Theor Appl Genet (1991) 83:24–32

Genetic architecture of growth curve parameters in chickens

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Received November 9, 1990; Accepted May 16, 1991
Communicated by E. J. Eisen

Summary. Genetic improvement in growth of poultry has traditionally proceeded via selection for body weight at a fixed age. Due to increased maintenance costs and reproductive problems of adult broiler breeders, the potential for genetic manipulation of the growth curve has been receiving increased interest. Research of both male and female progeny of a three-way diallel cross was used to investigate the inheritance of growth curve parameters. The Laird form of the Gompertz equation was used to determine growth curve parameters, and was suited to the juvenile growth data frequently collected from meat-type chickens. Growth rate exhibited significant heterosis due to both autosomes and the sex chromosomes. Age at inflection point also exhibited significant average heterosis, though only among females. Growth rate was also influenced by average line effects, as was age at inflection point. Maternal effects had no influence on growth curve parameters, while additive sex linkage was observed for growth rate. Phenotypic and genetic correlations were calculated among the growth curve parameters and suggest that specific breeding programs could alter the growth trajectory of the contemporary broiler chicken. Moderate heritabilities were observed for the growth curve parameters and support the hypothesis that the growth curve could be altered via genetic manipulation of early postnatal growth, especially during the first 14 days post-hatch.

Key words: Genetics – Growth curve – Body weight – Chickens

Introduction

Genetic improvement of growth in meat-type poultry stocks has been accomplished primarily by selection for body weight at a fixed age (Siegel and Dunnington 1987). Genetic alteration of body weight has resulted in increased feed intake (Barbato et al. 1983), feed efficiency (Siegel and Wisman 1966), body fat (Katanbaf et al. 1988a), age at sexual maturity (Zelenka et al. 1986), and generally decreased reproductive performance (Dunnington and Siegel 1985). Clearly, the rapid early growth rate that has benefitted meat production has been detrimental to the reproductive capacity of the bird (Dunnington and Siegel 1985). Further, selection for body weight at a fixed age has resulted in a bird that is fatter at any given age, but not necessarily at the same weight (Katanbaf et al. 1988b). The accumulated data from the aforementioned studies suggest that the generalized growth function has become dissociated from the developmental timing of its individual components. A breeding program that alters the shape of the growth curve to maintain optimal early growth and protein accretion, while attenuating later growth and fat deposition, would possibly resolve this problem.

An obvious method of changing the growth trajectory would be to select directly on the mathematical parameters of the growth curve itself. As early as 1945, Brody suggested that the asymptotic or mature weight, rate of attainment of mature weight, and the standardized age at which an animal attained the inflection point of the curve were quantities that could be manipulated by quantitative geneticists. Indeed, Marks (1980) has suggested that the main correlated response to selection for body weight at a fixed age in chickens is early relative growth rate between 0 and 14 days of age, subsequently described as early exponential growth rate by Ricklefs (1985). Ricklefs' (1985) function is roughly equivalent to the growth rate of animals described as an exponential power function (Gompertz 1825).

Marks (1978) and Ricklefs (1985) reported that selection for high 4-week body weight in quail resulted in a

100% increase in asymptotic, or mature, body weight (A), a 40% increase in initial specific growth rate (K), and a 28% increase in the inflection point of the curve (I). Therefore, selection for body weight at a fixed age has changed growth curve parameters, albeit in a non-specific fashion. Direct manipulation of the growth function has been attempted several times. Ricard (1975) showed that growth could be genetically manipulated in this manner by selecting for high body weight at 8 weeks of age and for low body weight at 36 weeks of age. In that experiment, however, neither the selection index used nor the degree of change of the growth curve parameters is clear. In this regard, Abplanalp et al. (1963) showed that 8- and 24-week body weight of turkeys coud be selected independently, but again the effects on the curve parameters were not determined. I have suggested using a different approach, an experiment to alter the growth curve of chickens by selecting divergently for exponential growth rate at either 14 or 42 days of age (Barbato 1990).

A variety of logistic, polynomial, and sigmoid curves have been fitted to the growth curve of chickens (Grossman and Bohren 1982). The logistic function appears to have the best mathematical fit (Grossman et al. 1985), while the Gompertz equation has the next best overall fit with biologically meaningful parameters (Ricklefs 1985). The original Gompertz equation, however, is a function of the asymptotic (or mature) weight of the bird. Meat-type poultry, on the other hand, rarely attain a mature body weight under ad libitum feeding conditions because they are usually processed or placed on a restricted, breeder-type feeding regimen between 4 and 12 weeks of age.

