POSTNATAL MORTALITY IN DANISH HOLSTEIN CALVES

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INTRODUCTION

Mortality of calves is a cost for dairy cattle farmers. Due to legislation reliable recordings of postnatal mortality are available for all Danish cattle today. This offers new possibilities for analysing and reducing the problem. Environmental improvements (e.g. management and housing) are one way to reduce mortality, but these improvements are in general not permanent. Instead, genetic improvements have a permanent effect. However, genetic improvements are only possible if genetic variation for mortality exists. Only few genetic studies of postnatal mortality are available (e.g. Erf *et al.,* 1990 (dairy cattle); Cundiff *et al.,* 1986 (beef cattle)) but they are all based on a scarce number of animals. In a review by Shook (1998), it was concluded that more genetic and economic studies of calfhood diseases and calfhood mortality were needed for making decisions about using these traits in breeding programs. Diarrhoea is believed to be the most important cause to early mortality from 1 to 30 days after birth but later (from 1 to 6 months after birth) respiratory diseases becomes the most important cause of mortality (Agerholm *et al.,* 1993; Menzies *et al.,* 1996). Different genes might therefore affect early and late postnatal mortality. The objective of this study was to estimate and test, if direct and maternal additive genetic variation for postnatal mortality exists and to estimate the genetic association between early and late postnatal mortality.

MATERIALS AND METHODS

Data. A new legislation in 1998 forced all Danish farmers to supply all living cattle with an unique identification and to record all births, transfers, deaths and slaughterings every week. These data have made it possible to trace all cattle from birth to death (or until it is exported). These records and all other information concerning Danish cattle are saved in the Danish Central Cattle Database. All Records from all Holstein calves born between January 1998 and October 2000 (1,249,041 records) were extracted from this database. Information on transfers, deaths, and slaughterings from birth until 180 days after birth was available for all these calves. The following editing procedures were carried out: Records of calves with unknown dam, unknown sire, or unknown maternal grand sire and records with dams younger than 21 months at calving were omitted. Records of triplets or more were not included. Herds should have more than 50 calves born in the period, and sires and maternal grand sires should both have more than ten records included. Finally, only calves that survived their first 24 hours were included. Four mortality traits were defined with different periods of risk: D1-14, D15-60,

D61-180, and D1-180. The numbers indicate the period of risk in days after birth. For each trait only calves, which were alive at the beginning of the period, were included. Mortality was defined as a binary trait: If the calf died in the period of risk it was given the value 1, and if it survived through the whole period it was given the value 0. Records of calves, which were slaughtered or exported before the end of the period of risk, were deleted for each trait. Calves, which were transferred from one herd to another within the country, were included in the analyses. Some herds were exporting all or a large proportion of their bull calves. In order to avoid an artificially high proportion of mortality in these herds, it was chosen to omit all records on bull calves from herds where more than 20% of the bull calves were exported in the period of risk. The mean and number of observations for the four mortality traits after editing are given in Table 1.

Table 1. Number of records and mean of mortality traits in the final dataset

Trait	No.	Mean
D1-14	841,921	0.0277
D15-60	767,070	0.0187
D61-180	750,596	0.0209
D1-180	788,189	0.0676

Statistical analyses. Although the mortality traits were binary all analyses were carried out with linear models. The analyses consisted of three parts: A) test of fixed effects using "proc GLM" in SAS, B) estimation of variance components with a univariate model, and C) estimation of variance components with a bivariate model. The analyses in part B and C were carried out with the AI-REML procedure (Jensen *et al.,* 1997).

In the analyses in part A a fixed effect of herd was absorbed. Fixed effects (and their interactions), which were significant ($p<0.05$) for just one of the traits, were chosen and used for all traits in the models in part B and C. This lead to the following fixed part of the model used for estimation of variance components in B and C: Birthyear-birthmonth-region-sex, calving age(parity), twin-sex, calving interval. For the trait D60-180 an effect of transfers from day 1- 29 and transfers from day 30-60 and their interactions with the season of transfer (in months) were included. An effect of calving age at second calving was not significant for any of the traits and thereby dropped.

The random effects in the univariate model in part B were: Herd-year-season, herd, herd-sex, sire of calf, sire of dam and the residual. Records were clustered to herd-year-season effects as described by Schmitz *et al.* (1991), with a maximum interval of 365 days and minimum desired cluster size of 20 records. Covariance between the sire of calf and the sire of dam was assumed but no covariances between all the other random effects were assumed. The number of sires of calves and sires of dams was 3553 and 5833. A part of these sires (828) was considered both as sires of calves and sires of dams. This resulted in 8558 different sires with records. The relationship matrix for these sires was build by tracing their sires and dams as far back as possible. This resulted in a relationship matrix consisting of 29,608 animals.

