Genetic analysis of growth curve parameters for male and female chickens resulting from selection on shape of growth curve

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ABSTRACT: The objectives of this research were to evaluate gender differences and selection on body weight as they affect growth curves of chickens. Marginal posterior densities of growth curve parameters were studied by Gibbs sampling on 10,671 male and female chickens originating from five lines. Line X+− was selected on low body weight at 8 wk (BW8) and high body weight at 36 wk (BW36), line X−+ on high BW8 and low BW36, X+− on high BW8 and BW36, X−− on low BW8, and X00 was an unselected control line. Growth was modeled by a Gompertz function. Heritabilities and genetic correlations among parameters of the Gompertz curve were estimated. Marginal posterior densities were drawn for parameters of the growth curve and for sexual dimorphism at ages ranging from hatching to 1 yr. Lines selected for a higher BW8 had higher initial specific growth rates (L), higher maturation rates (K), and lower ages at inflection (T1). Lines selected for a higher BW36 had higher asymptotic body weights (A). Estimates of A, L, and T1 were higher in males and K was higher in females. Difference between sexes for A was greater in the line selected for a lower BW8 and a higher BW36. Dimorphism for L and K was the lowest in lines X+− and X−−, respectively. The greatest difference in T1 was observed in the line selected for lower BW8 and BW36. Sexual dimorphism of body weight was lower at most ages in the lightest line. Before 15 wk, sexual dimorphism in X+− line was lower than in the line selected for higher BW8 and lower BW36. The increase in sexual dimorphism with body weight could be reduced by selecting animals on body weight at two ages instead of one, as is usually done in commercial lines.

Key Words: Chicken, Genetic Parameters, Gibbs Sampling, Growth, Sexual Dimorphism

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Introduction

Genetic evaluation of broilers has been based only on body weight at age of slaughter. This has resulted in modifications of body composition, precocity, and sexual maturity (Sauveur, 1988) and in an increase in sexual dimorphism (Reffay and Salichon, 1995). The overall kinetics of growth have also been modified (Timon and Eisen, 1969). Selecting on the growth curve could allow both selection on body weight and modification of sexual dimorphism. The latter may partly result from differences in precocity between sexes, that is, differences in physiological ages between males and females at the same chronological age (Ricklefs, 1985). Comparing body weight of animals at a given physiological age would make it possible to test this hypothesis. Sexual dimorphism may also result from a scale effect, with the difference between sexes being proportional to the mean of the trait.

Barbato (1991) and Mignon-Grasteau et al. (1999) showed that parameters of the Gompertz curve describing age-weight relationships in chickens wereheritable. Mignon-Grasteau et al. (1999) showed that genes controlling these parameters seemed partly to differ between sexes. Growth curves differed, but it was not possible to test the significance of these differences as distributions of the growth curve parameters remained unknown.

Therefore, response in growth curve parameters of male and female chickens to selection on body weights at 8 and 36 wk were compared. The objective of this research was to establish whether sexual dimorphism was modified by selection and due to differences in precocity or to a scale effect. A Bayesian estimation of
these parameters was used as in Varona et al. (1997) to provide the posterior density of these unknowns.

## Materials and Methods

### Animal Material

Data originated from a selection experiment on the form of the growth curve initiated by F. Ricard in 1960 on meat-type chickens. Line X+ was selected for high juvenile body weight at 8 wk (BW8) and low adult body weight at 36 wk (BW36). In contrast, line X− was selected for low juvenile body weight and high adult body weight. Line X00 was an unselected control line. In line X++, the animals were selected for both body weights at both ages, and, in the opposite line, X−, they were selected for low body weights at both ages. The first 14 generations were used in this study (i.e., those from 1960 to 1973). The description of this experiment and preliminary results of the selection can be found in Ricard (1975).

