated protein content was confirmed by Kjeldahl analysis.

### *Trial 1*

The purpose of this trial was to test the assumption of a two-stage growth response during refeeding. Birds were restricted to 50% of the expected *ad libitum* weight gain for 4 or 6 days, starting at 5 days of age. These groups were individually weighed daily up to the age of 21 days. The control birds were weighed at the age of 5, 9, 11, 14 and 21 days. All birds were then kept up to 49 days of age and weighed at weekly intervals.

### *Trial 2*

In this validation experiment, the applicability of the model and its parameters was tested in broilers subjected to feed restriction to a weight gain of 50% of the *ad libitum*-fed control during 7, 14, and 21 days followed by *ad libitum* feeding. The experiment was started with 7-day-old male chicks. Individual body weights and

TABLE 1. *Composition of the experimental diets*<sup>1</sup>

Diet No.	1	2	3
<u>Ingredient, g/kg</u>			
Corn	350	500	500
Sorghum	203	103	170
Soybean meal <sup>2</sup>	322	265	221
Fish meal	50	50	40
Soy soapstock	34	50	40
Dicalcium phosphate	16.0	21.2	18.5
Ground limestone	7.1	5.	5
Sodium chloride	2.5	2.5	2.5
DL-Methionine	1.8	.5	-
Vitamin mix <sup>3</sup>	2.5	2.5	2.5
Mineral mix <sup>3</sup>	1.0	1.0	1.0
<u>Calculated Composition</u>			
Metabolizable Energy,			
kcal/kg	2920	3200	3200
Protein, %	22.0	20.1	18.1
Sulfur amino acids, %	.93	.74	.63
Lysine, %	1.32	1.16	1.00

<sup>1</sup> Diets 1, 2 and 3 were fed to 0 - 3, 3 - 6 and 6 - 8-week-old chicks, respectively.

<sup>2</sup> Containing 44.5% protein.

<sup>3</sup> The vitamin mixture supplied (per kg feed): retinyl acetate, 2.7 mg; cholecalciferol, 42.5 mg; dl-a-tocopheryl acetate, 10 mg; menadione sodium bisulfite, 2 mg; riboflavin, 5 mg; Ca -pantothenate, 10 mg; niacin, 20 mg; cyanocobalamin, 0.01 mg; folacin, 0.5 mg; pyridoxine, 1.5 mg; biotin, 0.1 mg; choline chloride, 200 mg; ethoxyquin, 125 mg; bacitracin, 10 mg. The mineral mix supplied (in mg/kg feed): Manganese, 80; zinc, 50; iron, 25; copper, 2; iodine, 1.2; cobalt, 0.2; selenium, 0.1.

1 group feed intake on a group basis were recorded at weekly intervals.

2  
3 *Trial 3*

4  
5 The purpose of this trial was to test the model in chicks in which both severity  
6 and duration of restriction were varied. The experiment was started with 6-day-old  
7 chicks and included an *ad libitum*-fed control, and two levels of restriction to 75%  
8 and 50% of the *ad libitum* weight gain, respectively, each applied for 1 or 2 weeks.  
9 The trial was terminated at the age of 54 days.

10  
11 *Trial 4*

12  
13 The purpose of this trial was to test the model in three strains of broiler chicks:  
14 two fast-growing - Cobb and Anak, and a relatively slow-growing strain - LaBelle.  
15 For each strain the experiment included *ad libitum*-fed controls and others feed-  
16 restricted to 75% of the expected weight gain of the respective control. Feed  
17 restriction was applied for 6 days, starting at 6 days of age. The experiment was  
18 terminated at the age of 52 days.

19  
20 *Statistical analyses*

21  
22 The results were subjected to a one-way analysis of variance (Snedecor and  
23 Cochran, 1967) and multiple comparison tests (Duncan, 1955).

## RESULTS

### *Trial 1*

Following a 50% growth retardation (Fig. 1), weight gain during the first day of refeeding (day 10 and day 12 in the 4-day and 6-day restriction groups, respectively) was remarkably accelerated. On the other hand, the early compensation term  $H(R_\tau, t)$ , calculated by the model for the two restriction groups of this experiment (Fig. 2), remained significant until the end of the second day of refeeding rather than being limited to the first day only. Subsequently during refeeding, a slower growth acceleration component with a slower expression resulted in closing the gap in body weight by approximately 20 days and 49 days in the 4-day and 6-day restriction groups, respectively.

On the basis of the results of this trial, an alternative simplified compensation term was examined in which the right-hand exponent of equation 5 was dropped:

$$H(R_\tau, t) = 1 + \eta \sqrt{1 - R_\tau} \quad [11]$$

$$\text{for } t = \tau + 1 \text{ and,}$$

$$H(R_\tau, t) = 1 \text{ for } t > \tau + 1$$

Applying this model to the experimental results previously used for parameter estimation (Talpez *et al.*, 1991), hardly affected the model fit, and was therefore adopted for further use. The estimated new value for  $\eta$  was 1.0785.

### *Trial 2*

In this trial the predicted response to restriction of different durations, ranging from 1 to 3 weeks, was compared with model predictions. Gompertz parameters for

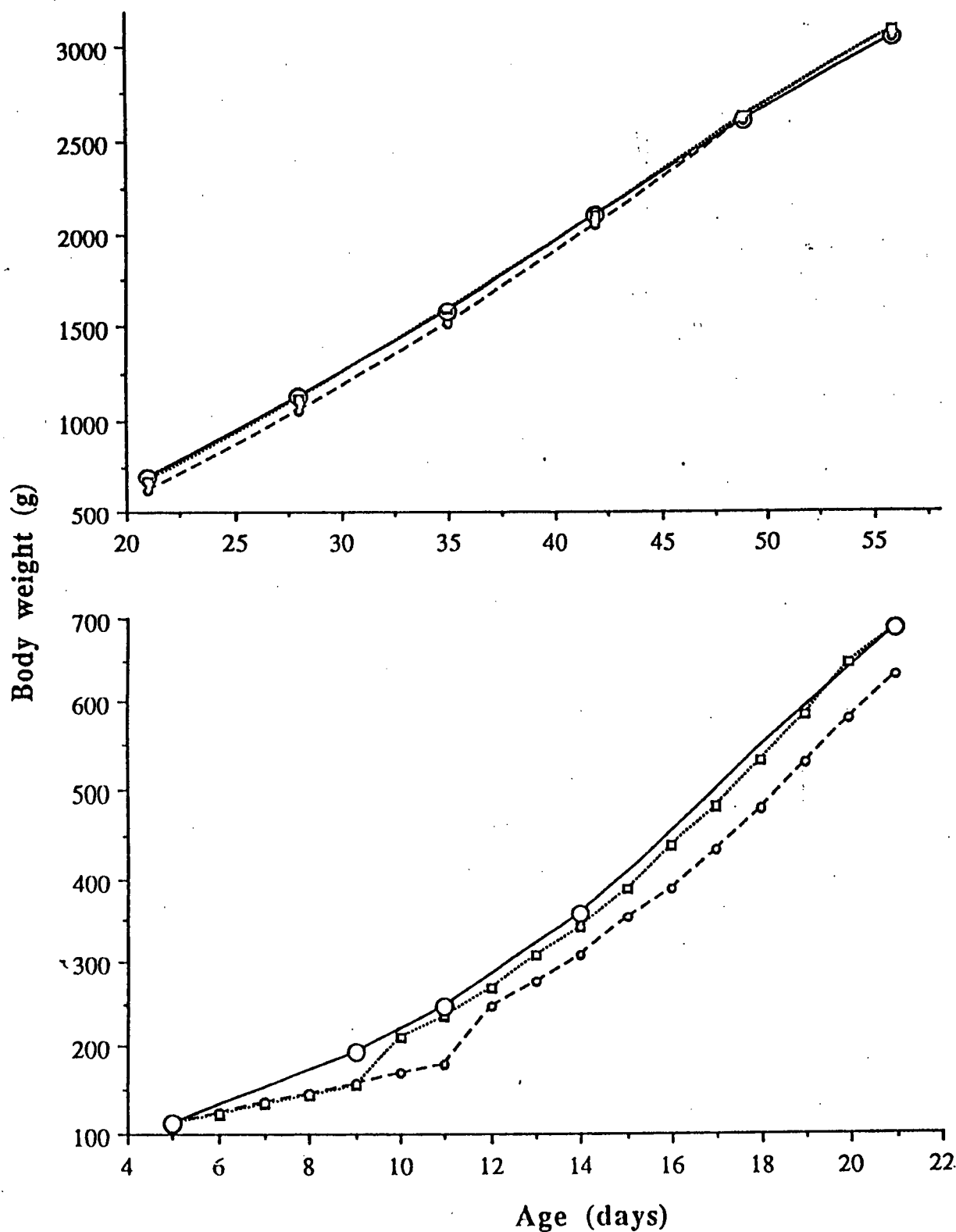


FIGURE 1. Detailed body weight response of broiler chicks to 4-day ( $\cdots\Box\cdots$ ) and 6-day ( $---\bigcirc---$ ) feed restriction periods starting at 5 days of age, compared with *ad libitum*-fed controls ( $—\bigcirc—$ ). For clarity the results are presented separately for the first 21 days (lower graph) and for 22-54 days (upper graph) of age (Trial 1).

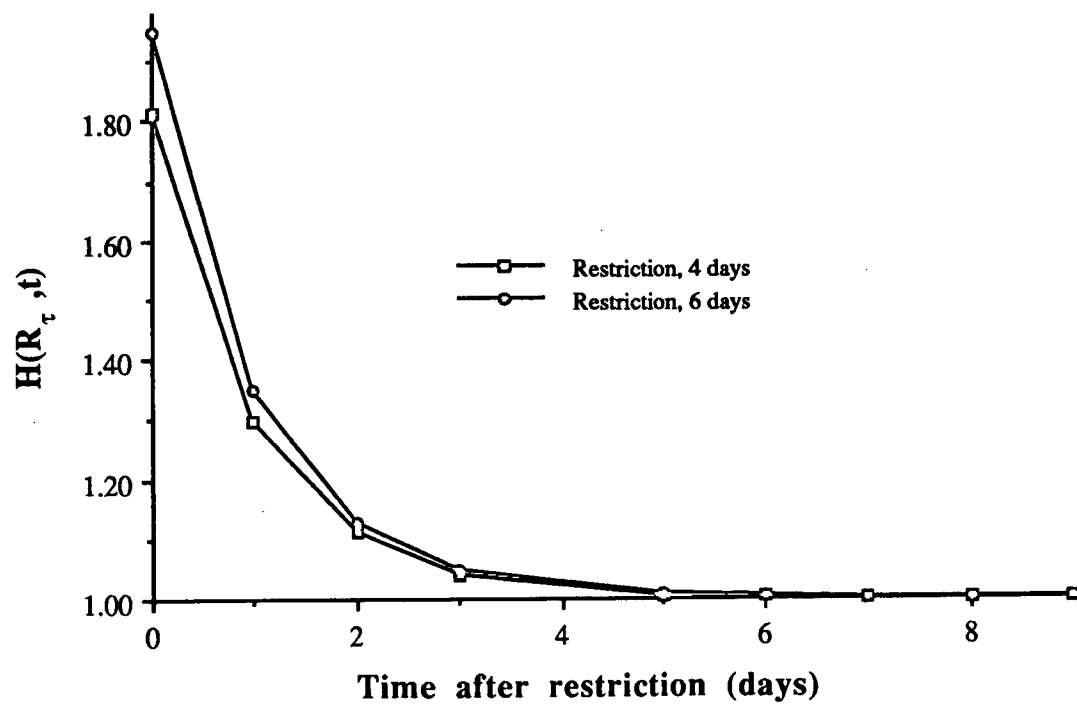


FIGURE 2. The early growth compensation response component calculated by the model (Trial 1).

calculation of body weight, estimated for the *ad libitum*-fed controls, are given in Table 2. As shown in Fig. 3, the model provided excellent estimates of body weights for the controls as well as for the 7- and 14-day restriction treatments. In the case of the 21-day restriction treatment, the model estimates were somewhat lower than those observed during the refeeding period, but the differences were less than 5% of the observed values (Table 3). Deviations in predicted feed intake and feed efficiency were less than 5% in all cases. Feed intake was lower and feed efficiency was higher in the restricted compared with the *ad libitum* - fed group. The hierarchies among the treatments were similar for the actual and predicted values for all three variables.

### *Trial 3*

In this trial, both length and severity of restriction were varied in a factorial fashion. For comparison, the results of the *ad libitum* controls were duplicated in the two parts of Fig. 3. Again the calculated Gompertz parameters (Table 2) provided good fit of the function (Eq. 1) for the control growth (Fig 3). The model also provided an excellent prediction of the temporal behavior of body weight in all four restriction treatment groups. As shown in Table 4, deviations of final body weights were less than 5% of the observed values. Deviations in predicted feed intake and efficiency values from the observed results were even less than 4%. Again the hierarchy of the predicted values was similar to that of the observed values within any one of the variables. Early age feed restriction always resulted in an improvement of feed efficiency.

### *Trial 4*

Among the three strains tested in this experiment, the slower growth rate of the

TABLE 2. *Gompertz parameters for body weight calculation, estimated for the ad libitum-fed controls in three separate trials.*

	$\omega$	$\beta$	$\gamma$
<u>Analytical integration</u>			
Trial 2, Cobb	4,346 $\pm$ 59.2	4.572 $\pm$ .047	.04421 $\pm$ .00070
Trial 3, Cobb	3,697 $\pm$ 61.3	4.498 $\pm$ .056	.04556 $\pm$ .00088
Trial 4, Cobb	4,949 $\pm$ 124.2	4.685 $\pm$ .042	.03771 $\pm$ .00081
Anak	4,812 $\pm$ 98.7	4.693 $\pm$ .037	.03841 $\pm$ .00069
LaBelle	3,146 $\pm$ 103.5	4.208 $\pm$ .052	.03799 $\pm$ .00115
<u>Numerical integration</u>			
Trial 2	4,840 $\pm$ 1.3	4.671 $\pm$ .001	.04594 $\pm$ .00005
Trial 3, Cobb	4,107 $\pm$ .1	4.594 $\pm$ .001	.04740 $\pm$ .00005
Trial 4, Cobb	5,415 $\pm$ .7	4.768 $\pm$ .001	.03916 $\pm$ .00005
Anak	5,282 $\pm$ .8	4.478 $\pm$ .001	.03980 $\pm$ .00003
LaBelle	3,354 $\pm$ .6	4.268 $\pm$ .001	.03929 $\pm$ .00003



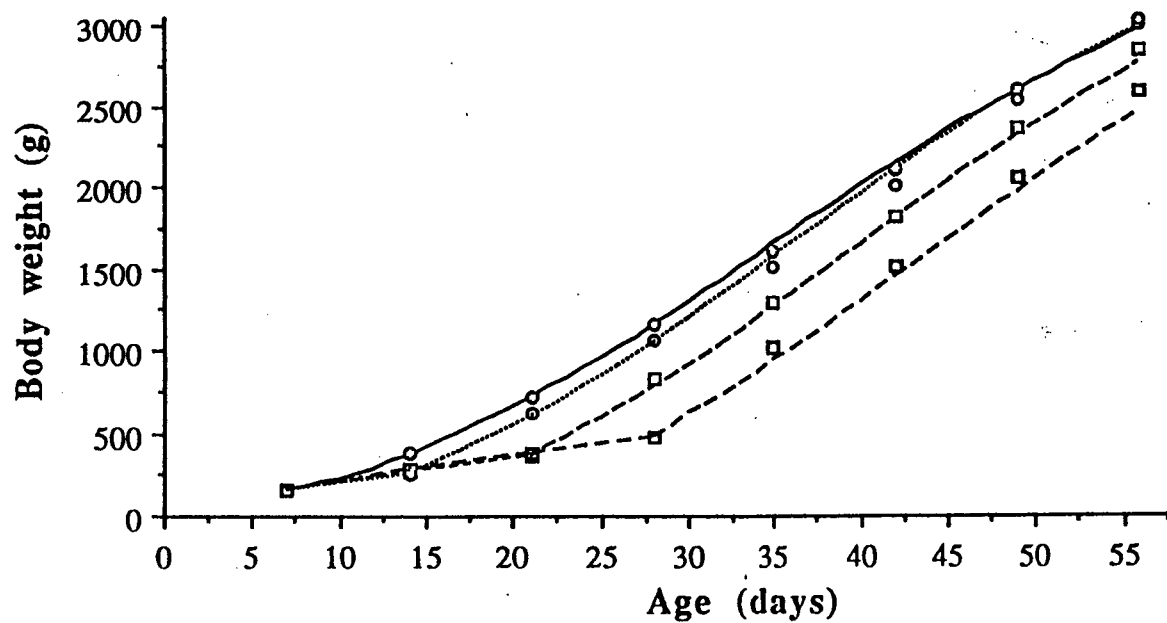


FIGURE 3. Observed (symbols) and predicted (lines) body weights during *ad libitum* feeding (○, —), and during and after feed restriction for 7 (○, ···), 14 (□, - - -) or 21 (□, - - -) days (Trial 2).

TABLE 3. Comparison between experimental results and model predictions of the response to early age feed restriction of different duration (Trial 2)<sup>1</sup>

Treatment	Weight gain			Feed intake			Feed efficiency		
	Actual	Predicted	$\Delta^2$	Actual	Predicted	$\Delta^2$	Actual	Predicted	$\Delta^2$
	---- g /47 d----	—%—		---- g /47 d----	—%—		-- gain/ feed--	—%—	
<i>Ad libitum</i>	2,837	2,807	-1.05	5,583	5,832	4.46	.508	.481	-5.31
Restriction, 7 d	2,855	2,817	-1.33	5,431	5,649	4.01	.526	.499	-5.13
14 d	2,679	2,593	-3.21	5,099	5,032	-1.31	.525	.515	-1.90
21 d	2,418	2,293	-5.16	4,446	4,392	-1.21	.544	.522	-1.04

<sup>1</sup> Initial body weight at 7 days of age was 156 g. The SE's were 25, 54, and .004 for observed body weight, feed intake and feed efficiency, respectively.

<sup>2</sup> 100 x (Predicted - Actual)/ Actual.

TABLE 4. *Comparison between experimental results and model predictions of the response to early age feed restriction of different severity and duration (Trial 3)<sup>1</sup>*

Treatment	Weight gain			Feed intake			Feed efficiency		
	Actual	Predicted	$\Delta^2$	Actual	Predicted	$\Delta$	Actual	Predicted	$\Delta$
	---- g /48 d-----		-%-	---- g /48 d-----		-%-	-- gain/ feed --		-%-
<i>Ad libitum</i>	2,397	2,392	-.21	4,934	5,079	2.94	.485	.471	-2.89
50% restriction, 7 d	2,327	2,395	2.92	4,665	4,850	3.97	.499	.494	-1.00
14 d	2,152	2,244	4.28	4,254	4,377	2.89	.506	.513	1.38
25% restriction, 7 d	2,425	2,437	.49	4,990	5,010	.41	.486	.486	0
14 d	2,293	2,340	2.05	4,578	4,658	1.75	.501	.502	.20

<sup>1</sup> Initial body weight at 7 days of age was 117 g. The SE's were 37, 69, and .005 for observed body weight, feed intake and feed efficiency, respectively.

<sup>2</sup>  $100 \times (\text{Predicted} - \text{Actual}) / \text{Actual}$ .

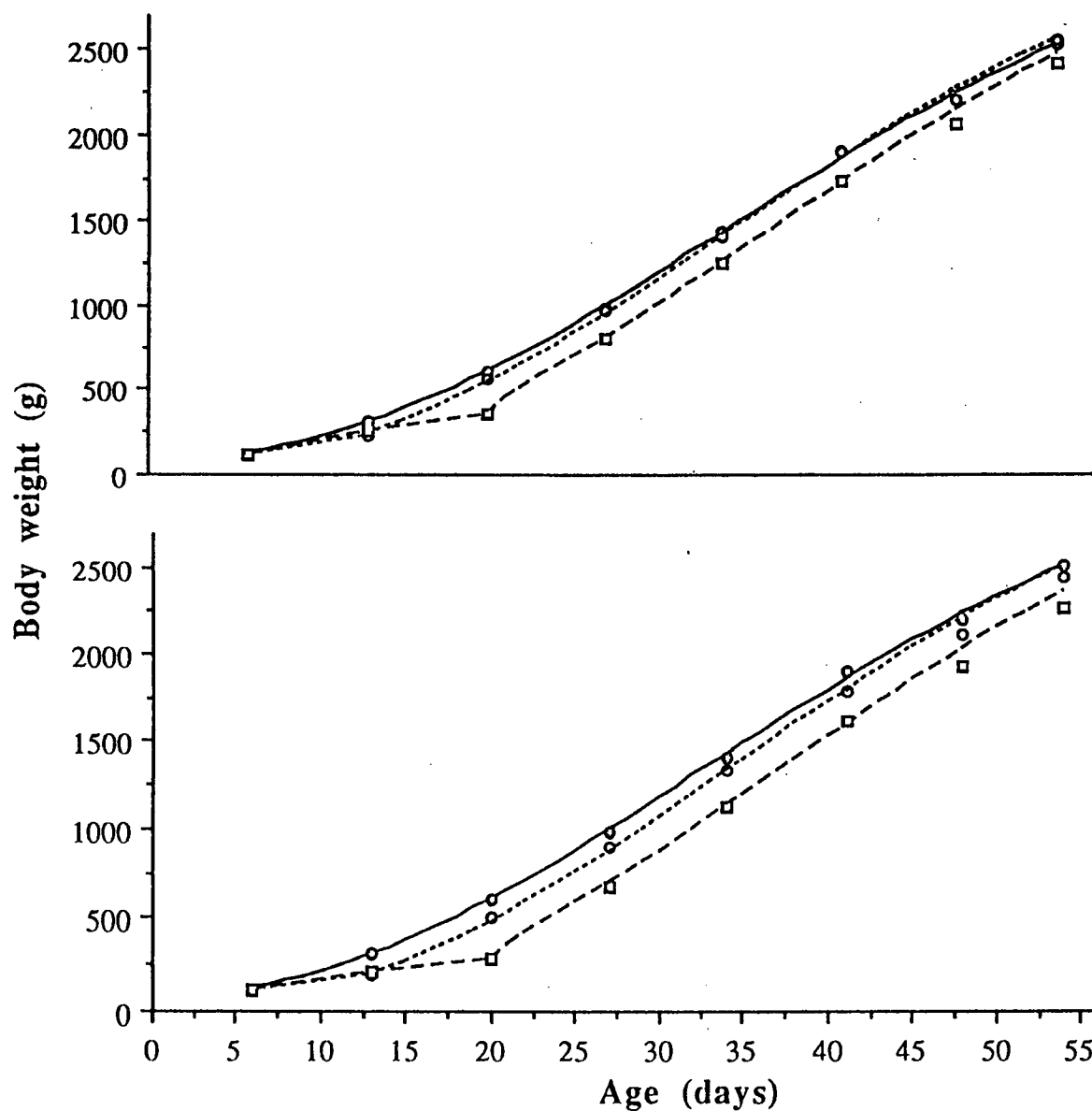


FIGURE 4. Observed (symbols) and predicted (lines) body weight during *ad libitum* feeding (○, —), and during and after feed restriction for 7 (○, ····) or 14 (□, - - -) days. Upper graph, 25% growth restriction; lower graph, 50% restriction (Trial 3).

