Kafkas Univ Vet Fak Derg
17 (3): 387-392, 2011
DOI:10.9775/kvfd.2010.3606

RESEARCH ARTICLE

Comparison of Different Models in Genetic Analysis of Dystocia

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Makale Kodu (Article Code): KVFD-2010-3606

Summary

The aim of this study was to compare threshold sire model (TS), threshold sire-maternal grandsire model (TS-MGS) and linear sire-maternal grandsire model (L) for genetic analysis of dystocia. Threshold models were based on Bayesian approach. In the study, a total of 19439 dystocia records from Holsteins in USA were used. The effects of calving year-season, sex of calf, parity of dam, sire of calf and herd effects were included in all models and also maternal grandsire effect of calf was included in only sire-maternal grandsire models. Variance-covariance estimates were greater in threshold models than in linear model. Estimates of heritability (±SE) of dystocia based on direct genetic effects (h^2_D) and maternal genetic effects (h^2_M) were 0.18±0.004 and 0.14±0.004 from TS-MGS and 0.12±0.003 and 0.09±0.003 from L, respectively. Heritability estimates based on direct genetic effects from TS was 0.20±0.009. Genetic correlation between direct and maternal genetic effect were -0.087±0.006 from the TS-MGS and -0.253±0.010 from L. It was concluded that the threshold models were better than the linear model in the analysis of dystocia. The higher heritability estimates on the underlying scale from threshold models should allow greater genetic improvement than those using linear model estimations.

Keywords: *Dystocia, Sire threshold model, Sire- maternal grandsire threshold model, Linear model, Holstein*

Buzağılama Güçlüğünün Genetik Analizinde Farklı Modellerin Karşılaştırılması

Özet

Bu çalışmanın amacı, buzağılama güçlüğünün genetik analizinde eşikli baba (TS), eşikli baba-ana tarafından büyükbaba (TS-MGS) ve doğrusal baba-ana tarafından büyükbaba (L) modellerini karşılaştırmaktır. Eşikli modeller Bayes yaklaşımına dayanmaktadır. Çalışmada, Amerika'daki Siyah Alacalara ait 19439 adet buzağılama güçlüğü kaydı kullanılmıştır. İstatistiksel modeller buzağılama yılı-mevsimi, buzağı cinsiyeti, annenin laktasyon sırası, buzağının babası ve sürü etkilerini içerirken, baba-ana tarafından büyükbaba modelinde, buzağının ana tarafından büyükbaba etkisi de bulunmaktadır. Buzağılama güçlüğü için eşikli modellerle tahminlenen varyans-kovaryanslar, doğrusal modelden tahminlenen değerlerden daha yüksek bulunmuştur. Doğrudan genetik etkiler (h^2_D) ile anaya ait genetik etkiler (h^2_M) kullanılarak elde edilen kalıtım derecesi tahminleri ve standart hataları TS-MGS ile sırasıyla 0.18±0.004 ve 0.14±0.004; doğrusal model ile 0.12±0.003 ve 0.09±0.003 olarak bulunmuştur. Doğrudan genetik etkilere ait kalıtım derecesi TS ile 0.20±0.009 olarak tahminlenmiştir. Ayrıca doğrudan ve anaya ait genetik etkiler arasındaki genetik korelasyon TS-MGS ile -0.087±0.006 bulunurken, doğrusal model ile -0.253±0.010 düzeyinde tahminlenmiştir. Sonuç olarak, buzağılama güçlüğünün analizinde eşikli model, doğrusal modele göre daha iyi kabul edilebilir. Doğrusal modelle karşılaştırıldığında eşikli modelle elde edilen daha yüksek kalıtım derecesi, daha yüksek genetik ilerleme sağlamaktadır.

Anahtar sözcükler: *Buzağılama güçlüğü, Baba eşikli modeli, Baba-ana tarafından büyükbaba eşikli modeli, Doğrusal model, Siyah Alaca*

INTRODUCTION

Dyscotia as a discrete trait is not distributed normally. However, linear models were applied in many studies for genetic parameter estimation of dystocia [1-3]. Theoretically, use of linear models is not appropriate for this kind of traits [4]. Gianola and Foulley [5], Gilmour [6], and Harville and Mee [7] proposed the threshold model techniques. Foulley et al.[8] and Janss and Foulley [9] extended the threshold methodology to multitrait analysis.

