Genetic Parameters for Fertility Related Disorders in Norwegian Red

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Abstract

Heritabilities and genetic correlations were estimated for the 4 most common fertility related disorders in Norwegian Red: retained placenta, cystic ovaries, silent heat and metritis. Each of the 4 disorders was analyzed separately with the first 5 lactations as correlated traits using multivariate threshold sire models. Heritability estimates ranged from 0.03 to 0.14, and were lowest for metritis and highest for cystic ovaries. The genetic correlations between lactations for cystic ovaries (0.74 – 0.95) was high and close to 1. So was also the correlations for retained placenta for lactations 2 through 5 (0.86 – 0.94), while the correlation between first and later lactations was lower (0.59 – 0.68). The genetic correlations between lactations were moderate for metritis (0.40-0.77) and silent heat (0.37-0.79). The results suggested that cystic ovaries can be considered to be the same trait genetically across lactations, while metritis and silent heat was different traits genetically across lactations and retained placenta in first lactation was genetically different from the subsequent lactations.

Key words: Retained placenta, cystic ovaries, silent heat, metritis, genetic correlations, heritability

Introduction

Healthy animals with good fertility are of great importance in dairy cattle production. Fertility related disorders were the only category of diseases that increased in Norway last year (Helsetjenesten for storfe, 2013). The most common fertility related disorders in Norway are retained placenta (**RP**), cystic ovaries (**CO**), silent heat (**SH**) and metritis (**MET**). Veterinary treatments have been recorded routinely in the Norwegian Dairy Herd Recording System (**NDHRS**) since 1978, but fertility related disorders have so far not been included in routine genetic evaluations.

Heringstad (2010) estimated heritabilities for and genetic correlations among SH, CO, RP and MET in first lactation Norwegian Red. The frequency of some of these traits may increase in later lactations. Differences in heritability for fertility disorders have been reported for estimates based on first lactation vs. all lactations (Uribe *et al.*, 1995, Zwald *et al.*, 2004). Heringstad *et al.* (2005) reported genetic correlation from 0.55 to 0.65 for RP across lactation 1 to 3. This suggests that fertility disorders may be considered to be genetically different across lactations.

The frequency of fertility related disorders are still low in Norway but with increasing frequencies it may become desirable to include this group of diseases in the breeding program for Norwegian Red. Using multiple lactations in genetic evaluation may be advantageous as some of these problems are more common in later lactations. The aim of this study was to estimate heritabilities for the 4 most common fertility related disorders in 5 lactations, and based on genetic correlations evaluate if these fertility related disorders genetically can be considered to be the same trait across lactations.

Material and Methods

Information on calving and fertility related health records from 780,114 Norwegian Red cows calving from January 2001 through December 2011, and sired by Norwegian Red AI bulls, was extracted from the NDHRS. Information from the first 5 lactations was used. The calving had to be within defined age intervals for the lactation record to be included: 20-36 months, 32-48 months, 44-60 months, 56-72 months and 68-84 months for first, second, third, fourth and fifth calving

respectively. Cows without a first lactation in the dataset were omitted. Lactation records were defined from the day of calving until 15 days before next calving, culling or 400 days after calving, whichever occurred first. Traits were defined for each lactation, giving a total of 20 traits; the 4 fertility disorders RP, CO, MET and SH in 5 lactations. Each trait was defined as binary, where 1 indicated that the cow had at least one veterinary treatment for that disorder in a given lactation, and 0 otherwise. For RP the treatment had to occur within the first 5 days after calving to be included, while for the other disorders all health records within the defined lactation were used. The numbers of cows in lactation 1 to 5 were 780,114, 489,903, 280,085, 138,938 and 58,461, respectively. There were a total of 27,185 animals in the pedigree file, which consisted of the 1,247 bulls with daughters in the dataset and their dams and sires traced back as far as possible.

Model

Each of the 4 fertility related disorders was analyzed separately, with the 5 lactations as correlated traits in a multivariate threshold sire model. Systematic effects for each trait were selected based on GLM analyses including the potential factors. Thus the following four models were used for the final analyses:

$$\boldsymbol{\lambda_{CO} = ys + ci + age + herd + sire + e}$$

$$\boldsymbol{\lambda_{SH} = ys + ci + herd + sire + e}$$

$$\boldsymbol{\lambda_{MET} = ys + cd + twin + herd + sire + e}$$

$$\boldsymbol{\lambda_{RP} = ys + ci + cd + twin + herd + sire + e}$$

where $\boldsymbol{\lambda_{CO}}$, $\boldsymbol{\lambda_{SH}}$, $\boldsymbol{\lambda_{MET}}$ and $\boldsymbol{\lambda_{RP}}$ are vectors of liabilities for each fertility disorder in 5 lactations. **ys** is the systematic effect of year and season of calving where the seasons were defined as follows: January-March, April-June, July-September and October-December. **age** is the systematic effect of age at calving within each lactation in months (described above). **ci** is the systematic effect of calving interval in previous lactation, divided into 6 categories: 1) ≤325 days, 2) 325-340 days, 3) 341-355 days, 4) 356-370 days, 5) 371-400 days and 6) >400 days. **cd** is the effect of calving difficulty in 4 categories (1=easy calving, 2=slight problems, 3=difficult calving, 4=not known). **twin** is the effect of twinning, recorded as 1 (single calf) or 2 (two or more calves). **Herd** is the effect of herd (17,677 levels), **sire** is the random effect of sire (1,247 sires), and **e** is the residual.

The analyses were done with a Bayesian approach using Gibbs sampler in the RJMC-routine of the DMU package (Madsen and Jensen, 2007). Total chain lengths of 300.000 iterations after 10.000 iterations burn in were used for all traits.

Results and Discussion

Frequency of fertility related disorders

The frequency of all traits were low (<4%) in all lactations (Table 1). The frequencies of RP and CO increased in later lactations, from 1.2% to 3.4% for RP and from 0.5% and 1.7% for CO. For MET the frequency was stable for the first 4 lactations, between 0.6% and 0.8%, and increased to 1.1% in the fifth lactation, while SH decreased in later lactations (from 2.7% to 1.7%). The latter can probably be because cows with SH have a higher risk of being culled due to fertility problems and will therefore not reach the next lactation. These frequencies were lower than those reported from other populations (e.g. van Dorp *et al.*, 1998; Koeck *et al.*, 2010), except for RP which frequency was similar to Austrian Fleckvieh (Koeck *et al.*, 2010).

Table 1. Mean frequency of cystic ovaries (CO), retained placenta (RP), metritis (MET) and silent heat (SH) in lactations 1 to 5.

Lactation	Frequceny (%)			
no	**CO**	**RP**	**MET**	**SH**
1	0.5	1.3	0.7	2.7
2	1.0	2.1	0.6	2.1
3	1.5	2.6	0.7	2.0
4	1.6	3.1	0.8	1.8
5	1.7	3.4	1.1	1.7

Heritabilities

Heritabilities of liability were low for all traits (Table 2-5), but seemed to increase slightly in later lactations. The SD was small, indicating fairly accurate estimates, but increasing with

Table 2. Posterior means (SD) of heritability of liability (diagonal) and posterior means of genetic correlations (below diagonal) for cystic ovaries (CO_i), in five lactations (i=1-5).

	CO1	**CO2**	**CO3**	**CO4**	**CO5**
CO1	*0.10* *(0.01)*				
CO2	0.91 (0.03)	*0.14* *(0.02)*			
CO3	0.84 (0.06)	0.95 (0.03)	*0.13* *(0.02)*		
CO4	0.86 (0.07)	0.93 (0.04)	0.92 (0.04)	*0.12* *(0.02)*	
CO5	0.74 (0.11)	0.85 (0.07)	0.85 (0.07)	0.90 (0.07)	*0.14* *(0.03)*

Table 3. Posterior means (SD) of heritability of liability (diagonal) and posterior means of genetic correlations (above diagonal) for silent heat (SH_i), in five lactations (i=1-5).

	SH1	**SH2**	**SH3**	**SH4**	**SH5**
SH1	*0.05* *(0.01)*				
SH2	0.79 (0.04)	*0.03* *(0.01)*			
SH3	0.60 (0.12)	0.83 (0.09)	*0.04* *(0.01)*		
SH4	0.37 (0.14)	0.65 (0.12)	0.77 (0.12)	*0.04* *(0.01)*	
SH5	00.44 (0.16)	0.77 (0.12)	0.59 (0.21)	0.44 (0.25)	*0.07* *(0.03)*

lactation. All first lactation estimates were in accordance with those reported by Heringstad (2010), except CO which was higher in the present study. In general, CO had the highest heritabilities, ranging from 0.10 to 0.14 (Table 2). These estimates were higher than those reported previously from threshold models of about 0.08 (Zwald *et al.*, 2004; Koeck *et al.*, 2010). Uribe *et al.* (1995) estimated heritabilities of 0.13 in first lactation cows, but 0.08 across lactations.

The lowest heritabilities were estimated for SH (Table 3) and MET (Table 4), ranging from 0.03 to 0.07 (SH) and 0.8 (MET). Few studies have published heritability of SH, but Koeck *et al.* (2010) reported a heritability of 0.012 for SH and anestrus estimated with a threshold model across the first 5 lactations, which is lower than the estimates reported here. Other threshold model heritability estimates for MET ranges from 0.06 (Koeck *et al.*, 2010) to 0.08 (Zwald *et al.*, 2004), which is slightly higher than our estimates.

