Correlated Responses in Body Composition to Divergent Selection for Exponential Growth Rate to 14 or 42 Days of Age in Chickens

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ABSTRACT Chicks divergently selected for 14-d (14H and 14L) or 42-d (42H and 42L) exponential growth rate (EGR) over five generations were used to determine correlated responses between growth at different ages and body composition. Regression coefficient estimates across five generations of selection were not significant for any line at either age for percentage total body water or protein. Genetic correlations between EGR from hatching to 14 d of age (EGR14) and 42-d percentage carcass fat were −0.18, −0.57, 0.63, and −0.79 among the 14H, 14L, 42H, and 42L lines, respectively. Genetic correlations between EGR from hatching to 42 d of age (EGR42) and 42-d percentage carcass fat were 0.09, −0.67, 0.50, and −0.75 among the 14H, 14L, 42H, and 42L lines, respectively. During the short-term selection experiment, selection for fast EGR14 or EGR42 increased fat at the age of selection. However, selection for fast EGR42 increased body weight and percentage fat at 42 d of age (DOA), whereas selection for fast EGR14 increased body weight but not fat at 42 DOA. Therefore, it is possible to simultaneously select for high body weight at, or near, the inflection point of the growth curve without increasing fat deposition or obesity by taking advantage of the lack of a genetic correlation between EGR14 and body fat percentage at later ages.

(Key words: selection, growth, body composition, correlated response)

INTRODUCTION

In selection experiments by McCarthy and co-workers (e.g., Hayes and McCarthy, 1976; McCarthy and Doolittle, 1977), it was shown that carcass fat of mice increased at, and beyond, the age of selection at early and late ages (35 and 70 d of age). These experiments led to the general hypothesis of McCarthy and Siegel (1983) that “selection for increased weight would be expected to increase the proportion of fat at all ages if the developmental pattern of fat deposition were to remain unaltered [italics added].”

Indeed, selection for body weight at a fixed juvenile age in chickens has consistently resulted in an increase in carcass fat at the age of selection (Pym and Solvyns, 1979; Barbato et al., 1983; Siegel and Dunnington, 1987). But, Katanbaf et al. (1988) showed that although carcass fat increased at the age of selection in lines divergently selected for 56-d body weight, the lines did not necessarily differ in fat content at the same body weight. Hence, carcass fat was considered to be a concomitant of selection for body weight at a particular age. These data are consistent with the hypothesis that selection for increased body weight is expected to increase the proportion of fat after the age of selection (McCarthy and Siegel, 1983).

However, as Anthony et al. (1991) pointed out, Katanbaf et al. (1988) performed their experiment on lines that were selected at or beyond the inflection point of the normal growth curve, as did Barbato et al. (1983) and McCarthy and Siegel (1983). Barbato (1992a) reported a divergent selection experiment, for high and low exponential growth rate (EGR) at 14 (14H, 14L) or 42 (42H, 42L) d of age (DOA), in which the growth curves of chickens were altered. Chicks selected for high EGR at 14 DOA had greater body weights than the 42H chicks at 14 d. By 42 d, 14H and 42H chicks had the same body weight. Chicks selected for low EGR at 14 DOA had lower body weights than the 42L chicks at 14 d, but by Day 42, 14L chicks were heavier than 42L chicks. This paper reports the correlated responses in total body composition among the aforementioned populations to selection for EGR at different ages.

Abbreviation Key: DOA = days of age; EGR = exponential growth rate; 14H = line selected for high EGR at 14 DOA; 14L = line selected for low EGR at 14 DOA; 42H = line selected for high EGR at 42 DOA; 42L = line selected for low EGR at 42 DOA.
MATERIALS AND METHODS

Base Population and Selection Criteria

Subsamples of the populations from a double divergent selection experiment previously described by Barbato (1992b) were used. Athens-Canadian randombred chickens were divergently selected for EGR to 14 or 42 DOA, resulting in four populations: 14H, 14L, 42H, and 42L. Selection intensities, response and correlated responses in body weight and growth were reported earlier (Barbato, 1992b). In Generations 0 through 5, 10 to 12 chicks, at 14 and 42 DOA, from each sex and population were killed by decapitation, exsanguinated, and placed in individual plastic bags that were sealed and frozen at −20 C for subsequent compositional analysis by using the assays reported in Barbato (1992a).

Linear regression and all subsequent analyses were performed with a combination of SAS software (1988).

Responses to Selection

Correlated responses to divergent selection for exponential growth rate to 14 DOA (EGR_{14}) and exponential growth rate to 42 DOA (EGR_{42}) were considered to be absolute body weight, carcass fat, carcass protein, and carcass water expressed as a percentage of body weight at 14 or 42 DOA. Responses were estimated as the linear regression of the least squares means of each line over generations, uncorrected for environmental trend.

Heritabilities and Correlations

Realized and correlated heritabilities (h^2) were estimated via the regression of the response, uncorrected for environmental trends, on cumulative selection differentials (Falconer, 1953; Carte and Siegel, 1970).