LaBelle strain compared with Cobb and Anak, is reflected in the lower value of  $\beta$  of the Gompertz equation (Table 2). This strain is also characterized by a lower final weight  $\omega$ . In all three strains, accelerated growth resulted in full compensation for body weight retarded during feed restriction. Furthermore, the compensation model with its determined parameters was equally effective in predicting body weight response in all three strains. Deviations of the predicted from the observed feed intake (Table 5) did not exceed 5% in any of the three strains, suggesting that the energy requirement model is similar for the three broiler strains. The agreements between the predicted and observed body weights and feed intake produced good predictions of feed efficiency as affected both by strain and feed restriction.

## DISCUSSION

It was shown previously (Talpaz *et al.*, 1991) when modeling the growth of chicks following early age feed restriction, that the inclusion of a two-phase response in the simulation model resulted in improved model fit to experimental results over the use of a single term. The initial model predicted a 2-day early response to restriction (Fig. 2). Results of Trial 1 (Fig. 1) confirmed the existence of the two-phase response but indicated that the early one was limited to the first day of refeeding. Furthermore, in agreement with the initial assumption of the dependence of the early compensation factor on  $R_t$ , the first day weight gain, acceleration was greater in the 6- than in the 4-day restriction group. These findings were taken into account by modifying Eq. 6 into Eq. 10, and re-evaluation of the parameters using the previous results.

According to modeling dogma, a simulation model should be validated prior to its implementation. Furthermore, conditions applied during the validation procedure should differ from those used in initial testing and parameter estimation, and cover a large range of conditions. The validation made in this study included feed restriction

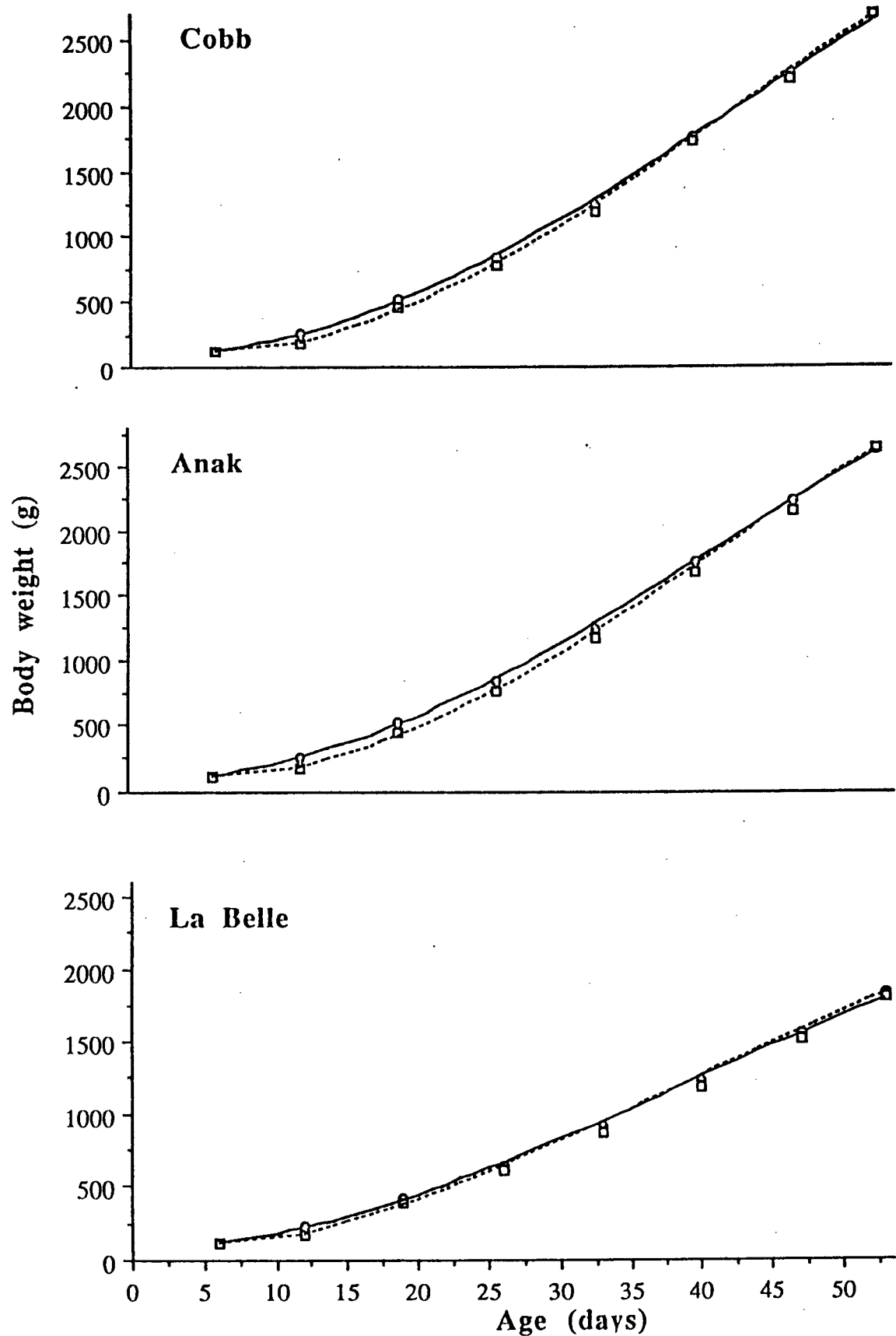


FIGURE 5. Observed (symbols) and predicted (lines) body weight during *ad libitum* feeding (○, —), and during and after feed restriction for 6 days (○ ····), in three different strains of broilers (Trial 4).

TABLE 5. *Comparison between experimental results and model predictions of the response to early age feed restriction in different broiler strains (Trial 4)*<sup>1</sup>

Treatment	Weight gain			Feed intake			Feed efficiency		
	Actual	Predicted	$\Delta^2$	Actual	Predicted	$\Delta^2$	Actual	Predicted	$\Delta^2$
	---- g /47 d----	—%—		---- g /47 d----	—%—		-- gain/ feed--	—%—	
Cobb, <i>ad libitum</i>	2,548	2,505	-1.69	5,106	4,901	-4.00	.499	.511	2.40
restricted	2,559	2,542	-.67	5,027	4,817	-4.18	.509	.528	3.73
Anak, <i>ad libitum</i>	2,517	2,487	-1.20	5,099	4,885	-4.20	.494	.509	3.04
restricted	2,528	2,515	-0.51	4,907	4,746	-3.28	.515	.530	2.92
LaBelle, <i>ad libitum</i>	1,721	1,679	-2.44	3,767	3,797	.80	.457	.442	-3.29
restricted	1,688	1,710	1.30	3,674	3,776	2.78	.459	.453	-1.31

<sup>1</sup> Initial body weight at 6 days of age was 112, 113, and 116 g for Cobb, Anak and Labelle, respectively. The SE's were 25, 40, and .003 for observed body weight, feed intake and feed efficiency, respectively.

<sup>2</sup>  $100 \times (\text{Predicted} - \text{Actual}) / \text{Actual}$ .

of durations considerably longer than the 6-day period used during parameter estimation. Furthermore, one of the the strains evaluated in the present study (LaBelle) exhibited a substantially slower growth rate than the Cobb strain used previously. An excellent agreement between the actual results and the corresponding predicted values is demonstrated visually in Figures 3 - 5. The largest deviation is observed for the group restricted for 3 weeks (Fig. 3), a duration considerably out of the expected range of implementation. The percentage of deviation of the predicted from the observed values in all three trials was  $-.43 \pm 2.41\%$ ,  $.95 \pm 3.07\%$ , and  $-.55 \pm 2.88\%$  for final body weight, feed intake and feed efficiency, respectively, indicating no consistent under- or overestimates by the model.

The success in predicting feed intake in the three broiler strains suggests that the model is not limited to particular genotypes. Moreover, the model inclusive of its parameters is also sufficient to predict the differences in feed efficiency among the strains. It should be pointed out, however, that although the model can be used equally well in layer strains, they have a lower specific need for energy of maintenance than broilers (Hurwitz *et al.*, 1978; Pinchasov *et al.*, 1991), suggesting some limitations in the applicability of the model parameters.

In the model as presented previously (Talpaz *et al.*, 1991) and as employed in the present study, the Gompertz growth equation has been used to describe normal growth. This equation is not, however, an essential part of the restriction-compensation model summarized in Eq. 4, and other formats which offer a good description of growth can be used.

The present results confirm the well documented previous observations (Plavnik and Hurwitz, 1985, 1988a,b; Plavnik *et al.*, 1986; McMurtry *et al.*, 1988) which suggested a practical advantage to early feed restriction in improving feed efficiency in male broiler chickens. The wide range of applicability which also includes different broiler strains, and the possibility of a reliable quantitative predictability of the response of both growth and feed efficiency, can be used within the algorithm



presented by Talpaz *et al.* (1988) and specifically modified in a later (Talpaz *et al.*, 1991) and in the present studies to select the most economic growth trajectory in broilers.

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## **Tibia Development in Turkeys and Chickens as Affected by Early- Age Feed Restriction\***

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*Running head*

BONE DEVELOPMENT IN BIRDS

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**ABSTRACT:** Tibia development was studied by histomorphometry in male chickens and turkeys fed *ad libitum* throughout the growing period, or subjected to an early-age (1-week) severe feed restriction for 6 and 14 days, respectively. In turkeys, the rates of longitudinal bone growth and epiphyseal width growth, both dependent on the activity of chondrocytes, proceeded rapidly and reached maxima at early ages of 24 and 11 days, respectively. Moreover, longitudinal bone growth and the growth of epiphyseal width were reduced during early-age feed restriction, in both chickens and turkeys. On the other hand, the rate growth of metaphyseal width, determined by activity of bone cells such as osteoclasts and osteoblasts, reached in turkeys a maximum at the relatively late age of 70 days, and was not influenced by feed restriction. Rhythmic temporal variation characterized the temporal behavior of the height of the hypertrophic and non-hypertrophic zones of the growth plate, the trabecular width, and the osteoclasts number at the subchondral region. Rhythmicity appeared similar in both *ad libitum*-fed and early-age feed-restricted birds of both species. The height of the non-hypertrophic epiphyseal cartilage appeared not to change, whereas the hypertrophic zone decreased with age and early-age feed restriction, demonstrating the importance of cartilage cell hypertrophy as a main determinant of longitudinal bone growth. Sexual maturation was accompanied by the disappearance of both hypertrophic and non-hypertrophic zones of the epiphyseal growth plate and the end of bone elongation. Trabecular width at the subchondral region increased while osteoclast number decreased with age, reflecting the net increase in bone mass, but were not affected significantly by early age feed restriction. Compensatory growth of the early-age feed-restricted birds, resulted in the complete recovery of affected bone variables within a few weeks of resumed *ad libitum* feed intake.

**KEY WORDS:** Epiphyseal growth plate, bone growth, birds, histomorphometry.

## INTRODUCTION

Bone growth proceeds at a rate distinct from that of other tissues such as muscle and some internal organs (Hurwitz *et al.*, 1991), and thus involves a specific regulatory system. Longitudinal bone growth is effected by the cellular activity of growth-plate chondrocytes. These cells, which are the products of cell division at the proliferative region of the growth plate, undergo extensive hypertrophy while secreting cartilaginous extracellular matrix. Following some degree of matrix calcification, the hypertrophied chondrocytes finally atrophy, the entire cartilaginous template is subjected to clastic resorptive activity and is replaced by osseous tissue following an invasion of blood vessels into the area. There is little quantitative information concerning the growth in width of the various bone regions and its regulation.

The structure of the growth plate in the young bird is fundamentally similar to that of the growing mammal (Leach and Gay, 1987; Wise and Jennings, 1973) despite some differences in cell number, size and density. In both mammals and birds, the structure of the growth plate is subject to various disorders such as chondrodystrophy, dyschondroplasia and rickets. However, detailed morphological information on the growth plate in birds approaching maturity was defined by Wise and Jennings (1973) as incomplete. Recent studies, such as those reviewed by Leach and Gay (1987) were conducted with only neonatal birds up to 6-7 weeks of age. Quantitative histomorphometry of turkey bone was carried out by Leblanc *et al.* (1986) but included only a few age points.

The rate of bone growth is determined genetically (Leblanc *et al.*, 1986) and programmed to vary with age. The effects of nutrition on bone growth have been recognized for many years (Sauveur, 1984). Calorie restriction in young (5-7 day old) chickens resulted in a marked decrease in body weight inclusive of various internal

organs (Plavnik and Hurwitz, 1983). During *ad libitum* feeding following such an early-age feed restriction in chickens, compensatory growth (Wilson and Osborn, 1960) resulted in full recovery of body weight, within a period of several weeks (Plavnik and Hurwitz, 1985, 1988a; Plavnik *et al.*, 1986). Following a 14-day feed restriction in turkeys, body weight which had been reduced to 50% of normal, even exceeded that of the *ad libitum*-fed birds by the age of 140 days (Plavnik and Hurwitz, 1988b).

Various hormones such as growth hormone, parathyroid hormone and 1,25 dihydroxyvitamin D<sub>3</sub> regulate bone growth (Hurwitz and Pines, 1992; Pines and Hurwitz, 1991). Also involved in bone growth regulation is a local paracrine-autocrine system including various growth factors such as insulin-like growth factor (IGF-I), epidermal growth factor (EGF) and various cytokines. Gonadal hormones are also involved in control of bone growth. Most importantly, these hormones appear to be responsible for the disappearance of the epiphyseal growth plate and termination of bone growth at the onset of sexual maturation (as reviewed by Leach and Gay, 1987). However, to the best of our knowledge, the precise time of closure of the growth plate, and its relationship to gonad development have not been evaluated in avian species.

The purpose of the present study was to relate age-dependent and nutrition-induced changes in various histomorphometric variables in bone, to bone growth. The study was conducted with two species - chicken (*Gallus domesticus*) and turkey (*Meleagris gallopavo*) - during normal development and following a short period of growth retardation induced by early age feed restriction.

## MATERIALS AND METHODS

### *Birds*

Day-old White Rock (Cobb strain) broiler chicks or British United Turkey poults were obtained from commercial hatcheries and raised for 4 weeks in battery brooders in constant-temperature rooms at 24°C. Subsequently, they were transferred to individual cages situated in temperature controlled (20°C) rooms. The birds were fed diets appropriate for their species and age (Hurwitz *et al.*, 1991) and designed to satisfy the recommendations of the National Research Council (1984).

At 7 days of age, the birds were assigned to the experimental groups, equalizing both means and variance of body weight within the groups. The experiments with either species included two treatment groups. One group was fed *ad libitum* throughout the study, and the other was subjected to early-age feed restriction. During the growth restriction period of 6 days in chickens and 14 days in turkeys, the restricted birds received a daily allowance of feed energy calculated to maintain 25% of their normal growth rate, according to the formula modified from Hurwitz *et al.* (1980). Following the restriction period, birds were fed *ad libitum* until the end of the experiment.

### *Specimen preparation*

At various time intervals, five birds from each treatment group were weighed and killed by neck dislocation. The right tibia of each bird was removed and its length was measured. The proximal end of each tibia was sectioned sagittally and the resulting slices were fixed in 4 % formaldehyde in 0.1 M phosphate buffer (pH 7.2), for 3 days. Specimens were then decalcified at room temperature in 10% ethylene



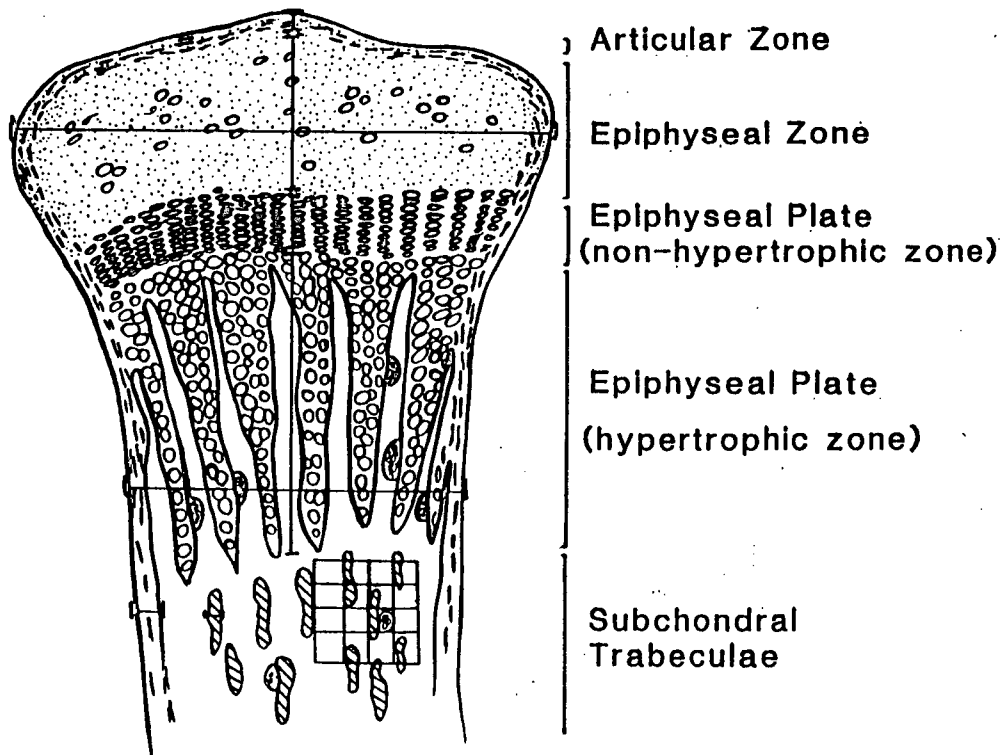
diaminetetraacetic acid (EDTA) in phosphate buffer, pH 7.4, for 2 to 8 weeks in proportion to their size, dehydrated in graded ethanol concentrations and embedded in paraplast (56°). Then, 6 µm sagittal sections were stained with hematoxiline-eosin (H&E).

Specimens for Scanning Electron Microscopy were fixed with 2.5% glutaraldehyde in phosphate buffered saline (PBS) containing 5% sucrose (18 h, 4°C), washed with PBS, dehydrated in increasing concentrations of ethanol followed by hexamethyldisilisan (HMDS, Fluka AG, Switzerland), and air dried. The specimens were then vacuum-coated with gold-palladium and were examined in a Jeal JSM 840 scanning electron microscope at 25 kv.

#### *Histomorphometric measurements*

Histomorphometric measurements were carried out with an Olympus microscope. Five animals were used for each age group and treatment, and three measurements of each variable were taken, of three different sections.

The measurements illustrated schematically in Figure 1 included: (i) height of the articular cartilaginous zone (not reported) ; (ii) height of the non-hypertrophic region of the epiphyseal growth plate containing the resting and the proliferative zones; (iii) height of the hypertrophic region of the growth plate containing cartilage and invading blood vessels; (iv) width of the epiphysis and (v) width of the metaphysis, just below the hypertrophic region; (vi) width of subchondral trabeculae; and (vii) the number of osteoclasts in the subchondral region.



**Figure 1.** Schematic representation of the histomorphometric measurements. The upper and the lower horizontal lines signify epiphyseal width and metaphyseal width, respectively. The grid at the lower part of the figure represents measurements of trabecular width and osteoclast number.

### *Statistical analyses*

The results were subjected to one-way analysis of variance (Snedecor and Cochran, 1967). The Gompertz growth equation was computed for some of the variables as described previously (Hurwitz *et al.*, 1991; Talpaz *et al.*, 1991).

