Stillbirths and Dystocia at First and Second Parities in Swedish Holstein Cattle

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Introduction

Stillbirths in Swedish Holstein (SLB) heifers have increased in the last 10-15 years. At first calvings around 9 % of the calves are born dead or die within the first day. This figure is almost twice as high as stillbirths of Swedish Red and White heifer calvings with 4.6 % (fig. 1). At later parities no breed differences in stillbirth rate seem to exist. In the Holstein population a variation in stillbirth rate among bulls as sires as well as maternal grandsires from 2 to 25% has been noted in large progeny groups (Philipsson et al. 1997). In a previous study it was shown that the problems of stillbirth appear to be somewhat different now compared with earlier experiences. The stillbirths do not seem to be as strongly related to high birth weights and difficult calvings as earlier (Steinbock, 1997). In SLB heifers more than half of the stillborn calves were now born at normal or easy calvings.

Without doubt there is a large genetic variation in stillbirths at first calvings. The issues are to what extent there is a genetic variation in stillbirth rate independent of dystocia and whether any genetic variation of importance exists at later parities. The objectives of this study were therefore to estimate genetic parameters for the direct and maternal effects in both stillbirth rate and dystocia at first and second parities separately, and also to analyse the influence on the estimates for stillbirth when the effects of dystocia were included in the model. Such information is basic for both correct definitions of traits to be utilized for use and selection of bulls, and for optimum designs of breeding programs aiming at minimizing the problems of dystocia and stillbirths, domestically and internationally.

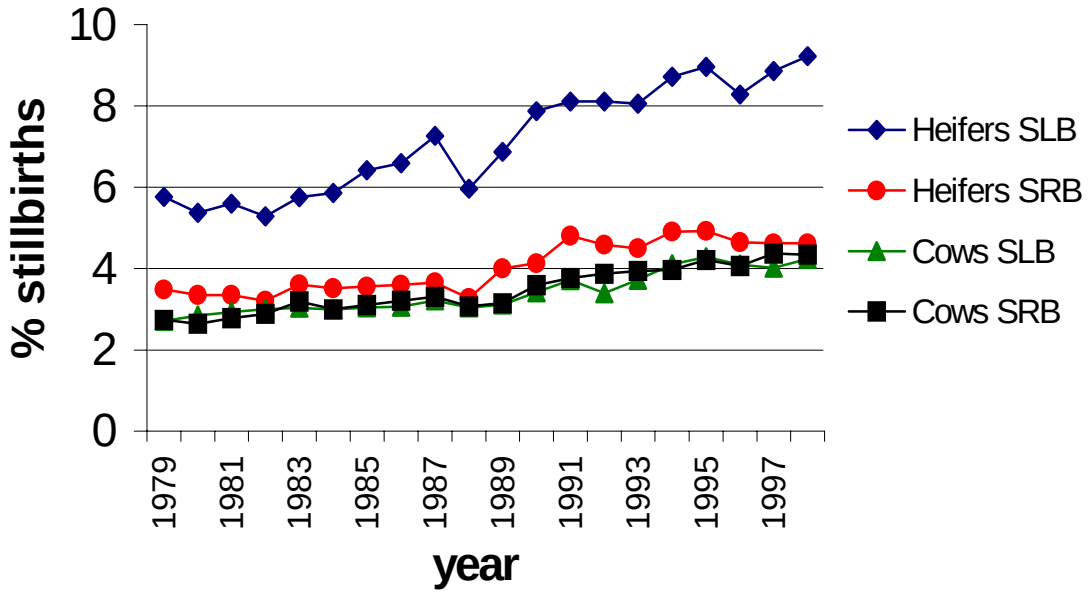

Figure 1. Stillbirth rates 1979-1998. Breed averages for Swedish Holstein, (SLB) and Swedish Red and White breed (SRB). Annual statistics from the Swedish Dairy Association.

Materials and Methods

Records were available from the Swedish Milk Recording Scheme on calves born between 1985 and 1996, sired by Holstein bulls. The analyses were conducted on data only comprising, single births, heifers between 20 and 38 months of age at calving, gestation lengths between 256 and 304 days and records with information of both sire and maternal grandsire. The material consisted then of 411 409 first, and 281 193 second calving records.

Stillbirths were defined as calves dead at birth or within 24 hours after birth. Dystocia was defined as calvings where the heifer/cow clearly needed assistance or when the calf had wrong position at birth.

Statistical model

$$Y_{ijklmnop} = \mu + a_i + b_j + c_k + d_l + f_m + s_n + g_o + hy_p + e_{ijklmnop}$$

where

$Y_{ijklmnop}$ denotes a 0 or 1 for live vs. stillborn and easy/normal vs. difficult calving
a_i = the fixed effect of the *i*th calving age of the dam (only first parity)
b_j = the fixed effect of the *j*th season of calving
c_k = the fixed effect of the *k*th sex of the calf
d_l = the fixed effect of the *l*th genetic group of sire
f_m = the fixed effect of the *m*th genetic group of maternal grandsire
s_n = the random effect of sire of calf
g_o = the random effect of maternal grandsire of calf
hy_p = the random effect of herd*year
$e_{ijklmnop}$ = random residual effect

Additionally, analyses were conducted with calving performance included as a fixed effect.