Grossman and Koops (1988) suggested a multiphasic function, which considered body weight to be an accumulation of two phases of growth. Production type data span the first phase of growth described by Grossman and Koops (1988), which gave results similar to the conventional Gompertz analysis. The age limitation imposed by broiler-type management schemes renders weight data ideal for the analysis proposed by Laird et al. (1965), which is a form of the Gompertz equation and takes into account the exponential decay of the specific growth rate of the animal based upon initial weight and inflection point parameters. Indeed, this model has been shown to be valid for the general case of avian growth (Laird 1966) as well as broiler chickens (Tzeng and Becker 1981), based upon the time frame of the model and the biological basis for growth acceleration/deceleration.

While direct genetic manipulation of the growth curve function is a potential solution to several anomalies of selection for body weight at a fixed age, there is insufficient data in the literature regarding heritabilities and/or the genetic architecture of growth curve parameters to estimate its feasability. Data from selected and inbred populations of mice (reviewed by Eisen 1976) have shown relatively small amounts of variation in growth curve parameters. Indeed, one of the few growth curve experiments involving chickens produced no evidence that selection for logistic growth curve parameters would alter the growth curve of chickens (Grossman and Bohren 1985). Less data exists regarding other aspects of the genetic architecture of growth curve parameters of the chicken. Zelenka et al. (1986) suggested that heterosis exists for developmental rates (as a percentage of weight at sexual maturity), but no direct estimates of heterosis of the growth curve parameters were made. The present experiment was designed to estimate directly the type and magnitude of genetic variation underlying the growth parameters of chickens using a diallel cross incorporating progeny of both sexes (Barbato and Vasilatos-Younken 1991).

Methods and materials

Design

Parental populations used in this study were a commercially developed sire pureline (CM), a broiler-type line selected for duration of fertility of frozen, then thawed semen (FS Ansah and Buckland 1983), and an unselected population of the Jersey Giant breed (JG). These stocks were specifically chosen for their range of growth rates. A complete set of F_1 crosses (including parental lines) were produced by pedigreed matings (1 male: 2 females) among 10 males and 20 virgin females per cross via artificial insemination. Number of progeny ranged from 40 to 80 chicks per pureline or cross ($n = 640$ chickens). Chicks of each sex were weighed at hatch and then weekly thereafter through 12 weeks of age. Chicks were placed in pens with shallow litter and fed ad libitum a 24% protein/3,300 kcal starter diet from hatch through 4 weeks of age; thereafter, they were fed a 20% protein/2,800 kcal grower diet.

Statistical analysis of growth curves

Growth curves were estimated via the Marquart algorithm for each individual in the study using the Laird form of the Gompertz equation (Laird et al. 1965). The Laird analysis was similar to that reported by Tzeng and Becker (1981), except that initial or hatching weight (W_0) was not estimated, but rather used as a constant, because preliminary analyses indicated a lack of concordance between estimated and observed values of W_0. This result was similar to the results of Tzeng and Becker (1981). The following equation describes the parameters and shape of the Laird growth curve:

$$W_t = W_0 e^{[(L/K)(1 - e^{-Kt})]},$$

where W_t is the weight of the animal at time t, L = instantaneous growth rate (day^{-1}), and K = exponential rate decay of L (day^{-1}).

The parameters of the inflection point, i.e., the age at the inflection point (T_i) and the weight of the animal at the inflection point (W_i), and the asymptotic weight (A) of the animal may be computed by the following equations:

$$T_i = (1/K) \log (L/K),$$

$$W_i = W_0 e^{((L/K) - 1)}, \text{ and}$$

$$A = W_i e.$$

Statistical analysis of genetic effects

Genetic analysis were based upon the diallel analysis of Gardner and Eberhardt (1966), modified to include maternal (Eisen et al. 1983) and sex-linked effects (Carbonell et al. 1983) as reported in Barbato and Vasilatos-Younken (1991).