The heritabilities of liability to RP ranged from 0.07 to 0.10 (Table 5). This is slightly higher than previously reported estimates of 0.06 (Koeck *et al.*, 2010) and 0.08 (Heringstad *et al.*, 2005). Heringstad (2010) reported a heritability of 0.06 for RP in the first lactation, which is the same as reported here.

Due to variation in models, definition of traits and no of lactations included, comparison across studies should be made with caution. Here, comparisons of heritability were only to other studies that used threshold models, but trait definitions and number of lactations included varies.

Genetic correlations

Genetic correlations (Tables 2-5) were in general moderate to high. The highest genetic correlation was found between the CO-traits (Table 2), ranging from 0.74 (CO1-CO5) to 0.97 (CO2-CO3). High genetic correlations indicate that CO can be assumed to be the same trait in different lactations, and a repeatability model could be used instead of the multi-trait model.

The genetic correlations were lower for SH (Table 3) and MET-traits (Table 4). For SH the genetic correlations ranged from 0.37 (SH1-SH4) to 0.83 (SH2-SH3), while for MET they ranged from 0.38 (MET4-MET5) to 0.77 (MET2-MET3). This suggests that SH and MET genetically are somewhat different traits when observed in different lactations. These estimates also had quite large SD.

Table 4. Posterior means (SD) of heritability of liability (diagonal) and posterior means of genetic correlations (above diagonal) for metritis (MET_i), in five lactations (i=1-5).

	MET1	**MET2**	**MET3**	**MET4**	**MET5**
MET1	*0.04* *(0.01)*				
MET2	0.56 (0.15)	*0.03* *(0.01)*			
MET3	0.64 (0.14)	0.77 (0.11)	*0.04* *(0.01)*		
MET4	0.51 (0.21)	0.67 (0.16)	0.53 (0.21)	*0.04* *(0.02)*	
MET5	0.67 (0.15)	0.44 (0.23)	0.40 (0.23)	0.38 (0.29)	*0.08* *(0.03)*

Table 5. Posterior means (SD) of heritability of liability (diagonal) and posterior means of genetic correlations (above diagonal) for retained placenta (RP_i), in five lactations (i=1-5).

	RP1	**RP2**	**RP3**	**RP4**	**RP5**
RP1	*0.07* *(0.01)*				
RP2	0.68 (0.06)	*0.08* *(0.01)*			
RP3	0.61 (0.07)	0.94 (0.03)	*0.09* *(0.01)*		
RP4	0.61 (0.08)	0.86 (0.05)	0.93 (0.04)	*0.10* *(0.01)*	
RP5	0.59 (0.11)	0.86 (0.06)	0.88 (0.05)	0.86 (0.07)	*0.10* *(0.02)*

For RP, all genetic correlations involving the first lactation were generally lower (0.57-0.71) than the genetic correlations for the second to fifth lactation (0.86-0.94) (Table 5). These genetic correlations were slightly higher than those reported by Heringstad *et al.* (2005), with estimates from 0.55 to 0.65 for RP in the three first lactations.

To our knowledge, few other studies have reported genetic correlations within disease across lactations for any of these fertility disorders as single traits. For RP, Heringstad *et al.* (2005) estimated genetic correlations in lactations 1 to 3 ranging from 0.55 to 0.65, while Schnitzenlehner *et al.* (1998) reported estimates for the genetic correlation for first and second lactation of 0.79. Mäntysaari *et al.* (1993) estimated genetic correlations between first and second lactation for MET (-0.58) and ovalutory disorders (0.60), while Pösö and Mäntysaari (1996) investigated the same disorders in lactations 1 through 3 and reported estimates of genetic correlations ranging from 0.09 to 0.62 for MET and 0.60 to 0.94 for ovulatory disorders. Nielsen *et al.* (1997) estimated genetic correlations for a combined reproduction disease trait across the three first lactations ranging from -0.01 to 0.19 in three Danish dairy breeds, indicating that fertility disorders are not genetically the same in different lactations, which is also supported by the results presented here. However, low to moderate genetic correlations between the fertility disorders (van Dorp *et al.*, 1998; Koeck *et al.*, 2010) may have great influence on the low genetic correlation of a composite fertility disorder trait across lactations.

Further reseach

Estimation of genetic parameters for a composite trait for fertility related disorders, as well as genetic correlations to other traits are if further interest. It is also interesting to investigate how information on fertility related disorders can be utilized for genomic evaluations.

Conclusions

Results suggest that it may be reasonable to use a repeatability model for CO lactation 1-5 and RP lactation 2-5, whereas SH and MET should be analyzed with multi-trait models.

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