Short communication: 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. Data on 1,747,500 lactations from 780,114 cows calving from January 2001 through December 2011 were analyzed using multivariate threshold sire models to estimate variance components for the 4 disorders in the first 5 lactations. The traits were defined as binary within lactation (0 = unaffected, 1 = affected), and each fertility-related disorder was analyzed separately with the 5 lactations as correlated traits. The mean frequency of affected cows ranged from 0.5 to 1.7% for cystic ovaries, 0.7 to 1.1% for metritis, 1.3 to 3.4% for retained placenta, and 1.7 to 2.7% for silent heat. Posterior means (standard deviations) of heritability of liability ranged from 0.02 (0.01) to 0.12 (0.01), and were lowest for silent heat and highest for cystic ovaries. Genetic correlations across lactation within disorder were positive and moderate to high, ranging from 0.79 to 0.95 for cystic ovaries, 0.40 to 0.75 for metritis, 0.53 to 0.94 for retained placenta, and 0.39 to 0.83 for silent heat.

Key words: retained placenta, cystic ovaries, silent heat, metritis, genetic correlation

Short Communication

Fertility-related disorders can decrease cow fertility and increase the calving interval, and they are of economic importance due to increased labor and veterinary costs and reduced production. Cystic ovaries (CO), metritis (MET), retained placenta (RP), and silent heat (SH) are the most common fertility-related disorders in Norway, and this category of diseases was the only category that increased in frequency in Norway in 2013 (Norwegian Cattle Health Services, 2014). Like many other disease traits, heritabilities of fertility-

related disorders are generally low. Heritability estimates from threshold models range from 0.05 to 0.08 for CO and 0.03 to 0.08 for MET (Zwald et al., 2004; Heringstad, 2010; Koeck et al., 2010), 0.06 to 0.08 for RP (Heringstad et al., 2005; Heringstad, 2010; Koeck et al., 2010), and 0.01 to 0.06 for SH (Heringstad, 2010; Koeck et al., 2010). Studies have shown that heritability varies between lactations; for example, Zwald et al. (2004) reported larger heritability estimates for CO and MET from the first lactation relative to estimates from all available lactations. Heringstad et al. (2005) reported a heritability of 0.08 for RP in lactations 1 to 3 in Norwegian Red, but the genetic correlations between lactations ranged from 0.55 to 0.65, indicating that the disorder genetically is not the same trait across lactations.

As some of the fertility-related disorders increase in frequency in the later lactations, it may be advantageous to use multiple lactations in genetic evaluations. The aims were to estimate heritabilities for CO, MET, RP, and SH in the first 5 lactations, and to evaluate whether these disorders can be considered to be genetically the same trait across lactations based on genetic correlations between the lactations within each disorder.

Information on calving and fertility-related health records were extracted from the Norwegian Dairy Herd Recording System. Information included up to 5 lactations from 780,114 cows sired by Norwegian Red AI bulls, calving from January 2001 to December 2011. Cows without first-lactation data were omitted from the data set. Age at calving had to be within defined intervals for the lactation record to be included (20–36 mo, 32–48 mo, 44–60 mo, 56–72 mo, and 68–84 mo for lactations 1 to 5, respectively). The definition of lactation was from the day of calving until 15 d before the next calving, culling, or 400 d after calving, whichever occurred first. The data set contained 20 traits, 5 lactations for each of the 4 disorders, where each trait was defined as a binary (0 = unaffected, 1 = affected). For RP, the veterinary treatment had to occur within the first 5 d after calving, whereas for the other disorders all health records within the defined lactation were

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Table 1. Number of records and mean frequency of cystic ovaries (CO), metritis (MET), retained placenta (RP), and silent heat (SH) in lactations 1 to 5 for Norwegian Red

Lactation no.	No. of records	Frequency (%)			
		CO	MET	RP	SH
1	780,114	0.5	0.7	1.3	2.7
2	489,903	1.0	0.6	2.1	2.1
3	280,085	1.5	0.7	2.6	2.0
4	138,938	1.6	0.8	3.1	1.8
5	58,461	1.7	1.1	3.4	1.7

used. Numbers of records and mean frequency for the traits are given in Table 1. In total, 27,185 animals were in the pedigree file, which consisted of the 1,247 bulls with daughters in the data set and their dams and sires traced back as far as possible.