Genetic correlations were estimated using the method of Falconer (1989), expressed as follows:

\[ r_A = \frac{\text{COV}_{XY}}{\left(\text{COV}_{XX} \cdot \text{COV}_{YY}\right)} \]

where COV_{XY} is the cross-covariance, and COV_{XX} and COV_{YY} are the offspring-parent covariances of each character separately. Phenotypic correlations were estimated as follows:

\[ r_P = \frac{\text{COV}_p}{\sigma_p \sigma_y} \]

where COV_p is the phenotypic covariance (with environmental covariance assumed to be zero), and \(\sigma_p\) and \(\sigma_y\) are the standard deviations of character x and y. Environmental correlations were estimated as follows:

\[ r_E = \frac{r_P - h_n h_x h_y}{e_x e_y} \]

where \(h_n = \sqrt{h_n^2}\) and \(e_n = \sqrt{1 - h_n^2}\).

No line-by-sex interactions were observed for any trait in any generation, therefore only the line estimates are presented.

Given the large number of variables being correlated, and the difficulty of assimilating such a large data set, we have presented the correlations of EGR_{14} and EGR_{42} with the body composition data using a radar plot. The radar plot has been drawn with each selected population having correlations represented on a vector having a range of |−1.0 to +1.0|. The four vectors (representing the four populations) have been drawn symmetrically to intersect at |−1.0|. This method of presentation makes it simple to observe large data sets for symmetrical (or asymmetrical) relationships among phenotypic, genetic, and environmental correlations.

RESULTS

Direct and Correlated Responses

Direct and associated responses in EGR_{14} and EGR_{42} to five generations of divergent selection for EGR_{14} and EGR_{42}, are presented in Figure 1. The direct response,
across five generations, was greater than the associated response for fast and slow EGR chicks at 14 DOA (14H: 0.014 versus 42H: 0.010; and 14L: −0.009, versus 42L: −0.008, respectively, Figure 1). At 42 DOA, the direct response, fast EGR, across five generations, was greater than the associated response (14H: 0.0072 versus 42H: 0.0081, Figure 1). At 42 DOA for slow EGR, the direct response was greater than the associated response when averaged through five generations (42L: 0.0010 versus 14H: 0.0016; Figure 1). At 14 DOA (EGR14), divergence of the 14H and 14L was significant (0.0064, Table 1). At 42 DOA (EGR42), divergence of 42H and 42L was significant (0.0062, Table 1). At 42 DOA (42H—42L), divergence of body weight per generation was greater than the associated response (14H: 0.0072 versus 42H: 0.008, respectively, Figure 1).

Regression coefficient estimates of direct and correlated responses are located on Table 1. The correlated responses, to divergent selection for EGR14 and EGR42, body weight are located on Table 1. The correlated regression coefficient estimates indicate that EGR and body weight not only moved in the same direction but with similar intensity (Table 1).

**Realized and Correlated Heritability Estimates**

Table 2 shows that realized heritability estimates for EGR14 were greater for the 14H line when compared to the 42H line. Realized heritability estimates for the 14L and 42L lines were 0.18 and 0.17, respectively, at 14 DOA (Table 2). Correlated heritability estimates for body weight were highest in the 42L line when compared to others. However, the response to selection was negative at 14 DOA (Table 1). The 14H and 42H lines had the highest correlated heritability estimates for percentage carcass water when compared to others, at 14 DOA. The 14H and 42L lines had the highest correlated heritability estimates for percentage carcass fat when compared to others at 14 DOA. Correlated heritability estimates for percentage carcass protein were highest among the low EGR lines at 14 DOA (Table 2).

At 42 DOA, realized heritability estimates were higher for the 14L line versus the 42L line for EGR42 (Table 2). Realized heritability estimates for the 14L and 42L lines were 0.04 and 0.06, respectively. Correlated heritability estimates for body weight were highest among the high EGR lines compared to the low EGR lines (Table 2). Among the 14H, 14L, 42H, and 42L lines, correlated heritability estimates for percentage carcass water and fat were 0.38, 0.37, 0.04, and 0, and 0.04, 0.36, 0.20, and 0.07, respectively. Correlated heritability estimates for percentage carcass protein were moderate to high for all lines at 42 DOA (Table 2) even though the response to selection was negative for all lines at 42 DOA (Table 1).

**Genetic, Phenotypic, and Environmental Correlations**

Genetic correlations ($r_A$) were positive and symmetrically similar between EGR (14 and 42) and body weight...