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In the Unites States from 1988 to 2001, a threshold sire model [10] was used for genetic evaluations of dystocia. Genetic evaluations for dystocia have been carried out since 1977 [11]. Instead of sire model, a sire-maternal grandsire (MGS) model [11] was implemented in 2002 for dystocia evaluation in USA. Adding MGS effect to the model is expected to improve accuracy by partially accounting for the genetic merit of the mates of the bull, and differences in the maternal ability of the dams [12].

The aim of this study was to compare threshold sire (TS), threshold sire-maternal grandsire (TS-MGS) and linear sire-maternal grandsire (L) models for heritability estimate of dystocia in Holsteins.

MATERIAL and METHODS

A total of 19439 dystocia records from American Holstein cows calving in 166 herds between years 1980 and 2001 in Columbia and Missouri States were used. Data were provided by National Association of Animal Breeders (NAAB). Dystocia scores were originally on a 1 to 5 scale [13], but we combined the last two categories because of less observation in the 5th category (1.7%). So that, in this study, dystocia scores used were 1 = no problem, 2 = slight problem, 3 = needed assistance, 4 = considerable force. Before analyzing data, some editing was performed in the data set. First of all, seasons were classified as summer (May to September) and winter (October to April). The dam's parity was combined into 3 levels (1, 2, and >3). In the models, fixed effects were sex of calf (male and female), the dam's parity and year-season effect (with 21 levels).

Statistical analyses were carried out using two different methods as Bayesian methodology for threshold models and Maximum Likelihood (ML) methodology for linear model.

Threshold Sire Model (TS): The assumed model for the underlying liability variable (l) for dystocia can be written as:

$$\mathbf{l} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{s} + \mathbf{Z}_2\mathbf{h} + \mathbf{e} \qquad (1)$$

where *l* is a vector of unobserved liability of dystocia; *b* is the vector of fixed effects, *s* is the vector of random sire effects; *h* is a vector of random herd effect; *e* is a vector of random residuals, and *X*, Z_1, and Z_2 denote the incidence matrices relating *l* to *b, s* and *h*, respectively.

The response of dystocia (y_i) for individual *i* was modeled with the following distribution:

$$\prod_{i=1} f(y_i \mid l_i) = \prod_{i=1}\left(\sum_{k=1}^{4} \mathrm{l}(t_{k-1} < l_i < t_k)\mathrm{l}(y_i = k)\right) \qquad (2)$$

where $l_i \sim N(\mathbf{x}_i\mathbf{b} + \mathbf{z}_{i1}\mathbf{s} + \mathbf{z}_{i2}\mathbf{h},\ \sigma_e^2 = 1)$, $\mathbf{x}_i, \mathbf{z}_{i1}$, and $\mathbf{z}_{i2}$ are row vectors for individual *i*, *k* represents dystocia scores (1, 2, 3 and 4), t is threshold value, and $t_0 = -\infty$ and $t_4 = \infty$. The values of t_1 and residual variance (σ_e^2) were fixed to zero and one, respectively. Flat prior distribution was assumed for thresholds t_2 and t_3.

Posterior estimates of sire variance (σ_s^2) was converted to direct genetic (σ_D^2) and phenotypic (σ_p^2) variances were calculated as:

$$\sigma_D^2 = 4\sigma_s^2 \quad \text{and} \quad \sigma_P^2 = \sigma_s^2 + \sigma_h^2 + \sigma_e^2 \qquad (3)$$

Threshold Sire-Maternal GrandSire model (TS-MGS): Because of significant maternal effect, a univariate sire-maternal grandsire threshold liability model was used. In matrix notation the model fitted can be written as:

$$l = Xb + Z_1s + Z_2m + Z_3h + e \qquad (4)$$

where *l* is a vector of unobserved liability of dystocia; *b* is the vector of fixed effects, *s* is the vector of random sire effects; *m* is the vector of random maternal grandsire effect of calf, *h* is the vector of random herd effect; *e* is the vector of random residuals, and *X*, Z_1, Z_2 and Z_3 denote the incidence matrices relating *l* to *b*, *s*, *m* and h, respectively.