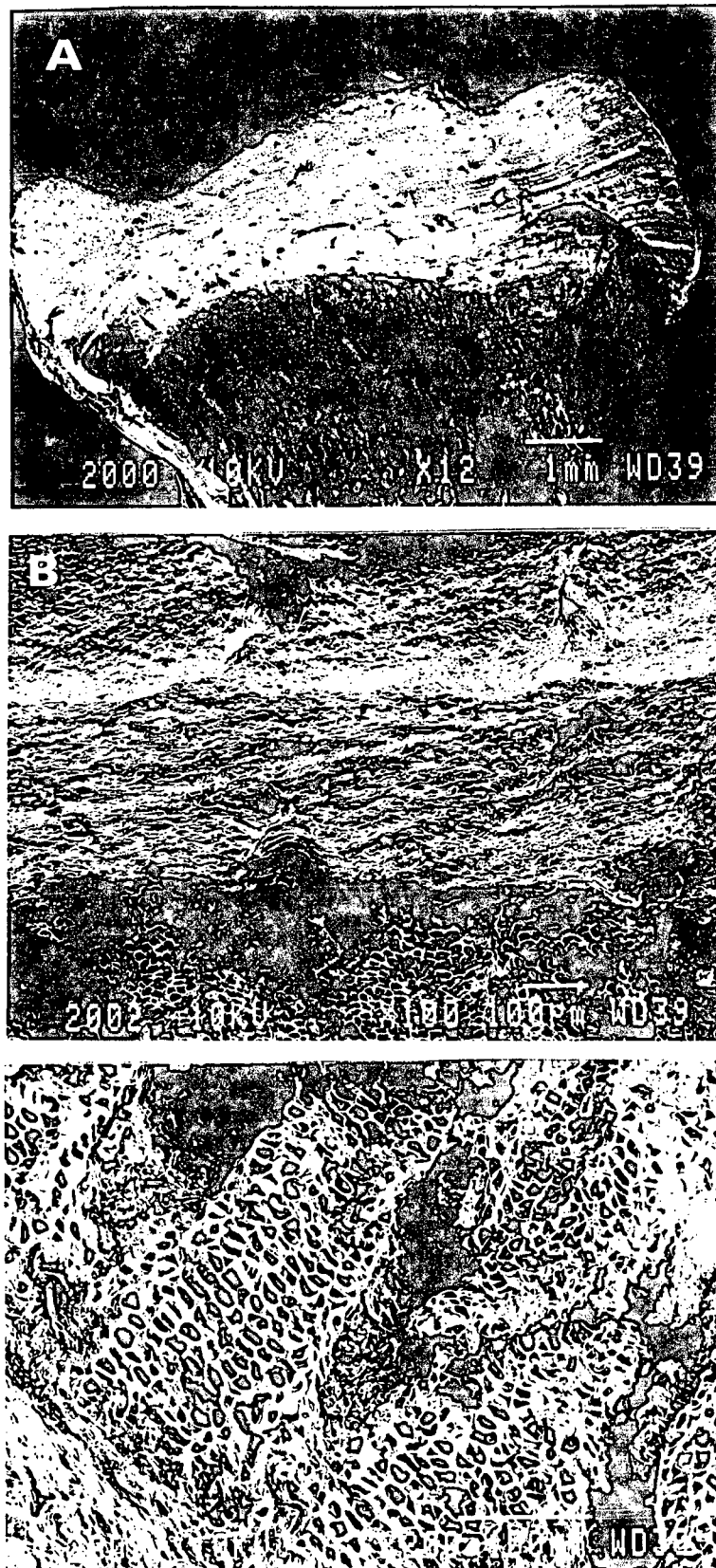
## RESULTS

### *Growth plate morphology*

The overall architecture of the head of the tibia including the articular cartilage, the growth plate region with the hypertrophic and non-hypertrophic regions, presented schematically in Figure 1, is distinguishable in the scanning electron micrograph (Figure 2A). At a higher magnification (Figure 2B), the imprints of the proliferative and especially those of the hypertrophic cells are visible. Figure 2C demonstrates the penetration of blood vessels into the hypertrophic region and the beginning of endochondral bone growth at the surfaces of the penetrating vessels.

### *Tibia growth*

In turkeys, tibia length (Figure 3) appeared to follow a sigmoidal curve (Hurwitz *et al.*, 1991) which could be described well by a Gompertz equation. By the age of 100 days, the length of the tibia exceeded 90% of its final dimension. The inflection point of tibia length was calculated as 24 days from the fitted Gompertz curve (Hurwitz *et al.*, 1991) and signifies the age of maximum growth. Epiphyseal width (Figure 3) appears to have reached a maximum value at approximately the same rate as tibia length. The inflection point calculated from the fitted Gompertz equation was 11 days. On the other



**Figure 2.** Scanning electron micrographs of the proximal tibia of a 3-week old *ad libitum*-fed turkey poult. A, the entire epiphyseal region (x12); B, the non-hypertrophic and upper part of the hypertrophic cartilage (x 100); C, details of blood vessel penetration of blood vessels into the hypertrophic zone (x200).

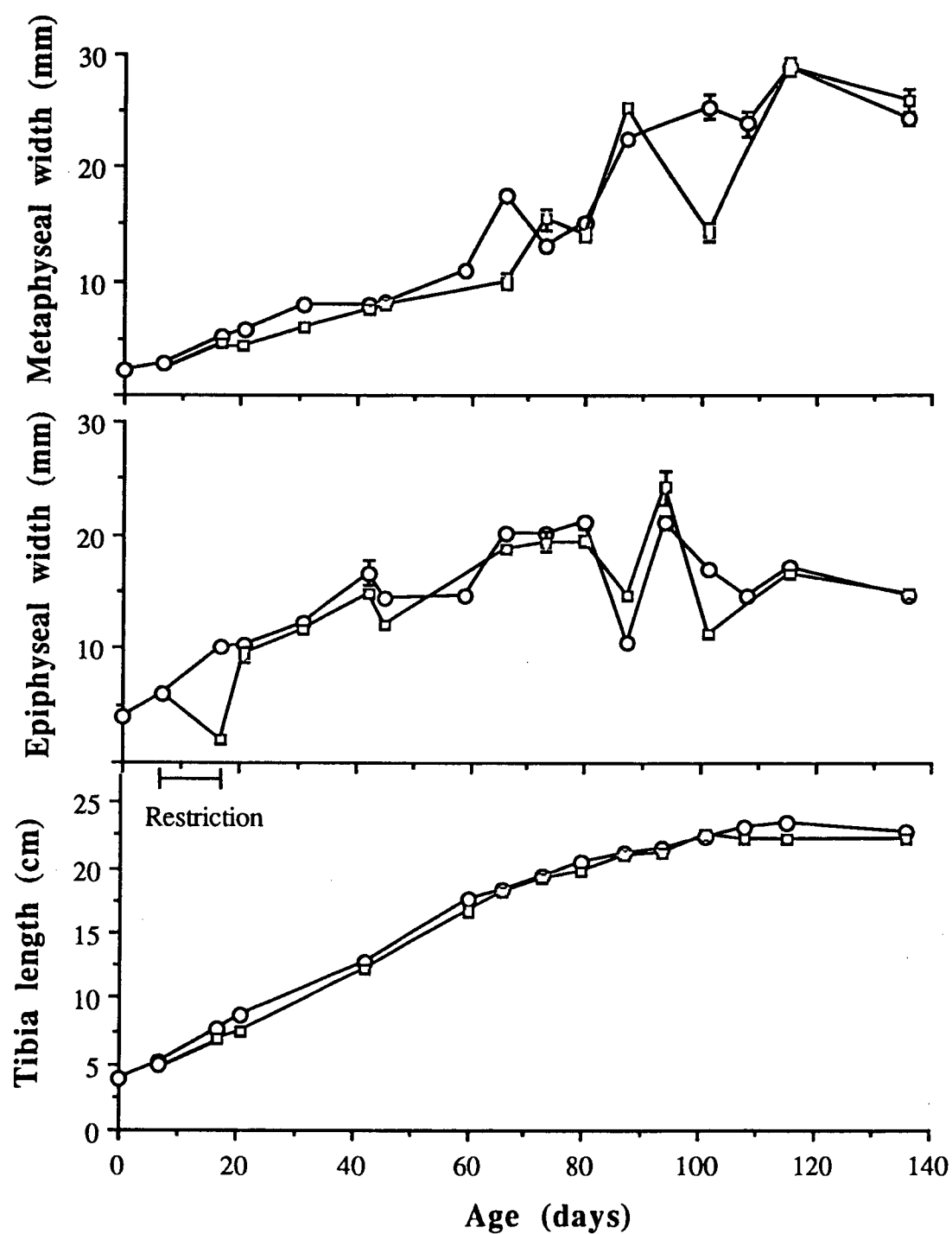
hand, metaphyseal width (Figure 3) appears to have increased at a slower rate, approaching maximal values only towards the end of the study, at an age exceeding 100 days. Maximum growth in width of the metaphysis occurred at 71 days.

In chickens, a maximal value of longitudinal tibia growth was not approached in the even at the age of 70 days (Figure 4). The width of the epiphysis and the metaphysis appears to have reached a maximum by approximately 30 days. The short period (6 weeks) of observation does not permit an exact comparison of the temporal changes in these variables with bone length.

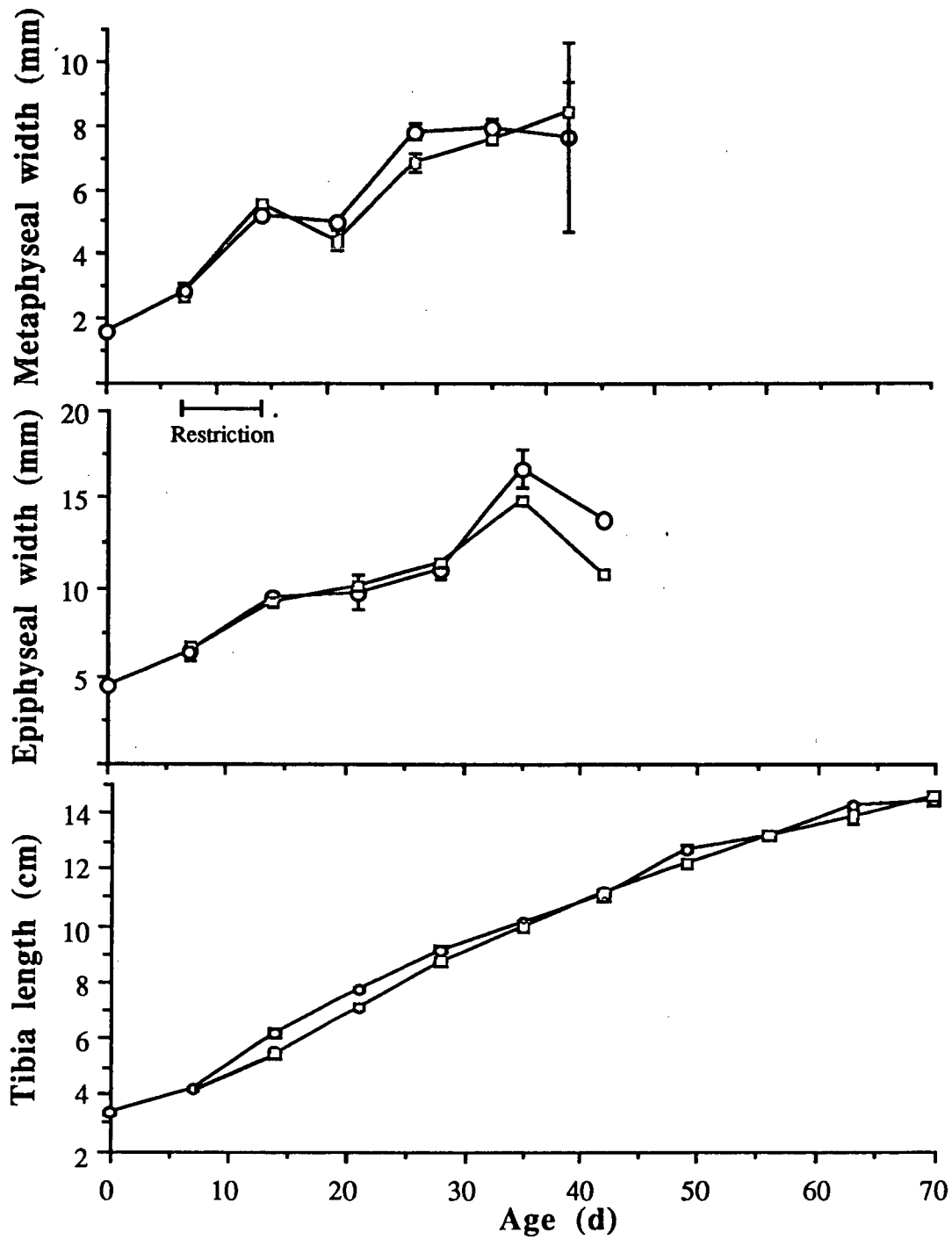
During feed restriction, longitudinal growth of the tibia was reduced in both species. Due to accelerated growth during refeeding, tibia length in the restricted birds reached that in the controls after 3-4 weeks of refeeding. Width of the epiphysis and metaphysis was slightly reduced at the end of the feed-restriction period in turkeys but not in chicks. The differences between the *ad libitum* -fed or feed-restricted turkeys appears to have disappeared after a few weeks of refeeding.

#### *The epiphyseal plate non-hypertrophic zone*

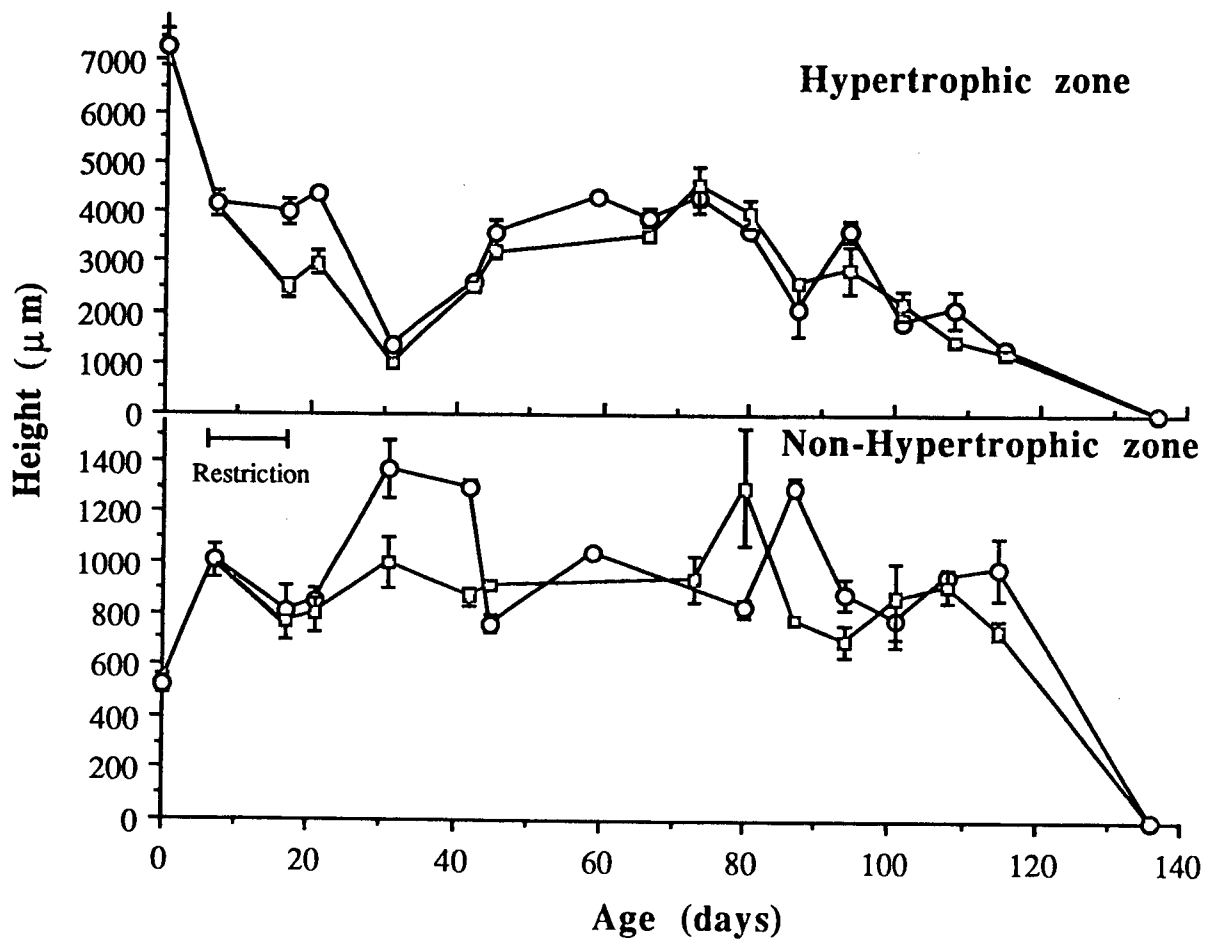
In the turkey, the height of the non-hypertrophic zone (Figure 5) did not follow any consistent pattern with age but between 110 and 140 days it disappeared completely. The height of the non-hypertrophic zone increased during the first week of life followed by little change in the next several weeks. This was followed by three peaks: one between 20 and 40 days of age, another at 70-90 days and a third at 100-120 days of age. This oscillatory behavior was moderately modified by feed restriction: peak activity was either quenched or its phase was changed; the 20-40 days peak was greatly quenched but the second and third peaks appeared one week earlier than in the *ad libitum* -fed birds.



**Figure 3.** Dimensions of bone growth in the *ad libitum*-fed (○) and feed-restricted (□) male turkey. Vertical bars represent the standard errors; horizontal bar signifies the period of feed restriction.



**Figure 4.** Dimensions of bone growth in the *ad libitum*-fed (○) and feed-restricted (□) male chicken. Vertical bars represent the standard errors; horizontal bar signifies the period of feed restriction.



**Figure 5.** Height of the hypertrophic (upper graph) and non-hypertrophic (lower graph) zones of the epiphyseal growth plate of *ad libitum* -fed (○) and feed-restricted (□) male turkeys. Vertical bars represent the standard errors; horizontal bar signifies the period of feed restriction.



In chickens, the height of the non-hypertrophic zone (Figure 6) increased during the first week of life, declined until 20 days of age and then increased again at 30-42 days. Early-age feed restriction caused a phase shift in the apparent rhythmic behavior.

#### *The hypertrophic zone*

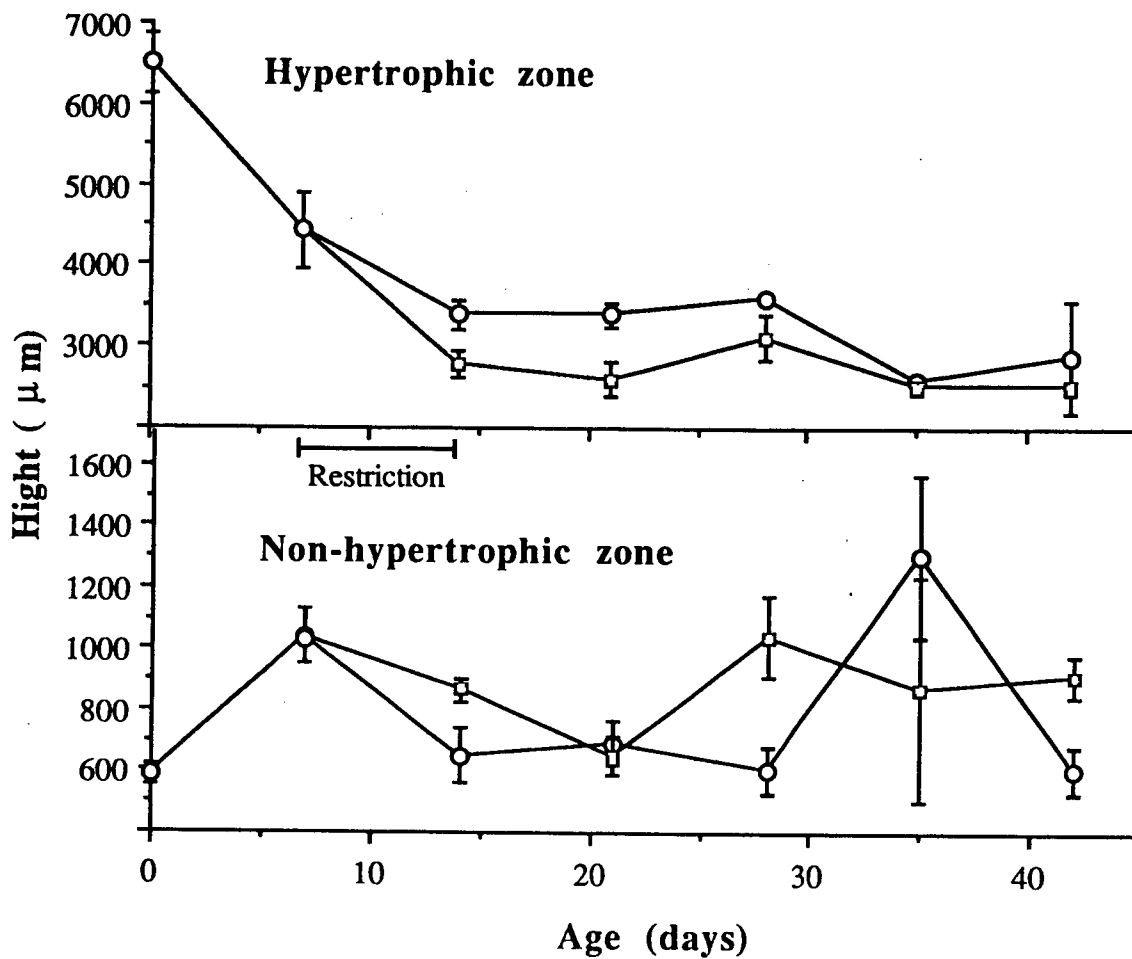
In the two species, the height of the hypertrophic zone (Figures 6 and 7) followed a general decrease during the growth period but exhibited similar rhythmic fluctuations in both treatments within species. In the turkey, in which bone histomorphometry was continued until maturation, this region disappeared completely by the age of 140 days.