All the animals were weighed on the day of hatching and at 8 wk of age. In one hatch per year, animals were weighed every second week until 16 wk and then monthly between 16 and 52 wk. Only animals of those hatches and their parents were used for this research. Thus, a total of 10,476 animals were included in the data file, of which 7,164 were measured regularly. The base generation was also included in the pedigree file, comprised of 10,671 records.

### Growth Model

The Gompertz function was used to describe the age-weight relationships for each bird. In this function, the body weight of an animal i at age j (BWij) was computed as

\[ BW_{ij} = A_i \times \exp[-B_i(e^{-K_i j})] + \varepsilon_{ij} \]  

where \( A_i \) is the asymptotic body weight of the animal i, that is, the weight at an infinite age. The parameter \( B_i \) is equal to \( \ln(A_i/BW_{0i}) \), where BW0i is the estimated hatching weight of animal i. Maturation rate is estimated by K. The latter is also called the exponential rate of decay of the specific growth rate, as 1/BW0i × dBWij/dt \( \propto e^{-K_i t} \). Finally, \( \varepsilon_{ij} \) is the residual of the estimation of body weight by the growth curve. Other parameters can be deduced from Eq. [1]. For the animal i, the initial specific growth rate (\( L_i \)) is defined as \( B_i \times K_i \), the age at inflection (\( T_{1i} \)) as \( 1/K_i \times \ln(B_i) \), and the body weight at hatching (\( BW_{0i} \)) as \( A_i \times \exp(-B_i) \) (Laird, 1966).

### Growth and Bayesian Estimation Procedure

The model that follows was proposed by Blasco and Varona (1998). The standard deviation of \( \varepsilon_{ij} \) at age j (\( \sigma_j \)) was also modelled by a Gompertz curve as in Blasco and Varona (1998) that is, using a notation similar to that used in Eq. [1]:

\[ \sigma_j = A_x \times \exp[-B_x \times e^{-K_x j}] \]  

where \( A_x, B_x, \) and \( K_x \) are the parameters of the Gompertz curve, and \( \mathbf{p}_x \) is the vector of these parameters (i.e., \( \mathbf{p}_x = (A_x \; B_x \; K_x) \)).

The prior distribution of parameters \( A_x, B_x, \) and \( K_x \) was taken to be multinormal, so that

\[
f(A, B, K | u_A, u_B, u_K, \beta_A, \beta_B, \beta_K, R, G) = N(X\beta_A + Z u_A, N X\beta_B + Z u_B, R \otimes I_N | X\beta_K + Z u_K)\]  

where \( u_A, u_B, \) and \( u_K \) are the vectors of breeding values for the 10,671 animals of the pedigree file; \( \beta_A, \beta_B, \) and \( \beta_K \) are the vectors of systematic effects, that is, generations (14 levels) and sex (2 levels); \( R \) and \( G \) are the 3 × 3 matrices of environmental and genetic (co)variance components of the growth curve parameters; \( X \) and \( Z \) are the common incidence matrices linking \( A, B, \) and \( K \) to the systematic and genetic effects, respectively; \( I_N \) is the identity matrix of dimension \( N \); and \( \otimes \) is the sign for direct Kronecker product.

Prior distributions of systematic effects are distributed as

\[
f(\beta_A, \beta_B, \beta_K) = N(m, V)\]  

where \( m \) and \( V \) are the subjective mean and variance for the prior beliefs, based on studies of the growth of meat-type chickens of Pasternak and Shalev (1983), Knizedova et al. (1985), Ricklefs (1985), Rogers et al. (1987), and Réminon (1993). Large variances were used in order to formalize a vague prior state of knowledge.

The prior distribution of breeding values follows a multivariate normal distribution:

\[
f(u_A, u_B, u_K | G) = N(0, A \otimes G)\]  

where \( A \) is the relationship matrix.

