Fitting animal models for the estimation of genetic parameters of birth weight in Aradi and Damascus goats, and their crosses in Saudi Arabia

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The goat population of the world increased between 1985 and 2009 by about 66% (Dubeuf and Boyazoglu 2009). Studies on genetic parameters in goats showed that body weights are moderately heritable (Rashidi et al. 2008) and genetic potential and maternal and environmental factors determine it (Mandal et al. 2006). However, BW is often considered as an early indicator of late growth and economic benefit (Portolano et al. 2002, Harrford et al. 2006). The major ways to increase the productive profit are—effective selection by accurate use of genetic parameters, improved environmental condition (Al-Shorepy et al. 2002) along with effective use of direct genetic variance—maternal effects become more important for early growth traits selection (Al-shorpy 2001).

Estimates of direct genetic, maternal, and environmental components of covariance became more important to make informative genetic evaluation and to develop multi-trait selection indices (Harnford et al. 2005, 2006, Mandal et al. 2008); now mixed model methodologies are methods of choice to estimate breeding values (Henderson 1988, Meyer 1989). Mandal et al. (2006, 2008) investigated importance of applying the most appropriate model for the estimation of covariance components and genetic parameters for the traits that are influenced by maternal effects in various species and breeds.

The objectives of this study are to estimate the covariance components and the genetic parameters for this trait, and to determine the most appropriate models of analysis for BW of Aradi and Damascus goats, and their crosses.

Breeding plan: Records (2,478) of BW of pure Aradi and of crossbred with Damascus kids were collected during the period from 2005 to 2010. Does (240) of Aradi goats were randomly divided into 2 groups, 120 does each, to be artificially inseminated from the semen of elite bucks of Aradi or Damascus breed. Damascus breed does (120) were inseminated with the semen of the same breed to produce purebred kids. Moreover, crossbred does of ½D½A were backcrossed with Damascus bucks to get the genetic group of ¾D¼A. Accordingly, the breeding plan permitted the production of 2 genetic groups ¼D¾A and ¾D¼A, in addition to the pure breeds AA and DD.

Statistical analysis: Data were analyzed using general linear model (SAS 1990) to determine the fixed effects to be included in the model. The statistical model included the fixed effect of location (2 levels: Qassim and Jouf), genetic group (4 levels: Aradi, Damascus, ½D½A, and ¾D¼A), year-season of kidding (20 levels: 4 seasons by different 5 years), sex (2 levels: male and female), and type of birth (3 levels: 1, 2 and 3). All these effects were significant (P<0.05) for body weight (BW), and all were included in the models that were later used to estimate genetic parameters.

Variances and covariance components were estimated by the restricted maximum likelihood (REML) using a derivative-free algorithm fitting an animal model (DFREML, Meyer 2001). Convergence of the REML solutions was assumed when the variance of function values (-2 logL) in the Simplex was less than 10^-8.

To ensure that a global maximum was reached, analyses were restarted for several rounds of iterations using results from the previous round as starting values. When estimates did not change, convergence was confirmed. Standard errors were calculated for the estimated parameters as a part of the DFREML program (Meyer 2001). Univariate animal models that fitted for the estimation of covariance components were the following:

\[ y = Xb + Z\alpha + e; \text{ model 1} \]
\[ y = Xb + Z\alpha + Z_cC + e; \text{ model 2} \]
\[ y = Xb + Z_a\alpha + Z_m m + e; \text{ no covariance (a,m) model 3} \]
\[ y = Xb + Z_a\alpha + Z_mm + e; \text{ with covariance (a,m) model 4} \]
\[ y = Xb + Z_a\alpha + Z_mC + Z_mm + e; \text{ no covariance (a, m) model 5} \]

where, b, a, m, c, and e are vectors of the fixed effects, the direct genetic effects, the maternal direct genetic effects, the permanent environmental effects of the dam, and the residual effects, respectively; X, incidence matrix for fixed effects; Z,
incidence matrix of random effects; e, residual effects normally and independently distributed (0, σ²e).

Model 1 included only the direct genetic effect of animal (a). Model 2 included (a) and the permanent environmental effect (c). Model 3 included (a), the maternal genetic value (m), assumed to be uncorrelated with (a). Model 4 was the same as model 3, but with (a) and (m) assumed to be correlated. Model 5 included (a), (m) uncorrelated with (a) and the permanent environmental effect (c). The model used by Dickerson (1947 and 1970), was applied in this study to estimate the total heritability (h²t) as follows:

\[ h^{2t} = \frac{h^2_a + 0.5h^2_m + 1.5}{\sigma^2_p}, \]

where, \( h^2_a \), heritability for direct genetic animal effect; \( h^2_m \), heritability for maternal genetic effect; \( \sigma_{am} \), estimate of covariance between direct and maternal genetic effects; and \( \sigma^2_p \), estimate of phenotypic variance.