In the bivariate models used in part C the effect of the sire of dam was excluded, as the effect was found to be small. Written in matrix notation the model was: $\mathbf{y}_i = \mathbf{X}_i\mathbf{b}_i + \mathbf{W}_i\mathbf{h}_i + \mathbf{Z}\mathbf{s}_i + \mathbf{e}_i$. Where $i=\{1,2\}$ is denoting the trait. $\mathbf{X}_i$, $\mathbf{W}_i$, and $\mathbf{Z}_i$ were design matrices relating effects to observations. The fixed effects in the model were given in $\mathbf{b}_i$ and the three random environmental effects were given in $\mathbf{h}_i$. The genetic effect of sire was given in $\mathbf{s}_i$ and the residual in $\mathbf{e}_i$. All random effects were assumed independent within each trait but covariance between traits was assumed for all random effects.

Estimates of heritabilities of direct and maternal effects and estimates of genetic correlation between direct and maternal effects were derived as in Manfredi *et al*. (1991). The total variation used to derive the heritabilities was defined as the sum of all variance components except variance components of herd, herd-sex, and herd-year-season. Heritabilities of binary traits depend on the frequency of the trait. In order to express heritabilities independent of the frequency they were transformed to the underlying scale by the formula of Dempster and Lerner (1950). Based on asymptotic theory all estimates were considered significantly different from the hypothesis, when they differed more than 1.96 times the standard error of the estimate.

RESULTS AND DISCUSSION

Univariate analysis. Estimates of heritabilities and genetic correlations between direct and maternal effects from the univariate analyses in part B are given in Table 2. The estimates of the heritability of the direct effects were significantly different from zero for all mortality traits. The estimates of direct heritability of D1-14, D15-60, D61-180, and D1-180 were 0.0044, 0.0026, 0.0123, and 0.0116. When transforming these heritabilities to the underlying scale, the heritability of D61-180 (0.100) was considerably higher than the other traits. Estimates of maternal heritabilities were significant for D1-14 and D1-180 only. Estimates of correlations between direct and maternal genetic effects have high standard errors. This was due to the structure of the data where most of the sires were only sires of calves or sires of dams. Only the estimated correlation for D61-180 (-0.503) was significantly different from zero.

Table 2. Estimates of phenotypic standard deviation, direct (D) heritability, maternal (M) heritability, and the correlation between direct and maternal effect [A]

	σ_P	$h^2_{D(o)}$	$h^2_{M(o)}$	$h^2_{D(u)}$	$h^2_{M(u)}$	$r_{D,M}$
D1-14	0.164	$0.0044_{\mp 0.0008}$	$0.0015_{\mp 0.0005}$	0.029	0.010	$0.210_{\mp 0.240}$
D15-60	0.135	$0.0026_{\mp 0.0006}$	$0.0005_{\mp 0.0003}$	0.023	0.004	$-0.195_{\mp 0.300}$
D61-180	0.143	$0.0123_{\mp 0.0015}$	$0.0006_{\mp 0.0005}$	0.100	0.005	$-0.503_{\mp 0.210}$
D1-180	0.251	$0.0116_{\mp 0.0014}$	$0.0024_{\mp 0.0009}$	0.043	0.009	$-0.294_{\mp 0.159}$

[A]Heritabilities are expressed on the observed scale (o) and the underlying scale (u). Standard errors are given in subscript.

Bivariate analysis. Estimates of correlations between traits from the bivariate analyses are given in Table3. The estimated genetic correlation between D1-14 and D15-60 was high (0.803). The estimated genetic correlation between D15-60 and D61-180 was moderate to high (0.589). The lowest estimate of genetic correlation was found between D1-14 and D61-180 (0.350), which was significantly different from 1. It indicates that different genes are responsible for early and late mortality. The estimates of residual correlations were all close to zero, but the interpretation of these correlations are not clear.

Table 3. Estimates of genetic and residual correlations for the direct effects of mortality[A]

	D1-14	D15-60	D61-180
D1-14		0.803 ± 0.073	0.350 ± 0.102
D15-60	-0.0038 ± 0.0016		0.589 ± 0.107
D61-180	0.0045 ± 0.0016	0.0021 ± 0.0016	

[A] Genetic correlations above diagonal and residual correlations below diagonal. Standard errors are given in subscript.

Postnatal mortality might be a potential trait to include in a breeding programme for dairy cattle. Even though the direct heritability is low the direct genetic variation is considerable. It is possible to gain reliable predictions of breeding values, as the number of offsprings per proven sire in general will be more than twice the number of milking daughters.

CONCLUSION

This study has shown that direct genetic variation for postnatal mortality exists. Maternal genetic variation seems to exist for D1-14 and D1-180, but the effect is small. No maternal genetic variation could be detected for the mortality after day 15. Moderate to high genetic correlations between the direct genetic effect of D1-14, D15-60, and D61-180 were estimated. Different genes seem to affect early (D1-14) and late mortality (D61-180) as the direct genetic correlation (0.350) was significantly less than 1.

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