Similarly, in both species, a significant diminution in the hypertrophic zone occurred during feed restriction; however, it disappeared after 2-4 weeks of refeeding.

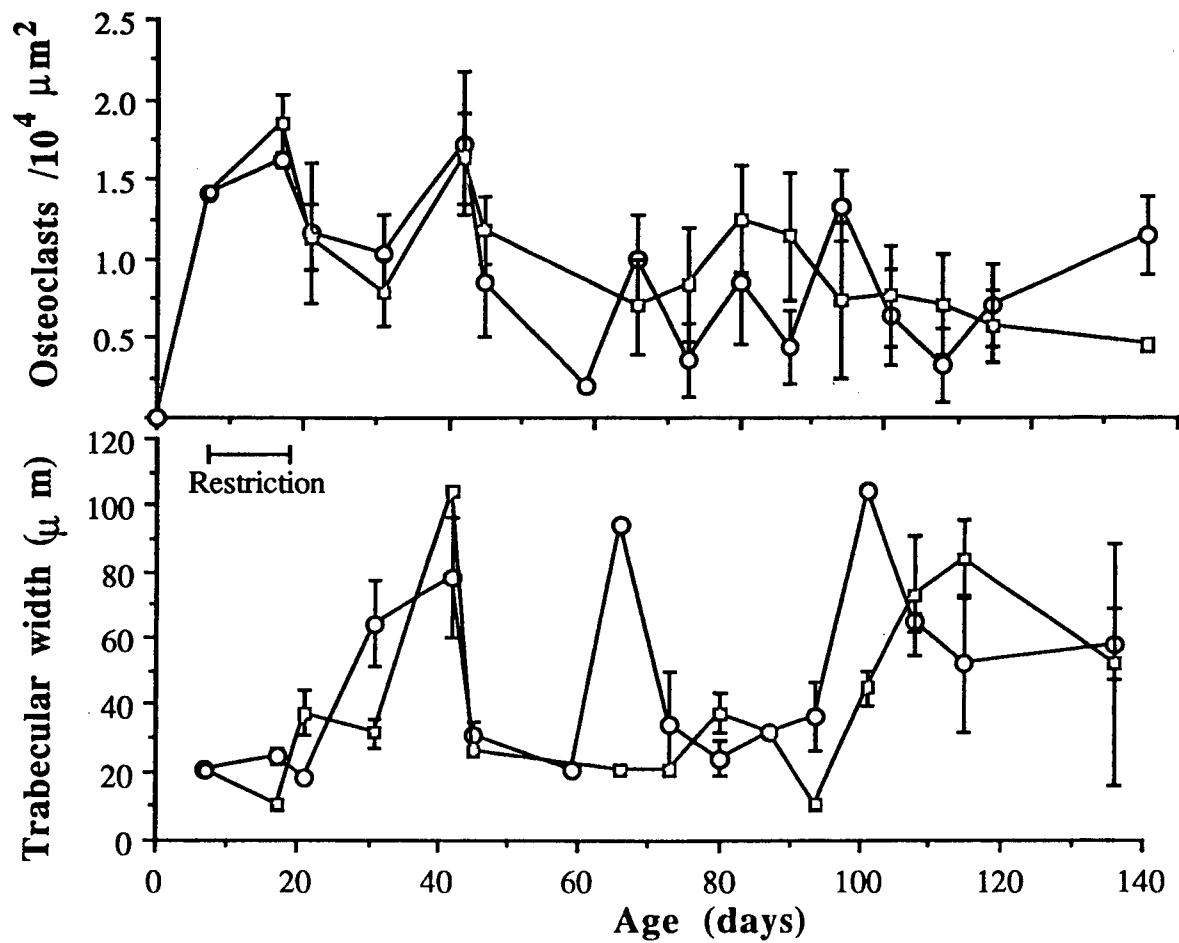
#### *Width of subchondral trabeculae*

In turkeys fed *ad libitum* (Figure 7), the width of subchondral trabeculae increased reaching a peak at the age of 25 to 42 days, followed by two additional peaks at 63 and 108 days of age. During the early-age feed restriction, the width of the trabeculae in turkeys was slightly reduced, but increased rapidly to reach peaks similar to those exhibited by the *ad libitum* -fed controls, albeit 1 to 2 weeks later. Except for this rhythmic activity, there was no consistent age-dependent change in the average trabecular width.

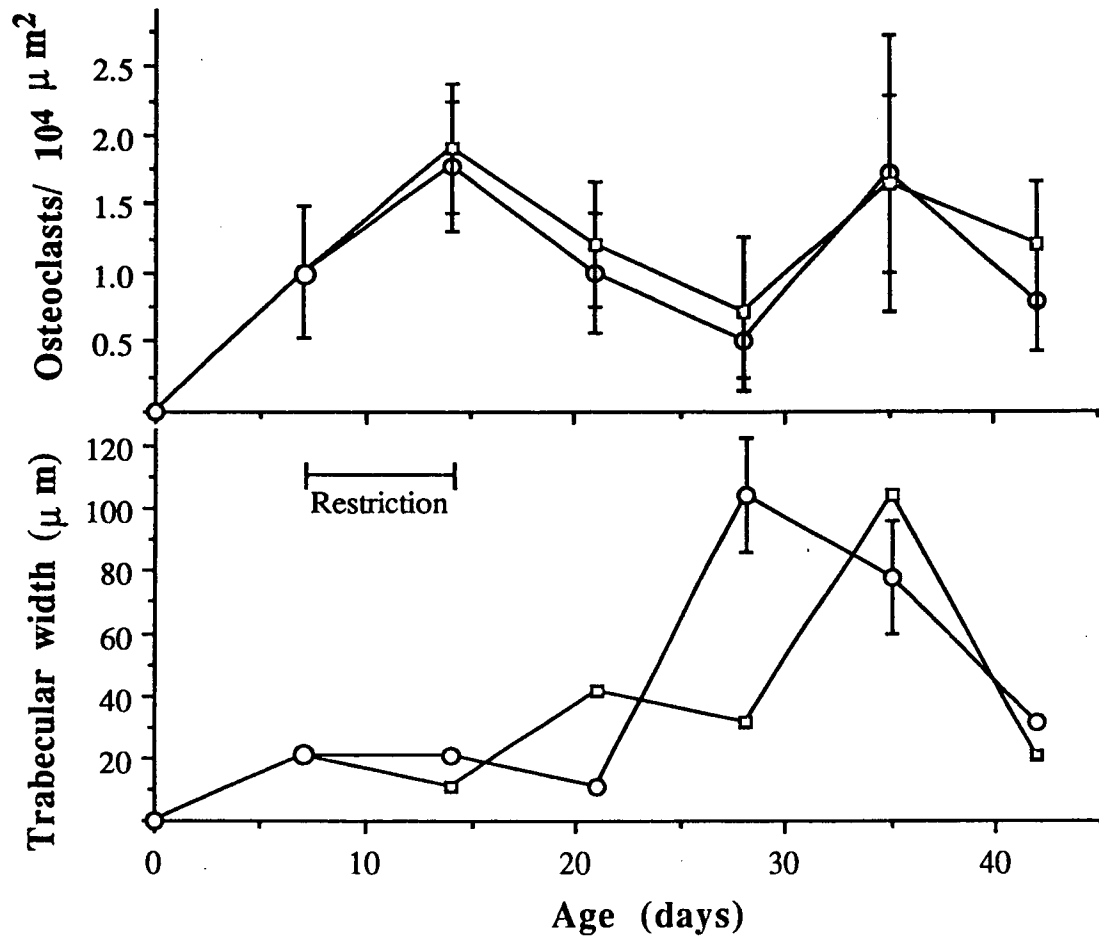
In chicks (Figure 8), a peak in trabecular width occurred at 28 days. Early-age feed restriction appears to have caused an initial reduction in trabecular width and a phase shift, with peak activity at 35 days in the restricted birds.



**Figure 6.** Height of the hypertrophic (upper graph) and non-hypertrophic (lower graph) zone of the epiphyseal growth plate of *ad libitum* -fed (○) and feed-restricted (□) male chickens. Vertical bars represent the standard errors; horizontal bar signifies the period of feed restriction.



**Figure 7.** Trabecular width and osteoclast number in the proximal subchondral region of the tibia of *ad libitum*-fed ( $\circ$ ) and feed-restricted ( $\square$ ) male turkeys. Vertical bars represent the standard errors; horizontal bar signifies the period of feed restriction.



**Figure 8.** Trabecular width and osteoclast number in the proximal subchondral region of the tibia of *ad libitum*-fed (○) and feed-restricted (□) male chickens. Vertical bars represent the standard errors; horizontal bar signifies the period of feed restriction.

### *Number of osteoclasts*

The number of osteoclasts per area (Figures 7 and 8) declined with age in the turkey but did not change appreciably in the chicken, while exhibiting a rhythmic fluctuation. Feed restriction did not alter the temporal pattern of the osteoclast number. When the pattern during the first 6 weeks of life is considered, a remarkable similarity between chickens and turkeys becomes apparent. Regardless of feeding schedule, osteoclast number peaked at 2 and 5 weeks of age. In the turkeys, additional peaks were evident between 60 and 100 days of age.

## DISCUSSION

In the turkey, both longitudinal growth and growth of epiphyseal width followed a similar temporal pattern, with growth maxima at 24 and 11 days, respectively, the earliest among the various organs examined (Hurwitz *et al.*, 1991). On the other hand, growth of metaphyseal width proceeded at a considerably different rate, with a maximum observed at the age of 70 days. The growth pattern of bone width is reminiscent of that of muscle, which reached a maximum rate at the age of approximately 80 days. The specific growth pattern of bone is probably the biological answer to the initial developmental need to create a proper body frame and a later need to enforce the existing frame in order to support the increasing muscle mass.

Bone elongation, epiphyseal width (in the turkey only) and metaphyseal width were slightly reduced by feed restriction. Due to compensatory growth, bone length and width approached normal after a few weeks of refeeding. This rapid rate of return to normal, exceeds that of body weight which, under the present conditions of feed restriction, is known to occur at 6-7 weeks of age in chickens and after 14-15 weeks in

turkeys (Plavnik and Hurwitz, 1988a,b).

Longitudinal growth and the increase in epiphyseal width are both the result of the activity of chondrocytes of the epiphyseal plate and those along the epiphyseal borders, whereas the change in width of the metaphysis is the result also of activity of bone cells - osteoblasts and osteoclasts - at both periosteal and endosteal surfaces. The difference in the age-dependence of the chondrocyte-associated and bone cell-associated growth suggests that the two cellular systems respond differently to similar humeral messages. On the other hand, the similarity in response to feed restriction suggests also a more generalized mode of regulation.

All cellular variables measured at the growth plate region in the two species exhibited a certain degree of rhythmic activity. Although a rigorous numerical analysis was not done, at least some of the exhibited rhythmic behavior patterns appear to be true biological phenomena rather than the result of random variation, since there was considerable similarity between the *ad libitum* -fed and feed-restricted birds, among the various variables and between the two species. The large change observed during the first week of life for all bone variables may be the consequence of the transition from the embryonal to the neonatal state. The disappearance of the cartilaginous growth plate, inclusive of the hypertrophic and the non-hypertrophic zones, occurred in male turkeys between 115 and 140 days of age, simultaneously with the large increase in testes size and the associated circulating testosterone level (Hurwitz *et al.*, 1991). No explanation can be offered, at present, for the observed oscillations in the various bone variables.

Of the two regions of the cartilaginous growth plate, the non-hypertrophic zone did not show any general change with age, except for a precipitous decline in turkeys between 110 and 136 days of age when the layer disappeared. On the other hand, from 24 days of age in turkeys, the height of the hypertrophic zone gradually declined,

parallel to the reduction in the rate of increase in tibia length. Furthermore, in both species, feed restriction did not affect the non-hypertrophic zone whereas it caused a significant decline in the height of the hypertrophic zone which later returned to normal together with bone length. These results with birds support the conclusions of Hunziker *et al.* (1987) and Hunziker and Schenk (1989), based on experimentation with rats, that longitudinal growth is determined by the magnitude of chondrocyte hypertrophy rather than the rate of their proliferation.

Despite considerable oscillations, trabecular width increased with age in both chickens and turkeys, reflecting the cumulative increase in bone mass and calcification. On the other hand, the number of osteoclasts appears to decrease with age as a function of the decrease in the rate of bone growth.

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## **Sexual Development in Male turkeys as Affected by Early-Age Feed Restriction\***

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*Running head*

SEXUAL DEVELOPMENT IN TURKEYS

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**ABSTRACT:** Testes development and plasma testosterone were evaluated in male turkeys subjected to an early age (1-week) severe feed restriction for 10 days or to *ad libitum* feeding throughout the growing period. Testes weight increased gradually up to the age of 42 days and at a more rapid rate between 42 and 108 days of age. A rapid increase in testicular weight at the age of 108 days marked the beginning of puberty. During refeeding, testes weight which had been depressed by feed restriction, rapidly attained normal values. Plasma testosterone initially decreased until the age of 63 days followed by about 30 days of little change and rapid increase in plasma testosterone starting at the age of 90 to 100 days. In turkeys previously subjected to feed restriction, plasma testosterone was higher than in controls fed *ad libitum* throughout most of the growing period. It is suggested that the higher testosterone levels may contribute to the induction of compensatory growth.

## INTRODUCTION

Calorie restriction in young (5-7 day old) turkeys resulted in a marked decrease in body weight. During *ad libitum* feeding following such an early-age feed restriction, compensatory growth (Wilson and Osborn, 1960) resulted in body weights even exceeding those of the *ad libitum*-fed birds by 140 days of age (Plavnik and Hurwitz, 1988a). However, compensatory growth was considerably more modest in females than in males (Plavnik and Hurwitz, 1988b). Furthermore, Howland (1975) showed that plasma testosterone level in rats subjected to feed restriction exceeded that of the control level during several days following *ad libitum* feeding, suggesting an involvement of this hormone in compensatory growth.

The purpose of the present study was to evaluate testicular development and plasma testosterone concentration in turkeys (*Meleagris gallopavo*) during normal *ad libitum* feeding and following a short early-age feed restriction, in an effort to evaluate the possible role of testosterone in induction of compensatory growth.

## MATERIALS AND METHODS

### *Birds*

Day-old British United Turkey males were obtained from a commercial hatchery and raised for 4 weeks in battery brooders in constant-temperature rooms at 24°C. Subsequently, they were transferred to individual cages situated in a temperature controlled (20°C) room. The birds were fed diets appropriate for their species and age

designed to satisfy the recommendations of the National Research Council (1984). Fluorescent lights provided continuous illumination throughout the study.

At 7 days of age, the birds were assigned to the experimental groups, equalizing both means and variance of body weight within the groups. The experiment included two treatment groups. One group was fed *ad libitum* throughout the study (control), and the other was subjected to early-age feed restriction. Following the restriction period, birds were fed *ad libitum* until the end of the experiment.

At various time intervals, five birds from each treatment group were weighed, bled between 9 and 10 AM and killed by neck dislocation. The testes were removed and their weights were recorded.

#### *Testosterone assay*

Plasma samples were collected and stored at -20°C until used. 1 ml plasma was spiked with <sup>3</sup>H testosterone (Amersham, Bucks, U.K.) and extracted with 8 ml of dichloromethane. After extensive shaking, a 4 ml aliquot of the lower organic phase was dried under nitrogen and reconstituted with 1 ml borate buffer. Samples of 100 µl were taken in duplicate for recovery determination (the recovery was between 70-80%) and for radioimmunoassay (RIA) of testosterone.

Testosterone was determined using a kit (Diagnostic Products Corporation, CA, USA). The assay was highly specific for testosterone but had a 34% cross-reaction with dihydrotestosterone and less than 0.1% cross-reactivity with cortisol, cortisone, estradiol, aldosterone and 5β-dihydrotestosterone. The sensitivity was 1pg/tube. All samples were assayed at one time.

### *Statistical analyses*

The results were subjected to analysis of variance (Snedecor and Cochran, 1967).

## RESULTS

At the end of the restriction period at the age of 17 days, body weights of the *ad libitum*-fed and feed restricted turkeys was 460 and 246 g, respectively. At 150 days of age, body weight was 13992 g and 14728 g in the controls and feed-restricted birds, respectively.

Testes weight was reduced by feed restriction. At the end of restriction the weights of the testes were 35 and 16 mg for the controls and feed restricted birds, respectively. At the age of 72 days testes weight of the restricted birds reached that of the controls; it remained similar to that of the controls until the end of measurements, at the age of 136 days.

From 7 to 17 days of age, testes weight as a fraction of body weight slightly increased and remained unchanged up to 42 days of age whence a more rapid development started. The rapid increase in testes weight starting at the age of 108 days, signifies puberty.

Plasma testosterone, significantly decreased in a parallel fashion in both treatments until the age of 63 days and remained low until 90- 100 days of age after which it rapidly increased. Early age feed restriction resulted in significantly higher plasma testosterone levels; this difference was observed at 9 out of the 11 sampling times.

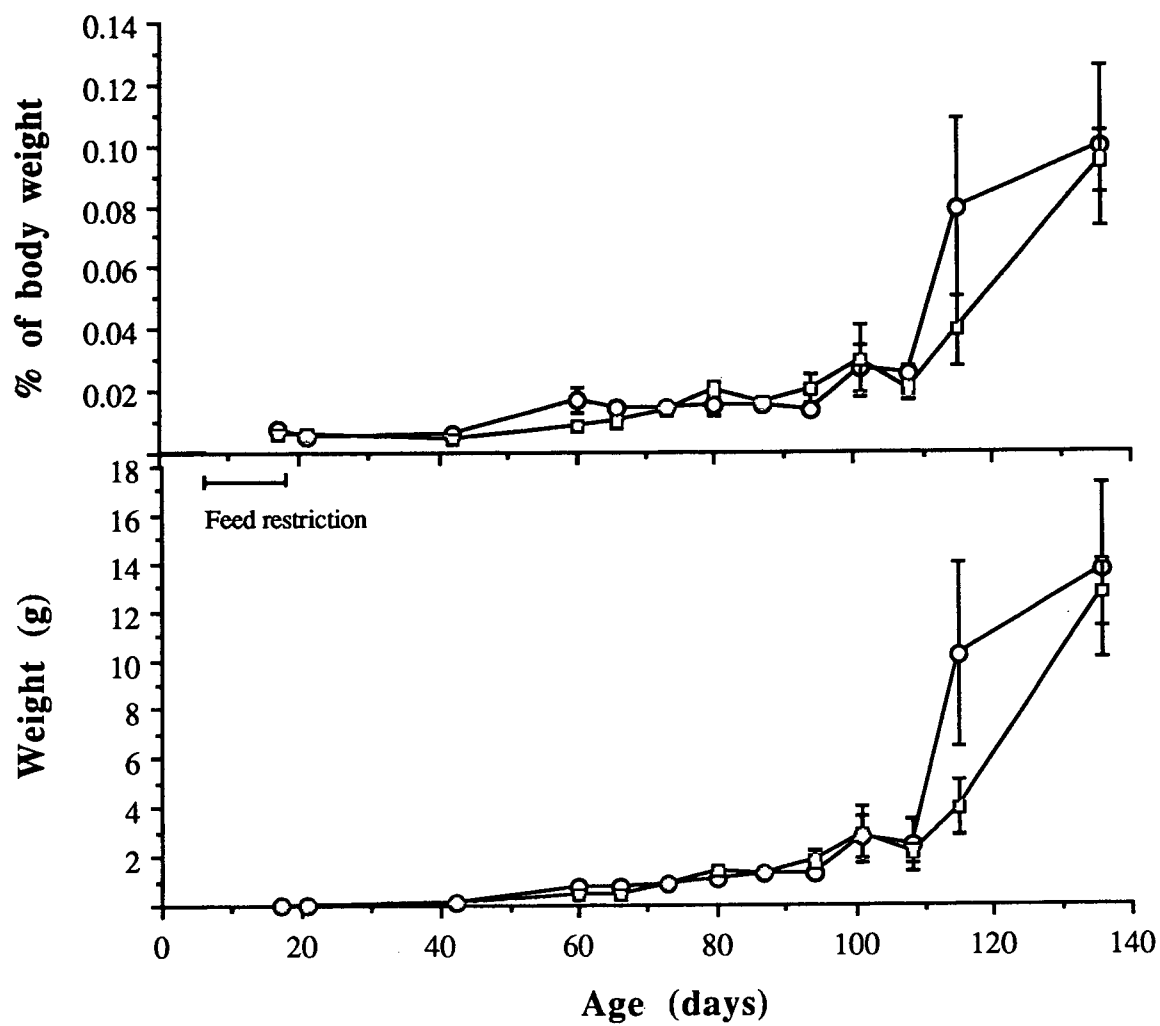


Fig. 1. Testes weight in turkeys in grams (lower graph) and relative to body weight (upper graph). ○ , ad libitum-fed; □ , feed restricted.