The general model representing the mean of a specific parental line or cross within a sex was given by Eisen et al. (1983), as follows:

$$Y_{ij} = \bar{y}_a + (l_i + l_j)/2 + m_j + \delta h_{ij}, \quad (1)$$

where:

Y_{ij} = the mean performance of sire line i crossed with dam line j ($\delta = 0$ for parental line progeny and $\delta = 1$ for crossbred progeny),
$\bar{y}_a$ = mean of the parental lines,
$m_j = \bar{y}_{.j} - \bar{y}_{j.}$,
$l_i = \bar{y}_{ii} - \bar{y}_a - m_j$,
$h_{ij} = \frac{1}{2}(\bar{y}_{ij} + \bar{y}_{ji} - \bar{y}_{ii} - \bar{y}_{jj})$,

and specific reciprocal effects (independent of m_j) were assumed to be zero in this model (i.e., $r^{**}_{ij} = 0$).

The term for heterosis (h_{ij}) has different expectations in males (p) and females (f) (Carbonell et al. 1983). They are as follows

$$h^p_{ij} = h^a_{ij} + h^s_{ij} + a\, a^h_{ij} \quad (2)$$

$$h^f_{ij} = h^a_{ij} + a\, a^h_{ij}, \quad (3)$$

where:

h^a_{ij} = specific heterosis of the autosomes,
h^s_{ij} = specific heterosis of the sex chromosomes (only in males), and
$a\, a^h_{ij}$ = specific additive-by-additive heterosis.

It follows, then, that

$$h^p_{ij} - h^f_{ij} = h^s_{ij}, \quad (4)$$

and one can obtain an estimate of heterotic effects due to heterozygosity of the sex chromosomes, which can only occur in the homogametic sex.

Line effects (l_i) were compared between males and females using the following relationships:

$$l_i = 2(g_i - h_i) \quad (5)$$

where:

g_i = average general combining ability for line i, and
h_i = average line heterosis.

Therefore, subtracting the average line effect of males from the average line effects of females:

$$l^p_i - l^f_i = 2(g^p_i - g^f_i), \quad (6)$$

assuming that $h^p_i = h^f_i$. The genetic relationships implied between these parameters, as defined by Carbonell et al. (1983), leads to the conclusion that

$$l^p_i - l^f_i = d^s + 2h^s_i - a^s_j \quad (7)$$

where:

d^s = intrapopulation dominant sex-linked effects,
h^s_i = heterotic sex-linked effects, and
a^s_j = additive sex-linked effects.

Therefore, significant differences between the sexes for line effect would not define the type of genetic variation influencing those differences, but would identify the source as being sex-linked.

The term m_j usually represents maternal effects in mammalian crossing systems, and sex-linkage is assumed to be zero.

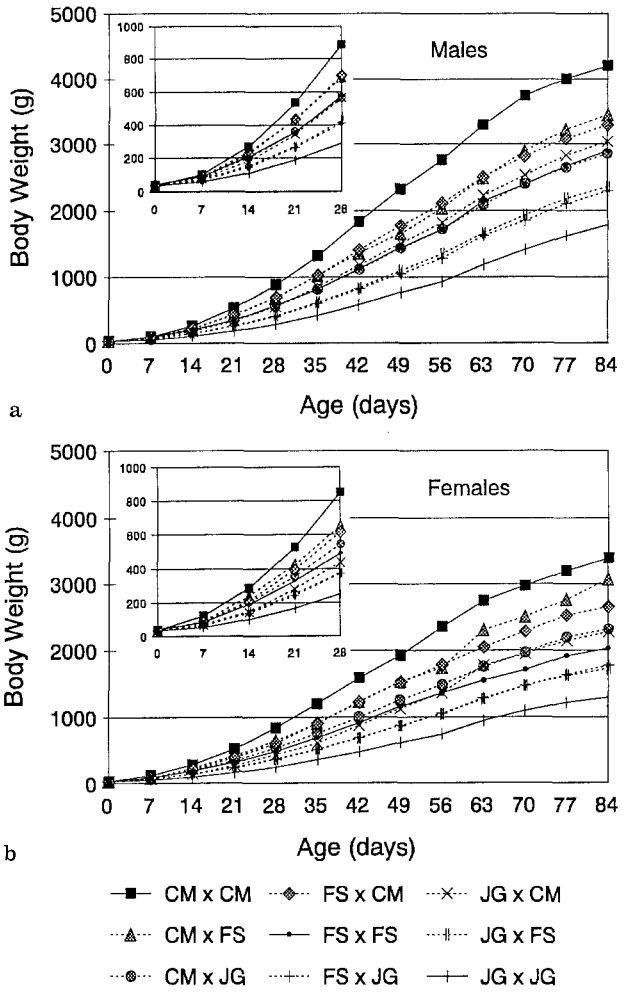

Fig. 1 A, B. Average growth curve of male (**A**) and female (**B**) chickens from a diallel experiment involving a commercially developed sire pureline (*CM*), a broiler-type line selected for duration of fertility of frozen, then thawed semen (*FS*), and an unselected population of the Jersey Giant breed (*JG*) [the sire line is denoted first and the dam line second for a particular mating]

