

Genetic parameters for anovulation and pregnancy loss in dairy cattle

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ABSTRACT

The objectives were to estimate heritabilities and genetic variances for anovulation at ~50 d in milk and pregnancy loss occurring between first and second pregnancy diagnoses after artificial insemination. Data were originally collected for trials on reproductive management. Anovulation data consisted of 5,818 records from 13 studies in 8 herds with an overall prevalence of 23.3%. A Bayesian approach using Markov Chain Monte Carlo methods was used in mixed threshold models for both traits. The statistical model for anovulation included fixed effects [parity, herd-study-treatment, and body condition score (BCS)], covariates (inbreeding and milk yield), and random effects (sire and residual). A second statistical model included all terms in the first model except BCS. In addition, 2 bivariate, mixed sire models were used to analyze anovulation with BCS and anovulation with milk yield. The posterior mean heritability estimate for anovulation was 0.171 [posterior standard deviation (PSD) = 0.052]. Correlations of anovulation with milk yield were as follows: genetic = 0.168, PSD = 0.187; residual = -0.046, PSD = 0.022; and phenotypic = -0.036. Bivariate analysis of BCS with anovulation showed a genetic correlation (-0.301, PSD = 0.177) and phenotypic correlation (-0.192, PSD = 0.019). Pregnancy-loss data consisted of 3,775 records from 14 studies in 8 herds with an overall prevalence of 14.4%. Analysis of pregnancy loss used a sire-maternal grandsire threshold model with embryo survival as the subject of analysis. Independent variables consisted of fixed effects (parity and herd-study), covariates (embryo and maternal inbreeding), and random effects (sire of embryo, maternal grandsire of embryo, and residual). In addition, separate sire models were analyzed using embryo as the subject and cow as the subject of analysis. The sire-maternal grandsire model yielded a heritability for direct effect of 0.489 (PSD = 0.221) and for maternal effects of 0.166 (PSD = 0.113). In this

study, the breeding value variance for embryo effects was 3 times the breeding value variance for maternal effects, indicating that, at the level of breeding values, the embryo's ability to survive has a greater effect on pregnancy loss than does the cow's ability to maintain the pregnancy. These results suggest that genetic improvement of reproductive performance could be enhanced by selection for fundamental measures such as abnormally long periods of postpartum anovulation and pregnancy loss. Larger studies of these traits are needed to obtain parameter estimates with greater precision.

Key words: anovulation, heritability, pregnancy loss, reproduction

INTRODUCTION

Reproductive success requires proper coordination of many physiological events and insemination practices, some of which are accomplished naturally, but increasingly are managed artificially. Protocols for synchronization of ovulation have been researched and promoted since the mid-1990s (Pursley et al., 1995). Genetic studies of reproductive performance have been conducted over many years, and a national genetic evaluation for female fertility was implemented in 2003 (Van Raden et al., 2004). Traditionally, genetic studies of reproductive performance have been based on time intervals and conception results. Current genetic evaluations for reproduction in the United States are based on days from calving to conception and in Europe, on days from calving to first insemination and nonreturn to first insemination. Approaching genetic improvement of reproduction with the use of physiologic measures and intermediate events in addition to the traditional measures has the potential to increase genetic gain (Darwash et al., 1997; Royal et al., 2002a). Two key challenges that limit reproduction in lactating dairy cows are a delayed return to cyclicity after calving (anovulation) and loss of the pregnancy following conception. Genetic improvement related to these specific reproductive obstacles could be an effective method for improving reproduction of dairy cows.

Anovulation is normal for cattle following parturition. The physiological basis of anovulation has been

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described by Wiltbank et al. (2002). Anovulation at the end of the voluntary waiting period causes extended calving intervals and reduced reproductive efficiency in both synchronized and nonsynchronized reproductive management systems. A survey of 18 herds found that for insemination at observed estrus, anovulation was associated with a delay of 8 d in time of first insemination and a delay of 30 d in time to pregnancy (Walsh et al., 2007). Other studies have found that 21-d pregnancy rates in cows exhibiting anovulation were decreased to 65 to 83% of the rates observed in normally cycling cows (Galvão et al., 2004; Chebel et al., 2006; Walsh et al., 2007). Anovulation was associated with decreased probability of conception (20% in one study and 6 to 10% in another), and the effect was similar whether insemination followed a detected estrus or a synchronization program (Gumen et al., 2003; Walsh et al., 2007). Anovulation also has been associated with increased pregnancy losses (Gumen et al., 2003; Galvão et al., 2004; Santos et al., 2004b; Sterry et al., 2006b). Thus, anovulation at the end of the voluntary waiting period is a trait of economic value on commercial dairy farms because of its effect on overall fertility and reproductive efficiency. Risk factors for anovulation include difficult calving, twin calving, low BCS, displaced abomasum, and subclinical ketosis (Walsh et al., 2007). Genetic correlation estimates have indicated that greater BCS were genetically associated with better reproductive performance (Pryce et al., 2000; Veerkamp et al., 2000; Dechow et al., 2001; Pryce et al., 2001; Banos et al., 2004). Previous genetic studies have indicated that anovulation at the end of the voluntary waiting period (Petersson et al., 2007) or days to onset of luteal activity (**OLA**; Darwash et al., 1997; Veerkamp et al., 2000; Royal et al., 2002a,b) had greater heritabilities (0.16 to 0.30) compared with traditional measures of reproduction. Longer time to first ovulation was found to have a positive (unfavorable) genetic correlation (0.36) with calving interval (Royal et al., 2002b). The OLA is a continuous trait that has generally been obtained by multiple measurements of progesterone in milk or from blood samples. Anovulation at the end of the voluntary waiting period is a binomial trait related to OLA but might be more practical to obtain on commercial dairies (Petersson et al., 2007). Thus, selection for less frequent anovulation at the end of the voluntary waiting period could be used to improve reproductive performance in dairy cattle.

Pregnancy loss contributes to extended calving intervals in all reproductive management systems. However, it is especially important in systems with extensive adoption of synchronization protocols because when the service rate is fixed by the reproductive management system, pregnancy rate per 21 d is determined only

by conception rate and pregnancy loss (Santos et al., 2004c). In general, pregnancy losses result from mortality of the embryo or from failure of the cow to establish and maintain a pregnancy. Pregnancy outcomes based on ultrasound beginning at 30 d after AI have a high reliability (Fricke, 2002). Early pregnancy loss, before 27 d after insemination, cannot be detected because of difficulties in examining the viability of embryos, and these losses are indistinguishable from conception failure. No previous studies have determined heritability specifically related to loss of pregnancy following accurate detection of a viable embryo.

This study involved collaboration between the disciplines of reproductive physiology and quantitative genetics. The objectives of this study were to estimate heritabilities and other genetic parameters for anovulation at ~50 DIM and pregnancy loss occurring between first and second pregnancy diagnoses after AI. In addition, we estimated sire effects for both traits and maternal grandsire (**MGS**) effects for pregnancy loss. For pregnancy loss, ancestral relationships are described in relation to the embryo. The dam is the cow observed for pregnancy, sire of embryo is the service sire for the dam, and MGS of embryo is the sire of the dam. This analysis allowed estimation of heritability for pregnancy loss from both embryonic and maternal perspectives.