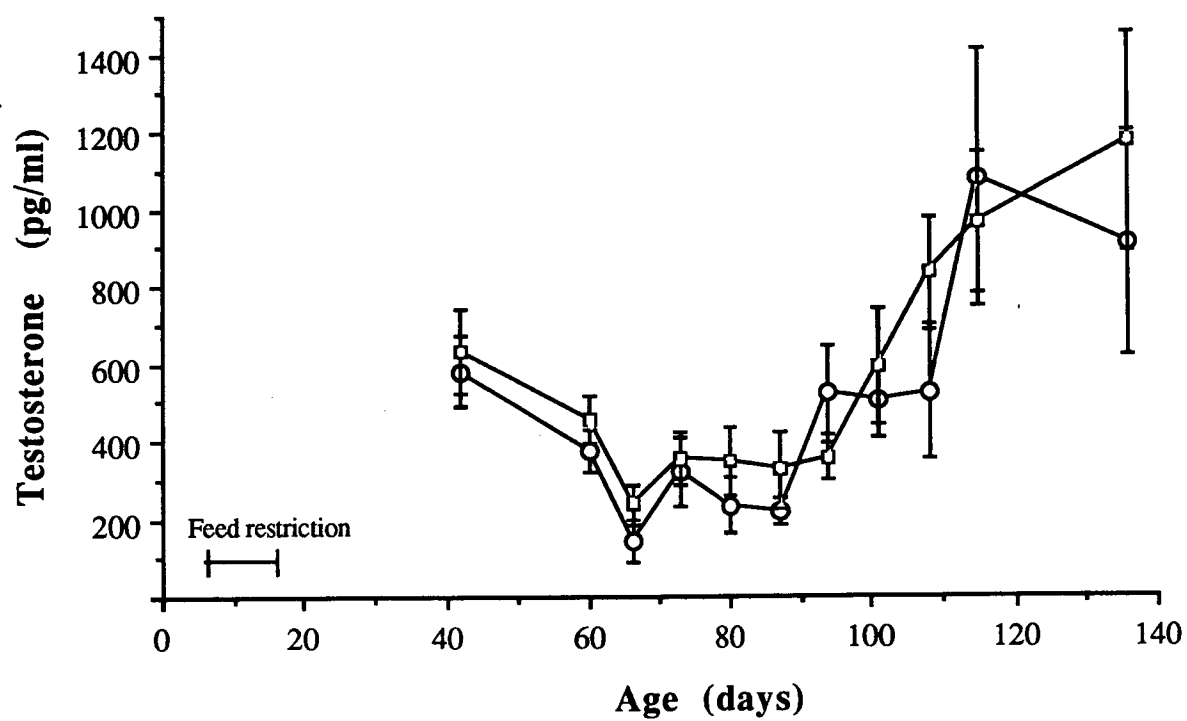


Fig. 2. Plasma testosterone in turkeys.  $\circ$ , ad libitum fed;  $\square$ , feed restricted.



## DISCUSSION

Bachman *et al.* (1987) showed that in chickens plasma concentration of dihydrotestosterone was less than 10% of that of testosterone. It may be further recalled that in the present assay system, dihydrotestosterone cross-reacted with the antibody at an efficiency of 39% of testosterone. Therefore, the measured levels of testosterone included a residual contribution of dihydrotestosterone. The testosterone values at puberty measured in this study are, however, remarkably similar to adult values measured by Cecil and Bakst (1988).

Testes development proceeds, at an early age slowly, similarly to the human (reviewed by Santen, 1986). However, unlike the human where testicular weight increases from the age of 6 years to adulthood in proportion to somatic growth, testes growth in the turkey proceeds from 42 days on at a rate exceeding that of body weight, specially at puberty. The initial decrease in testes weight as a consequence of feed restriction is well in accord with previous studies (Hulet and Brody, 1986). However, compensatory growth of the testes during refeeding results in a rapid restoration of normal testes weights.

The high plasma testosterone level early in postnatal life is similar to that observed in humans (Winter *et al.*, 1976), and may be of extragonadal origin. The time-dependent reduction in plasma testosterone is opposite to the direction of the temporal changes in testicular development. This trend and the earlier increase in testosterone than testes weight, is consistent with a relative independence of the control of testosterone secretion from that of the bulk of the testes weight, the spermatogenic tissue.

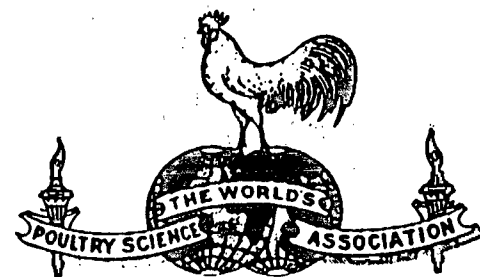
The higher testosterone level of the feed-restricted-refed turkey is in agreement with the early observations on the rat (Howland *et al.*, 1975). Moreover, the present results

show that in the turkey the high testosterone concentration is sustained for a long period after feed restriction and may be responsible for at least part of the observed compensatory growth response in males.

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THE EFFECT OF QUANTITATIVE FEED RESTRICTION IN YOUNG POULTS  
ON THE PERFORMANCE OF MEAT-TYPE TURKEYS

M. Lecher and coworkers - Kibbutz Ma'ale Gilboa  
I. Plavnik - Volcani Center, Agric. Res. Org.

The idea of a mild feed restriction in poults during their second week of age has been proposed and studied by Plavnik et al. This new management tool was applied on 5 flocks in the turkey farm of Kibbutz Ma'ale Gilboa, in order to test it under commercial conditions, with emphasis on studying the effects of such feed restriction on (1) body weight at marketing (in spite of the initial loss of weight gain), (2) feed utilization and (3) the incidence of leg problems.

The brooder house was divided into 2 equal rooms, in one of which feed restriction was employed (the room involved was changed from flock to flock). The latter was performed from 8 to 15 days of age in males and from 8 to 14 days in females. The actual amount of feed allotted depended on the weight of the poults at one week of age and on the planned marketing age. At the end of the feed restriction period the male poults averaged about 82% of the weight of the controls and the females about 88%. After brooding, the turkeys were transferred to 4 sheds, 2 sheds per treatment.

### Results

The following table is based on only 3 flocks, since in one trial the control birds suffered severely from turkey coryza and the results of the 5th flock have not yet been summarized.

As shown by the data presented in the table, early short-term and mild feed restriction did not affect marketing weights, improved feed utilization by 2.9% and the production index by 8.6%, and decreased mortality and the incidence of leg problems.

Flock No.	74		80		81	
Hatch	Sept. 1989		Oct. 1990		Dec. 1990	
Treatment	Exptl.	Control	Exptl.	Control	Exptl.	Control

**Mortality**

Brooder house	11.7	17.8	2.2	2.2	2.6	3.2
Growing shed	4.6	4.6	5.5	8.5	3.5	4.5

**Marketing****Males**

Age (days)	134	138	135	137	141	140
BW (kg)	15.35	16.05	15.77	15.96	15.30	15.39

**Females**

Age (days)	103	101	101	103	104	102
BW (kg)	7.94	7.98	7.76	7.91	7.29	7.17

Feed utilization	2.71	2.80	2.82	2.83	2.78	2.94
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Production index	364	322	367	356	379	343
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**Effect of Feed Restriction on Growth, Feed Efficiency,  
Carcass Composition and Metabolic Hormones in Chickens and  
Turkeys**

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## USDA - FINAL REPORT

A series of experiments were conducted to further evaluate the effect of early in life feed restriction on feed efficiency and carcass composition in broiler chickens. In addition, a series of experiments were conducted to determine the extent of metabolic hormone adaptations to feed restriction in broiler, meat-type chickens and turkey poults. In this report each experiment will be reported individually. Manuscripts, reporting the results of these experiments, are in various stages of preparation.

## EXPERIMENT 1.

INTRODUCTION. Early work by Plavnik and Hurwitz (1985) and Plavnik et al., (1986) demonstrated that the most efficient compensatory growth occurred after a 6-day restriction period beginning when the chicks were 6 days of age. Longer restrictions of 10,12, 14 and 28 days did not result in compensatory growth patterns with regards to weight gain. Some of this data showed that while compensatory gain was somewhat reduced, there was a suggestion of continued improvement in feed efficiency and further decreased abdominal fat. The present study was initiated to investigate the effects of two restriction periods, 6 and 12 days, on weight gain, feed efficiency, body composition, and to examine the changes in body composition at weekly intervals during a 56 day growth period.

MATERIALS AND METHODS. Two hundred and sixteen day old male Ross broiler chicks were raised in battery brooders and fed a standard starter diet (Table 1) until 6 days of age. On day 6 chicks were divided into 3 treatment groups of 8 replicates (9 chicks/replicate) each. The treatments were as follows: control, restricted (6 days), and restricted (12 days). Restriction was conducted as previously described (Plavnik et al., 1986), i.e., 40 kcal ME/chick/day. At the end of restriction period, chicks were fed ad lib until the end of the 56 day trial. The starter diet (Table 1) was fed until chicks were 28 days of age at which time the grower diet was fed (Table 1). Starting at day 7 of age and continuing at weekly intervals to 56 days of age, one chick from each treatment replicate (a total of 8 chicks/treatment) was randomly selected for body composition determination.



TABLE 1. COMPOSITION OF DIETS.

Ingredients	Diets	
	Starter	Grower
	-----	-----
Soybean meal (49%)	33.00	24.22
Fish meal	3.00	-----
Ground corn meal	56.70	70.60
Corn oil	3.60	1.50
Dicalcium phosphate	1.80	2.00
Limestone	1.10	1.00
DL Methionine	.19	.36
Iodized salt	.25	.25
Vitamin mix	.25	.25
Mineral mix	.10	.10
Calculated analysis		
Crude protein %	23.0	18.1
ME kcal/kg	3106	3088
Lysine %	1.3	.9
Sulfur amino acids %	.9	1.0
Calcium %	1.0	.9
Phosphorus %	.8	.7

All calculations made using NRC, Nutrient Requirements for Poultry, Eighth Edition, 1984.

In preparation for chemical analysis for body composition, chickens were weighed, eviscerated, the viscera emptied and the empty carcass, viscera and abdominal fat pad weighed. From 3 weeks of age to the end of the experiment, all chickens were picked prior to evisceration. The empty carcass plus fat pad were frozen, autoclaved, and homogenized. Dry matter, ash and nitrogen were determined according to AOAC procedures. Fat content of viscera and carcass aliquot was determined according to the method of Folch et al.(1957).

SUMMARY OF RESULTS. 1. As shown in Figure 1, eight week live weight gain for both 6 and 12 day restricted chickens exceeded that of control fed chickens. Also five and six week gains of 6 day restricted chickens exceeded that of control birds during the same periods. Chickens in all treatments exhibited a plateau of gain between 4 and 6 weeks. Chickens restricted for 12 days had the greatest weekly weight gain, 708 g, during the last week of the experiment.

2. Table 2 presents the overall live weight gains from 14-56 days and 21-56 days. The 12 day restricted chickens gained less ( $p<.05$ ) than the other two groups from 14-56 days. Both 6 and 12 days restriction resulted in improved feed efficiency ( $p<.05$ ). There were no treatment differences in live weight gain in birds from 21-56 days, and both levels of restriction again improved feed efficiency ( $p<.05$ ). Results shown in Table 3 demonstrate that empty carcass weight differs at both 21 and 56 days ( $p<.05$ ), but carcass gain for the same period did not change.

3. Table 4 presents results that show that the effect of restriction on gains of carcass fat, ash and water from 21 through 56 days. Fat gain decreased in restricted chickens and carcass water gain increased ( $p<.05$ ) during the 35 day experimental period. Ash gain was unchanged. As shown in Table 5, only visceral tissue changed during the experimental period, with an increased weight gain of viscera in 12 day restricted chickens.

4. The weekly gains in carcass fat are presented in Figure 2. These results show that in the restricted chickens there is an unusual pattern of fat deposition following feed restriction. Fat gain in the control birds is substantially greater, during the period up to 7 weeks, than that observed in restricted chickens, but the pattern shows a dramatic increase in fat gain

TABLE 2. EFFECT OF 6 OR 12 DAY FEED RESTRICTION ON LIVE WEIGHT GAIN AND FEED EFFICIENCY.

Treatment	Live Weight Gain		
	14-56 d --g--	F/G	21-56 d --g--
Control	2805	2.37 <sup>a</sup>	2489
6 d Restriction	2793	2.04	2495
12 d Restriction	2304 <sup>a</sup>	1.97	2397

<sup>a</sup> Significant Treatment Effect (P<.05)

2.46<sup>a</sup>

2.12

1.97

TABLE 3. EFFECT OF 6 OR 12 DAY FEED RESTRICTION ON CARCASS WEIGHT GAIN.

Treatment	Carcass Weight		Total Gain
	21 d	56 d	
	----- g -----		-----
Control	491 <sup>a</sup>	2618 <sup>a</sup>	2127
6 d Restriction	309 <sup>a</sup>	2430 <sup>a</sup>	2121
12 d Restriction	118 <sup>a</sup>	2118 <sup>a</sup>	2000

<sup>a</sup> Significant Treatment Effect ( $P < .05$ ).

TABLE 4. EFFECT OF 6 AND 12 DAY FEED RESTRICTION ON CARCASS FAT, ASH AND WATER GAIN FROM 21-56 DAYS.

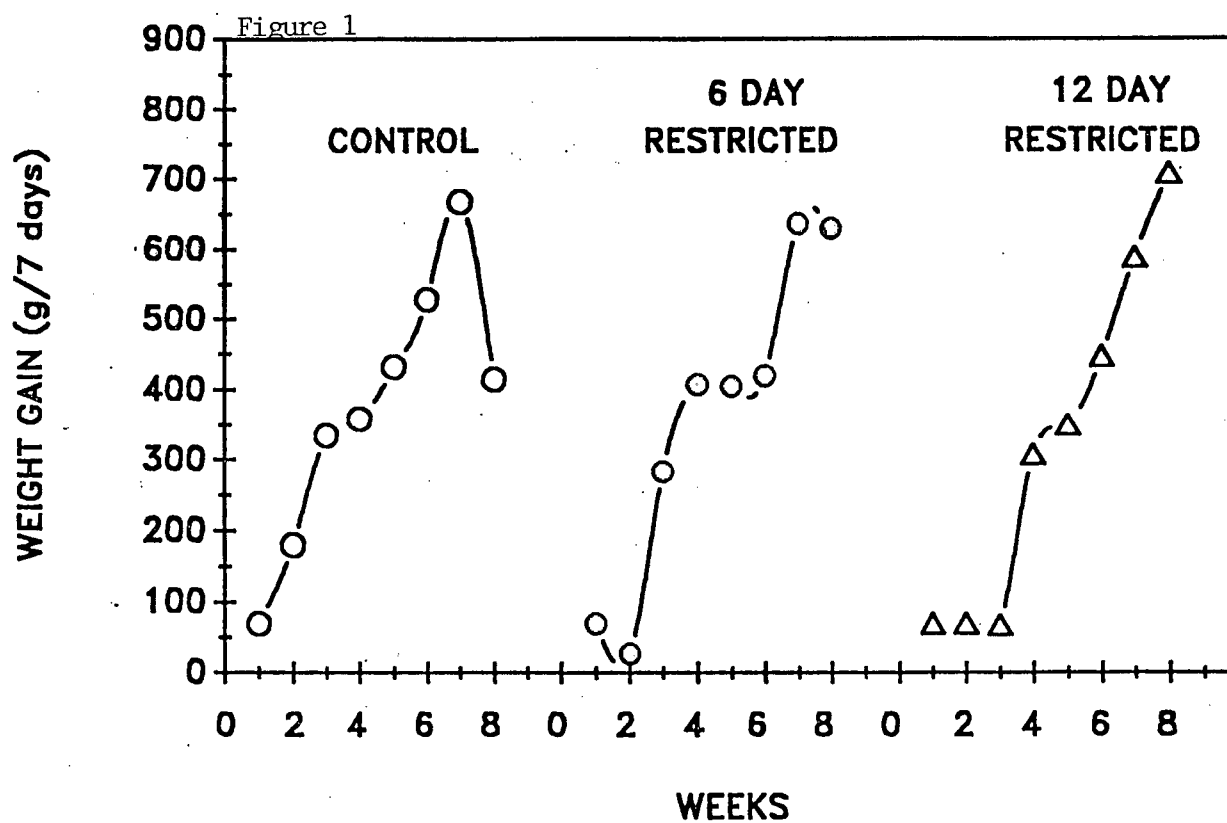
Treatment	Fat	Ash	Water
	----- g/kg carcass gain -----		
Control	171	33	635
6 d Restriction	157	30	644
12 d Restriction	158	31	652 <sup>a</sup>

<sup>a</sup> Significant Treatment Effect ( $P < .05$ ).

TABLE 5. EFFECT OF 6 AND 12 DAY FEED RESTRICTION ON GROWTH OF VISCERA, LIVER AND ABDOMINAL FAT PAD FROM 21 TO 56 DAYS.

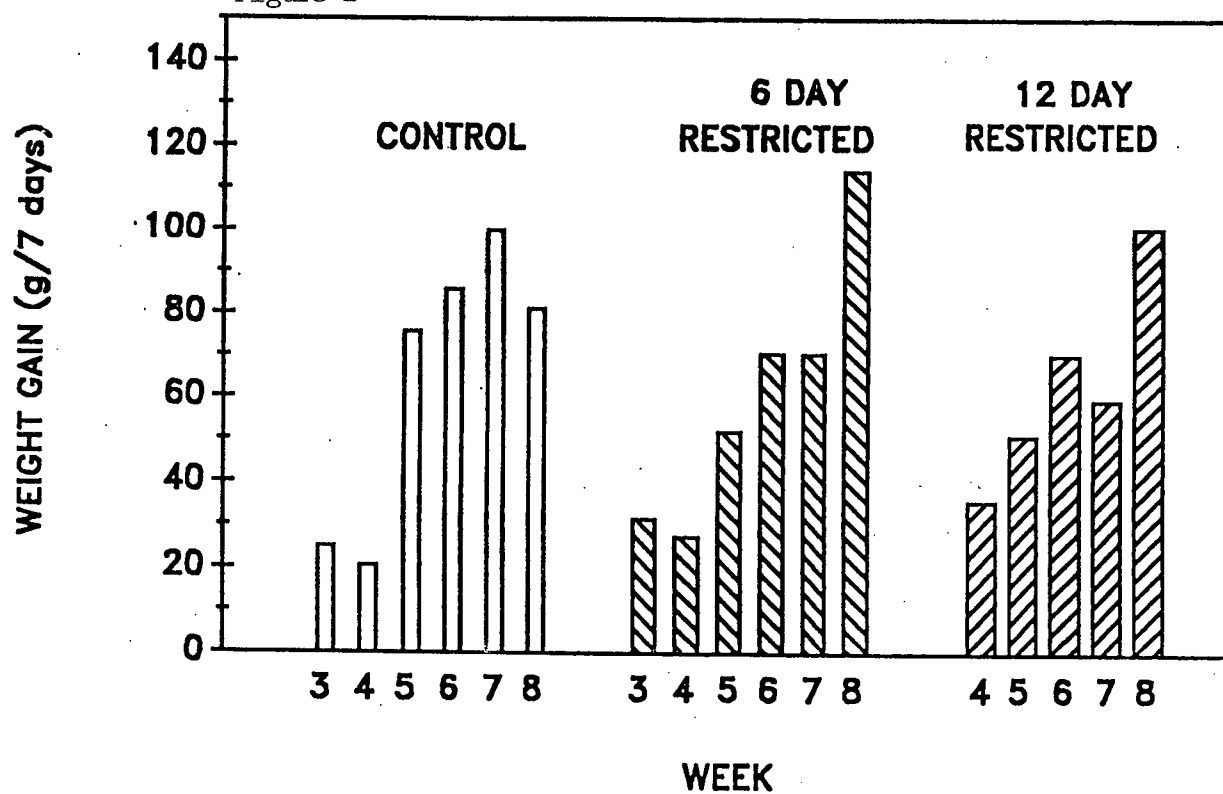
Treatment	Viscera		Liver		Fat Pad	
	g	g/kg BW	g	g/kg BW	g	g/kg BW
Control	124	58	51	24	43	20
6 d Restriction	136	64	50	24	44	21
12 d Restriction	158 <sup>a</sup>	80 <sup>a</sup>	58	29	37	18

<sup>a</sup> Significant Treatment Effect ( $P < 0.05$ ).



LIVE WEIGHT GAIN, G/BIRD/WEEK, OF BROILER CHICKS FED DIETS AD  
LIBITUM AND RESTRICTED.

Figure 2



FAT GAIN, G/BIRD/WEEK, OF BROILER CHICKS FED DIETS AD LIBITUM AND RESTRICTED.



in restricted birds during the final week of the experiment. Fat gain during this final week in the restricted chicks equaled or exceeded the highest level of gain in the control birds. This observation is particularly important since overall gain in carcass fat (Table 4) decreased in restricted chickens.

CONCLUSIONS. These results demonstrate that there is no practical advantage in increasing early feed restriction from 6 to 12 days. However, both periods of early feed restriction change the composition of tissue gain, and as demonstrated by the weekly changes in fat gains, the patterns of those tissue component gains may be dramatically different from that suggested by just measuring overall changes in tissue composition. Restriction appears to depress lipogenic activity immediately following restriction, but fat synthesis may then exceed normal levels later in the growth cycle.

## EXPERIMENT 2.

INTRODUCTION. As the potential of using early feed restriction to make significant improvements in feed efficiency and reducing carcass fat became evident. However, the response to early feed restriction has been equivocal. It has been suggested that one plausible explanation for the varied response to early in life feed restriction was the effect of genetic background. To investigate that possibility, the effect of early feed restriction on four commercial strains of meat-type broiler chicks was conducted.