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**TABLE 1. Regression coefficient estimates of direct and correlated responses in body weight and carcass composition among lines divergently selected for exponential growth rate (EGR) at 14 (14H and 14L) or 42 (42H and 42L) d of age**

<table>
<thead>
<tr>
<th>Line</th>
<th>EGR (ln/g/d$^{-1}$)</th>
<th>Body weight (g/generation)</th>
<th>Water (g/100 g BW/generation)</th>
<th>Fat (g/100 g BW/generation)</th>
<th>Protein (g/100 g BW/generation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14H</td>
<td>0.0031</td>
<td>4.2</td>
<td>0.17</td>
<td>0.17</td>
<td>0.43</td>
</tr>
<tr>
<td>14L</td>
<td>−0.0054</td>
<td>−5.0</td>
<td>0.11</td>
<td>−0.23</td>
<td>0.37</td>
</tr>
<tr>
<td>14H—14L</td>
<td>0.0064*</td>
<td>9.2</td>
<td>0.06</td>
<td>0.04</td>
<td>0.05</td>
</tr>
<tr>
<td>42H</td>
<td>0.0031</td>
<td>3.5</td>
<td>0.20</td>
<td>0.14</td>
<td>0.31</td>
</tr>
<tr>
<td>42L</td>
<td>−0.0031</td>
<td>−3.2</td>
<td>−0.20</td>
<td>−0.03</td>
<td>0.51*</td>
</tr>
<tr>
<td>42H—42L</td>
<td>0.0062</td>
<td>6.7</td>
<td>0.40</td>
<td>0.17</td>
<td>−0.20</td>
</tr>
</tbody>
</table>

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1EGR = ln b − lna/t, where $t =$ time (d), $a =$ BW at hatching, and $b =$ BW at 14 or 42 d of age (EGR14 and EGR42, respectively).

*($P < 0.05$); **($P < 0.01$) indicate regressions that were significantly different from zero.
(at 14 DOA and at 42 DOA) for all lines (Figures 4 and 5). Genetic correlations between EGR\textsubscript{14} and 42-d percentage carcass fat were negative for all lines except the 42H line. Percentage fat measured between EGR\textsubscript{42} and 42-d, Figure 5, clearly shows positive correlations ($r_x$) of 0.09 and 0.50 for 14H and 42 lines, respectively. Genetic correlations between EGR\textsubscript{14} and 42-d percentage carcass protein were positive for the 14H, 14L, 42H, and 42L lines (0.36, 0.24, 0.22, and 0.05, respectively). Genetic correlations between EGR\textsubscript{42} and 42-d percentage carcass protein were positive for the 14H (0.10) and 14L (0.27) lines and negative for the 42H (−0.06) and the 42L (−0.34) lines. Genetic correlations between EGR\textsubscript{14} and 42-d, percentage carcass water were positive for all lines. Between EGR\textsubscript{42} and 42-d, genetic correlations for percentage water were negative for the high lines.

Examination of both figures indicates that phenotypic correlations ($r_P$) between EGR\textsubscript{14} and 42-d body weight and carcass components and between EGR\textsubscript{42} and 42-d body weight and carcass components moved in the same direction but, in most cases, was of slightly less magnitude.

Environmental correlations ($r_E$) between EGR\textsubscript{14} and 42-d body weight for 14H, 14L, 42H, and 42L chicks were
The current data indicate that fast growing lines of chickens whose growth curves differ also differ in fat content. The results with the 42H line support the hypothesis that percentage fat increases at the age of selection; however, percentage body fat in the 14H line does not continue to increase after 14 DOA. Barbato (1992a) demonstrated that different genes or gene combinations are involved in determining growth rate to different ages. The results of this experiment imply that some proportion of the genes that differ among these lines are involved in the development of adipose stores. Further, the increase in adiposity of the 42H line at later ages is probably related to the decrease in age at sexual maturity and decrease in egg production in hens of that line (Kerr et al., 2001).

These data have clear implications for current attempts to identify quantitative trait loci or specific genes involved in growth and fat deposition (e.g., Plotsky et al., 1993; van Kaam et al., 1999). As elegantly shown by Cheverud’s group (Cheverud et al., 1996; Vaughn et al., 1999), different QTL in mice, and by association different genes, influence growth rate to different ages. Further, fat deposition is a dynamic process, whereby animals that have increased fat deposition at very early ages are not necessarily those that will have the greatest fat at more mature stages of life. One might suspect that selection for EGR14 has resulted in a genetic dissection of cell division and later cell growth, as suggested by the work of Winick and Noble (1965) and as attempted in mice by Atchley et al. (2000).

By selecting on components of the growth curve, we have altered the traditional genetic correlation between body weight at a given age and fat deposition. Figures 4 and 5 clearly illustrate the differential correlated responses in carcass compositions between selection for fast growth at 14 d of age versus fast growth at 42 d of age. In the 14H population, fast early growth is not necessarily those that will have the greatest fat at more mature stages of life. One might suspect that selection for EGR14 has resulted in a genetic dissection of cell division and later cell growth, as suggested by the work of Winick and Noble (1965) and as attempted in mice by Atchley et al. (2000).

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line allows for identical genetic improvement in body weight as the commercial practice while avoiding the negative concomitants of obesity (this paper), reproduction (Kerr et al., 2001), and susceptibility to ascites (Barbato, 1997). Further, the double, divergent selection experiment described herein provides us with an ideal model to identify genetic markers or genes for accelerated growth, fat deposition, and fitness traits by using modern molecular techniques.

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REFERENCES