Each of the 4 fertility-related disorders was analyzed separately, with the 5 lactations as correlated traits in a multivariate threshold sire model. In matrix notation, the model can be written as $\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_h\mathbf{h} + \mathbf{Z}_s\mathbf{s} + \mathbf{e}$, where $\mathbf{y}$ is a vector of unobserved liabilities, $\boldsymbol{\beta}$ is a vector of systematic effects (described below), $\mathbf{h}$ is a vector of random herd-5-year effects (30,583 levels), $\mathbf{s}$ is the random effect of sire (1,247 levels), $\mathbf{e}$ is the vector of residual, and $\mathbf{X}$, $\mathbf{Z}_h$, and $\mathbf{Z}_s$ are the corresponding incidence matrices. The systematic effects were year-season of calving (seasons defined as January–March, April–June, July–September and October–December) (44, 41, 37, 33 and 29 levels for lactations 1 to 5 respectively) and age at calving in months (17 levels). Herd-5-year classes were defined by using 2 periods of approximately 5 yr each (2001–2006 and 2007–2011). Heritability was calculated as

$$h^2 = \frac{4\sigma_{sire}^2}{\sigma_{sire}^2 + \sigma_{herd}^2 + \sigma_{residual}^2},$$

where σ_{sire}^2 , σ_{herd}^2 , and $\sigma_{residual}^2$ are the sire variance, herd-5-year variance, and residual variance, respectively. A

Bayesian approach using Gibbs sampler in the RJMC routine of the DMU package (Madsen and Jensen, 2007) was used for analyses. Based on Raftery and Lewis convergence statistics using the BOA package (Smith, 2003), we decided to use a total chain length of 300,000 iterations after 10,000-iteration burn-in for all traits.

The mean frequencies were low in all lactations for all disorders, less than 4% (Table 1). For 2 of the disorders (CO and RP), frequency increased in later lactations, and for CO the frequency was 3 times as high in the fifth lactation (1.7%) as in the first lactation (0.5%). For RP, the frequency more than doubled from the first lactation (1.3%) to the fifth lactation (3.4%). For MET, the frequency was stable (0.6–0.8%) in the first 4 lactations but increased in the fifth lactation (1.1%). The frequency of SH decreased with increasing lactations, from 2.7 to 1.7%. In general, these frequencies were lower than disease frequencies reported in other studies; for CO, frequencies range from 3.1% (Canadian Holstein, van Dorp et al., 1998) to 13% (Finnish Ayrshire, Mäntysaari et al., 1993), whereas for MET they range from 2.5% (Finnish Ayrshire, Pösö and Mäntysaari, 1996) to 21% (US Holstein, Zwald et al., 2004). Koeck et al. (2010) reported a frequency for SH of 6.3% in Austrian Fleckvieh. For RP, the frequencies were more similar to those presented in the present study, where most range between 1.3% (Canadian Holstein, van Dorp et al., 1998) and 5.8% (Austrian Simmental, Schnitzenlehner et al., 1998), although Lin et al. (1989) presented frequencies for RP in US Holstein of 8.5 and 12.7% for second-lactation cows and older cows, respectively.

Heritabilities of liability were low for all traits (Tables 2, 3, 4, and 5). The posterior mean ranged from 0.02 (SH2 and SH3; where SH2 = SH in lactation 2) to 0.12 (CO2), with small SD (0.01–0.02) for all traits, indicating accurate heritability estimates. All first-lactation estimates were in accordance with those reported by Heringstad (2010), which were based on partly the same data set as in the present study.

Table 2. Posterior mean (SD) of heritability of liability (diagonal), genetic correlations (below diagonal), and herd correlations (above diagonal) for cystic ovaries (CO i), in 5 lactations ($i = 1-5$), with 95% highest posterior density interval given in brackets

	CO1	CO2	CO3	CO4	CO5
CO1	0.08 (0.01) [0.06–0.11]	0.92 (0.02) [0.89–0.96]	0.88 (0.03) [0.83–0.93]	0.79 (0.03) [0.73–0.86]	0.76 (0.05) [0.66–0.85]
CO2	0.91 (0.04) [0.83–0.97]	0.12 (0.01) [0.09–0.14]	0.97 (0.01) [0.94–1.00]	0.93 (0.02) [0.88–0.98]	0.90 (0.02) [0.83–0.97]
CO3	0.83 (0.06) [0.70–0.94]	0.95 (0.02) [0.90–0.99]	0.11 (0.01) [0.08–0.14]	0.94 (0.03) [0.89–0.99]	0.86 (0.04) [0.78–0.94]
CO4	0.88 (0.06) [0.77–0.97]	0.94 (0.03) [0.89–0.99]	0.93 (0.04) [0.85–0.99]	0.09 (0.02) [0.06–0.12]	0.88 (0.05) [0.80–0.97]
CO5	0.79 (0.09) [0.61–0.94]	0.90 (0.07) [0.77–0.98]	0.92 (0.06) [0.79–0.99]	0.91 (0.06) [0.79–0.99]	0.09 (0.02) [0.06–0.13]