In a Bayesian context, priors are needed for each parameter. The prior distribution of \( \mathbf{p}_x \) is taken to be uniform, and inverted Wishart distributions are taken as priors for the matrices \( R \) and \( G \), so that

\[
f(R | S_R, \nu_R) \propto |R| \exp[-1/2(\nu_R+p+1)]\exp[-1/2 \times \text{tr}(R^{-1} S_R^{-1})]\]  

\[
f(G | S_G, \nu_G) \propto |G| \exp[-1/2(\nu_G+p+1)]\exp[-1/2 \times \text{tr}(G^{-1} S_G^{-1})]\]  

where \( S_R, \nu_R, S_G, \) and \( \nu_G \) are the hyperparameters of the distributions of \( R \) and \( G \) respectively, with \( \nu_R, \) and
\(\nu_G = -4\). The terms \(S_G\) and \(S_R\) are null matrices of order 3.

**Statistical Methods**

In a Bayesian context, marginal posterior densities of each unknown, given the data, are used to estimate unknowns. The joint posterior density is

\[
f(A, B, K, u_A, u_B, u_K, \beta_A, \beta_B, \beta_K, R, G, p; | y) \propto f(y | A, B, K, p) \times f(A, B, K | u_A, u_B, u_K, \beta_A, \beta_B, \beta_K, R, G) \times f(u_A, u_B, u_K | G) f(p, p) f(\beta_A, \beta_B, \beta_K) f(R) f(G)
\]

where all distributions were known, and the likelihood was a product of normal functions, with mean and variance defined in [1] and [2], respectively. Details on the algorithm used by the Gibbs Sampler for the computation of these terms can be found in Blasco and Varona (1998).

**Gibbs Sampler**

Conditional distributions were derived from [8] to apply the Gibbs sampling technique following Varona et al. (1997) and Blasco and Varona (1998). Because there was no information on the genetic parameters of growth curve in chickens, flat priors were used for the (co)variance matrices \(R\) and \(G\). One long chain of 100,000 iterations and one short chain of 40,000 iterations were performed. The comparison of results obtained with the short and the long chain using the same prior helped to check whether convergence had been reached.

After graphical analysis of the convergence (Bottcher and Gibson, 1997), the first 20,000 iterations of each chain were discarded as burn-in (i.e., the period after which convergence is considered to be reached). Moreover, as the autocorrelation between successive iterations was greater than .9 for each parameter, only 1 iteration out of 20 was kept. This thus led to samples of 4,000 and 1,000 points for the long and short chains, respectively.

This analysis provided estimates of genetic parameters of parameters \(A\), \(B\), and \(K\) (Table 1). At each iteration and for each combination of systematic effects (i.e., line \(\times\) sex \(\times\) generation), samples were taken from the distribution of \(A\), \(B\), and \(K\). They were used to derive the phenotypic values of \(L\), \(T_1\), and \(BW_0\). For each parameter, these values were averaged over all the iterations to check the effect of selection on the growth curve parameters (Table 2). In order to obtain comparable values, only the first 10 generations of selection were taken into account for the selected lines. All the generations were used for the control line. Distributions of the mean of each parameter over the iterations were also drawn for males at the 10th generation of selection (i.e., year 1969 for lines \(X++\), \(X+-\), and the control and 1973 for lines \(X++\) and \(X--\)).

**Sexual Dimorphism**

Distributions of the mean of sexual dimorphism for all the parameters of the growth curve were drawn from the computed phenotypic values of each line \(\times\) sex \(\times\) generation combination and expressed as a difference from the control line. At each iteration and for each parameter, the difference between sexes was compared between each selected line and the control line, and the probability for this difference being higher in each selected line than in the control line was computed. Absolute sexual dimorphism is computed (e.g., for parameter \(A\) in line \(X++\)) as follows:

\[
[A_m(X--) - A_f(X--)] - [A_m(X00) - A_f(X00)] \tag{9}
\]

Its relative value was computed as

\[
\frac{[A_m(X--) - A_f(X--)]}{0.5 \times [A_m(X--) + A_f(X--)]} - \frac{[A_m(X00) - A_f(X00)]}{0.5 \times [A_m(X00) + A_f(X00)]} \tag{10}
\]

where subscripts \(m\) and \(f\) stand for male and female, respectively. The evolution of sexual dimorphism of body weight at a given chronological age was also investigated. Sexual dimorphism at ages ranging from 0 to 52 wk was computed from [9] and [10] using the mean estimates of the growth curve parameters.