The total heritability was estimated to show the effect of adding covariance between the direct genetic effects to the total heritability, as compared to the direct heritability. However, the environmental parameter estimated was the ratio of variance associated with the dam to the total phenotypic variance.

The method described by Mood et al. (1963) and Rao (1973) to calculate the ratio of likelihood was used to compare models. The ratio 2 [log \( \hat{E}_i \) - log \( \hat{E}_j \)] is approximately distributed as chi-square with a degree of freedom equal to the parameters in the models i and j, where \( \hat{E} \) is the value of likelihood function for the model, after the convergence criterion was reached. Moreover, in \( i = j \) there is no comparison between the 2 models. Thus, the variance and covariance structure is as follows.

\[
\begin{bmatrix}
a \\
m \\
c \\
e
\end{bmatrix} =
\begin{bmatrix}
\Lambda \sigma^2_a & \sigma_{am} & 0 & 0 \\
\sigma_{am} & \Lambda \sigma^2_m & 0 & 0 \\
0 & 0 & 1 \sigma^2_c & 0 \\
0 & 0 & 0 & 1 \sigma^2_e
\end{bmatrix}
\]

where, A, numerator relationship matrix; \( \sigma^2_a \), direct genetic variance; \( \sigma^2_m \), maternal genetic variance \( \sigma_{am} \), covariance between a and m; \( \sigma^2_c \), variance due to permanent environmental effect; In, identity matrix of order equal to the records trait; \( \sigma^2_e \), the error variance.

The convergence criterion used in this analysis was the variance of likelihood in the current set of simplex value in the derivative free REML algorithm (Boldman et al. 1995). The desired range of this variance at the convergence is 10⁻⁶ to 10⁻¹⁰. The estimation program should be restarted repeatedly with the converged values and the prior simplex information until convergence occurred to the same values that were supplied as starting values. This approach should ensure that a global, rather than a local, maximum likelihood estimate is obtained. All estimates were based on restarted system.

Likelihood ratios for testing models are presented in Table 1. Significantly, more likelihood value was obtained when the permanent and the maternal component were added to direct genetic component only, assuming no covariance between direct genetic and maternal genetic effects (Model 5). Moreover, the permanent environmental effect added significant information in (Model 2). As a result, the covariance between the direct and the maternal genetic effects in model 4 added extra information, when compared to Model 3 without covariance. The estimates of genetic, environmental, and phenotypic parameters that were obtained in the five animal models are presented in (Table 2).

The estimates of direct genetic heritability \( h^2_a \) for BW were moderate and accurately estimated as 0.43, 0.23, 0.21, 0.18 and 0.22 for models 1,2,3,4 and 5, respectively. The result obtained by model 5 was in agreement with the estimates reported by Zhang et al. (2008). Furthermore, the estimates of the maternal direct genetic effect \( h^2_m \) as a proportion of the total phenotypic variance, were 0.17, 0.15, and 0.03 of the models 3, 4 and 5, respectively. The estimate of \( h^2_m \) by model 5 was 0.03, which is lower than those by Al-Shorepy et al. (2002) and Rashidi et al. (2008).

The estimates of \( h^2_a \) and \( h^2_t \) by the different models used were different, but all models were closely the same in both ends. This was in accordance with the observation that \( h^2_t \) was reduced by a half when the maternal genetic effect was added in model 3 (Table 2). The estimates of \( h^2_m \) from the models 3 and 4 were close: 0.17 and 0.15, respectively. Model 5 showed a difference in the estimate of \( h^2_m \) (0.03), which was higher than in those estimates in models 3 and 4 (0.17 and 0.15). Vice versa, the maternal genetic effect showed 3% and 15% up to 17% of the phenotypic value in the models 5, 4 and 3, respectively. The increase in \( h^2_t \) estimates, from 0.30 in model 3 to 0.32 in model 4, was not significant, which

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Table 1. The differences between the likelihood function of two different animal models, asymptotically distributed as chi-square, to test the difference between models

<table>
<thead>
<tr>
<th>H₀</th>
<th>H₁</th>
<th>χ²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–2</td>
<td>Mod2&gt;mod1</td>
<td>26.92*&lt;a</td>
</tr>
<tr>
<td>1–3</td>
<td>Mod3&gt;mod1</td>
<td>20.05*&lt;b</td>
</tr>
<tr>
<td>1–4</td>
<td>Mod4&gt;mod1</td>
<td>20.56*&lt;b</td>
</tr>
<tr>
<td>1–5</td>
<td>Mod5&gt;mod1</td>
<td>27.25*&lt;c</td>
</tr>
<tr>
<td>2–4</td>
<td>Mod4&lt;mod2</td>
<td>6.36*&lt;a</td>
</tr>
<tr>
<td>2–5</td>
<td>Mod5&lt;mod2</td>
<td>0.33*&lt;a</td>
</tr>
<tr>
<td>3–4</td>
<td>Mod4&lt;mod3</td>
<td>0.51*&lt;a</td>
</tr>
<tr>
<td>3–5</td>
<td>Mod3&lt;mod5</td>
<td>7.20*&lt;a</td>
</tr>
</tbody>
</table>