MATERIALS AND METHODS

Data

Data were previously collected for studies of reproductive management between 2001 and 2006 and were provided for genetic analysis by researchers in California and Wisconsin. Most trials dealt with protocols for synchronization of ovulation or estrus. Only commercial herds were represented in the California trials, whereas data from commercial herds and one research herd were available in Wisconsin. Sire identification for all cows and embryos was included in the original research data, obtained from management files in individual herds, or recovered from the USDA Animal Improvement Programs Laboratory (**AIPL**; Beltsville, MD). To construct the relationship matrix among bulls, ancestral pedigree information for at least 3 generations was obtained from the AIPL for sires of cows (for both traits) and sires of embryos (for pregnancy loss). Inbreeding coefficients for cows in both studies were obtained from the AIPL database. To calculate inbreeding coefficients for embryos in pregnancy-loss data, up to 6 generations of available pedigree information for both parents was obtained from AIPL. Inbreeding coefficients were calculated using the Proc Inbreeding procedure in SAS (SAS Institute, 2002).

Table 1. Anovulation summary by herd

Herd	Cows (no.)	Anovulation (%)	Method	Days postpartum ¹	Reference
1	466	30.0	Progesterone	53 and 65	Silva et al., 2007a
2	1,682	18.1	Progesterone	51 and 65 41 and 56 51 and 63 44 and 58	Cerri et al., 2004 Galvão et al., 2004 Santos et al., 2004a Santos et al., 2004b
3	811	25.6	Progesterone	51 and 65 41 and 56 51 and 63	Cerri et al., 2004 Galvão et al., 2004 Juchem et al., 2008
4	232	7.3	Progesterone	37 and 51	Bruno et al., 2005 Bruno et al., 2008
5	711	17.2	Progesterone	51 and 65 44 and 58 37 and 51	Cerri et al., 2004 Santos et al., 2004a Bruno et al., 2005 Bruno et al., 2008
6	976	41.7	Ultrasound	42	Juchem et al., 2002
7	706	13.6	Progesterone	49	Chebel et al., 2006
			Ultrasound	51 and 65	Souza et al., 2007 Cunha et al., 2005
8	234	24.8	Ultrasound	53 and 65	Sterry et al., 2006b
Total	5,818	23.3			

¹Different studies within the same herd measured anovulation at different times. The statistical analysis accounted for this by including herd-study-treatment in the model.

Anovulatory status was determined at the end of the voluntary waiting period in all herds. Anovulation was determined by circulating progesterone concentrations or by visualization of a corpus luteum using transrectal ultrasonography. In most herds, low serum progesterone concentrations at both of 2 measurement times 12 to 15 d apart were used to define anovulation (Table 1). In 2 herds, a single observation was used to determine ovulatory status at 14 d after 2 treatments with prostaglandin F₂ α , a time when ovular cows should have a corpus luteum. Anovulation data consisted of 5,818 records of Holstein cows from 13 studies in 8 herds. Previous reports derived from these data are listed in Table 1. The BCS were recorded by the methods of Wildman et al. (1982) or Ferguson et al. (1994) on a scale that ranged from 1 (emaciated) to 5 (obese). The BCS were recorded variously from once shortly before first insemination to 3 times from the first week to ninth week postpartum. When a record included more than one BCS, data analysis used the score nearest (before or after) timed AI. Milk production was recorded from DHIA sample-day milk yields. Data analysis used the sample day nearest (before or after) timed AI for Wisconsin herds or the average of the first 3 monthly sample days for California herds. No adjustment was made to milk yields for cows in estrus on DHIA sample day. Average milk yield was 42.9 kg/d, and standard deviation was 9.4 kg.

Pregnancy loss was determined by an initial pregnancy diagnosis 26 to 33 d after AI followed by determination of loss of that embryo at a subsequent diagnosis 14 to 39 d later (Table 2). The median interval between

initial and subsequent pregnancy diagnoses was 28 d. Initial pregnancy diagnoses were performed using transrectal ultrasonography, and later diagnoses were by ultrasonography or palpation per rectum. Pregnancy loss data consisted of 3,775 records from 14 studies in 8 herds plus routinely recorded data in one research herd. Previous reports derived from these data are listed in Table 2. Animals for this study were Holstein cows or crossbreds (56 cows) that were daughters of Holstein bulls, 3 Jersey bulls, or 9 crossbred bulls.

Statistical Analysis

A Bayesian approach using Markov Chain Monte Carlo methods was used in threshold models for both anovulation and pregnancy loss (Sorensen et al., 1995; Sorensen and Gianola, 2002). Draws from the posterior distribution of the parameters were obtained using a Gibbs sampler (Sorensen and Gianola, 2002). For each model, 100,000 iterations were performed, and the last 50,000 iterations were used for genetic parameter estimation. Posterior means and standard deviations of parameter estimates are reported. The models assumed an underlying normally distributed liability. Bounded uniform priors were used for all fixed effects; for example, parity, and normal priors were assumed for all random effects; for example, sire. Cows with liability greater than the threshold (zero) were affected (i.e., anovular or lost pregnancy), and cows with liability less than the threshold were nonaffected. A cumulative normal density function was used to link liability with probability of anovulation or pregnancy loss (either 0

Table 2. Pregnancy loss summary by herd

Herd	Cows (no.)	Pregnancy loss (%)	Method	Pregnancy check (d post AI) ¹	Reference
1	169	11.8	Ultrasound	31 and 66	Silva et al., 2007b
2	500	15.2	Ultrasound and palpation	30 and 58 27 and 41	Cerri et al., 2004 Galvão et al., 2004 Santos et al., 2004b Santos et al., 2004a
3	278	11.2	Ultrasound and palpation	31 and 45 30 and 58 27 and 41 28 and 67	Cerri et al., 2004 Galvão et al., 2004 Juchem et al., 2008
4	168	14.9	Ultrasound and palpation	28 and 67	Bruno et al., 2005 Bruno et al., 2008
5	201	16.9	Ultrasound and palpation	30 and 58 27 and 41 28 and 67	Cerri et al., 2004 Santos et al., 2004a Bruno et al., 2005 Bruno et al., 2008
6	319	20.4	Ultrasound and palpation	27 and 41 31 and 60	Juchem et al., 2002 Chebel et al., 2006
7	674	7.6	Ultrasound	33 and 54	Brusveen et al., 2008 Souza et al., 2007 Cunha et al., 2005
8	199	21.6	Ultrasound	26–33 and 68	Sterry et al., 2006a
9	1,267	15.5	Ultrasound	32 and 64	Research herd
Total	3,775	14.4			

¹Different studies within the same herd measured pregnancy loss at different times.

or 1). The residual variance was set to 1.00. Identification of dams of cows for anovulation and identification of maternal granddams of embryos (dams of cows) for pregnancy loss were not available for many records; for this reason, analyses were done with sire models rather than animal models. In all models for both traits, random sire effects were assumed to be normally distributed as follows: $\mathbf{s} \sim N(0, \mathbf{A}\sigma_s^2)$, where \mathbf{s} = vector of sire effects, \mathbf{A} = sire additive numerator relationship matrix, and σ_s^2 = sire variance. Heritabilities (h^2) for the sire models were calculated for each Monte Carlo Markov chain iteration as

$$h^2 = \frac{4 \times \sigma_s^2}{\sigma_s^2 + \sigma_e^2},$$

where σ_s^2 and σ_e^2 are the sire and residual variances, respectively.