**Results**

**Convergence and Estimation of Genetic Parameters**

The short and long chains run with the same prior converged to similar estimates of the genetic parameters in a low number of iterations. Both chains were thus pooled to compute the effective lengths of chains. Monte Carlo standard errors for the genetic parameters were between .01 and .03. The effective sample size ranged from 96 to 214.

Heritabilities were high for all three parameters (i.e., from .43 to .60). The genetic correlation of \(A\) with \(B\) was highly positive, whereas the genetic correlations of \(K\) and \(A\) or \(B\) were moderately negative.
Table 2. Estimates of the mean (± standard deviations) of the line × generation fixed effects of the parameters of the growth curve over the iterations, on the first 10 generations of selection

<table>
<thead>
<tr>
<th>Line and sexa</th>
<th>A, g</th>
<th>B</th>
<th>K × 10^3, d⁻¹</th>
<th>L × 10^3, d⁻¹</th>
<th>T₁, d</th>
<th>BW₀, g</th>
</tr>
</thead>
<tbody>
<tr>
<td>X++</td>
<td>M</td>
<td>3,960 ± 288</td>
<td>4.56 ± .13</td>
<td>1.94 ± .20</td>
<td>8.85 ± .88</td>
<td>79.0 ± 9.3</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>2,984 ± 332</td>
<td>4.26 ± .16</td>
<td>1.96 ± .21</td>
<td>8.31 ± .82</td>
<td>75.0 ± 9.6</td>
</tr>
<tr>
<td></td>
<td>µ</td>
<td>3,472 ± 579</td>
<td>4.41 ± .21</td>
<td>1.95 ± .21</td>
<td>8.58 ± .87</td>
<td>77.0 ± 9.7</td>
</tr>
<tr>
<td>X−</td>
<td>M</td>
<td>3,016 ± 279</td>
<td>4.29 ± .13</td>
<td>2.47 ± .20</td>
<td>10.62 ± .88</td>
<td>59.2 ± 5.1</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>2,245 ± 224</td>
<td>4.01 ± .14</td>
<td>2.53 ± .21</td>
<td>10.15 ± .71</td>
<td>55.0 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>µ</td>
<td>2,630 ± 461</td>
<td>4.15 ± .20</td>
<td>2.50 ± .21</td>
<td>10.38 ± .83</td>
<td>57.1 ± 5.2</td>
</tr>
<tr>
<td>X++</td>
<td>M</td>
<td>3,806 ± 237</td>
<td>4.51 ± .08</td>
<td>2.27 ± .07</td>
<td>10.22 ± .36</td>
<td>66.5 ± 2.8</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>3,033 ± 210</td>
<td>4.27 ± .08</td>
<td>2.28 ± .08</td>
<td>9.75 ± .34</td>
<td>64.3 ± 2.2</td>
</tr>
<tr>
<td></td>
<td>µ</td>
<td>3,397 ± 446</td>
<td>4.39 ± .14</td>
<td>2.28 ± .08</td>
<td>9.97 ± .42</td>
<td>65.6 ± 2.8</td>
</tr>
<tr>
<td>X−</td>
<td>M</td>
<td>2,978 ± 357</td>
<td>4.40 ± .09</td>
<td>1.79 ± .21</td>
<td>7.90 ± .96</td>
<td>83.8 ± 9.5</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>2,175 ± 288</td>
<td>4.13 ± .10</td>
<td>1.84 ± .22</td>
<td>7.61 ± .88</td>
<td>78.0 ± 9.2</td>
</tr>
<tr>
<td></td>
<td>µ</td>
<td>2,553 ± 515</td>
<td>4.26 ± .17</td>
<td>1.82 ± .22</td>
<td>7.75 ± .93</td>
<td>80.2 ± 10.8</td>
</tr>
<tr>
<td>X00</td>
<td>M</td>
<td>3,341 ± 317</td>
<td>4.40 ± .11</td>
<td>2.18 ± .14</td>
<td>9.59 ± .62</td>
<td>68.6 ± 5.1</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>2,449 ± 272</td>
<td>4.11 ± .13</td>
<td>2.23 ± .14</td>
<td>9.17 ± .58</td>
<td>63.9 ± 5.0</td>
</tr>
<tr>
<td></td>
<td>µ</td>
<td>2,877 ± 535</td>
<td>4.25 ± .19</td>
<td>2.21 ± .14</td>
<td>9.37 ± .63</td>
<td>65.2 ± 5.1</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td>3,384 ± 451</td>
<td>4.42 ± .14</td>
<td>2.15 ± .29</td>
<td>9.50 ± 1.21</td>
<td>70.9 ± 12.2</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td>2,543 ± 436</td>
<td>4.14 ± .15</td>
<td>2.19 ± .29</td>
<td>9.05 ± 1.12</td>
<td>66.3 ± 10.2</td>
</tr>
</tbody>
</table>