Sire and MGS effects were assumed to be correlated, and follow the multivariate normal distribution as,

$$\begin{pmatrix}\mathbf{s}\\ \mathbf{m}\end{pmatrix} \mid \mathbf{G}_0, \mathbf{A} \sim N(\mathbf{0}, \mathbf{G}_0 \otimes \mathbf{A}) \qquad (5)$$

where G_0 is the sire-MGS covariance matrix

$$\mathbf{G}_0 = \begin{bmatrix} \sigma^2_s & \sigma_{s,m} \\ \sigma_{s,m} & \sigma^2_m \end{bmatrix} \qquad (6)$$

where σ^2_s is sire variance, σ^2_m is MGS variance, $\sigma_{s,m}$ is covariance between sire and MGS effects and *A* is the additive genetic relationship matrix among sire and MGS.

Direct genetic variance (σ^2_D), additive maternal genetic variance (σ^2_M) and direct-maternal covariance (σ_{DM}) were calculated as [12,14]:

$$\begin{bmatrix}\sigma^2_D\\ \sigma_{DM}\\ \sigma^2_M\end{bmatrix} = \begin{bmatrix}4 & 0 & 0\\ -2 & 4 & 0\\ 1 & -4 & 4\end{bmatrix}\begin{bmatrix}\sigma^2_s\\ \sigma_{sm}\\ \sigma^2_m\end{bmatrix} \qquad (7)$$

Phenotypic variance (σ^2_p), heritabilities (h^2) and genetic correlation (r_{DM}) between direct and maternal effects were calculated as:

$$\sigma^2_p = \sigma^2_s + 2\sigma_{sm} + \sigma^2_m + \sigma^2_h + \sigma^2_e \quad (8)$$

$$h^2_D = \sigma^2_D/\sigma^2_p, \quad h^2_M = \sigma^2_M/\sigma^2_p \quad \text{and} \quad r_{DM} = \sigma_{DM}/\sqrt{\sigma^2_D\sigma^2_M} \quad (9)$$

Threshold models (TS and TS-MGS) were run by using a fortran program written by Chang [15]. All threshold models were performed 1.000.000 cycles. First 100.000 cycles were determined as a burn-in period. Effective sample size, posterior mean and standard deviation for each parameter estimate were obtained by R Project [16].

Prior distributions were uniform for the *b* and multivariate normal distributions for sire, maternal grandsire and herd effects.

Posterior distributions for herd, sire and residual variances were set to be inverted chi-squared distributions:

$$f(\sigma^2_h) \sim \chi^{-2}(\nu_h, \sigma^2_{h_0}) \quad f(\sigma^2_s) \sim \chi^{-2}(\nu_s, \sigma^2_{s_0}) \quad f(\sigma^2_e) \sim \chi^{-2}(\nu_e, \sigma^2_{e_0}) \quad (10)$$

where ν_h, ν_s and ν_e are the degrees of freedom parameters, and $\sigma^2_{h_0}$, $\sigma^2_{s_0}$ and $\sigma^2_{e_0}$ are the scale parameters.

Posterior conditional distribution for the thresholds (t_2 and t_3) was uniform [17]. The posterior conditional distribution for the underlying liability was truncated normal distribution as described by Sorensen et al.[17].

Linear Model (L): Univariate linear model was also fitted for dysctocia using an AI-REML algorithm [18]. In linear model, dystocia was assumed as a continuous trait. This model included the same effects as TS-MGS model. Variance components were estimated with univariate REML procedure in DFREML program [19]. Approximate standard errors of these variables were obtained by a first-order Taylor series expansion of the average information matrix of the estimated (co)variance components.