However, there is evidence for sex-linkage in chickens, and the expectations are as follows (Carbonell et al. 1983):

$$m^{*p}_j = M_j \quad (8)$$

$$m^{*f}_j = M_j - a^s_j \quad (9)$$

where M_j = average maternal effect, and additive sex linkage can then be estimated by the equation:

$$a^s_j = m^{*p}_j - m^{*f}_j. \quad (10)$$

Estimates of these comparisons were made by constructing the appropriate contrasts among the least squares means of the purelines and F_1 crosses. As Eisen et al. (1983) pointed out, the number of linear contrasts exceeds the degrees of freedom in the experiment, but the contrasts were developed *a priori* to determine the relative importance of a limited number of effects in the model, i.e., average line effects, heterosis, heterosis of the sex chromosomes, additive sex linkage, and maternal effects. Heritabilities and genetic correlations were calculated within sex subclasses using the method of Griffing (1956).

Table 1. Least square means of growth curve parameters for male and female (in parentheses) progeny, sire* dam subclasses, and line of sire and dam

Sire \ Dam	CM	FS	JG	$\bar{y}_{i.}^{*}$	$\bar{y}_{i.}$
Hatching weights (g)[a,b]					
CM	39 (41)	36 (35)	37 (36)	37 (36)	37 (38)
FS	37 (37)	42 (43)	34 (34)	35 (35)	38 (38)
JG	38 (37)	38 (39)	37 (36)	38 (38)	38 (37)
$\bar{y}_{.i}^{*}$	38 (37)	37 (37)	35 (35)	$\bar{y}_a = 39$ (40)	
$\bar{y}_{.i}$	38 (38)	39 (39)	36 (35)	$\bar{y}_c = 37$ (36)	
				SE = 0.4–1.5	
L (instantaneous growth rate; day^{-1})					
CM	0.1832 (0.1742)	0.1619 (0.1750)	0.1444 (0.1516)	0.1532 (0.1633)	0.1632 (0.1669)
FS	0.1655 (0.1622)	0.1417 (0.1312)	0.1285 (0.1243)	0.1470 (0.1433)	0.1452 (0.1392)
JG	0.1447 (0.1399)	0.1231 (0.1173)	0.1023 (0.0976)	0.1339 (0.1286)	0.1234 (0.1183)
$\bar{y}_{.i}^{*}$	0.1551 (0.1511)	0.1425 (0.1462)	0.1365 (0.1380)	$\bar{y}_a = 0.1424$ (0.1343)	
$\bar{y}_{.i}$	0.1645 (0.1588)	0.1422 (0.1412)	0.1251 (0.1245)	$\bar{y}_c = 0.1447$ (0.1451)	
				SE = 0.0015–0.0065	
K (rate of decay of L; day^{-1})					
CM	0.0382 (0.0378)	0.0342 (0.0606)	0.0308 (0.0355)	0.0325 (0.0481)	0.0344 (0.0446)
FS	0.0350 (0.0364)	0.0313 (0.0314)	0.0276 (0.0286)	0.0313 (0.0325)	0.0313 (0.0321)
JG	0.0308 (0.0335)	0.0267 (0.0279)	0.0223 (0.0230)	0.0288 (0.0307)	0.0266 (0.0281)
$\bar{y}_{.i}^{*}$	0.0329 (0.0349)	0.0305 (0.0443)	0.0292 (0.0321)	$\bar{y}_a = 0.0306$ (0.0307)	
$\bar{y}_{.i}$	0.0347 (0.0359)	0.0307 (0.0400)	0.0269 (0.0290)	$\bar{y}_c = 0.0309$ (0.0371)	
				SE = 0.0016–0.0070	