aM = males; F = females; µ = mean of line with both sexes grouped together.

Estimates of the Parameters of the Growth Curve

As all marginal a posteriori distributions were approximately normal, only the mean and standard deviation are presented in Table 2. Distributions of the means of the parameters for males at the 10th generation of selection are shown in Figures 1 and 2. Distributions were similar for females. Lines selected for high body weight at 8 wk showed higher estimates of initial specific growth rate (L) and maturation rate (K), and lower estimates for age at inflection (T₁). In contrast, lines selected for high body weight at 36 wk showed the highest values for asymptotic body weight (A) and for B. It should be noted that, for parameters L, K, and T₁, the line closest to the control line was X++. Finally, the extreme values of BW₀ were found in X++ and X−, respectively.

Differences between sexes were also observed. Males had higher average A, L, T₁, and BW₀, and lower K.

Differences Between Sexes in Estimated Growth Curve Parameters

Table 3 shows the probability for differences between sexes for parameters of the growth curve being higher in the selected line than in the control line. The distributions of the differences between sexes for estimated A and T₁ are also shown in Figures 3 and 4. Sexual dimorphism for asymptotic body weight (A) was lower than in the control line in lines X−, X++, and X−−, and higher in line X++. However, when considering sexual dimorphism as a relative difference, line X−− had a higher dimorphism, whereas X++, X−−, and X++ were lower than the control dimorphism. Only line X−− had a probability slightly higher than 50% to have a greater dimorphism than the control line for parameter B, whereas probabilities in lines X++, X−−, and X++ were lower (Table 3). When sexual dimorphism was computed as a relative value, the ranks of the different lines remained almost unchanged. For the maturation rate (K), lines X++ and X−− had less dimorphism than the control line.

For BW₀, absolute dimorphism was higher in lines X−−, and X++, and lower for X++ and X++. For L, X−− and X++ were higher than X00. Only X−− line was higher than X00 for T₁. However, when relative sexual dimorphism was considered, line X−− was also higher than X00 for T₁.

Discussion

Convergence

The effective size of sample was in the same range as in Hofer and Ducrocq (1997), Varona et al. (1998), and Ugarte et al. (1996). According to the limit suggested by Su et al. (1997), convergence seemed to be reached, as the Monte Carlo errors were lower than 3% of the mean of the genetic parameters. The estimates depended very little on the priors (data not shown).