MATERIALS AND METHODS. For this study day old male chicks were obtained from 4 suppliers of broiler chicks: Ross, Arbor Acres, Cobb and Petersen. Feed restriction was carried out as described in Experiment 1. Body weight determinations and feed intakes were determined weekly. Blood samples were analyzed for growth hormone and insulin-like growth factor-1 (IGF-1) at selected intervals prior to, during and after feed restriction.

RESULTS. As shown in Table 6 final body weights between control and restricted chicks were not different except for Petersen chicks. Within this strain, restricted chicks were not able to fully compensate in body weight gain by 56 days of age ( $P < .05$ ). It is unknown as to why chicks from this particular strain are not as responsive to feed restriction as others.

Table 6  
Effect of 6-day feed restriction on body weight gain (g) from 6-56 days  
of age in 4 broiler strains

Treatment	Strain			
	1	2	3	4
Control	2805	2324	2538	2516
Restricted	2705	2066 <sup>b</sup>	2456	2372

<sup>a</sup> Significant strain effect ( $P < .05$ )

<sup>b</sup> Significant treatment effect ( $P < .05$ )

Strain 1 = Ross

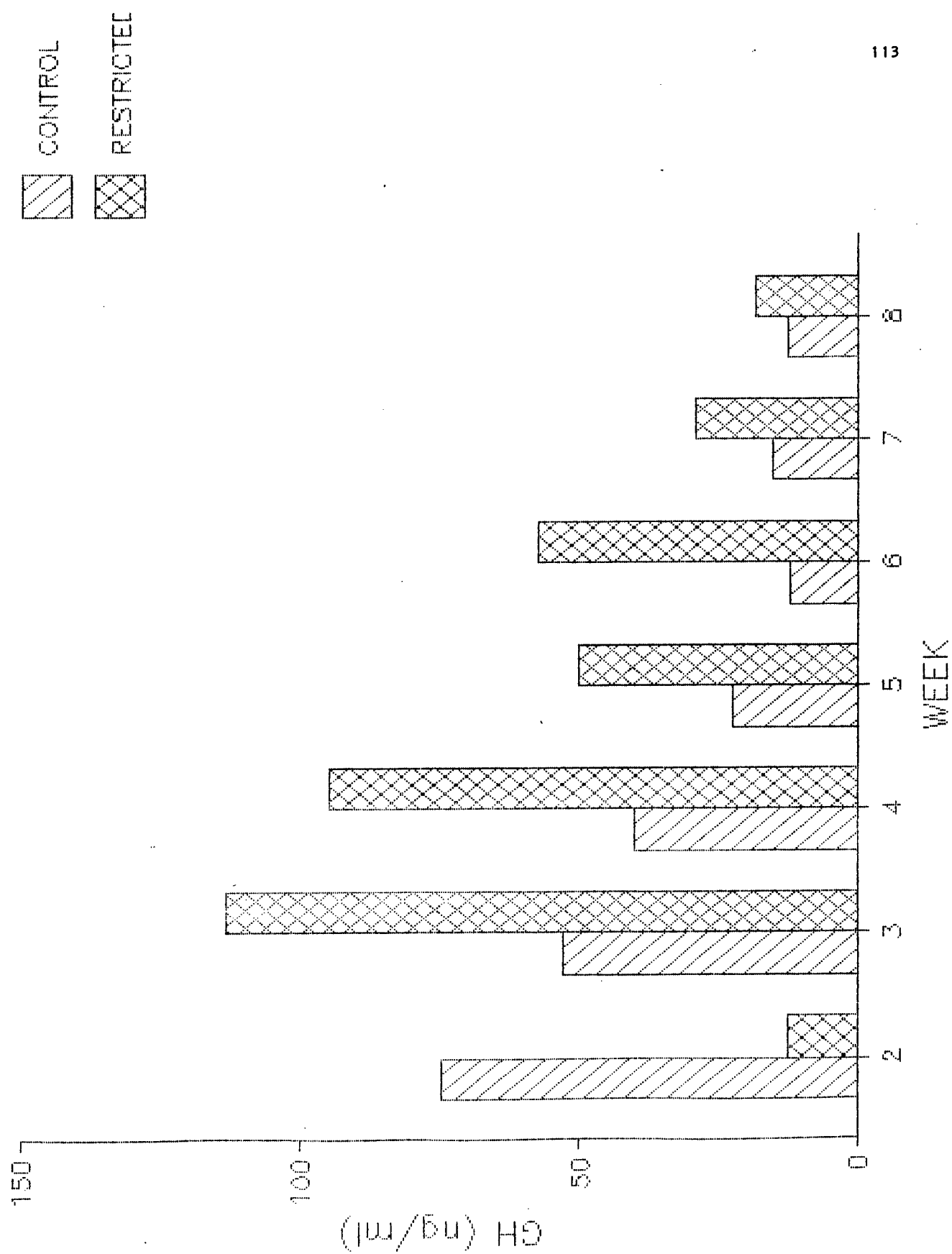
Strain 2 = Peterson

Strain 3 = Cobb

Strain 4 = Arbor Acres

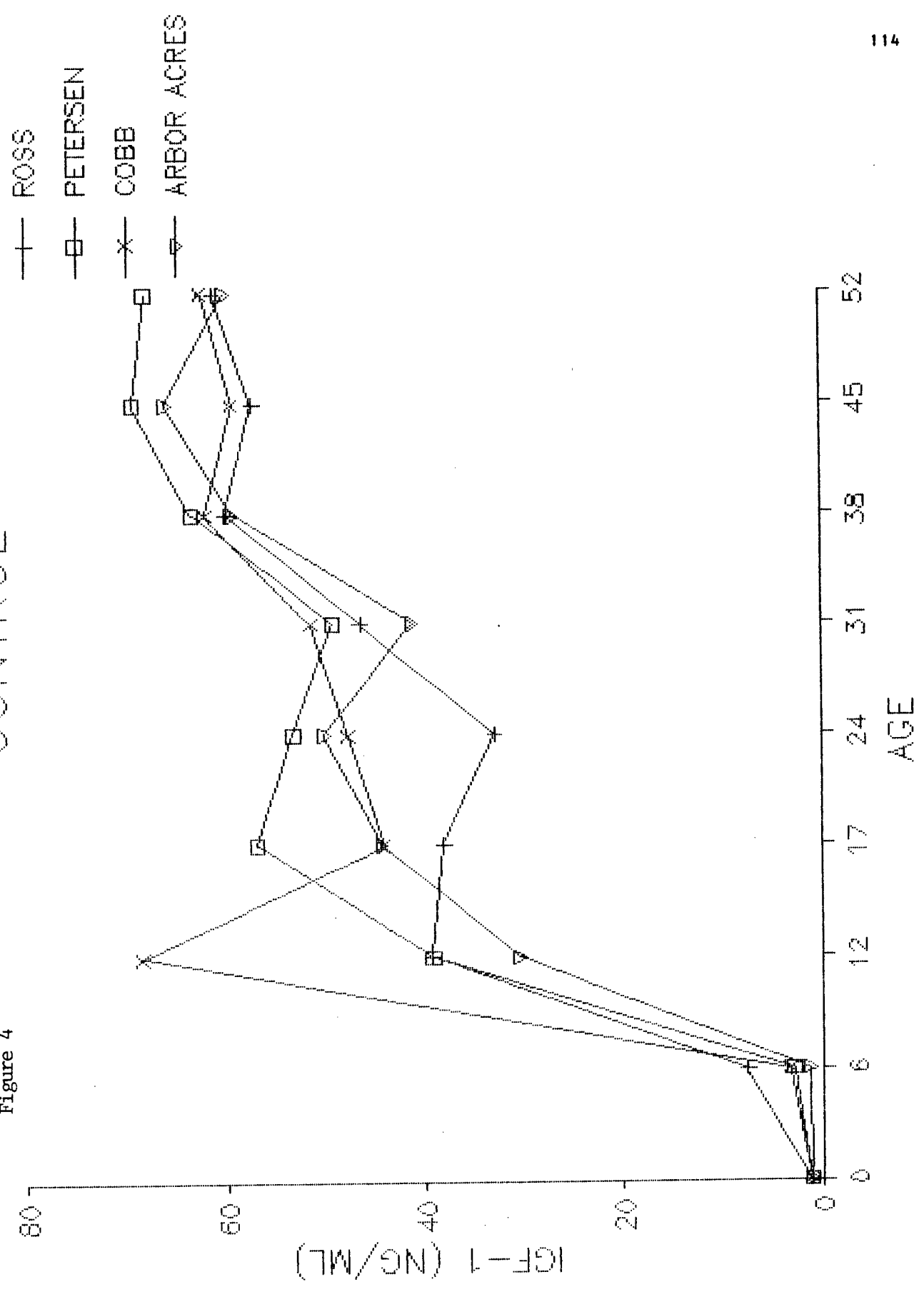
The growth hormone response to feed restriction is shown in Figure 3. The data shown represent an average of the four strains at each sampling time. There was no significant difference between strains in circulating growth hormone concentrations. However, a significant ( $P<.05$ ) treatment effect was observed. Growth hormone was suppressed by feed restriction (week 2), and the normal decline (controls) in circulating growth hormone was retarded in restricted chicks (weeks 3-6). Circulating insulin-like growth factor-1 levels were unaffected by feed restriction except for the period during feed restriction (Figures 4 and 5).

CONCLUSIONS. It is apparent that the response to early in life feed restriction is influenced to some extent by the genetic background of the broiler chicken. If early in life feed restriction is going to be utilized in commercial production systems, then a full evaluation of the type of bird used needs to be considered. It is clear that part of the response (compensatory growth and reduced carcass fat) to early in life feed restriction can be explained in part to changes in circulating growth hormone. Our laboratory has previously reported this phenomena (McMurtry et al., Comp. Biochem. Physiol. 91A:67. 1988). Early feed restriction delays the normal decline in growth hormone secretion associated with maturity. Feed restriction allows the bird to be exposed to elevated growth hormone levels for a longer period of time, possibly explaining the compensatory growth response observed in restricted birds and the reduction in carcass fat. This response is most likely not being mediated through the growth hormone/insulin-like growth factor-1 axis.



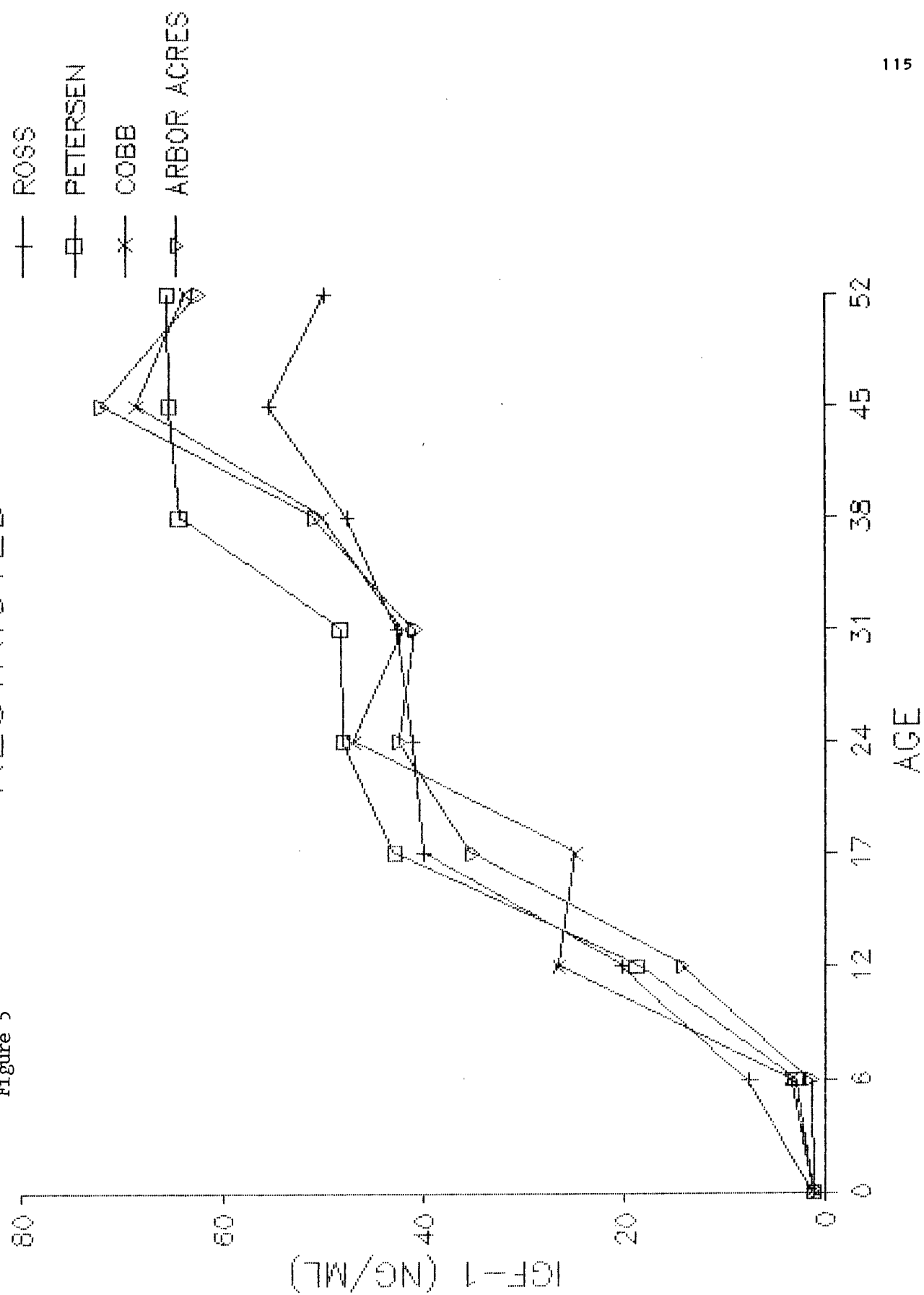
# CONTROL

Figure 4



# RESTRICTED

Figure 5



## EXPERIMENT 5.

**INTRODUCTION.** Previous studies by Hurwitz and Plavnik have shown that male turkey poults undergo two periods of rapid body weight gain. The first occurs at around 10-11 weeks of age, followed by a decline in growth at 12 weeks of age. A second growth spurt occurs around 15-16 weeks of age. The initial peak in growth is thought to be associated with the completion of skeletal growth. Increased muscle development, especially breast muscle, may account for the rapid growth occurring during weeks 15-16.

The purpose of this experiment was to examine whether there is an endocrine basis for the changes in growth rate discussed above. Endocrine profiles of growth hormone (GH) and insulin-like growth factor-I (IGF-1) were examined. In addition the effect of early feed restriction on body weight gain, feed efficiency and hormone profiles were examined.

**MATERIALS AND METHODS.** Three hundred-twenty British United male poults were divided into 4 groups: control, restricted 80%, restricted 70%, and restricted 50% of normal intake. Poults were restricted fed from day 7 to 17 post-hatch. Commercial diets that met the NRC guidelines for growing male turkeys were used throughout the experiment. Individual blood samples were collected at weekly intervals for 20 weeks, starting on day 7, eight to ten birds per treatment. Turkeys were cannulated at three time periods for serial blood sampling: 8-9 weeks of age, 11-12 weeks of age and 14-15 weeks of age. Birds were sampled three days post-operative. Blood was drawn at 15 minute intervals for 6 hr period at each age. Five-six birds from control and restricted 50% were cannulated. Liver samples were also collected at various ages for enzyme determinations.

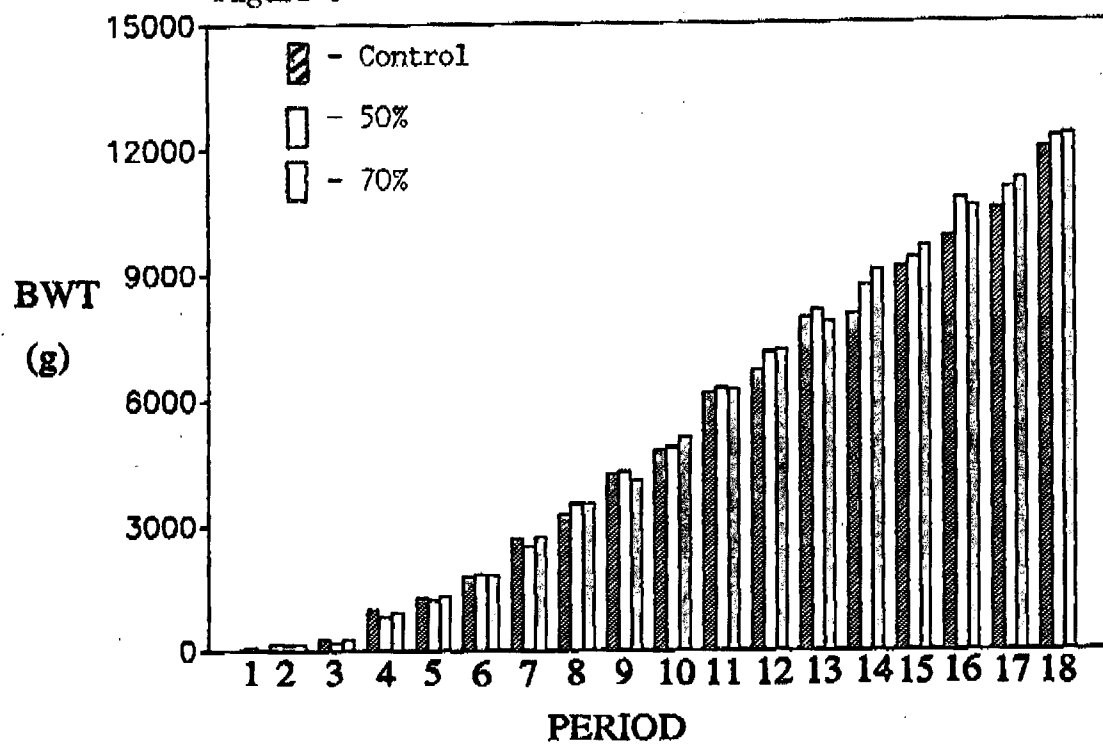
**RESULTS.** This experiment ended September, 1991, therefore, sample and data analyses are incomplete. The 80% restricted group performed the same as controls during the restriction period, so this group was eliminated from the experiment. As shown in Figure 6, the two restricted groups (70 % and 50% of intake) had greater body weights than controls at the end of the experimental period (19 weeks of age).

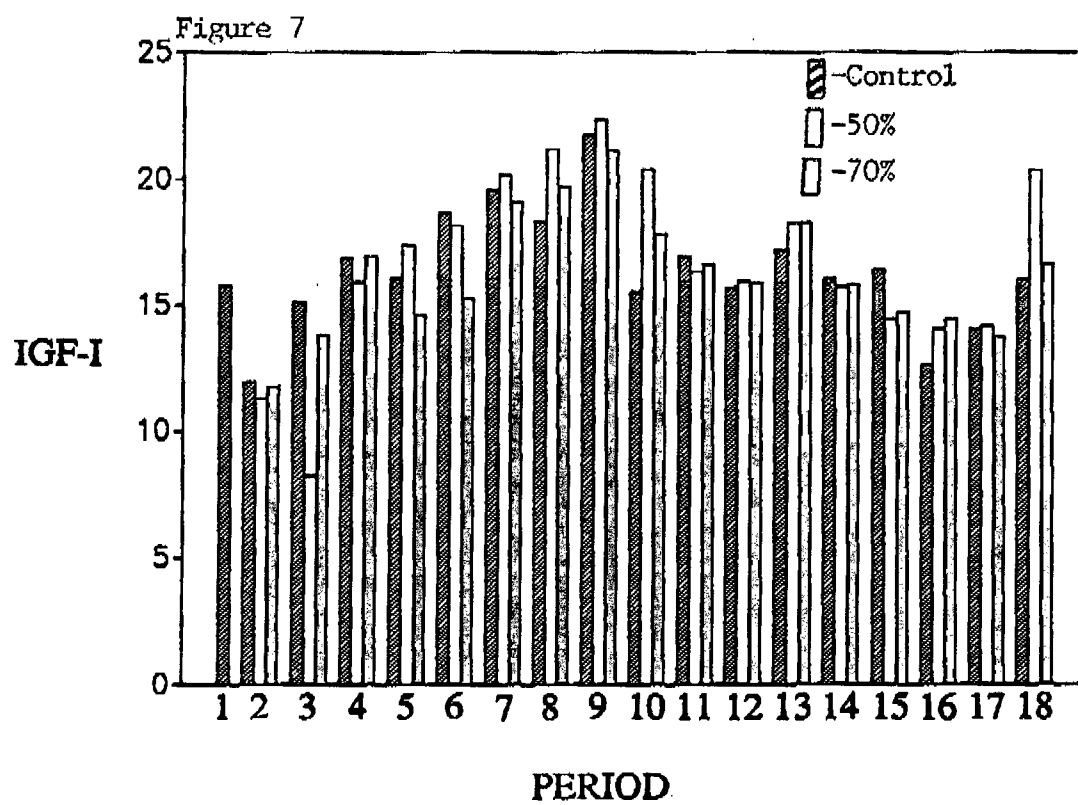
Weekly insulin-like growth factor determinations are shown in Figure 7. During restriction (period 3) the 50% of intake group had significantly lower IGF-1 concentrations, which returned to normal following refeeding (period 4). Overall, both restricted groups had greater IGF-1 concentrations over the experimental period than controls. Of interest, the elevated IGF-1 levels in the 50% group at period 18.

Samples to be analyzed include growth hormone determinations on the weekly samples and the serial bleedings. Liver enzymes will be determined. Further statistical analyses will conclude this study.