Probabilities (ETA) for anovulation or pregnancy loss in sires' daughter groups were estimated as follows (Heringstad et al., 2008): $\Pr[y_i = 1 \mid \mu, \bar{s}_i] \approx 1 - \Phi(\mu + \bar{s}_i)$, where y_i = binary response variable with value 0 if cow was unaffected and 1 if affected, μ = population mean liability of trait in question, \bar{s}_i = posterior mean of liability for sire i , and $\Phi(\cdot)$ = cumulative normal density function. Sire probabilities are a nonlinear transformation of sire liabilities. Sires rank in the same order on both scales, but the probability scale is more easily interpreted.

Anovulation. Analysis of anovulation used presence or absence of cyclicity as the dependent variable in a mixed, threshold sire model. The model was

$$L_{ijklm} = p_i + hst_j + bcs_k + b_1F_{ijklm} + b_2my_{ijklm} + s_l + e_{ijklm} \quad [1]$$

where L_{ijklm} = liability for cow $ijklm$, p_i = fixed effect of parity (first or later), hst_j = fixed effect of herd-study-treatment (41 classes), bcs_k = fixed effect for BCS (9 levels; 8 increments of 0.25 and unknown), b_1 = regression of liability on inbreeding, F_{ijklm} = cow inbreeding coefficient, b_2 = regression of liability on milk yield, my_{ijklm} = cow milk yield (kg/d near end of voluntary waiting period), s_l = random sire effect, and e_{ijklm} = random residual. The BCS levels and their frequencies are shown in Figure 1. Data were also analyzed using a second model that excluded BCS (model 2). The regressions of liability for anovulation on inbreeding and milk yield were not significant, and they were removed from the final models. Data were restricted to Holstein sire families with 5 or more daughters. In addition, bulls with fewer than 5 daughters were included if they were also a sire or son of a bull with 5 or more daughters. Among the 1,522 sires in the analysis, 1,360 had daughters in the data, and the rest were ancestors of these sires or MGS of the cows. Parameters for model 1 and model 2 were estimated using Binary_Sire.F90 software of Y. M. Chang that was previously applied by Heringstad et al. (2008).

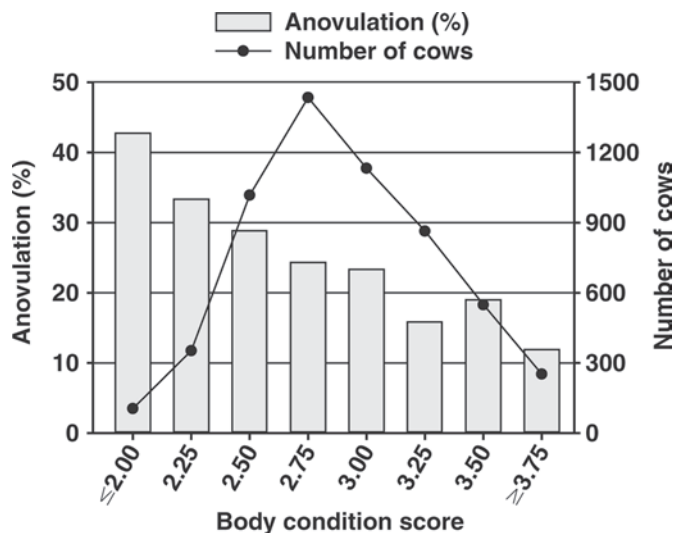


Figure 1. Observed prevalence of anovulation (bars) and number of cows (line) by category of BCS. An additional 118 records without BCS had 13.6% prevalence.

In addition, bivariate, mixed sire models were used to analyze 2 traits simultaneously [anovulation with milk yield (model 3, equation [2]) and anovulation with BCS (model 4) to estimate variances for each trait and covariances between traits. The model for milk yield was

$$L_{ijklm}, C_{ijklm} = p_i + hst_j + bcs_k + s_l + e_{ijklm}, \quad [2]$$

where C_{ijklm} is the correlated trait [milk yield (model 3) or BCS (model 4)] for cow $ijklm$, and the other terms are as described previously. Milk yield was analyzed with a linear model assuming Gaussian residuals using the software *Bivar_Binary_Linear.F90* (Heringstad et al., 2005). The bivariate analysis for milk yield was limited to 5,152 daughters of 1,412 sires in 7 herds with milk yield data. Model 4 for analysis of BCS was similar to model 3 (equation [2]), but the bcs term was not included. The residual variance for BCS was set to 1. The BCS was analyzed with an ordered categorical model with 8 categories (Figure 1) using the software *Bivar_Ordered_Binary.F90* (Heringstad et al., 2006). The bivariate analysis for BCS was limited to 5,700 records with BCS observations.

Pregnancy Loss. Analysis of pregnancy loss used a sire-MGS threshold model with embryo survival as the subject of analysis. The model was

$$L_{ijklm} = p_i + hs_j + b_1 Fe_{ijklm} + b_2 Fm_{ijklm} + s_k + mgs_l + e_{ijklm}, \quad [3]$$

where L_{ijklm} = liability for embryo $ijklm$, p_i = fixed effect for parity of dam (first or later), hs_j = fixed effect of herd-study (23 classes), b_1 = regression of liability on inbreeding of the embryo, Fe_{ijklm} = embryo inbreeding coefficient, b_2 = regression of liability on maternal inbreeding, Fm_{ijklm} = maternal inbreeding coefficient, s_k = random effect for sire of embryo, mgs_l = random effect for MGS of embryo, and e_{ijklm} = random residual. The random MGS effects were assumed to be distributed normally as $\mathbf{m} \sim N(0, \mathbf{A}\sigma_{mgs}^2)$, where \mathbf{m} = vector of MGS effects, \mathbf{A} = MGS additive numerator relationship matrix, and σ_{mgs}^2 = MGS variance; also the covariance between sire and MGS effects, $\sigma_{s,mgs}$, was estimated. Parameters for model 5 (equation [3]) were estimated using the software *Binary_Sire_MGS.F90* (Heringstad et al., 2007). A total of 1,881 bulls were included in the analysis: 1,286 were sires of cows; 650 were sires of embryos, including 122 bulls that were both sires of cows and sires of embryos; and the remainder were ancestral sires.

Direct (embryo) and maternal (dam of embryo) effects and their covariance were derived from the sire and MGS variances and their covariance (Wiggans et al., 2003; Heringstad et al., 2007). Heritabilities for direct (h_D^2) and maternal (h_M^2) effects were calculated for each iteration of the sire-MGS model as

$$h_D^2 = \frac{\sigma_D^2}{\sigma_P^2},$$

$$\text{and } h_M^2 = \frac{\sigma_M^2}{\sigma_P^2},$$

where σ_D^2 is the direct variance, σ_M^2 is the maternal variance, and σ_P^2 is the phenotypic variance. The phenotypic variance was calculated as $\sigma_P^2 = \sigma_s^2 + 2\sigma_{s,mgs} + \sigma_{mgs}^2 + \sigma_e^2$ (Heringstad et al., 2007). For an individual bull, direct effect on the liability scale was equal to that bull's sire effect and maternal effect was MGS effect $- 0.5 \times$ that same bull's sire effect. These relationships derive from the fact that sire (of embryo) influences only the embryo, whereas MGS (of embryo, also sire of dam) influences both the cow and the embryo. The MGS influence on the embryo, in contrast to the sire influence, is one generation removed, thus the coefficient 0.5 on the MGS variance.