[a] Terms $\bar{y}_{i.}^{*}$ ($\bar{y}_{.i}^{*}$) represent the sire (dam) marginal means excluding the parental lines, and terms $\bar{y}_{i.}$ ($\bar{y}_{.i}$) represent the sire (dam) marginal means including the parental lines

[b] $\bar{y}_a$ = parental line mean, $\bar{y}_c$ = crossbred mean, and SE = range of subclass standard errors

Results

Figure 1 illustrates the mean growth curves for male (Fig. 1 A) and female (Fig. 1 B) chickens from each pureline and hybrid population. The least square means of growth curve parameters for all male and female progeny of each population are presented in Table 1, the least square means of the derived parameters at the inflection point (i.e., T_i and W_i) are presented in Table 2.

Heterosis

The average heterotic effects and percentage heterosis for male and female progeny for all growth curve parameters (including those at the inflection point) are presented in Table 3. Significant average heterosis was observed for hatching weight (W_0), instantaneous growth rate (L), and age at point of inflection (T_i). Instantaneous growth rate also exhibited significant positive heterosis among all crosses; however, there were significant sex differences in the degree of heterosis. This suggests that there was negative heterosis due to the sex chromosome among male progeny, because male heterosis was less than female heterosis and males are the homogametic sex. Average heterosis was also significant and in the negative direction for age at inflection point, indicating that the crosses reached their average inflection point 3 to 7 days before the pureline parents. Specific heterosis was observed for rate of decay among CM × FS progeny, and sex differences were significant. There was also heterosis for asymptotic weight (A) and weight at the inflection point (W_i) among CM × JG progeny, although it was expressed mostly among female progeny and always in the negative direction.

Line effects

Average line effects for male and female progeny are presented in Table 4. There were no significant line effects on hatching weight, asymptotic nor weight at the inflection point. There were line effects for instantaneous growth rate among all lines, with differences due to sex among the CM and FS line effects. Line effects for rate of decay were observed among the CM and FS lines, but there were sex differences for line effects. Significant line effects were observed among all lines for age at inflection point, with sex differences being significant among the CM and FS line effects. Differences in line effects due to sex are suggestive of sex-linked variation, although the particular source of the variation may be intrapopulational

Table 2. Least square means of inflection point parameters for male and female (in parentheses) progeny, sire* dam subclasses, and line of sire and dam

Sire \ Dam	CM	FS	JG	$\bar{y}_{i.}^{*}$	$\bar{y}_{i,}$
W_i (weight at inflection point (g))[a]					
CM	1,871 (1,524)	1,685 (1,047)	1,535 (1,033)	1,610 (1,040)	1,697 (1,201)
FS	1,583 (1,343)	1,463 (1,160)	1,383 (1,004)	1,483 (1,174)	1,476 (1,169)
JG	1,613 (947)	1,485 (1,211)	1,389 (999)	1,549 (1,079)	1,496 (1,052)
$\bar{y}_{.i}^{*}$	1,598 (1,145)	1,585 (1,129)	1,459 (1,019)	$\bar{y}_a = 1{,}574$ (1,228)	
$\bar{y}_{.i}$	1,689 (1,271)	1,544 (1,139)	1,436 (1,012)	$\bar{y}_c = 1{,}547$ (1,098)	
				SE = 47–207	
T_i (age at inflection point (days))					
CM	43 (41)	47 (36)	51 (42)	49 (39)	47 (40)
FS	45 (44)	49 (48)	57 (52)	51 (48)	50 (48)
JG	51 (45)	58 (55)	70 (65)	55 (50)	60 (55)
$\bar{y}_{.i}^{*}$	48 (45)	53 (46)	54 (47)	$\bar{y}_a = 54$ (51)	
$\bar{y}_{.i}$	46 (43)	51 (46)	59 (53)	$\bar{y}_c = 52$ (46)	
				SE = 1–5	

[a] Same notation as in Table 1.