Figure 6





## COMPARISON BETWEEN CONSTANT-PROTEIN, CALORIE-RESTRICTED AND PROTEIN-RESTRICTED, CALORIE-RESTRICTED DIETS ON GROWTH, *IN VITRO* LIPOGENESIS AND PLASMA GROWTH HORMONE, THYROXINE, TRIIODOTHYRONINE AND SOMATOMEDIN-C (Sm-C) OF YOUNG CHICKENS

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**Abstract**—1. We studied the effects of calorie-restricted, constant-protein and calorie-restricted, protein-restricted diets on growth and *in vitro* metabolism of male chickens from select (Cobb Line 500) and byproduct (Cobb female line) lines of broiler chickens.

2. Chickens consumed 40, 60, 80 or 100% of a prescribed formula for dietary energy (body weight in  $g^{0.70} \times 16.7$  kJ) in the presence of set (CEP) or varied dietary protein (VEP).

3. Chickens fed VEP were heavier ( $P < 0.05$ ) at all energy intakes than chickens fed CEP. Slope analysis of data for *in vitro* lipogenesis showed a significant difference between the two treatment series.

4. Plasma growth hormone was inversely related ( $P < 0.05$ ) to Sm-C. Growth hormone levels were greater in chickens on a low plane of energy nutrition (40%) than on the maximum plane (100%).

5. Plasma Sm-C levels (pooled across energy series) were greater in the select than in the byproduct line. There were no differences in plasma  $T_3$  between the two lines. There was a significant increase ( $P < 0.05$ ) in  $T_3$  and a decrease in the  $T_4/T_3$  ratio accompanying an increase in dietary energy.

6. Restricting dietary carbohydrate and protein compromises anabolic processes more than restricting carbohydrate alone.

### INTRODUCTION

Defining the exact roles of protein *per se* and carbohydrate calories may be difficult because the addition of protein to a diet normally involves deleting carbohydrate. Likewise, addition of carbohydrate to diets involves substitution of the carbohydrate for some other nonprotein energy source. In most cases, this technique involves substitution of carbohydrate for fat. An effect noted under these conditions may involve a sparing of resultant protein for anabolic processes rather than its use as a substrate for synthesis of glucose for glucose-dependent tissues.

Nutritional deficiencies and the therapy of these deficiencies modulate plasma somatomedins which mediate the effects of growth hormone (Prewitt *et al.*, 1982; Maes *et al.*, 1984). These somatomedins (also known as insulin-like growth factors I and II; Sm-C and Sm-A) are required for both skeletal and muscular growth and may be responsible for repartitioning of dietary energy from fat to lean tissue growth. In addition, protein and nonprotein calories are required to maintain IGF homeostasis (Daughaday, 1981). Cree and Schalch (1985) reported that a diet limiting in an essential amino acid (lysine) reduced somatomedins in rats. Schalch and Cree (1985) reported a similar finding in total nutrient-restricted rats. Yang *et al.* (1987) expanded upon these two

previous studies by restricting dietary energy and maintaining a constant protein intake and found a similar decrease in somatomedins. Thus, circulating somatomedins reflect the two conditions responsible for changes in protein metabolism, dietary amino acid balance and the ratio of dietary energy to protein.

In a previous study (Rosebrough *et al.*, 1988), we found an inverse relationship between the growth of chickens from 14 to 28 days and subsequent growth hormone levels. In contrast, we found a highly significant relationship between plasma Sm-C and relative breast muscle size (breast muscle as a percent of body weight). We are unsure if Sm-C merely reflects states of protein anabolism in the chick or if the hormone plays an active part in protein accretion. Furthermore, we could not adequately define the role of protein and energy in this study because of the confounding of carbohydrate and protein contents of the diets used in the study.

We conducted the present study to define what effects dietary energy and protein restrictions have on endocrine regulators of growth. The findings from previous studies in our laboratory suggest that although energy restrictions may decrease circulating hormone levels and change rates of *de novo* lipogenesis, these effects require certain levels of dietary protein for expression.

## MATERIALS AND METHODS

## Diets

We formulated a diet containing 200 g protein/kg diet which served as the basal diet for the constant energy:protein series (CEP). The restriction series was based on body weight in  $g^{0.7} \times 16.7 \text{ kJ} \times 100, 80, 60$  or 40%. We also formulated low-protein (50 g protein/kg) and high-protein (750 g protein/kg) diets which were mixed to provide a constant protein intake with a varied energy:protein intake ratio (VEP). A constant amount of dietary protein was fed to all groups in VEP as opposed to a varied amount of protein in CEP. The composition of the basal diets is shown in Table 1.

## Animals

Male broiler chickens were purchased from either a select (Line 500) or nonselect (female line) from Cobb which is a participant in the National Poultry Improvement Plan. As such, chickens were certified as *Pullorum*, *Typhoid*, *Mycoplasma meleagridis* and *Mycoplasma gallisepticum* free. All chickens were held under a quarantine which was certified by the station veterinarian and observed daily for healthiness. One authorized animal caretaker was assigned to maintain chickens over the course of the experiments. In addition, the research protocols were approved by the Beltsville Agricultural Research Center Poultry Care Committee. Chickens were raised under common starter conditions until 14 days of age (average weight 300 g) and then randomly assigned to dietary treatment groups for a 14 day experimental period. Each dietary treatment was replicated four times and each replicate consisted of a pen of six chickens. A preliminary experiment indicated that the formula body weight in  $g^{0.7} \times 16.7 \text{ kJ}$  described median daily voluntary feed intake of chicks growing from 14 to 35 days of age. Chicks were weighed every 3 days and feed intake adjusted according to growth of the group fed the 200 g protein/kg diet at body weight in  $g^{0.7} \times 16.7 \text{ kJ}$ . The diet formulations are shown in Table 1. Chickens were raised in Petersime battery brooders in an environmentally controlled room maintained at 22°C and fed their respective diets until the termination of the experiment. A 12 hr light-dark cycle (0600–1800 hr light; 1800–0600 hr dark) was maintained, and water was allowed on an *ad libitum* basis.

## Assays

A portion of each liver was homogenized in 50 mM HEPES–3.3 mM mercaptoethanol (pH 7.5) and centrifuged for 60 min at 50,000 g. The supernate was stored at –80°C until the activities of malic enzyme (ME, EC 1.1.1.40; Hsu and Lardy, 1969), isocitrate dehydrogenase (NADP)

(ICD, EC 1.1.1.42; Cleland *et al.*, 1969), and fatty acid synthetase (FAS, no EC number; Mersmann *et al.*, 1973) were measured. Activities are expressed as micromoles of product formed per minute under the assay conditions (Rosebrough and Steele, 1985).

Another portion of each liver was sliced (50–75 mg) with a Stadie–Riggs hand microtome. Duplicate slices were incubated at 37°C for 2 hr in Hanks' balanced salts (HBSS; Hanks and Wallace, 1949) supplemented with 10 mM HEPES, 1% bovine serum albumin and 20 mM [2-<sup>14</sup>C] sodium acetate. The slices were then extracted and treated (Rosebrough and Steele, 1985).

Both T<sub>3</sub> and T<sub>4</sub> were estimated with commercially available kits (Immuchem Corp, Carson CA). Both T<sub>3</sub> and T<sub>4</sub> standards were prepared in charcoal-stripped chicken sera. Apparent somatomedin-C (Sm-C) was estimated by slightly modifying the method of Huybrechts *et al.* (1985) by using a human Sm-C radioimmunoassay (Nichols Diagnostics, San Juan Capistrano, CA). The assay was validated by noting both parallelism between a pool of chicken plasma and the standards and recovery of a known quantity of human Sm-C (Amgen) from the plasma pool (Fig. 1). Values for Sm-C were calculated on the basis of units per ml.

## Statistical analyses

Metabolic activities are expressed on the basis of relative liver size of the chicken (liver as percent of body weight  $\times$  metabolic activity per g liver) to account for changes in metabolic activity relative to independent changes in liver size. Values for chickens from a particular pen were averaged to derive a pen mean because we considered the pen of chickens subjected to a particular treatment as the experimental unit. Because of an initial lack of homogeneity of error variances for enzyme activities, data were subjected to natural log transformations to develop pooled estimates of error variances. The data were subjected to regression analyses by the method of least squares. Treatment effects were compared by noting significant differences among slopes (Remington and Schork, 1970).

## RESULTS

## Growth

Body weights are presented in Fig. 2 and the efficiencies of feed utilization in Table 2. Differences among treatment groups for final body weights and food utilization were similar at a 100% energy intake. Regression analysis gave a common slope ( $6.3 \times g^{-1} \times \text{dietary energy}^{-1}$ ) which did not differ significantly ( $P < 0.05$ ) from the slopes for each energy series within a line. In contrast, the intercept, which denotes an overall dietary treatment effect

Table 1. Composition (g/kg diet) of the diets

Ingredient	Dietary protein		
	50	200	750
Corn meal	450	700	
Soya-bean meal		100	127
Soya protein*	13	100	788
Glucose	450		
Corn oil		20	20
Sand	27	15	
L-Methionine		5	
Dicalcium phosphate	40	40	40
Limestone	10	10	10
Selenium premix†	1	1	1
Mineral premix†	1	1	1
Vitamin premix†	5	5	5
Iodized salt	3	3	3
Calculated energy level (MJ)	13.0	13.0	13.2

\*Alpha protien (900 g/kg crude protein); Nutritional Biochemical, Cleveland, OH.

†For a description of these premixes see Rosebrough and Steele (1985).

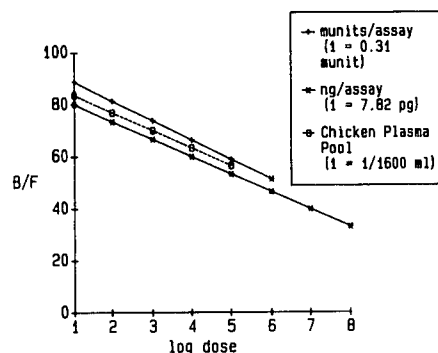


Fig. 1. Validation of the use of a heterologous assay to estimate somatomedin-C (Sm-C) in chicken plasma.

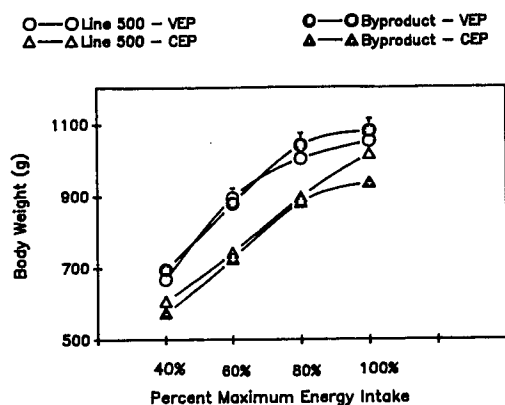


Fig. 2. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on growth of two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

pooled across lines, was significantly ( $P < 0.05$ ) different for CEP compared to VEP (340 g vs 481). Thus treatment differences were most noticeable during conditions of energy restriction.

#### *In vitro* lipogenesis

Comparisons of rates of lipogenesis revealed little effect of either diet or genetic background when chickens were restricted to 40% of the maximum allowed energy intake. As the dietary energy intake increased, lipogenic rates rose ( $P < 0.05$ ) in both lines of chickens (Fig. 3). In contrast to growth data, both dietary treatments and lines affected rates of *in vitro* lipogenesis. The slope for by-products was greater than for the Line 500 chickens receiving either CEP ( $15.5 \times \mu\text{mol}^{-1} \times$  vs  $9.2 \times \mu\text{mol}^{-1} \times$  dietary energy $^{-1} \times \text{kg body wt}^{-1}$ ) or VEP ( $13.4 \times \mu\text{mol}^{-1}$  vs  $11.5 \times \mu\text{mol}^{-1} \times$  dietary energy $^{-1} \times \text{kg body wt}^{-1}$ ).

*Sm-C*. There were slight though significant differences in slopes for energy quality (CEP vs VEP;  $1.24 \text{ munits} \times \text{ml}^{-1} \times$  dietary energy $^{-1}$  and

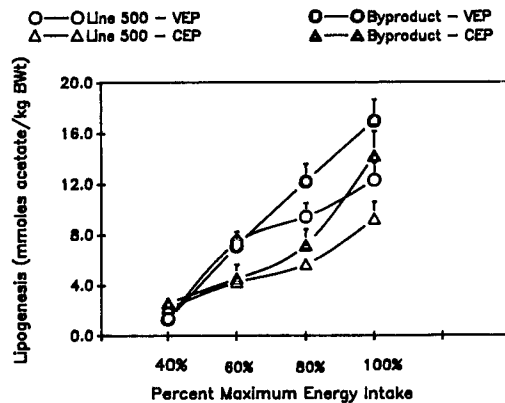


Fig. 3. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on lipogenesis ( $[2-^{14}\text{C}]$  acetate incorporation into hepatic fatty acids by liver explants *in vitro*) in two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

$1.72 \times \text{ml}^{-1} \times$  dietary energy $^{-1}$ , respectively) when data were pooled across the nonsignificant effect of line (Fig. 4). When compared at partial energy intakes ( $< 80\%$ ), the regression lines were parallel, but lower at all points for CEP. All slopes were tested for the effect of dietary energy and were found to be significantly ( $P < 0.05$ ) greater than zero.

*GH*. Immunoreactive GH concentrations decreased significantly ( $P < 0.05$ ) in all treatment groups as dietary energy increased from 40 to 100% (Fig. 5). Comparisons of slopes revealed no line effect. Pooling data across lines revealed a significant ( $P < 0.05$ ) effect of energy quality on GH (CEP vs VEP;  $-265 \text{ pg} \times \text{ml}^{-1} \times$  dietary energy $^{-1}$  and  $-338 \text{ pg} \times \text{ml}^{-1} \times$  dietary energy $^{-1}$ , respectively). Figure 6 depicts the significant negative correlation between plasma GH and *Sm-C* noted in this experiment. It should be noted, however, that a third-order

Table 2. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on growth of two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) to give varied protein and energy intakes. Mean values with their standard errors for four pens per group

Dietary treatments			Select				Non-select			
			Body wt gain (g)		Wt gain/feed (g/g)		Body wt gain (g)		Wt gain/feed (g/g)	
Level	Energy/protein (kJ/g)		Mean	SE	Mean	SE	Mean	SE	Mean	SE
VEP	100%	65.1	754	25	0.78	0.03	741	24	0.77	0.02
	80%	54.7	704	14	0.80	0.02	704	23	0.89	0.03
	60%	43.4	596	12	1.03	0.02	580	12	1.00	0.02
	40%	30.8	369	9	0.97	0.02	396	15	1.04	0.04
CEP	100%	65.1	716	10	0.74	0.01	636	19	0.66	0.02
	80%	65.1	598	13	0.76	0.02	585	11	0.74	0.01
	60%	65.1	444	16	0.77	0.03	428	14	0.74	0.02
	40%	65.1	308	14	0.81	0.04	275	13	0.72	0.03
Analyses of variance										
Genetic background (Select vs non-select)			$P < 0.05$		NS		NS			
Dietary energy levels			$P < 0.01$		$P < 0.01$		$P < 0.01$		$P < 0.01$	
Energy quality (VEPS vs CEP)			$P < 0.01$		$P < 0.01$		$P < 0.01$		$P < 0.01$	

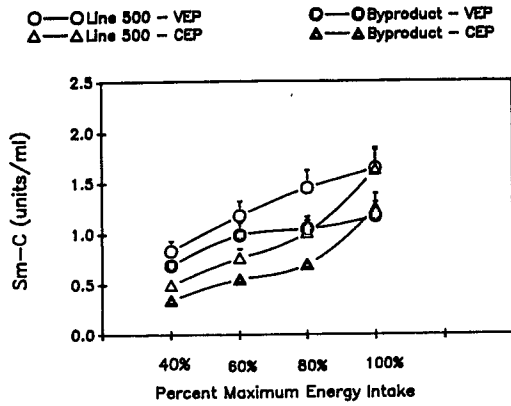


Fig. 4. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on plasma growth hormone concentrations in two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

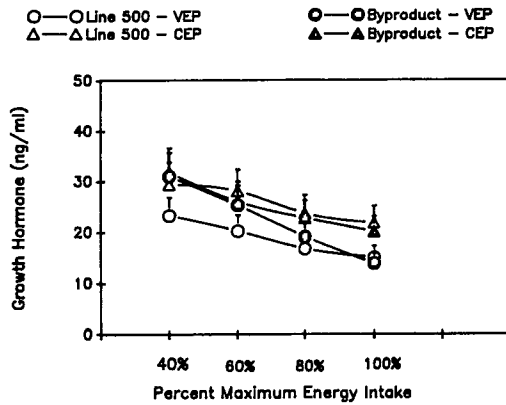


Fig. 5. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on plasma somatomedin-C (Sm-C) concentrations in two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

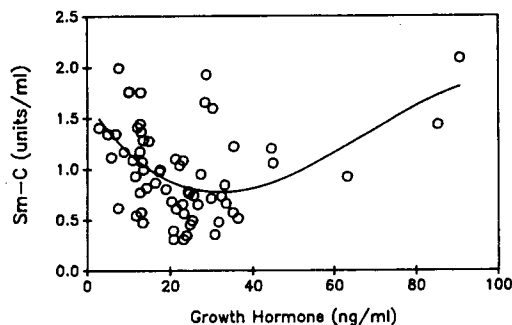


Fig. 6. Overall comparison of the relationship between plasma growth and somatomedin-C (Sm-C) in chickens.

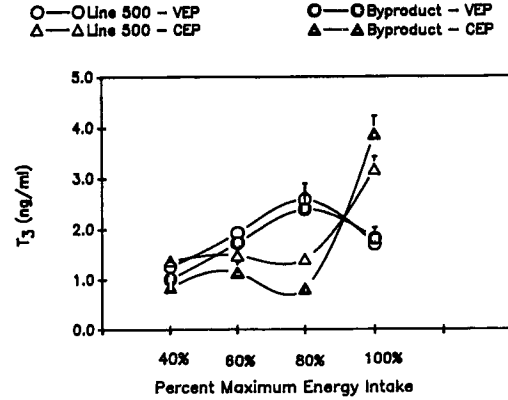


Fig. 7. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on plasma triiodothyronine ( $T_3$ ) concentrations in two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

polynomial best fit this data set and that a simple linear relationship was not significant.

**Thyroid hormones.** Triiodothyronine ( $T_3$ ) increased ( $P < 0.05$ ) in both VEP and CEP as dietary energy increased; however, there was no significant line effect (Fig. 7). The rate of increase for CEP ( $25.5 \text{ pg} \times \text{ml}^{-1} \times \text{dietary energy}^{+1}$ ) was significantly ( $P < 0.05$ ) than for VEP ( $21.8 \text{ pg} \times \text{ml}^{-1} \times \text{dietary energy}^{-1}$ ). Data for thyroxine ( $T_4$ ) is expressed as the ratio of  $T_4:T_3$  (Fig. 8). The ratio for all treatment groups was essentially equal at the 100% energy intake level. The slopes for CEP and VEP could not be compared because the ratio for CEP remained unchanged until energy was increased to 80%, while the ratio

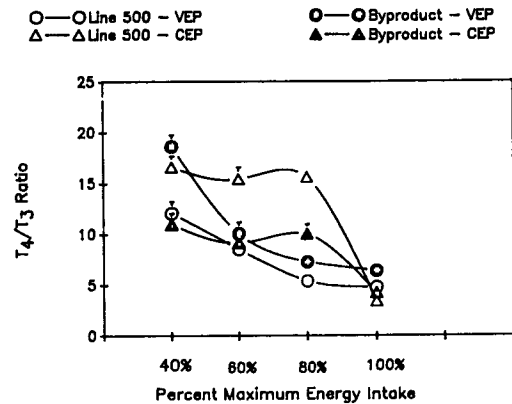


Fig. 8. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on the ratio of the ratio of thyroxine to triiodothyronine ( $T_4/T_3$ ) in two lines of chickens. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

decreased in VEP with the first increment of dietary energy (40–60%).

Figures 9–11 summarize the effects of lines and dietary treatment on the activities of certain enzymes implicated in *de novo* lipogenesis. None of the activities could be described by either first- or second-order plots, although all activities summed across lines increased with increases in energy (both VEP and CEP). Fatty acid synthetase activities for both energy series and lines were equal at the 100% intake level. Both lines and dietary treatments differed ( $P < 0.05$ ) at the 80% level (VEP > CEP; byproduct > Line 500). Malic enzyme activity was not affected by genetic background but was greater ( $P < 0.05$ ) in VEP than in CEP. Isocitrate dehydrogenase activity was inconsistent in both lines of chickens and did not appear to be under dietary regulation in the present study.

#### DISCUSSION

Species differences in responses to diet composition are interesting because we have speculated that carbohydrate and protein calories have specific effects on intermediary metabolism in the chicken. Comparing our findings with those of Tanaka *et al.* (1983) is difficult because in their studies, caloric intakes were not equal among their force-fed groups and the relationships among the energy constituents changed. None the less, the results of the present study seem to reflect those of Tanaka *et al.* (1983), which indicated that in the face of a constant carbohydrate intake, an increase in protein calories would result in a decrease in lipogenesis. Although many groups have reported that high-protein diets enhance lean tissue development in chickens, few groups have offered any biochemical logic for the decrease in lipogenesis accompanying the feeding of high-protein diets. Yeh and Leveille (1969) found an inverse relationship between the level of the dietary protein

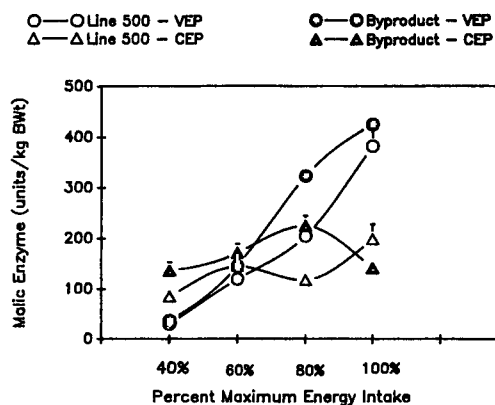


Fig. 10. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on hepatic malic enzyme activity (EC 1.1.1.40). Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

and the subsequent rate of *in vitro* lipogenesis and speculated that an increase in the dietary protein level decreased the flow of substrates through glycolysis and increased the production of glucose from substrates that were formerly in the pathways leading to fat synthesis. Yeh and Leveille (1969) also compared slopes in varied protein, iso-carbohydrate and varied carbohydrate dietary treatment groups and found slight differences in favor of protein as a regulator of lipogenesis. Interpretation of most results (including those in the present report) must be tempered with the knowledge that the formulation of diets always results in a compromise.