In addition to the sire-MGS model, separate sire models were used to analyze the data with embryo as the subject of analysis (model 6, equation [4]) and cow

(i.e., dam of embryo) as the subject of analysis (model 7, equation [4]). The models were

$$L_{ijkl} = p_i + hs_j + b_1 F_{ijkl} + s_k + e_{ijkl}, \quad [4]$$

in which b_1 is the regression of liability on inbreeding coefficient, F_{ijkl} is the inbreeding coefficient of the subject individual, s_k is the sire of the subject individual, and the other terms are as described previously. Parameters for model 6 and model 7 (equation [4]) were estimated using the Binary_Sire.F90 (Heringstad et al., 2008).

RESULTS AND DISCUSSION

Anovulation

The overall prevalence of anovulation in the data for this study was 23.3%. Observed anovulation percentages within herds ranged from 7.3 to 41.7 (Table 1). These results correspond closely to a survey of 18 herds with 1,341 cows in Ontario in which overall prevalence was 19.5% and within-herd prevalences ranged from 5 to 45% (Walsh et al., 2007). Prevalence of anovulation at the end of the voluntary waiting period ranged from 18 to 34% in 5 previous studies (Gumen et al., 2003; Cerri et al., 2004; Galvão et al., 2004; Chebel et al., 2006; Walsh et al., 2007). Observed prevalences of anovulation in the current study were 29.6% for 2,318 primiparous cows and 19.1% for 3,500 multiparous cows. Previous reports showed that anovulation in primiparous cows was 1.6 to 1.9 times that of multiparous cows (Gumen and Wiltbank, 2002; Cerri et al., 2004; Santos et al., 2004b; Chebel et al., 2006; Silva et al., 2007a).

Heritability, sire variance, and covariance estimates for the 4 anovulation models are shown in Table 3. The heritability and sire variance estimates were similar among all models; values from the univariate analysis (model 1, equation [1]) that included BCS were intermediate. The frequency distribution of sire evaluations, including ancestral bulls, for daughter anovulation from the sire model with BCS (model 1) is in Figure 2. The sire predictions ranged from 15 to 31%, with a median of 23%. The ETA from models 1, 2, and 4 for 12 bulls with 50 or more daughters are shown in Table 4. For all 3 models, sires 10 and 12 had the smallest and sire 9 the largest probability of daughter anovulation (Table 4). In general, the 3 models provided similar predictions of daughter prevalence of anovulation for the 12 bulls. Genetic correlation of anovulation with daily milk yield obtained from the bivariate analysis (model 3, equation [2]) was 0.168; however, the posterior standard deviation (PSD) was large (0.187). The residual correlation was -0.046 (PSD = 0.022), and the phenotypic correlation was -0.036. Phenotypically, anovulation was

nearly independent of milk yield, but genetically, there was a tendency for high milk yield to be associated with greater anovulation rates.

Only one previous study has estimated heritability for delayed ovulation, e.g., beyond 45 or 50 d, recorded as a binary variable. Petersson et al. (2007) defined an occurrence of anovulation when thrice-weekly milk progesterone tests remained continuously below 3 ng/mL for more than 45 d postpartum. The definition of anovulation by Petersson et al. (2007) was similar to our study, the main differences being in the time (45 d vs. 50 to 60 d) and method of determination. Heritability for delayed ovulation estimated by Petersson et al. (2007) was 0.203 (SE = 0.054) based on 1,212 records; their result was similar to our estimate of 0.171 (PSD = 0.052), which was based on 5,818 records. Frequent milk progesterone testing, as in the Petersson study, is costly and not practical for management in commercial herds. A typical reproductive management protocol in commercial herds can include routine palpation per rectum or transrectal ultrasound to observe recovery from parturition, ovarian structures, and pregnancy diagnosis. These methods could enable detection of anovulation without progesterone testing. Thus, although most of the data used in our study were obtained by one-time analysis of progesterone, it seems likely that anovulation based on routine commercial methods might provide similar data.

Three studies of postpartum interval to OLA have been reported (Darwash et al., 1997; Veerkamp et al., 2000; Royal et al., 2002a). The OLA was determined variously from daily or twice or thrice weekly milk progesterone tests from 7 to 60 d or more than 100 d postpartum. The OLA was recorded as days from calving to the first (or first of 2 successive) milk progesterone tests >3 ng/mL. Average OLA intervals (\pm SD) were 26.5 (\pm 11.9), 29.5 (\pm 16.8), and 25 d (geometric mean) based on 1,737, 420, and 1,212 records, respectively (Darwash et al., 1997; Veerkamp et al., 2000; Royal et al., 2002a). Accounting for the time from ovulation to the rise of milk progesterone and the interval between successive progesterone tests, ovulation was estimated to occur around 5 to 6 d before OLA (Darwash et al., 1997).

The original genetic study of OLA involved 1,737 records of 1,137 cows from 147 paternal half-sib groups in 12 herds. Heritability estimates were 0.28 for OLA and 0.21 (95% confidence interval = 0.05–0.33) for logarithm of OLA (Darwash et al., 1997). A study of 420 first-lactation cows in an experimental herd found heritability for OLA was 0.16 ± 0.10 (Veerkamp et al., 2000). In data from 1,212 lactations of 1,080 cows that were daughters of 169 sires in 8 herds, the heritability estimate for log OLA was 0.16 ± 0.05 (Royal et al.,

Table 3. Genetic parameter estimates for anovulation from 4 statistical models¹

Model	Anovulation			Correlated trait			Covariance ²		
	h^2	σ^2 Sire	σ^2 Residual	h^2	σ^2 Sire	σ^2 Residual	Sire	Residual	
1: Univariate with BCS	0.171 (0.052)	0.045 (0.014)	1						
2: Univariate without BCS	0.182 (0.057)	0.055 (0.019)	1						
3: Bivariate anovulation and milk yield	0.192 (0.063)	0.051 (0.018)	1	0.171 (0.040)	2.08 (0.50)	46.56 (0.97)	0.055 (0.064)	-0.310 (0.149)	
4: Bivariate anovulation and BCS	0.160 (0.048)	0.042 (0.013)	1	0.340 (0.062)	0.093 (0.019)	1	-0.019 (0.012)	-0.186 (0.019)	

¹Posterior SD for the estimates are in parentheses.²Covariance of anovulation with the correlated trait.

2002a). The estimates for these 3 studies were pooled, resulting in a heritability of 0.18 ± 0.03 based on 3,282 records (Royal et al., 2002a). Heritability estimates for anovulation and OLA in all of these studies, including our study, were remarkably similar. In addition, they were greater than estimates based on traits such as days to first insemination, days open, or calving interval, for which heritability estimates were typically less than 0.10 and often less than 0.05 (Hayes et al., 1992; Dechow et al., 2001; Pryce et al., 2001; VanRaden et al., 2004; Goodling et al., 2005). Genetic correlation of log OLA with calving interval was 0.36, indicating that sires whose daughters have delayed OLA tend to have extended calving intervals (Royal et al., 2002b). However, other factors also contribute to variation in calving interval.