Table 3. Heterosis (h_{ij}) and percentage heterosis (h_{ij}%) for growth curve parameters by cross and sex

	Cross	W_0 (g)		L (day^{-1})		K (day^{-1})		W_i (g)		T_i (days)	
		Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
h_{ij}	CM × FS	−4.2*	−5.9*	0.0013+	0.0159*	−0.0002+	0.0139*	−33	−147	1	−5*
	CM × JG	−0.6	−2.3*	0.0019+	0.0099*	0.0006	0.0042	−56	−271*	−6*	−9*
	FS × JG	−3.3*	−2.8*	0.0038	0.0064*	0.0004	0.0020	8	28	−4	−6*
	$\bar{h}_{ij}$	−2.7*	−3.8*	0.0023+	0.0107*	0.0003	0.0067	−27	−130	−3	−7*
h_{ij}%	CM × FS	−10	−14	1	10	−1	40	−2	−11	0	−10
	CM × JG	− 1	− 5	1	7	2	13	−3	−22	−10	−19
	FS × JG	− 9	− 8	3	6	1	4	1	3	− 3	− 6
	$\bar{h}_{ij}$%	− 7	− 9	2	8	1	19	−1	−10	− 4	−12

* Significant heterosis ($P < 0.05$)
\+ Significant sex* heterosis interaction ($P < 0.05$)

Table 4. Line effects on growth curve parameters for male and female progeny

Line	W_0 (g)		L (day^{-1})		K (day^{-1})		W_i (g)		T_i (days)	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
CM	−2.1	0.1	0.0370*+	0.0644*	0.0069+	0.0333*	321	88	−9*+	−22*
FS	−0.1	−1.0	0.0084+	−0.0089*	0.0024+	−0.0229*	−315	20	−9*+	2
JG	2.2	0.8	−0.0453*	−0.0555*	−0.0093	−0.0104	− 6	−108	17*	20*

* Significant line effects ($P < 0.05$)
\+ Significant sex* line interaction ($P < 0.05$)

Table 5. Maternal (M_i) and sex-linked (a_s) effects for growth rate parameters

	Line	W_0 (g)	L (day^{-1})	K (day^{-1})	W_i (g)	T_i (days)
M_i	CM	2.2	0.0039	0.0007	− 24	− 2.4
	FS	3.0*	−0.0091	−0.0002	204	3.6
	JG	−5.2*	0.0052	0.0009	−179	− 1.2
a_s	CM	1.0	0.0284*	0.0270*	−233	−14*
	FS	−0.6	−0.0149	−0.0252*	291	10*
	JG	−0.4	−0.0135*	−0.0017	58	5

* Significant genetic effect ($P < 0.05$)

Table 6. Phenotypic (below diagonal) and genetic (above diagonal) correlations among growth related traits

Trait	W_0	W_{14}	W_{42}	L	K	W_i	T_i
W_0		−0.64	−0.60	−0.44	−0.50	−0.61	0.65
W_{14}	0.31		0.43	0.52	0.43	0.93	−0.42
W_{42}	0.25	0.88		0.36	0.29	0.56	−0.30
L	−0.08	0.84	0.88		0.36	0.70	−0.38
K	−0.01	0.74	0.72	0.92		0.59	−0.28
W_i	0.16	0.08	0.48	−0.09	−0.41		−0.63
T_i	−0.01	−0.65	−0.58	−0.80	−0.93	0.58	

Table 7. Heritabilities of growth curve parameters and representative body weights as estimated from the diallel table

Trait	Heritability
W_0	0.39
W_{14}	0.49
W_{42}	0.22
L	0.48
K	0.28
W_i	0.12
T_i	0.18

dominant sex-linkage, heterosis of the sex chromosomes or additive sex-linked effects (Carbonell et al. 1983; Barbato and Vasilatos-Younken 1991).

Maternal effects and additive sex linkage

Maternal and additive sex-linked effects are presented in Table 5. Significant maternal effects were observed among the FS and JG lines for hatching weight.

Additive sex-linkage, on the other hand, was observed for all lines for instantaneous growth rate. Sex linkage was also observed among the CM and FS lines for rate of decay and age at inflection point.

Phenotypic and genetic correlations

Phenotypic and genetic correlations among the growth curve parameters and selected body weights at three ages are presented in Table 6. Phenotypic correlations between W_0, W_{14} and W_{42} were positive, with the correlation between W_{14} and W_{42} being +0.88. These body weights were chosen because traditional selection for body weight among commercial poultry breeders occurs at, or near, 42 days of age. Further, in an independent study, body weight at 14 days was shown to be an important determinant of growth at later ages (Barbato 1990). Phenotypic correlations show that there is a large positive relationship between body weight at 14 and 42 days of age, and both L and K. Body weight at 14 days of age essentially had no correlation to W_i, both measures of post-juvenile growth. Body weight at 42 days of age had moderate correlations with W_i, whereas weight at either age was negatively correlated with T_i (age at inflection point). Among the growth parameters themselves, L and K were highly positively correlated with each other, and each was negatively correlated with W_i and T_i. W_i and T_i were positively correlated, which is not entirely surprising because W_i and T_i are functions of L and K.