Schlach and Cree (1985) found that a reduction in the energy intake from fat calories decreased body

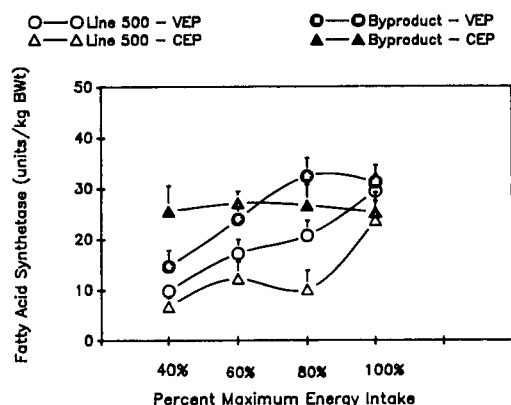


Fig. 9. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on hepatic isocitrate dehydrogenase (NADP) (EC 1.1.1.42). Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

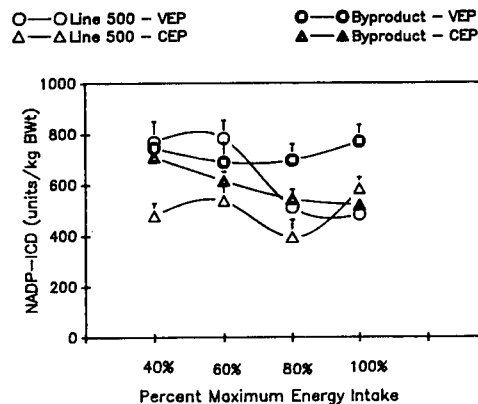


Fig. 11. Comparison between constant-protein, varied energy and varied-protein, varied energy feeding regimens on the activity of the fatty acid synthetase complex. Select (Cobb line 500) and non-select (byproduct) lines of chickens were fed daily either a constant amount of protein and graded levels of energy (VEP) (40, 60, 80 or 100%  $\times$  body weight  $g^{0.7} \times 16.7$  kJ) or graded amounts of a single diet (CEP) to give a varied protein and energy intake. Values presented are means and SEs for four observations per point.

weight and  $T_3$  and  $T_4$  in young rats. In contrast a later study from this same group (Yang *et al.*, 1987) indicated that although a reduction in carbohydrate energy also decreased body weight, there were no changes in either  $T_3$  or  $T_4$ . This study seemed to imply that in rats: (1) growth could be separated from thyroid function and (2) growth retardation was related to the quantity of the dietary energy rather than the quality of the energy sources in the diet.

Variation in product moments for an individual animal could block more subtle treatment differences because growth hormone (GH) release is pulsatile (Buonomo *et al.*, 1984; Vasilatos-Younken and Zarkower, 1986). Although stress may play a part in GH secretion in mammals, all birds were treated in a similar fashion: therefore, stress effects were presumably spread across all treatments. In contrast, circulating somatomedins (Sm-A and Sm-C) are known to be bound to specific binding proteins and are more stable indicators of growth status than GH. In a previous study concerning relative muscle size (RMS) in nutritionally deprived chickens, we found a positive correlation between RMS and Sm-C (Rosebrough *et al.*, 1988). In the present study, a significant relationship was observed between body weight and Sm-C. Because of the length of this study, growth *per se* cannot be separated from overall energy intake and subsequent Sm-C values. For example, maintaining a constant protein intake in the face of varied energy intakes (VEP) will result in a greater response in both body weight and Sm-C to dietary energy than if both dietary protein and energy are varied together (CEP).

At first, the apparent lack of a positive relationship between GH and Sm-C presents a conflict because of the putative control of Sm-C by GH (Roberts *et al.*, 1986). A large body of work indicates that low GH levels are found in rapidly growing birds (Burke and Marks, 1982; Stewart and Washburn, 1983). It is also apparent to us that natural levels of GH have a relationship to growth that is dissimilar to levels attained by artificial means. For example, Leung *et al.* (1986) and Vasilatos-Younken *et al.* (1988a, b) increased body weights of chickens with exogenous GH.

Sm-C may act selectively to regulate GH gene expression. Yamashita *et al.* (1986) found that stimulation of GH secretion by GHRH could be prevented by simultaneously treating cells with Sm-C. Furthermore, this group also reported that Sm-C suppresses both basal and GHRH-dependent GH secretion in pituitary adenoma cells. Several recent studies provide evidence for nutrient control of Sm-C and ultimately GH levels. Refeeding a diet containing nonessential amino acids increased Sm-C in rats less than did refeeding a diet containing essential amino acids (Isley *et al.*, 1984; Clemons *et al.*, 1985a, b).

Malic enzyme activity and  $T_3$  concentrations possibly support hormone action at the cellular level (Oppenheimer *et al.*, 1978). A depression in either  $T_3$  binding or circulating levels (as is the case in the present study) would result in decreased enzyme activity and subsequent *de novo* lipogenesis. Another plausible explanation is that ME activity reflects NADPH utilization and does not regulate fatty acid synthesis. The enzyme activities measured in this

study suggest that isocitrate dehydrogenase functions in both lipid and protein metabolism by providing both a residual capacity for the production of reducing equivalents during a period of decreased malic enzyme activity and a coreactant for transamination. In addition, competition may exist between acetyl CoA carboxylase and the aconitase-isocitrate dehydrogenase pathway for limited cytoplasmic citrate. Thus, the requirement for  $\alpha$ -ketoglutarate as a co-reactant for transamination during increased protein intake would depress citrate levels to a point where activation of acetyl CoA carboxylase would not be expected to occur. Clark *et al.* (1979) reported that avian acetyl CoA carboxylase did not appear to be under the control of phosphorylation-dephosphorylation as is the rat enzyme and that the avian enzyme is particularly sensitive to citrate levels. The report seems to offer some support for our contention of the role of high-protein diets as regulators of lipogenesis via citrate availability.

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***In vitro* Lipogenesis and Glucose Production in Chickens  
Exposed to Protein/ Amino Acid Restriction**

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## FINAL REPORT

There are findings characterizing physiological responses to meal feeding and intermittent feeding in rodents. Both these feeding regimens increase *de novo* lipogenesis, carcass and liver fat, and improve dietary energy utilization. Early reports showed that periodic feeding regimens increase *in vitro* lipid metabolism in chickens but do not improve energetic efficiency. We found that intermittent-feeding cycles or daily changes in the dietary protein level also change lipid metabolism. The alteration in metabolism is similar to the intermittent-feeding response noted in older chickens.

An overall comparison between the effects of diet composition and feeding regimens reveals that feeding low-protein diets as a part of an alternating-high-low-protein feeding cycle and refeeding fasted chickens result in similar responses. In both cases, *de novo* lipid synthesis increases. In contrast, feeding high-protein diets and fasting chickens decrease *de novo* lipogenesis. It is of interest if both these types of nutritional regimens can be used on a chronic basis to adapt intermediary metabolism in the broiler to further increase lipogenesis during the respective refeeding and low-protein phases of the two feeding programmes.

The purpose of the following section was to examine *in vitro* lipogenesis and glucose production in chickens subjected to chronic or acute bouts of either an intermittent- or alternating-protein feeding regimen. The overall hypothesis was that an alternating-protein diet series would result in a metabolic response similar to the refeeding response noted during the intermittent-feeding regimen. Circulating metabolic hormone levels were also measured during chronic or acute dietary treatment regimens. We were especially interested in the adaptation chickens subjected to repeated bouts of high-low-protein feeding and fasting-refeeding compared to chickens subjected to single bouts of these two regimens.

Broiler chickens growing from 7 to 28 d of age were fed: (1) a 210 g protein/kg control diet for the entire experimental period, (2) an intermittent regimen (210 g protein/kg diet for either 1 or 2 d followed by a 1-d fast) or 3) a daily change in the dietary protein level from 120 to 300 g/kg diet. Treatment variables examined were *in vitro* lipogenesis, glucose production, circulating concentrations of insulin, triiodothyronine (T<sub>3</sub>), and thyroxine (T<sub>4</sub>) to determine the effects of chronic or acute dietary

treatments. Feeding the 300 g protein/kg diet or withholding feed for 1 d decreased ( $P<0.05$ ) *in vitro* lipogenesis compared to controls. Feeding the 120 g protein/kg diet or refeeding a 210 g protein/kg diet for 1 or 2 d increased ( $P<0.05$ ) *in vitro* lipogenesis compared to controls. Glucose production was affected in the same manner. Fasting decreased ( $P<0.05$ ) plasma insulin and  $T_3$  and increased  $T_4$ . Both refeeding or a low-protein diet increased  $T_3$ . Refeeding increased and a low-protein diet decreased insulin. Chronic use (7-28 d of age) of either an alternating-protein or intermittent-feeding regimen caused greater responses compared to acute bouts (single cycle) of either of the regimens.

Although the level of dietary protein and of limiting amino acids influence both lean and adipose tissue development in broiler chickens, interpretation of effects can be confounded by dietary energy intake. Interpretation of the effects of both dietary carbohydrate and protein on lipogenesis in chickens is further complicated, even in isocaloric diets, by difficulties in dietary formulations. For example, an increase in dietary protein is normally accompanied by a decrease in the dietary carbohydrate level. The importance of dietary carbohydrate was illustrated by Clark *et al.* (1980) who found little relationship between the level of the dietary fat and *de novo* lipogenesis when

chicks were provided the same amount of dietary carbohydrate in all dietary treatments. In contrast, the role of dietary protein in the regulation of lipid metabolism in the chick appears to be more complex than that of dietary fat. For example, it can be shown that an improvement in dietary protein quality (addition of the limiting amino acid lysine to a low protein diet) depressed *de novo* lipogenesis in chicks. Confounding the role of amino acid supply as a regulator of lipogenesis in all birds was the observation that turkey poults overeat diets containing a wide calorie:protein ratio ( $>17:1$ ), even when these diets are supplemented with limiting amino acids to meet requirements (NRC, 1984). The net effect is still an increase in lipogenesis even in the presence of an ideal dietary amino acid profile.

Thus, in defining the mechanisms surrounding the role of dietary protein in avian lipid metabolism, one must look farther than just meeting the amino acid requirement for a particular stage of growth. The liver is main site of *de novo* lipid synthesis in birds as well as a major point of degradation and transamination of dietary amino acids. We have found active net synthesis of glucose (gluconeogenesis) in chicks fed high-protein diets ( $>23\%$ ) and have speculated that carbon skeleton rearrangement

during gluconeogenesis directed substrates from lipid synthetic pathways and required energy which could be used for lipogenesis. We are unaware any of work describing gluconeogenesis, lipogenesis and endocrine function under defined energy and protein intakes because some form of limited feeding regime is necessary to separate the effects of dietary protein and energy intake. The experiments described in the next section were designed to test the effects of absolute protein and energy intakes on avian carbohydrate and lipid metabolism.

Ross male broiler chicks growing from 14 to 28 days of age were fed 14 and 20% protein diets (4 Kcal day<sup>-1</sup>/BWt<sup>0.66</sup>) or 20 and 28% protein diets (2.8 Kcal day<sup>-1</sup>/BWt<sup>0.66</sup>) in a 2 x 2 factorial arrangement to determine the effects of protein and energy intakes on *in vitro* lipogenesis (IVL) and net glucose production (NGP). Plasma concentrations of insulin, glucagon, thyroid hormones (T<sub>3</sub> and T<sub>4</sub>) and somatomedin-C (Sm-C) were estimated by radioimmunoassay. There was a significant (P<0.05) decrease in IVL in the chicks given the higher daily protein intake. The higher protein intake increased (P<0.05) NGP while the lower energy intake decreased (P<0.05) NGP. Insulin, both thyroid hormones and Sm-C were affected by dietary energy and protein intakes.

Diets containing large energy to protein ratios (>13 MJ, 72 MJ/kg crude protein) promote high rates of *in vitro* lipogenesis as well as *de novo* carcass lipid synthesis by the liver. Diets with small energy to protein ratios (<13 MJ, 56 MJ/kg crude protein) promote lean broiler carcasses. We have recently examined energy to protein ratios and have found different possible interpretations of results. For example, feeding a very small energy:protein diet (13 MJ, 43 MJ/kg crude protein) will result in a very lean carcass (crude protein as a % of dry matter) when compared to results attained by feeding a larger energy:protein diet (13 MJ, 65 MJ/kg crude protein). When the data were analyzed on the basis of grams of nitrogen deposited over the course of the experiment, it became obvious to us that the diet containing a larger energy: protein ratio favoured the deposition of more carcass protein as well as lipid. Thus, diets containing very small energy:protein ratios may promote lean carcasses by naturally restricting feed intake. Bartov (1979) has also proposed that the excretion of excess excess amino acid nitrogen would require energy.

Both the quantity (crude protein concentration) and quality (amino acid composition relative to the required balance) of the dietary protein affect the body composition of chicks. The effective protein level can be calculated according to the lysine content

relative to the crude protein level. It was assumed that a completely effective protein contained 60 g of lysine per kg of protein. Although the crude protein level (g/kg diet), as well as the effective dietary protein level (% of the requirement of the first limiting amino acid present in the diet), may regulate lipogenesis, it is unknown if the effect is due to the presence of a limiting amino acid at the ribosomal level or to a shift in cellular metabolism caused by a need to process excess amino acid carbon. In addition the process of converting gluconeogenic amino acid carbon into glucose carbon, reducing equivalents (NADH-NADPH) normally required for the addition of acetyl residues during *de novo* lipogenesis are utilised and may be unavailable for lipogenesis.

The purpose of the experiments that follow was to examine lipid and carbohydrate metabolism in chickens fed additional amounts of the the limiting amino acid lysine as either lysine.HCl or soya-bean meal. The null hypothesis tested was that lysine supplementation of a low-protein diet would influence metabolism similarly to a diet containing a greater amount of crude protein. In the former case, the effective protein level (g lysine per kg of crude protein) would increase and in the latter case, the effective level would remain at 60 g lysine per kg of crude protein. A maize-soya-bean meal basal diet was formulated to be marginally adequate for all essential amino acids with the exception of lysine. Crystalline lysine.HCl was then added to give a total of 12g/kg of diet.

Chickens were fed diets containing either 12.8 MJ, 150 g crude protein/kg or 12.8 MJ, 200 g crude protein/kg to determine differences in metabolism. The diet containing 12.8 MJ, 150 g crude protein/kg contained either 8 or 12 g lysine/kg. Treatment variables examined were *in vitro* lipogenesis, glucose production and hepatic enzyme activities to compare metabolism in chicks fed a low-protein, lysine-supplemented diet and a diet formulated to contain the required amount of lysine from intact protein. Growth was similar in chicks fed diets containing either 12.8 MJ, 154 g crude protein-12 g lysine/kg or 12.8 MJ, 200 g crude protein/kg. Net glucose production was greater ( $P < 0.05$ ) in liver explants from chickens fed diets containing either 12.8 MJ, 154 g crude protein-12 g lysine/kg or 12.8 MJ, 200 g crude protein/kg than in explants from chickens fed 12.8 MJ, 150 g crude protein-8 g lysine/kg. Pyruvate use for glucose production was greater ( $P < 0.05$ ) in chickens fed a diet containing 12.8 MJ, 150 g crude protein-8 g kg lysine/kg. The data in this study suggest that crystalline and "natural" lysine additions to chick diets may influence metabolism differently.

We have also used a severe nutrient restriction (6 d at maintenance energy intake beginning at 6 d of age) to study both short and long-term repletion patterns in broilers (Rosebrough *et al.*, 1986). In some cases, there is a disproportional reduction in abdominal fat in the 7 wk old broiler which can be attributed to a severe energy restriction during the second week of life. We have noted a 4 fold increase in the apparent rate of lipid synthesis during the short-term repletion period (5 d following relief from restriction). Thus, physically restricting a balanced diet containing a required level of protein does not appear to cause long-term growth retardation. This finding contrasts with those of Lauterio and Scanes (1987) describing an elegant pattern in the development of hormone metabolic profiles that can be severely altered by offering diets containing less than 100 g crude protein/kg. These authors also indicated that when protein restrictions were begun early in life, body weight gain did not approach that of controls several weeks after repletion. All studies, including our own, should be cautiously interpreted because repleted chickens never consume the same amount of feed as controls during a corresponding time period.

The purpose of the experiments in the following section was to examine the adaptation of chickens during repletion from low- and adequate-protein diets. A secondary purpose was to alter lipogenesis by dietary treatments and to then determine if a  $\beta$ -adrenergic agonist decreased *in vitro* lipogenesis equally. An equal decrease on a percentage basis. Allred and Roebrig (1973) established that acetyl coenzyme A carboxylase (AcCbx) activity could be increased or decreased *in vitro* depending upon dibutyl cyclic adenosine monophosphate (cAMP) levels. Peptide hormones such as catecholamines or their synthetic analogs such as isoproterenol interact with cell membrane receptors to change intracellular concentrations of cAMP which change metabolic processes. It is true that describing the absolute mechanism involving cAMP and lipogenesis in chick liver appears to be more involved than just presuming the existence of a cAMP-dependent protein kinase-catalyzed phosphorylation of a rate-limiting enzyme (AcCbx) as is common in describing regulation in rat liver. For example, the level of cellular citrate is particularly important in the regulation of the interconversion of the protomeric to the polymeric form of AcCbx with the latter form being the so called active form. Therefore, regulation of glycolytic flux and subsequent formation of citrate influences *de novo* lipogenesis.

Experiments were conducted with Cobb male broiler breeder chickens to determine the metabolic effects of two levels of dietary crude protein fed during a repletion

regimen on subsequent responses to a  $\beta$ -adrenergic agonist (isoproterenol) *in vitro*. Day-old chickens were fed diets containing either 12 or 20% crude protein until 14 d of age and then switched to the opposite diets until 28 d. In Exp 1, chickens were killed at 28 d and in Exp 2, at intervals following the switch at 14 d. *In vitro* lipogenesis (IVL) and lipolysis (IVLY) were determined as the incorporation of 10 mM [2- $^{14}$ C]acetate  $\pm$   $10^{-4}$  M isoproterenol into hepatic lipids and glycerol release from adipose tissue over the course of a 2-hr incubation at 37 C, respectively. Certain hepatic enzyme activities (isocitrate dehydrogenase; ICD, malic enzyme; ME, and fatty acid synthetase; FAS) were determined during the 14 to 28-day repletion period. Both IVL and IVLY were greater at 28 d when chickens were fed a 12% than a 20% protein diet. Neither 0 to 14-d dietary treatment influenced 28-d metabolic parameters. In contrast, the 12% protein diet depressed 14-d weight (160 g vs 320 g;  $P < .05$ ) and gain during the 14 to 28-d period regardless of protein repletion levels (508 g vs 626 g;  $P < 0.05$ ). Both processes were at their highest point 4 d following the dietary switch. Isoproterenol decreased IVL 70% in all dietary treatment groups. Stimulated lipolysis was not affected by nutritional status and was 2x the unstimulated rate. Enzyme activities followed trends noted in explants. It can be concluded from this study that most metabolic responses to severe dietary challenges occur within 1 to 3 d and that a steady state is reached within 4 d.



## **f. COOPERATION**

The research program conducted in the two laboratories was coordinated. For example different strains of broilers were tested in both locations in order to obtain a larger range of conditions applicable in either country. Work with turkeys in the U.S. was started as a joint venture of the U.S.A. team and one of the Israeli coinvestigators (I. Plavnik) during a sojourn in Beltsville. Contact was kept for consultations between the teams.

## **g. EVALUATION OF RESEARCH ACHIEVEMENTS**

Following the initial establishment of the feed restriction methodology and a basic optimization algorithm in the previous BARD projects, the main practical achievement of the present study is the establishment of the response to feed restriction under a large variety of conditions in terms of mode, severity and duration of feed restriction and in various strains. Formulation of the compensatory growth into a mathematical model is used to simulate the responses of body weight and feed efficiency so that the appropriate regimen can be selected on the basis of economic profitability. This economic optimization is facilitated by an improved computer program.

The simulated response to feed restriction was appropriately validated under laboratory conditions. Early-age feed restriction was field-tested in turkeys and found to be economic beneficial: Feed efficiency was improved by 2.9% and the productive index by 8.6%. A major American broiler producer (4 million broilers per week) adopted the method, and according to their nutritionist an improvement of 1 to 2 percentage points in feed efficiency is constantly obtained with a benefit of several million dollars annually. Details have not been disclosed due to commercial reasons.

At least a part of the mechanism of compensatory growth may be associated with growth hormone and/or testosterone. The latter explains the better response to feed restriction observed in males.

## h. PUBLICATION LIST\*

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\* Does not include abstracts, papers submitted for publications or in preparation.