Genetic Parameters of the Gompertz Function Parameters

Heritabilities of the growth curve parameters were higher than those estimated by REML in the same lines.
Selection modifies sexual dimorphism

by Mignon-Grasteau et al. (1999) (i.e., .49 for K and .44 for A when grouping both sexes together). In the latter study, information provided by relatives could not be used to estimate the Gompertz function parameters. Moreover, the model of analysis differed between the previous and the present studies. In the latter, maternal effects, which accounted for 5 to 10% of the variance in Mignon-Grasteau et al. (1999), were not taken into account. This should have resulted in increased heritabilities. However, our estimates were also in the same range as those obtained by Grossman and Bohren (1985) in chickens and by Gebhardt-Henrich and Marks (1993) and Akbas and Oguz (1998) in quails. High positive genetic correlation between A and B on the one hand, and negative correlation between B and K on the other, could be expected from the mathematical relationship between the parameters \( A = BW_0 \times \exp(B) \) and \( B = L/K \), respectively. The negative correlation between A and K could also be expected, because a rapid decrease in growth rate after inflection resulted in a lower asymptotic body weight. The residual correlation between A and B was very high.

**Estimates of the Gompertz Curve Parameters**

Most, but not all, of the mean estimates of individual phenotypic values were similar to those estimated in the same lines by Mignon-Grasteau et al. (1999). In lines X+ and X−, age at inflection was consistently higher (between 7.4 d for X+ males and 12.8 d for X− females) than with nonlinear regression estimates.

**Figure 1.** Distributions of the parameters of the Gompertz curve A, B, and K in males at the 10th generation of selection.

**Figure 2.** Distributions of the parameters of the Gompertz curve L, T, and BW₀ in males at the 10th generation of selection.
This increase in $T_I$ was consistent with the objective of selection in these lines (i.e., reducing initial growth rate). These differences between studies most probably resulted from better fitting of the growth curve in the present research, as the current procedure uses information on related animals. Moreover, current techniques allowed estimation of parameters of the Gompertz function with a good precision for animals that were not regularly measured, whereas this information had to be treated as missing with nonlinear regression.

Differences in Estimated Growth Curve Parameters Between Sexes

Comparing asymptotic body weights of males and females was of particular interest to study the extent of differences in precocity between sexes, as $A$ was directly proportional to body weight at inflection ($A = BW/e$). At the point of inflection, males and females were of the same physiological age, i.e., 37% of adult body weight. At this age, a low dimorphism was observed in line X++, which was also the line where the smallest difference was observed in $T_I$ (i.e., 2.2 d). However, sexual dimorphism cannot be reduced to differences in precocity. If this were the case, classification of lines on age at inflection and on asymptotic body weight would be the same, and this was not true. For example, similar differences in $T_I$ were observed between sexes in lines X-- and X++ (4.0 d and 3.8 d, respectively), but with very different values of parameters, resulting in much greater dimorphism for $A$ in line X++ than in line X-- (976 g and 771 g, respectively). Furthermore, the difference in $T_I$ in line X-- was close to the difference in the control line (3.8 d and 4.7 d, respectively), but inflection occurred much sooner in line X++ and thus dimorphism of $A$ was much less in this line (i.e., 771 g) than in line X00 (i.e., 892 g). Finally, in spite of a considerable difference between sexes for $T_I$ (5.8 d) in line X--, the curve was so flattened and growth so slow that, even at older ages, sexual dimorphism of body weight was moderate (i.e., 703 g).

Scale effects were not the only cause of sexual dimorphism, as differences between lines remained when sexual dimorphism was computed as a proportion (Figure 5b). Moreover, this probably explained why lines ranked differently for $A$ and for sexual dimorphism of $A$.