Age of the dam was divided into seven classes; 20-22, 23-24, 25-26, 27-28, 29-31, 32-34 and 35-38 months at calving. The calving seasons were August – September, October, November – February, March – April and May –July. The groupings of sires and maternal grandsires were based on year of birth of the bulls and on proportion of Holstein Friesian genes expressed in quartiles. The relationship matrix for sires and maternal grandsires in the data set, contained in total 2995 equations for data from first, and 1905 equations for data from second parity.

The data was analysed with both linear mixed models and with threshold models. Variance components from the linear mixed models were estimated using a restricted maximum likelihood (REML) procedure, (Jensen & Madsen, 1993). In a Bayesian analysis inference from the threshold model was based on a posterior distribution achieved with the Gibbs sampling technique (Jensen, 1994). In the threshold model the event of a stillbirth was assumed to be the result of a normally distributed variate, a liability value. The liability values for stillbirths were created by data augmentation, as described by Sorensen (1996). They were replaced by values drawn from normal distributions conditional on the other effects in the model, the standard linear methodology could hence be applied.

Uniform, improper prior distributions were used for fixed effects. As vague priors, properly scaled variance components estimated in the linear analyses were used for herd-year variance and for genetic and environmental (co)variances. The environmental variance was assumed to be 1 and the threshold was set to 0. A chain with 11 000 samples for the first and 14000 samples for second parities were used. The first 300 samples were regarded as burn-in period and discarded.

Variances, covariances and heritabilities for the direct and maternal effect were calculated as:

$$\sigma^2_{Ad} = 4V_s$$
$$\sigma_{AdAm} = 4COV_{smgs} - \tfrac{1}{2}\sigma^2_{Ad}$$
$$\sigma^2_{Am} = 4V_{mgs} - {}^1/_4\sigma^2_{Ad} - \sigma_{AdAm}$$
$$h^2_{Ad} = 4\sigma_s^2 / \sigma_p^2$$
$$h^2_{m} = \sigma^2_{Am} / \sigma_p^2$$

The genetic correlation between direct and maternal effects was calculated as:

$$r_{g,AdAm} = \sigma_{AdAm} / \sqrt{\sigma^2_{Ad} * \sigma_{AdAm}}$$

where subscripts Ad and Am represent the direct and maternal additive effects respectively. V (variances) and COV (covariances) with appropriate subscripts of maternal grandsires and sires were from the output both from REML procedure and the Gibbs sampling.

For comparison the heritabilities from linear analyses were transformed to the underlying scale according to Dempster and Lerner (1950).

Results and discussion

The incidences of stillbirth and dystocia, heritabilities for the maternal and direct effects, and genetic correlations between direct and maternal effects are presented in Table 1.

Table 1. Estimates of heritabilities for direct and maternal genetic effects and genetic correlations between these effects at first and second parities for stillbirths and calving performance from the linear analyses, transformed values from the linear analyses, and estimates for stillbirths from threshold analyses

Trait		Inci-	h^2_{direct}			$h^2_{maternal}$			$r_{gdir/mat}$	
	Parity	dence	Linear	Transf	Thresh	Linear	Transf	Thresh	Linear	Thresh
Stillbirth	1	0.071	0.038	0.14	0.12	0.028	0.11	0.082	-0.13	-0.12
	2	0.027	0.007	0.04	0.044	0.003	0.020	0.022	-0.083	-0.12
Stillb. calving	1	0.071	0.020	0.072		0.013	0.047		-0.16	
perf. fixed eff.	2	0.027	0.003	0.022		0.001	0.005		0.49	
Calving	1	0.083	0.044	0.14		0.036	0.12		-0.10	
difficulty	2	0.051	0.0032	0.014		0.0008	0.004		-0.038	

For the first parity the Gibbs chain reached equilibrium distribution almost immediately. For the second parity, the chain was more unstable but seemed also to have reached equilibrium distribution relatively early.

The estimated heritabilities from the Bayesian analyses correspond well with the transformed heritabilities from the linear method. Compared to previous Swedish estimates from linear analyses heritability values for stillbirth rate at first calving have almost doubled, which is partly an effect of the increased incidence level. The heritabilities estimated for second parity were less than a third of the values for first parity when the threshold model was used, and of course even less when estimated on the visible scale. Because of the low frequency of stillbirths at second calving the genetic contribution to a visible variation in stillbirths at second parity is negligible. Also for calving difficulty at second parity, a genetic contribution to the visible variation seemed negligible. Here the difference between parities in frequency of difficult calving was smaller. Hence, as the transformed heritability suggest, the genetic variation is likely to be very small and not suitable for use in the selection against calving difficulties.

When adjusting for dystocia score in the model for stillbirths, heritabilities for direct and maternal effects at first parity calvings were reduced to half of the values obtained when no adjustment was applied. Thus, a significant genetic variation in stillbirth rate is independent of the incidence of dystocia. A consistent but small antagonistic genetic effect was found to exist between the direct and maternal effects at first parity, which indicates the necessity to evaluate bulls as both sires and maternal grandsires of calves.

Conclusions

From this study it could be concluded that the genetic variation in both stillbirth rate and dystocia at first calving is substantial in the Holstein breed both when measured as direct and maternal traits, while the corresponding genetic variation at second parity seems negligible. About half of the stillborn calves were born without difficulty and a significant genetic variation in liveability of these calves was found. The results shown have clear implications for the design of breeding strategies to reduce stillbirths and dystocia and for the needs to clearly define traits for genetic evaluation. Further research will be done to

estimate the correlation between stillbirths at first and second parity and to repeat the study on the SRB breed where the incidence levels at first parity are much less than in the Holstein Breed.

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