Time to first ovulation has been related to timing of the postpartum negative energy balance nadir and return to positive energy balance (Staples et al., 1990; Beam and Butler, 1999; Butler, 2000; Wathes et al., 2007). The BCS is widely used in dairy herds to evaluate energy balance of cows. In the current data, observed prevalences of anovulation by categories of BCS are shown in Figure 1. These trends correspond closely with other studies (Gumen et al., 2003; Santos et al., 2004b; Silva et al., 2007a). Previous phenotypic studies have shown strongly greater prevalences of anovulation associated with lower BCS (Cerri et al., 2004; Galvão et al., 2004; Chebel et al., 2006; Walsh et al., 2007). In our univariate analysis of anovulation (model 1), BCS had a significant, albeit small, effect on anovulation. Heritability of BCS in our data (model 4) was 0.340 (PSD = 0.062). Heritabilities for BCS in previous studies have ranged from 0.16 ± 0.04 to 0.35 ± 0.05 and averaged 0.24 (Pryce et al., 2000; Dechow et al., 2001; Pryce et al., 2001; Banos et al., 2004). In the current study, bivariate analysis of BCS with anovulation (model 4) found a genetic correlation -0.301 (PSD = 0.177), residual correlation -0.186 (PSD = 0.019), and phenotypic correlation -0.192 (PSD = 0.019). The modest phenotypic correlation between anovulation and BCS might seem to be inconsistent with the strong stepwise decreases in anovulation as BCS increased (Figure 1). However, anovulation at the lowest level of BCS was 42.7%, far less than 100%, indicating only a moderate association. Further analysis of our results indicated that BCS was not a good diagnostic for anovulation. For example, the sum of specificity and sensitivity can provide an indication of the accuracy of a diagnostic test (200% = perfect test; 100% = random chance). Selection of the optimal threshold from our data (BCS ≤ 2.75) produced only 60% specificity (probability of correctly diagnosing anovular cows) and 51.8% sensitivity (probability of correctly diagnosing cycling cows)

Table 4. Sire ETA for daughter anovulation and BCS for bulls with 50 or more daughters

Sire	Daughters (no.)	Granddaughters (no.)	ETA 1 ¹ (%)	ETA 2 ² (%)	Bivariate model ETA (model 4)	
					Anovulation (%)	BCS (points)
1	153	70	20.8	18.9	23.1	0.113
2	96	0	22.1	22.8	23.9	0.009
3	82	0	19.9	19.9	21.0	0.101
4	73	0	25.7	24.4	25.5	-0.050
5	71	2	26.3	26.1	26.4	0.144
6	71	0	24.1	23.6	24.5	-0.123
7	68	1	21.2	22.7	21.5	-0.019
8	64	111	25.3	25.9	23.2	0.230
9	59	0	28.5	31.0	28.0	0.003
10	53	0	17.7	16.5	19.3	0.188
11	52	0	18.9	19.2	20.2	-0.069
12	50	28	15.3	16.8	16.8	0.025

¹ETA 1 = Univariate sire model with BCS (model 1).

²ETA 2 = Univariate sire model without BCS (model 2).

for a sum of less than 112%. Thus, although cows with low BCS were at greater risk for anovulation, it is clear that BCS alone does not completely explain anovulation in individual cows. Another indication of the weak genetic association between anovulation and BCS is provided in Table 4: Including BCS (ETA 1) or not including BCS (ETA 2) in the statistical model did not substantially change bulls' ETA for daughter anovulation. In addition, among the bulls in Table 4, substantial differences were observed in rank of ETA for BCS compared with ETA for anovulation. This result is consistent with the moderate genetic correlation estimate between these traits.

Genetic correlation estimates of BCS with reproduction in previous studies span a wide range because of sampling errors from modest numbers of records and varying reproductive measures, among other factors. One report estimated genetic correlation of OLA with energy balance derived from feed intakes during the first 15 wk of lactation. Genetic correlation estimates were -0.60 without adjustment for milk yield and -0.49 after adjustment, indicating that positive energy balance was genetically associated with earlier OLA (Veerkamp et al., 2000). These estimates were based on only 420 cows, so standard errors were large. In a data set of 1,023 lactations with 124 sire families, genetic correlation of BCS with log OLA was -0.84, indicating that cows with low BCS were more likely to exhibit delayed OLA (Royal et al., 2002b). Using 19,042 records, genetic correlation was estimated for calving interval with BCS, which was recorded one time during lactation at the time of type classification. Estimates were -0.40 ± 0.09 or -0.48 ± 0.01 by different statistical models without considering yield and -0.22 ± 0.11 with yield adjustment in both models (Pryce et al., 2000; Pryce et al., 2002). Genetic correlations of calving interval

with BCS by month of lactation decreased as lactation advanced, ranging from -0.88 in mo 1 to -0.60 in mo 3 and below -0.050 after mo 4 (Pryce et al., 2000). A study of data sets that ranged from 5,190 to 10,728 records found little difference between genetic correlations with or without adjustment for yield (Dechow et al., 2001). Genetic correlations with BCS in early lactation averaged -0.61 for days to first insemination and -0.16 for number of services per conception (Dechow et al., 2001). The largest study of genetic correlation for BCS with calving interval was based on 150,301 records, and the largest study of genetic correlation for BCS with nonreturn at 56 d was based on 190,407 records (Banos et al., 2004). Genetic correlations of BCS with calving interval ranged from -0.31 to -0.37 ± 0.07 and with nonreturn ranged from -0.10 to -0.32 ± 0.10 . These studies indicate that a moderate portion but not

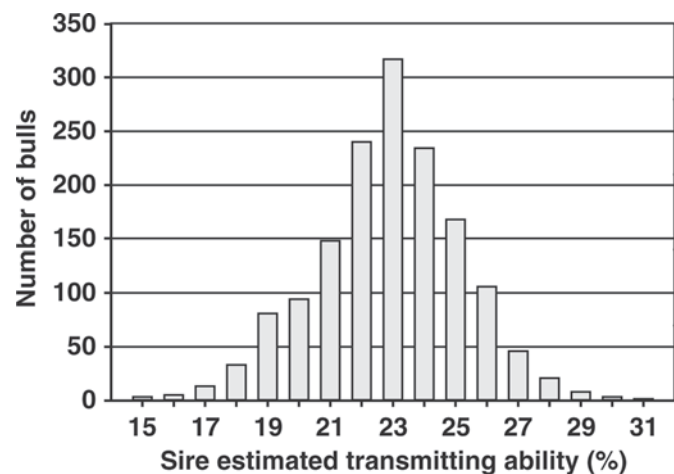


Figure 2. Frequency distribution of sire evaluations for daughter anovulation from the univariate model with BCS (model 1, equation [1]).

all of the genetic variation in reproductive performance is associated with BCS and that high production associated with inadequate energy intake are common causes of both reduced BCS and impaired reproductive performance.

The moderately high heritability of BCS and its genetic correlation with some reproductive traits indicate a potential usefulness of BCS as an adjunct for genetic improvement of reproduction. Results from the current study indicate that BCS has only modest to moderate phenotypic and genetic correlations with anovulation. Our results further indicate that anovulation has a moderate heritability that could be useful in direct selection to improve reproduction in dairy cattle. For individual selection, assuming equal selection intensities and generation intervals and using even optimistic assumptions for heritability of BCS (0.36) and genetic correlation of BCS with anovulation (-0.50 , our estimate was -0.30), direct selection against anovulation (heritability = 0.17) would produce nearly 40% [$0.17^{0.5} / (0.50 \times 0.36^{0.5}) = 1.37$] greater selection response than indirect selection through BCS. For progeny selection, the advantage of direct selection over indirect selection is even greater. As the number of progeny becomes large, accuracy of selection approaches 1.0 and response of direct selection relative to indirect selection approaches the reciprocal of genetic correlation between the traits. Again assuming an optimistic genetic correlation of BCS with anovulation (-0.50), the response of direct selection against anovulation would approach twice that of indirect selection for BCS as the number of progeny becomes large. Clearly, indirect selection on BCS would be much less effective than direct selection against anovulation. A combination of direct and indirect selection would be most effective, especially for individual selection or when progeny numbers are small.