More importantly, sexual dimorphism of body weight at a given physiological age was very different from sexual dimorphism at a given chronological age (i.e., the economically important trait). It varied with age, as shown in Figure 5 for absolute and relative sexual dimorphism. At 8 wk, relative dimorphism was smaller in line X++ than in line X00, probably as a result of the decrease in difference in precocity. Absolute sexual dimorphism was also smaller in this line than in line X--. But in both lines sexual dimorphism was increased. This difference supported the hypothesis that commercial selection for higher body weight at age of slaughter has contributed to the increase in sexual dimorphism. Such a selection was probably halfway between lines X++ and X--. However, the difference between those two lines suggests that sexual dimorphism could probably be reduced by selecting for both juvenile and adult body weight. A greater decrease would require reducing the scale effect and thus body weight. This was corroborated by the estimation of genetic correlation between sexual dimorphism and body weight, which was higher in males than in females (.69 and .48 at 8 wk, respectively, as estimated by Mignon-Grasteau et al., 1999).

Conclusion

Selection on body weight at 8 and 36 wk modified the whole growth curve, as all the parameters describing the growth curve evolved with selection, with high ini-

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Table 3. Probabilities (%) that the differences between sexes are higher in the selected lines than in the control line

<table>
<thead>
<tr>
<th>Trait</th>
<th>Dimorphism treated as</th>
<th>X++</th>
<th>X+-</th>
<th>X+-</th>
<th>X--</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Absolute difference(^a)</td>
<td>50.5</td>
<td>11.1</td>
<td>20.0</td>
<td>29.8</td>
</tr>
<tr>
<td></td>
<td>Relative difference(^b)</td>
<td>29.3</td>
<td>27.0</td>
<td>4.6</td>
<td>66.2</td>
</tr>
<tr>
<td>B</td>
<td>Absolute difference</td>
<td>38.0</td>
<td>22.5</td>
<td>16.4</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td>Relative difference</td>
<td>32.7</td>
<td>26.4</td>
<td>12.5</td>
<td>51.5</td>
</tr>
<tr>
<td>K</td>
<td>Absolute difference</td>
<td>77.9</td>
<td>33.4</td>
<td>62.9</td>
<td>23.1</td>
</tr>
<tr>
<td></td>
<td>Relative difference</td>
<td>76.1</td>
<td>38.9</td>
<td>63.1</td>
<td>19.8</td>
</tr>
<tr>
<td>L</td>
<td>Absolute difference</td>
<td>56.7</td>
<td>39.4</td>
<td>59.9</td>
<td>6.2</td>
</tr>
<tr>
<td></td>
<td>Relative difference</td>
<td>75.2</td>
<td>25.9</td>
<td>55.7</td>
<td>17.0</td>
</tr>
<tr>
<td>TI</td>
<td>Absolute difference</td>
<td>32.7</td>
<td>43.1</td>
<td>31.8</td>
<td>86.1</td>
</tr>
<tr>
<td></td>
<td>Relative difference</td>
<td>23.7</td>
<td>55.4</td>
<td>32.4</td>
<td>76.2</td>
</tr>
<tr>
<td>BW0</td>
<td>Absolute difference</td>
<td>13.0</td>
<td>74.2</td>
<td>7.7</td>
<td>77.6</td>
</tr>
<tr>
<td></td>
<td>Relative difference</td>
<td>13.0</td>
<td>75.7</td>
<td>8.3</td>
<td>82.8</td>
</tr>
</tbody>
</table>

\(^a\)Absolute difference is computed with Eq. [9].
\(^b\)Relative difference is computed with Eq. [10].
Figure 3. Distribution of the mean sexual dimorphism of A as a difference from the control line.
Figure 4. Distribution of the mean sexual dimorphism of $T_1$ as a difference from the control line.
Selection modifies sexual dimorphism

Figure 5. Probability that sexual dimorphism (A, absolute; B, relative) of body weight is higher in selected lines than in the control line.

Implications

Reducing sexual dimorphism implies decreasing both the scale effect and the difference in precocity between males and females. The former implies decreasing mean body weight, which has economic consequences. The latter could be achieved by selecting on both juvenile and adult body weight, thus modifying the growth curve. Such an objective could be achieved without im-
important modification of the body weight at slaughter age.

**Literature Cited**