RESULTS

Distribution of births according to parity, sex of calves and dystocia subgroups is presented in *Table 1*.

Table 1 shows that most of dystocia records (90%) were coded as 1 and 2, meaning that there is no or slight problem. Percentage of dystocia problem (scores 3+4) was 18.53% in the first parity, while it was 8.21% and 7.60% in the second and third parities, respectively *(Table 1)*. Approximately 10% of the births (dystocia scores "3" and "4") required considerable assistance. Frequency of dystocia problems in male calves was roughly more than 2 times in female calves *(Table 1)*.

Estimates of variance components obtained from both threshold and linear models were summarized in *Table 2*. Estimates of herd variance (σ^2_h), sire genetic variance (σ^2_s) and direct genetic variance (σ^2_d) and were 0.258, 0.066 and 0.264 from TS, 0.259, 0.066 and 0.264 from TS-MGS, respectively. Those from L were 0.015, 0.004 and 0.016, respectively.

Table 3 illustrates the effective sample sizes for sire, herd and MGS variance components and threshold values (t_2 and t_3). The effective sample sizes were computed by using the algorithm proposed by Geyer [20].

The effective sample sizes for variance components and threshold values were ranged from 5230 to 198500.

Table 1. *Frequencies of dystocia groups for parity and sex*
Tablo 1. *Laktasyon sırası ve eşey için buzağılama güçlüğü frekansları*

Parity	Sex	Dystocia					Total (3+4)	Percent (3+4)
		1	2	3	4	Total		
1	Male	1381	431	298	252	2362	550	23.29
	Female	1506	344	189	94	2133	283	13.27
	Total	2887	775	487	346	4495	833	18.53
2	Male	2087	398	168	139	2792	307	11.00
	Female	2248	300	93	50	2691	143	5.31
	Total	4335	698	261	189	5483	450	8.21
3	Male	3875	657	268	212	5012	480	9.58
	Female	3765	445	156	83	4449	239	5.37
	Total	7640	1102	424	295	9461	719	7.60
Total		**14862**	**2575**	**172**	**830**	**19439**		
Percent		**76**	**14**	**6**	**4**	**100**		

Table 2. *Posterior means and standard deviations (SD) of variance components of dystocia from TS, TS-MGS and L*

Tablo 2. *Buzağılama güçlüğü için TS, TS-MGS ve L kullanılarak elde edilen varyans komponentlerine ait ortalamalar ve standart sapmalar (SD)*

Variable	Threshold Models				L	
	TS		TS-MGS			
	Mean	SD	Mean	SD	Mean	SD
σ^2_h	0.258	0.037	0.259	0.040	0.015	0.030
σ^2_s	0.066	0.010	0.066	0.010	0.004	0.009
σ^2_m	-	-	0.062	0.010	0.003	0.009
σ_{sm}	-	-	0.028	0.002	0.001	0.001
σ^2_D	0.264	0.011	0.264	0.011	0.016	0.008
σ^2_M	-	-	0.202	0.011	0.012	0.011
σ_{DM}	-	-	-0.020	0.002	-0.004	0.003

Table 3. *Effective sample sizes of variance components and threshold values*

Tablo 3. *Varyans komponentleri ve eşik değerlerine ait etkili örnek büyüklükleri*

Variances	TS	TS-MGS
σ^2_s	7811	5624
σ^2_h	147676	134856
σ^2_m	-	5230
t_2	193000	198500
t_3	140110	120520

direct and maternal genetic effects. Estimates of variance-covariance components were similar to those obtained by Ramirez-Valverde et al.[22] and Weller et al.[23]. Heritability estimates from threshold models were higher than those from linear model. Previous studies have the same tendency [18,24]. Steinbock et al.[25] estimated direct and maternal heritabilities as 0.06 and 0.05. Jamrozik et al.[26] using linear model estimated these parameters as 0.08 and 0.14, respectively. Varona et al.[27] determined that threshold models gave slightly higher estimates than linear models, particularly when dystocia was analyzed

Table 4. *Estimates of heritabilities and genetic correlations with standard errors*