*adegree of freedom=1, bdegree of freedom=2, cdegree of freedom=3; H₀= null hypothesis; H₁= alternative hypothesis.
Table 2. Estimates of genetic, environmental, and phenotypic parameters, obtain by five models

<table>
<thead>
<tr>
<th>Item</th>
<th>Mod1</th>
<th>Mod2</th>
<th>Mod3</th>
<th>Mod4</th>
<th>Mod5</th>
</tr>
</thead>
<tbody>
<tr>
<td>$h^2_a$</td>
<td>0.43</td>
<td>0.23</td>
<td>0.21</td>
<td>0.18</td>
<td>0.22</td>
</tr>
<tr>
<td>$h^2_m$</td>
<td>$\sigma_{am}/\sigma^2_p$</td>
<td>$-$</td>
<td>$-$</td>
<td>0.17</td>
<td>0.15</td>
</tr>
<tr>
<td>$r_{am}$</td>
<td>$-$</td>
<td>$-$</td>
<td>0.0</td>
<td>0.041</td>
<td>0.0</td>
</tr>
<tr>
<td>$r^2_c$</td>
<td>0.43</td>
<td>0.23</td>
<td>0.30</td>
<td>0.32</td>
<td>0.24</td>
</tr>
<tr>
<td>$r_{p}$</td>
<td>$-$</td>
<td>$-$</td>
<td>0.0</td>
<td>0.26</td>
<td>0.0</td>
</tr>
<tr>
<td>$\sigma^2_r$</td>
<td>$-$</td>
<td>0.10</td>
<td>$-$</td>
<td>$-$</td>
<td>0.08</td>
</tr>
<tr>
<td>$\sigma^2_C$</td>
<td>0.54</td>
<td>0.53</td>
<td>0.53</td>
<td>0.53</td>
<td>0.53</td>
</tr>
<tr>
<td>$\sigma^2_e$</td>
<td>0.30</td>
<td>0.31</td>
<td>0.33</td>
<td>0.34</td>
<td>0.31</td>
</tr>
</tbody>
</table>

$h^2_a$, heritability for direct genetic animal effect; $h^2_m$, heritability for maternal genetic effect; $\sigma^2_{am}/\sigma^2_p$, covariation between direct and maternal genetic effect as a proportion of phenotypic variance; $h^2_v$, heritability for the total genetic contribution of the animal (total heritability); $r_{am}$, correlation between direct and maternal genetic effect; $\sigma^2_C$, relative contribution of permanent environmental effects; $\sigma^2_p$, phenotypic variance; $\sigma^2_e$, residual variance.

may be due to the lower contribution of covariance between the direct genetic and the maternal genetic effects; Table 2 showed an addition of covariance effect between the maternal genetic and the direct genetic effects on the total genetic contribution. The $h^2_m$ and $r_{am}$ estimates, by applying model 4, were 0.15 and 0.26 respectively, had an effect on the estimate $h^2_v$, which increased from 0.30 in model 3 to 0.32 in model 4. For practical purposes, the permanent environmental effect, the maternal effect, and the genetic correlation between maternal and direct effects could be of concern. The permanent environmental effects explain 8% in model 5 up to 10% in model 2.

It can be concluded that model 5 was the most appropriate model for the explanation of the variation in the BW, the simple model 2 gave close results to those obtained by model 5. The maternal effect, and the covariance between maternal and direct genetic effect, had a significant contribution to the explanation of the total genetic variance.

SUMMARY

Records (2,478) for birth weight (BW) of Aradi and crossbred with Damascus kids were collected from 2005 until 2010. REML procedure was used to estimate the genetic, phenotypic, and environmental parameters through using 5 different animal models: the first included solely the direct genetic effect of animal (a); the second included (a), and the permanent environmental effect (c); the third included (a), the maternal effect (m), assumed to be uncorrelated with (a); the fourth included (a) and (m) assumed to be correlated with (a); the fifth included (a) and (m) uncorrelated with (a) and the permanent environmental effect. The random animal direct genetic (a), the permanent environmental(c), and the maternal genetic (m) effects and covariance between (a) and (m) were sequentially added to the models. Estimates of total heritability from different models ranged from 0.23 to 0.43. Estimates of maternal genetic variance ranged from 0.03 to 0.17% of the phenotypic variance. Estimates of correlation between (a) and (m) ranged from 0.0 in models 3 and 5 to 0.26 in model 4. Permanent environmental effects showed a difference that ranged from 15% in model 5 to 18% in model 2 of the total phenotypic variance. Among all the models used, model 5 was the most appropriate to explain the variation in a continuous trait. The maternal effect and covariance between maternal and direct genetic effects have a significant contribution in the explanation of total genetic variance.

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