Table 3. Posterior mean (SD) of heritability of liability (diagonal), genetic correlations (below diagonal), and herd correlations (above diagonal) for metritis (MET i), in 5 lactations ($i = 1-5$), with 95% highest posterior density interval given in brackets

	MET1	MET2	MET3	MET4	MET5
MET1	0.04 (0.01) [0.02–0.05]	0.87 (0.03) [0.81–0.93]	0.77 (0.05) [0.67–0.86]	0.76 (0.08) [0.61–0.91]	0.71 (0.08) [0.54–0.86]
MET2	0.57 (0.13) [0.31–0.81]	0.03 (0.01) [0.01–0.04]	0.85 (0.06) [0.75–0.96]	0.82 (0.06) [0.71–0.94]	0.86 (0.06) [0.73–0.96]
MET3	0.59 (0.15) [0.28–0.84]	0.74 (0.13) [0.50–0.96]	0.03 (0.01) [0.02–0.05]	0.74 (0.08) [0.58–0.89]	0.81 (0.07) [0.66–0.94]
MET4	0.48 (0.23) [0.07–0.89]	0.75 (0.14) [0.48–0.95]	0.55 (0.20) [0.16–0.91]	0.03 (0.01) [0.01–0.05]	0.68 (0.13) [0.45–0.92]
MET5	0.72 (0.13) [0.47–0.93]	0.47 (0.21) [0.05–0.82]	0.42 (0.27) [–0.05–0.86]	0.40 (0.23) [–0.03–0.82]	0.06 (0.02) [0.02–0.10]

The highest heritabilities were found for CO, ranging from 0.08 to 0.12 (Table 2), which is in agreement with previously reported heritability estimates for CO (e.g., Zwald et al., 2004; Koeck et al., 2010). The lowest heritabilities of liability were estimated for SH (0.02–0.04; Table 5) and MET (0.03–0.06; Table 3). Very few studies have published heritability of SH, so comparisons are sparse. Koeck et al. (2010) reported a heritability from a threshold model of 0.012 for SH and anestrus in the first 5 lactations, which is slightly lower than the estimates reported here. Threshold model heritability estimates for MET range from 0.06 (Koeck et al., 2010) to 0.08 (Zwald et al., 2004), which is in accordance with our estimates. The heritabilities of liability to RP ranged from 0.06 to 0.09 (Table 4). This is in agreement with previous estimates of 0.06 (Koeck et al., 2010) and 0.08 (Heringstad et al., 2005).

Generally, the genetic correlations (Tables 2 to 5) among lactations within each disorder were positive and moderate to high. The highest genetic correlations were found between the CO traits (Table 2), with posterior mean ranging from 0.79 (CO1–CO5) to 0.95 (CO2–CO3), and the upper bounds of the 95% highest posterior density (HPD) intervals were all >0.94. This was higher than genetic correlations of ovulatory disorders between lactations reported previously, ranging from 0.60 to 0.94 (Mäntysaari et al., 1993; Pösö

and Mäntysaari, 1996). However, those studies included anestrus, subestrus, and other infertilities in addition to CO, and comparison is therefore difficult.

The posterior means of genetic correlations for SH ranged from 0.39 (SH1–SH4) to 0.83 (SH3–SH4), whereas for MET, the posterior means of genetic correlations ranged from 0.40 (MET4–MET5) to 0.75 (MET2–MET4). The 95% HPD intervals for the genetic correlations among these traits were relatively wide, indicating uncertain estimates, especially for the later lactations. For MET5, the 95% HPD interval of the genetic correlations to MET3 and MET4 even included zero. Previous genetic correlation estimates for MET between lactations range from –0.58 to 0.62 (Mäntysaari et al., 1993; Pösö and Mäntysaari, 1996). In those studies, the standard errors were also large.

The posterior means of genetic correlations for RP showed a difference between the correlations involving the first lactation (0.53–0.69) and the correlations among the second to fifth lactation (0.84–0.94; Table 4). 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 3 first lactations. Schnitzenlehner et al. (1998) reported a genetic correlation for RP in the first and second lactation of 0.79, which is higher than the corresponding estimate of the present study.