Tablo 4. *Kalıtım derecesi ve genetik korelasyonlar ile bunlara ait standart hatalar*

Genetic Parameters	TS	TS-MGS	L
Direct heritability	0.20±0.009	0.18±0.004	0.12±0.003
Maternal heritability	-	0.14±0.004	0.09±0.003
Direct and maternal genetic correlation	-	-0.087±0.006	-0.253±0.010

Heritabilities from TS and TS-MGS threshold models and L were given in *Table 4*. The heritability estimates of dystocia (±SE) on the basis of direct genetic effects (h^2_D) were 0.20±0.009 and 0.18±0.004 from both threshold models (TS and TS-MGS) and 0.12±0.003 from L. Estimates of maternal heritability (h^2_M) were 0.14±0.004 and 0.09±0.03 from TS-MGS and L, respectively. Genetic correlations between direct and maternal genetic effects were -0.087±0.006 and -0.253±0.010 from TS-MGS and L, respectively.

DISCUSSION

The current studies of genetic improvement for high meat and milk production in cattle has a potential to cause a relative discordance between dam and fetus, and this leads to increase the dystocia problems [21]. We found in this study that dystocia was affected by both

together with birth weight in a bivariate linear-threshold analysis. Because threshold model equations are nonlinear and involve normal probability functions, computational complexity and computing resources required are greater than those in a linear model analysis [22,23,28].

Effectiveness of MCMC mixing after burn-in was determined by effective sample size of the samples [14,28]. The effective sample sizes give an estimate on the information of the MCMC samples in terms of an equivalent number of independent samples [29]. Umari et al.[30] suggested 100 as the minimum effective sample sizes for reliable statistical inference. In the present study, effective sample sizes for threshold values were found to be higher than those for the variance components (except herd variance components). The results were similar with other MCMC studies [14,27,31,32].

Genetic correlation between direct genetic and maternal genetic effect was found as -0.087±0.006. Similarly

in some studies [25,33] negative genetic correlations were reported between direct genetic and maternal genetic effects for dystocia. In TS-MGS and linear model, negative genetic correlations between direct and maternal effects showed an antagonistic genetic relationship between them. The estimates given by Hansen et al.[34] were not all negative, but they were all near zero, ranging from -0.13 to 0.14. Jamrozik et al.[35] did not report correlation between direct and maternal effects [36]. Philipsson et al.[37] suggested that female calves of a small size are likely to be born easily, but as adults have more difficulties in birth because of the reduced pelvic opening dimensions. This can be biological explanation for negative relationship between direct and maternal effects [38].

Since dystocia has a discrete nature and it's analysis by threshold model would better account for the probabilistic structure of the data [5], threshold models have been reported better than linear models for estimation of genetic parameters of dystocia [22,27]. However, some researchers reported that there are only a slightly advantage of the threshold models to linear models especially if the amount of information for fixed effects is small [39,40]. As a result, threshold models have a theoretical advantage over linear model for categorical traits and could be applied to analysis of categorical traits by using Bayesian methodologies with Markov Chain Monte Carlo algorithm although computations are more difficult with threshold models than with linear model. Threshold model is computationally harder than linear models [23]. However, recent developments in computer hardware and software, threshold model can be applied easily. The including MGS effect in the model provide information from the maternal contribution to dystocia. The TS-MGS model provides more information about both direct (sire) and maternal grandsire dystocia effects. TS-MGS model provide information about the maternal contribution to dystocia and improve the accuracy of evaluations. If MGS information is known, it is better to develop a statistical model including MGS; otherwise sire model can be used. Upon those results, the methodology would be implemented for routine genetic evaluation of dyctocia data in Turkey.

Acknowledgment

TUBITAK (The Scientific & Technological Research Council of Turkey) and Ege University Research Institute are gratefully acknowledged for financial support.

The authors wish to thank Dr. Daniel GIANOLA and Dr. Kent WEIGEL in USA for helps and providing data and Dr. Ignacy MISZTAL for his provision of CMMAT2 fortran subroutines used in this study.

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