



## 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

Received April 22, 2014.

Accepted November 4, 2014.

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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  $\boldsymbol{\lambda} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}_h\mathbf{h} + \mathbf{Z}_s\mathbf{s} + \mathbf{e}$ , where  $\boldsymbol{\lambda}$  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  $\sigma_{sire}^2$ ,  $\sigma_{herd}^2$ , 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]

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