Genetic correlations differed from the phenotypic correlations. There were negative genetic correlations between W_0 and subsequent body weights, whereas the correlation between W_{14} and W_{42} was +0.43. W_0 also had negative genetic correlations with all growth curve parameters except T_i. Both W_{14} and W_{42} had moderate to large positive genetic correlations with the other growth curve parameters (except T_i, which was negative). In each case, however, W_{14} had genetic correlations with the curve parameters that were 40–50% higher than those of W_{42}.

Heritability estimates of the growth curve parameters along with body weights at representative ages derived from the diallel are presented in Table 7. All representative body weights had moderate heritabilities. Heritability estimates of the growth curve parameters were also moderate, although the heritability of L was 2–4 times as large as any other parameter.

Discussion

Average growth curve parameters (Tables 1 and 2) were slightly smaller than those obtained by Tzeng and Becker

(1981), who found L = 0.189, K = 0.037, and W_i = 1743. However, their values were obtained from pedigreed male broilers, which would be roughly comparable to the CM line (L = 0.1832, K = 0.0382, and W_i = 1871). Hatching weights were smaller in their report than in this study (29 versus 39 g, respectively), because they estimated W_0 as a parameter of the model, whereas here the actual hatching weight was a constant. In either case, the Laird form of the Gompertz equation provides a growth model suited to the broiler chicken. The utility of fitted growth curves in animal breeding has been of questionable value due to the long generation intervals resulting from the time required to achieve asymptotic weights (Fitzhugh and Taylor 1971; Rutledge et al. 1972). The Laird equation ignores this problem and concentrates on the portion of the growth curve important for domestic meat-animal production.

Estimates of direct genetic effects on growth curve parameters in animals are few in number, and, to my knowledge, no previous estimates have been made using chickens as the experimental animal. Laird and Howard (1967) compared the growth of inbred lines of mice and their F_1 hybrids and observed significant heterosis for growth rate. Kidwell and associates (Kidwell and Howard 1969, 1970; Kidwell et al. 1969) performed a four-way diallel cross with mice and found low estimates of general and specific combining ability for the growth parameters of the Gompertz equation, although crossbreds always had greater L than inbreds. In agreement with these studies, the instantaneous growth rate (L) of chickens exhibited significant heterosis. This result is contrary to the genetic architecture of body weight, which exhibits little heterotic character after hatching (Barbato et al. 1983; Katanbaf et al. 1988 a; Barbato and Vasilatos-Younken 1991). Significant sex differences in average heterosis were observed for L, indicating heterosis for the sex chromosomes. While sex* heterosis interactions are not common among mammals (White et al. 1970), sex* breed interactions were observed for growth rate in mice, although the source of the interaction was not elucidated (Kidwell et al. 1969). The growth rate parameter L, then, must have different physiological implications than solely absolute body weight at a given age. The most obvious variables are protein and fat deposition, which exhibit marked heterosis at early ages in quail (Wyatt et al. 1982) and chickens (Barbato et al. 1983; Katanbaf et al. 1988 b). The observation that F_1 crosses of male and female chickens of the same high- and low-weight lines (although originating from different generations) exhibit different patterns of heterosis for abdominal fat pad size and breast weight (Barbato et al. 1983; Katanbaf et al. 1988 b) is suggestive of the existence of sex* heterosis interactions for these traits.

Line effects, a measure of general combining ability without line heterosis (Eisen et al. 1983), were significant for L, K and T_i in this study. This result is in marked contrast to the work by Kidwell et al. (1969), who found that general combining ability effects were not significant in mice. They also found highly significant maternal effects for growth rate, whereas no maternal effects were significant in this experiment.

There is a dearth of information regarding phenotypic or genetic correlations between growth increments, based upon weight or age. Further, as Ricklefs (1985) noted, correlations between growth increments are highly dependent upon the ages chosen and provide no information on the patterns of variation in growth curves. In Chamber's (1990) recent review of the genetics of growth in the fowl, most genetic correlations between body weights obtained 1 week apart were high (> 0.9), whereas lower correlations were observed between weights separated by several weeks. Chambers also observed that correlations between weight at a given age and previous gain were substantially higher than weight at a given age and subsequent gain (0.9 versus 0.6).