Pregnancy Loss

Average prevalence of pregnancy loss in this analysis was 14.4%. Prevalence of pregnancy loss within herds ranged from 7.6 to 21.6% (Table 2). Among 7 previous studies, prevalence of pregnancy loss ranged from 6 to 20% (Cerri et al., 2004; Galvão et al., 2004; Santos et al., 2004b; Starbuck et al., 2004; Chebel et al., 2006; Sterry et al., 2006a; Brusveen et al., 2008). In a survey of 14 published trials, pregnancy loss averaged 12.8% of 4,780 pregnancies (Santos et al., 2004c). Losses after 60 d are less prevalent; Vasconcelos et al. (1997) reported 17% pregnancy loss from 28 to 56 d and 2% from 56 to 98 d. In addition, Santos et al. (2004c) concluded that fetal losses (>42 d) are less prevalent than embryonic losses.

Parity, embryo inbreeding, and maternal inbreeding effects were not significant and were not included in the analyses reported here. Observed prevalence was 13.0% among 1,637 primiparous cows and 15.0% among 2,138 multiparous cows in our data. By chi-square test, pregnancy losses did not differ between parities ($P > 0.10$). In previous studies, parity has not been a significant factor in pregnancy losses (Cerri et al., 2004; Chebel et al., 2004; Galvão et al., 2004; Starbuck et al., 2004; Sterry et al., 2006a,b; Brusveen et al., 2008).

Sire-MGS Model 5. The sire variance for pregnancy loss was 0.149 (PSD = 0.063) and the MGS variance was 0.0112 (PSD = 0.0069). The covariance between sire and MGS effects was -0.0097 (PSD = 0.0275), resulting in a correlation between sire and MGS effects of -0.237 . Pregnancy loss ETA for sires of embryos, MGS of embryos, and maternal effects are in Table 5 for bulls with 50 or more matings (embryos); as shown in the table, these bulls had few or no daughters. The ETA for sire of cow, MGS of embryo, and maternal effects are shown in Table 6 for bulls with 30 or more daughters; as shown in the table, these bulls had few or no matings. As expected, the ETA for MGS varied more for bulls with large numbers of daughters (Table 6) than for bulls with few daughters but large numbers of matings (Table 5). For bulls with more than 30 daughters (Table 6), the MGS ETA were estimated directly from daughter performance and relationships with other bulls. Conversely, for bulls with few or no daughters but with more than 50 matings (Table 5), the MGS ETA were estimated indirectly via the sire of embryo effects and the correlation between sire and MGS effects; consequently, MGS effects for these bulls were regressed more strongly toward the population average than was true for bulls with larger numbers of daughters. The ETA frequency distribution for sires of embryos for all bulls is shown in Figure 3, and ETA ranged from 4 to 31%.

From the standpoint of observation and analysis, pregnancy loss is evaluated in terms of sire and MGS effects. However, the underlying biological causes of pregnancy loss are factors associated with mortality of the embryo and factors associated with the ability of the cow to maintain an established pregnancy. For an individual record, it cannot be determined whether pregnancy loss is the result of embryo factors or maternal factors. Nevertheless, the relative importance of embryo and maternal factors can be partitioned on a population basis. Sire of embryo can only influence embryo survival and accounts for 1/4 of the additive genetic variance in embryo survival. Sire of cow contributes to both embryo and maternal factors accounting for 1/16 of the additive genetic variance in embryo survival and 1/4 of additive genetic variance in maternal

Table 5. Sire, maternal grandsire (MGS), and maternal ETA for pregnancy loss for sire-MGS model 5 (equation [3]) and sire of embryo model 6 (equation [4]) for bulls with more than 50 matings

Sire	Matings	Daughters	Sire-MGS model			Sire of embryo model
			Sire ETA	MGS ETA	Maternal ETA	Sire ETA
13	98	0	16.1	14.3	13.5	16.6
14	88	0	10.3	14.6	17.8	9.7
15	87	0	15.5	14.2	13.7	15.4
16	86	1	14.5	14.3	14.2	14.0
17	85	0	11.3	14.5	16.1	11.4
18	82	0	18.5	14.2	12.3	18.7
19	69	1	13.8	14.3	14.6	14.7
20	54	0	6.8	14.8	20.3	6.2
21	51	12	17.5	14.6	13.3	17.4

maintenance of pregnancy. Accordingly, variances and covariances for embryo and maternal effects can be derived from the sire and MGS variances and covariances (Luo et al., 1999; Wiggans et al., 2003). The variance of breeding values for embryo effects was 0.558 (PSD = 0.252), and the variance of breeding values for maternal effects was 0.189 (PSD = 0.129). The phenotypic variance was 1.1408. Heritability for embryo (direct) effect was 0.489 (PSD = 0.221), and the heritability for maternal (dam of embryo) effect was 0.166 (PSD = 0.113). The covariance between embryo and maternal effects was -0.292 (PSD = 0.173), and the corresponding correlation was -0.868 (PSD = 0.190) with a wide 95% confidence interval (-0.985 to -0.283). The strong negative correlation between embryo and maternal effects is illustrated by the inverse rankings of sire ETA and maternal ETA in Tables 5 and 6. Because only 122 bulls out of 1,881 in the analysis were sires of both embryos and cows, the data structure was poorly suited to estimating this correlation. Altogether, these 122 bulls were sires of 1,015 embryos and 721 cows. Any over- (or under-) estimation of the direct effect necessarily led to under- (or over-) estimation of the maternal effects for individual bulls. The fact that so few bulls were sires of both cows and embryos was due to the relatively

short time during which these data were collected. In future studies of pregnancy loss, a longer period of data collection would be advised to allow a larger percentage of bulls to appear in the data as both sires of embryos and sires of cows.

In this study, the breeding value variance for embryo effects was 3 times the breeding value variance for maternal effects, indicating that, at the level of breeding values, the embryo's ability to survive had a greater impact on pregnancy loss than did the cow's ability to maintain pregnancy or other maternal effects. This result suggests that selection of sires to improve embryo survival would have a greater effect on pregnancy loss than would selection of sires to improve maternal effects. Also, it indicates that the opportunity to reduce pregnancy loss by selection for sire of cow, i.e., MGS of embryo, effects would be limited. However, PSD for these variance and covariance estimates were large. The correlation between embryo and maternal effects was large and negative, indicating that sires and dams that have favorable breeding values for embryo survival tend to have unfavorable breeding values for maternal effects. If, in fact, this correlation is as strong as found here, selection to improve embryo survival alone would be accompanied by deterioration in maternal effects.