Table 4. Posterior mean (SD) of heritability of liability (diagonal), genetic correlations (below diagonal), and herd correlations (above diagonal) for retained placenta (RP i), in 5 lactations ($i = 1-5$), with 95% highest posterior density interval given in brackets

	RP1	RP2	RP3	RP4	RP5
RP1	0.06 (0.01) [0.04–0.07]	0.89 (0.03) [0.84–0.95]	0.93 (0.03) [0.88–0.98]	0.84 (0.04) [0.76–0.93]	0.84 (0.05) [0.74–0.93]
RP2	0.69 (0.06) [0.56–0.80]	0.07 (0.01) [0.05–0.08]	0.96 (0.02) [0.93–0.98]	0.92 (0.03) [0.86–0.99]	0.87 (0.05) [0.77–0.96]
RP3	0.60 (0.07) [0.47–0.74]	0.92 (0.03) [0.86–0.98]	0.08 (0.01) [0.06–0.10]	0.92 (0.03) [0.86–0.98]	0.88 (0.05) [0.80–0.98]
RP4	0.60 (0.08) [0.45–0.74]	0.84 (0.05) [0.74–0.94]	0.94 (0.03) [0.89–0.99]	0.09 (0.01) [0.06–0.11]	0.83 (0.06) [0.70–0.93]
RP5	0.53 (0.10) [0.32–0.73]	0.84 (0.06) [0.72–0.95]	0.87 (0.06) [0.76–0.98]	0.86 (0.07) [0.73–0.97]	0.09 (0.02) [0.05–0.12]

Table 5. Posterior mean (SD) of heritability of liability (diagonal), genetic correlations (below diagonal), and herd correlations (above diagonal) for silent heat (SH_{*i*}), in 5 lactations (*i* = 1–5), with 95% highest posterior density interval given in brackets

	SH1	SH2	SH3	SH4	SH5
SH1	0.04 (0.01) [0.03–0.05]	0.96 (0.01) [0.95–0.97]	0.93 (0.01) [0.92–0.94]	0.90 (0.01) [0.88–0.93]	0.88 (0.02) [0.85–0.92]
SH2	0.78 (0.06) [0.65–0.89]	0.02 (0.01) [0.02–0.03]	0.98 (0.01) [0.96–0.99]	0.96 (0.01) [0.95–0.98]	0.94 (0.02) [0.91–0.98]
SH3	0.58 (0.11) [0.37–0.78]	0.78 (0.10) [0.59–0.97]	0.02 (0.01) [0.01–0.03]	0.98 (0.01) [0.96–1.00]	0.97 (0.02) [0.93–1.00]
SH4	0.39 (0.14) [0.13–0.64]	0.64 (0.11) [0.42–0.87]	0.83 (0.09) [0.95–0.98]	0.03 (0.01) [0.01–0.04]	0.96 (0.02) [0.93–0.99]
SH5	0.45 (0.18) [0.14–0.82]	0.54 (0.20) [0.16–0.92]	0.51 (0.22) [0.10–0.88]	0.47 (0.18) [0.11–0.81]	0.04 (0.02) [0.01–0.08]

The posterior means of herd correlations between lactations were positive and high (0.71–0.98) for all the fertility-related disorders (Tables 2 to 5). The posterior means of residual correlations (results not shown) between lactations were low for all disorders, 0.06 to 0.31, –0.04 to 0.14, 0.11 to 0.19, and –0.05 to 0.19 for CO, MET, RP, and SH, respectively.

The main challenge with fertility-related disorders is their low frequency and definitions of the traits. In Norway, only veterinary treatments of disease are recorded and, in the analyses, a cow was considered “affected” if she had one or more veterinary treatments for the given disorder during the lactation. For some disorders, such as RP, this covers most of the actual cases of the disorder because it is easy to discover. Other disorders are more challenging and likely include more false negatives, such as SH, for example. Some cases of SH may not be discovered by the farmer and therefore not treated (and consequently, not recorded), or the disorder is discovered but the cow is culled instead of treated. The actual incidence of disease is therefore probably greater than that indicated by the records, valuable information is lost, and genetic analyses may be less accurate. In Tables 2 to 5, it can be seen that the SD and 95% HPD intervals for the genetic correlations between lactations were large for MET and SH. This may reflect the low frequency and complexity of these traits, relative to CO and RP, which have low SD and smaller 95% HPD intervals.

Genetic correlations between lactations within disorder were positive and moderate to high. Moreover, our results suggest that it is reasonable to assume that CO in lactations 1 to 5 is genetically the same trait, whereas neither MET nor SH can be considered the same trait across lactations.

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