In this experiment, phenotypic correlations between L and K were 0.92 and were similar to those reported in mice (0.83; Kidwell et al. 1969). They also observed small negative correlations between initial weight and both L and K, which were identical to the relationships in this report. Phenotypic and genetic correlations among growth curve parameters were often different in sign, as well as magnitude, suggesting that the sources of the genetic and environmental variation influencing the traits do so via different physiological or biochemical systems. These data are consistent with those summarized by Chambers (1990), in that genetic correlations among body weight and/or growth increments with abdominal fatness were often of different sign and/or magnitude.

Hypothetically, if one selected for L as a method for increasing meat yield in broilers as suggested by Ricklefs (1985), one might predict that W_{14}, W_{42}, K, and W_i would increase and that T_i would decrease as a result of the genetic correlations. Selection for W_{42} (the norm in the breeding industry) would be associated with similar changes, which agrees with the general hypothesis of Ricklefs (1985) and Marks (1980). If, however, selection pressure was placed on W_{14}, the genetic correlations among the growth curve parameters would be in the same direction, but larger, suggesting a more rapid accumulation of alleles influencing the correlated traits and hence greater response! These relationships have yet to be tested empirically.

More rapid progress for a given trait can be achieved by selecting on a highly correlated trait if the trait under consideration is difficult to measure with precision (i.e., measurement errors increase environmental variation), resulting in lower heritability. Indeed, the heritability of W_{14} is more than twice that of W_{42} in this experiment.

El-Ibiary and Shoffner (1951) estimated that heritability in the broad sense (estimated from full-sibs) for body weight was greater at 14 days than at 42 days (0.42 versus 0.32), although the estimates were biased by maternal and dominance variation. Unfortunately, half-sib estimates yielded heritability estimates that were less than 0.15 for body weight at each age, perhaps due to the small number of sires (i.e., 8) included in the experiment. Fujishima (1966) concluded that there was adequate additive variation for selection at 14 days (25–30%), although details concerning later growth were lacking. Heritability estimates obtained in this experiments are biased from maternal and dominance variation because non-inbred parentals were used in the diallel cross. It should be pointed out, however, that maternal effects were non-existent after hatch (Barbato and Vasilatos-Younken 1990), so the majority of the bias was probably due to dominance variation. In any case, all of the growth curve parameters exhibited moderate heritabilities similar to those reported for mice. Rutledge et al. (1972) found that the weighted mean growth rate of mice was moderately heritable (0.28). Moderate heritability estimates for growth curve parameters were obtained by Eisen et al. (1969) and Timon and Eisen (1969), although these estimates may also have been inflated due to maternal and dominance variation. Moderate heritabilities for the growth rate constant of Rhode Island Red and White Leghorn chickens were obtained by Grossman and Bohren (1985), but the large standard errors of the heritabilities warranted caution in interpreting the results.

The importance of the growth curve of animals, separate from weight at a given age, has long been known to have important evolutionary and fitness implications (e.g., Gould 1966). Not surprisingly, as selection pressures for production have become more intense, fitness relationships have become important among domestic species. Certainly, it is the inverse relationship between selection for body weight at a fixed age (usually during the juvenile period) and reproductive performance that has revived discussions regarding the use of growth trajectories of body components and their relationship to the composite growth curve in contemporary breeding programs (see Katanbaf et al. 1988a; Famula et al. 1988). The present study has attempted to delineate the genetic architecture of growth curve parameters in chickens by using a diallel cross among diverse populations. The data indicate that growth curves of chickens may be amenable to genetic manipulation and that this may be accomplished by independently selecting on heritable parameters of the curve or by indirectly selecting on the basis of genetic correlations. Further, due to the heterosis that is exhibited by certain portions of the curve, it should be possible a comprehensive breeding plan that maximizes early growth and minimizes the deleterious effects of selection for body weight at a fixed age.

Acknowledgements. Special thanks to C. Hadnot for help in all phases of this work and to J. Chambers, N. Anthony and two anonymous reviewers for comments and criticisms. This research was supported by a grant from the Pennsylvania Department of Agriculture to the author (Contract Number ME48005).

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