Table 6. Sire, maternal grandsire (MGS), and maternal ETA for pregnancy loss for sire-MGS model 5 (equation [3]) and sire of cow model 7 (equation [4]) for bulls with more than 30 daughters

Sire	Daughters (no.)	Matings (no.)	Sire-MGS model			Sire of cow model
			Sire ETA ¹	MGS ETA	Maternal ETA	Sire ETA
22	77	0	15.5	12.8	12.3	10.8
23	49	8	10.1	16.2	18.5	19.1
24	48	19	24.2	14.0	10.5	14.1
5	46	0	8.6	13.7	17.1	11.6
1	43	0	15.5	14.6	13.9	14.2
25	43	12	8.2	15.5	19.9	16.6
26	36	0	7.8	14.5	18.8	13.6
27	32	0	14.7	14.1	14.1	13.9
28	31	0	14.6	14.6	14.8	15.1

¹Sire ETA in the sire of cow model is comparable to MGS ETA in the sire-MGS model.

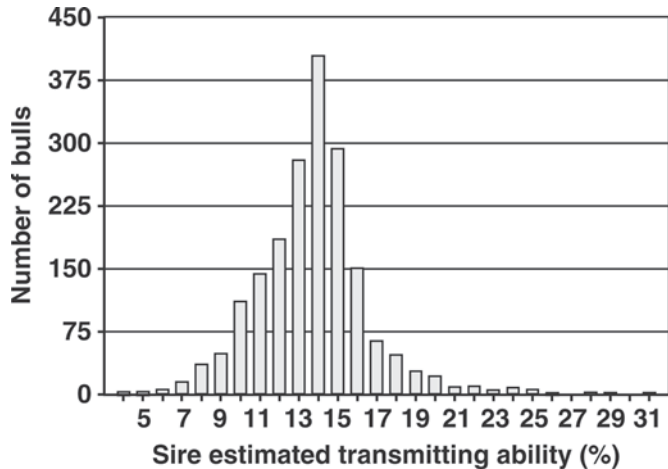


Figure 3. Frequency distribution for sire of embryo evaluations for pregnancy loss based on the sire-MGS model (model 5, equation [3]).

Because additive genetic variance for embryo effects is larger and sire of embryo effects are observed a generation earlier, selection to reduce pregnancy loss perhaps should emphasize embryo effects over maternal effects. However, maternal effects must not be disregarded because they are expressed at each pregnancy (embryo effects are expressed only once) and because genetic covariance between embryo and maternal effects is negative. Optimal improvement for pregnancy loss could be attained by a selection index that accounts for these genetic parameters and economic factors.

Sire of Embryo and Sire of Cow Models. The variance for sire of embryo (model 6, equation [4]) was 0.192 (PSD = 0.056), and the variance for sire of cow (model 7, equation [4]) was 0.0411 (PSD = 0.0142). Heritability for sire of embryo was 0.638 (PSD = 0.158),

whereas heritability for sire of cow was 0.157 (PSD = 0.052). The ETA for sires of embryos are in Table 5 for bulls with more than 50 matings, and ETA for sires of cows are in Table 6 for bulls with more than 30 daughters. The frequency distributions of ETA for sire of cow and sire of embryo are in Figures 4 and 5 for all bulls. These variance estimates and frequency distributions confirm the finding of the sire-MGS model that sire of embryo effects are substantially greater than sire of cow effects.

A concern about the sire-MGS model 5 (equation [3]) in this study was validity of the results because of the large number of parameters estimated from a comparatively small data set. The simpler sire of embryo (model 6) and sire of cow (model 7) models were used to assess validity of the sire-MGS results. In Table 5 are ETA for both the sire-MGS and sire of embryo models for bulls with more than 50 matings. The sire ETA are in close agreement between the 2 models. In Table 6 are ETA for sire-MGS and sire of cow models for bulls with more than 30 daughters. Comparing MGS ETA in the sire-MGS model with the sire ETA in the sire of cow model shows differences ranging from 0.1 to 2.9%. However, rankings of the bulls' ETA for the 2 models from high to low correspond closely (Table 6, rankings not shown); only 2 of the 9 bulls differ between the 2 rankings. The variance estimate for sire in the sire-MGS model was smaller than in the sire of embryo model. The variance estimate for MGS in the sire-MGS model was much smaller than the sire variance in the sire of cow model. The models were not expected to produce identical results because the 2 sire models do not account for nonrandom mating or for the covariance between sire and MGS effects. We conclude that, except for the sire-MGS covariance, results from

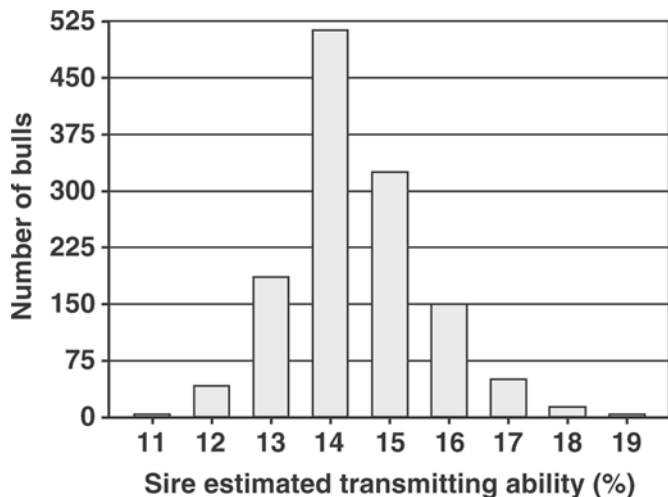


Figure 4. Frequency distribution of sire evaluations for pregnancy loss based on the sire of cow model (model 7, equation [4]).

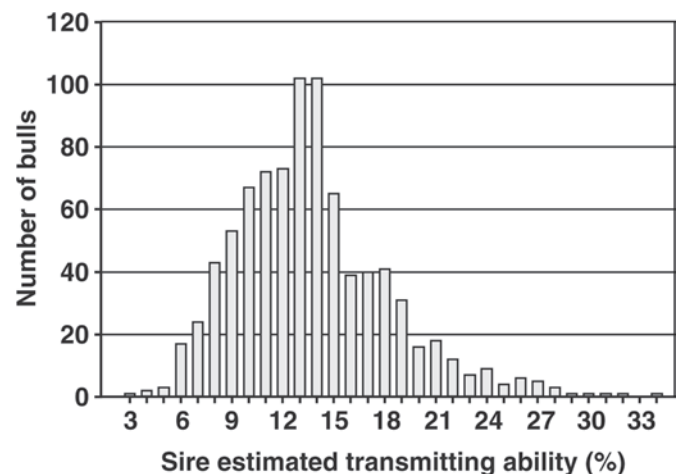


Figure 5. Frequency distribution of sire evaluations for pregnancy loss based on the sire of embryo model (model 6, equation [4]).

the sire-MGS model are credible but not exceedingly precise given the limited number of records.

The heritability estimates for embryo effects seem unrealistically large for both sire-MGS and sire of embryo models. These large estimates might be caused by the few bulls with extremely large ETA for embryo effects (Figures 3 and 5). We examined the data for an explanation of these results, but no cause was apparent. In particular, connections between sires and herds were many and appeared adequate for simultaneous estimation of sire and herd effects.

Errors in the initial diagnosis of pregnancy would create an upward bias in prevalence of pregnancy loss. For pregnancy diagnosis at 27 d after first AI, sensitivity (probability of correctly diagnosing a pregnant cow) was 96.8% and specificity (probability of correctly diagnosing a nonpregnant cow) was 91.7% (Silva et al., 2007b). Diagnoses at later stages are even more accurate than at 27 d (Fricke, 2002). Using these data from 27-d pregnancy diagnosis indicates that 8.3% of nonpregnant cows would be misdiagnosed as pregnant and most likely would be found nonpregnant at a 60-d diagnosis; these would be classified in the data as lost pregnancies. In contrast, about 3.2% of pregnant cows would be misdiagnosed as nonpregnant at 27 d, but they would not appear in the data because pregnancy loss required an initial diagnosis of pregnancy. The latter type of misdiagnosis would have no apparent effect on the results. The former would result in an inflated prevalence of pregnancy loss. Errors in pregnancy diagnosis are likely independent of sires and MGS, and they would appear to have little effect on genetic parameter estimates.

No previous studies have directly estimated genetic parameters for pregnancy loss. A large study of genetic parameters for 70-d nonreturn rate involved 1,739,055 records from DHIA sources (VanRaden and Miller, 2006). The data involved 1,251 bulls with ≥ 100 daughters and ≥ 500 first services as mating bulls. Their study did not have access to confirmed cases of pregnancy and the subsequent loss of pregnancy. Instead, the 70-d nonreturn rate measured the combined effects of fertility and pregnancy loss. They estimated heritability for the MGS effects to be 0.01, and heritability for the sire of embryo effect was not estimable. The current study was limited to additive genetic effects because of a relatively small number of records, whereas VanRaden and Miller (2006) estimated, and found significant, dominance effects and sire by MGS interactions.

Among genetic factors, pregnancy loss can be caused by homozygous lethal recessive genotypes. Inbreeding increases homozygosity, and some early losses might be caused by homozygous recessive loci. Therefore, inbreeding would be expected to increase the probability

of embryonic death. VanRaden and Miller (2006) found that 70-d nonreturn percentage decreased by 1% for each 10% increase in embryo inbreeding. Neither effects for cow inbreeding (which ranged from 0.5 to 29%) nor embryo inbreeding (which ranged from 0.21 to 17.3%) were significant in the current study. The lack of significance for inbreeding in the current study might have resulted from the relatively small number of observations. One known example of a recessive lethal is deficiency of uridine monophosphate synthase, which causes embryonic death at about 40 d after insemination (Shanks et al., 1992). This allele has been nearly eliminated from Holstein AI bulls by removing carriers of the defective allele, so it is an unlikely cause of pregnancy loss in our data. Recently, a homozygous recessive genotype in the STAT5A gene has been discovered that increases mortality at around 5 to 7 d after insemination (Khatib et al., 2008). Mortality due to STAT5A precedes initial pregnancy diagnosis, so this gene could not contribute to pregnancy loss in our study. It is likely that there are other unidentified lethal recessives that influence embryonic survival. Data similar to this study, but on a much larger scale and with the addition of DNA analysis, could identify markers for lethal recessives or other embryonic and maternal genetic mechanisms for pregnancy loss.

Pregnancy loss can occur through many mechanisms. Among aborted fetuses submitted to a veterinary laboratory, 56% were attributable to an infectious agent and the remainder had undetermined causes (Santos et al., 2004c). Infection by various bacteria such as *Brucella* spp., *Leptospira* spp., and *Salmonella* spp.; protozoa such as *Neospora caninum*; and viruses such as bovine viral diarrhoea virus and infectious bovine rhinotracheitis virus can cause abortion (Murray, 2006). The extent of infections as a cause of pregnancy loss between 30 and 60 d of gestation is unknown. Risk factors for pregnancy loss include mastitis, uterine health, and progesterone concentration. Cows experiencing mastitis during the first 45 d of lactation were more likely to lose pregnancy (22.6 vs. 12.3%) between 31 and 45 d after AI (Chebel et al., 2004). Poor uterine health, indicated by retained placenta, pyometra, and even subclinical endometritis for example, was detrimental to embryo survival (Santos et al., 2004c). Although embryonic mortality occurred before luteal regression, pregnancy loss from the fifth to ninth week was greater for cows in the lowest quartile for progesterone concentration at wk 5 (Starbuck et al., 2004).

Previous research has found no significant effect on pregnancy loss caused by insemination number (Chebel et al., 2004; Starbuck et al., 2004; Sterry et al., 2006a; Brusveen et al., 2008) or insemination protocol (estrus detection vs. timed AI; Cerri et al., 2004; Chebel et

al., 2004; Santos et al., 2004b,c; Starbuck et al., 2004; Sterry et al., 2006a,b; Brusveen et al., 2008). In addition, no effect was found for milk production, parity, or DIM at the time of insemination (Chebel et al., 2004; Starbuck et al., 2004). In individual experiments, no effect was observed for heat stress (Chebel et al., 2004), follicle size, or size of corpus luteum (Starbuck et al., 2004). The BCS was significant in one study (Starbuck et al., 2004) but not in another (Sterry et al., 2006a).

Most research has shown a negative effect of anovulation on pregnancy loss. Pregnancy loss in anovular cows was nearly twice that in cycling cows (Galvão et al., 2004; Santos et al., 2004b,c; Sterry et al., 2006b; Stevenson et al., 2006). By contrast, Chebel et al. (2006) found no effect of anovulation on pregnancy loss. The current study included 1,752 cows for which we had both pregnancy loss and anovulation records. Of the 301 cows that were anovular, 17.9% lost a pregnancy, whereas 1,451 records were of cycling cows with a pregnancy loss of 13.0%. A chi-square test of independence was significant ($P = 0.025$), indicating a modestly greater frequency of pregnancy loss in anovular compared with cycling cows. These data were too few to warrant estimation of genetic correlation between anovulation and pregnancy loss. No previous studies have estimated this correlation. This area needs further examination.

Several large studies of reproductive management had pregnancy loss and anovulation data available for analysis in this study; however, they could not be utilized because of a lack of DHI-compatible cow identification in the databases. Future interdisciplinary collaborations between reproductive physiologists and geneticists would be facilitated by recording cow registration or eartag numbers when large-scale reproductive studies are conducted in DHIA herds.

CONCLUSIONS

The objectives of this study were to estimate genetic parameters for anovulation at ~50 DIM and loss of confirmed pregnancies between first and second pregnancy diagnoses after insemination. The heritability of anovulation was 0.171. Genetic correlation estimates were 0.168 for anovulation with milk yield and -0.301 for anovulation with BCS. Accordingly, selection against delayed onset of ovulation can be accomplished much more effectively by direct selection on anovulation than by indirect selection on yield or BCS. The heritability of embryo effects for pregnancy loss was 0.489 and for maternal effects was 0.166. Breeding value variances were 0.558 for embryo effects and 0.189 for maternal effects. In regard to the underlying genetic components of pregnancy loss, embryo effects appear to have a greater effect than maternal effects. Genetic reduction

in pregnancy loss would best be accomplished by an index that includes both direct and maternal effects. Both of these traits exhibited greater heritabilities than are typical of traditional measures based on components of the calving interval or nonreturn rate. These data were recorded by skilled technicians in controlled experiments. Heritabilities for these traits would likely be less in data collected under field conditions. These results suggest that genetic improvement of reproductive performance could be enhanced by selection against extended periods of postpartum anovulation and pregnancy loss. Larger studies of these traits are needed to obtain parameter estimates with greater precision.

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