# Genetic parameters for hoof lesions in Canadian Holstein cows estimated from hoof trimming records

N. Chapinal,\*<sup>¥</sup>A. Koeck,<sup>£</sup>A. Sewalem,<sup>#</sup> D. F. Kelton,\* S. Mason,<sup>§</sup>

G. Cramer, <sup>\$</sup>F. Miglior<sup>†#</sup>

<sup>\*</sup>Department of Population Medicine, University of Guelph, <sup>¥</sup>Animal Welfare Program, University of British Columbia, <sup>£</sup>Centre for Genetic Improvement of Livestock, University of Guelph, <sup>†</sup>Guelph Food Research Centre, Agriculture and Agri-Food Canada, <sup>#</sup>Canadian Dairy Network, <sup>§</sup>Alberta Milk, <sup>§</sup>Cramer Mobile Bovine Veterinary Services

### ABSTRACT

The objectives were to estimate the co(variance) components of hoof infectious and horn lesions using data collected by hoof trimmers in Canada, and the genetic correlations of hoof health traits with feet and legs conformation traits (including locomotion score) recorded by breed classifiers. Hoof health was recorded as presence or absence of specific hoof lesions in each hoof. Lesions were classified into infectious (digital and interdigital dermatitis, foot rot, and heel erosion), horn lesions (sole and toe ulcer, sole hemorrhage, and white line disease) and others lesions (interdigital hyperplasia, fissures, thin soles, and corkscrew claw). A total of 34,905 hoof health records from 27,179 cows and 365 herds, collected by 18 different hooftrimmers in Ontario, Alberta and British Columbia, were analyzed using linear animal models. In addition, 5 feet and leg conformation traits (foot angle, heel depth, bone quality, rear leg side view and rear leg rear view) and locomotion from primiparous cows were considered (n = 11,419and 6,966 cows, for conformation traits and locomotion, respectively). At least 1 lesion was found in nearly 40% of the hoof trimming records. The heritability estimates for hoof health lesions ranged from 0.01 for front horn lesions to 0.09 for rear infectious lesions. Despite the low heritability estimates, there was a large variability in sire relative breeding value for resistance to hoof lesions. There was a positive genetic correlation between the occurrence of front and rear infectious lesions (0.77), and front and rear horn lesions (0.61), but not between infectious and horn lesions (0.08). The heritability of the conformation traits ranged from 0.04 for rear leg rear view to 0.22 for bone quality, whereas the heritability for locomotion was 0.03. The genetic correlations between the hoof health and the conformation traits were low to moderate, yet most of the estimates were associated with high SE. In conclusion, although hoof lesions are low heritable traits, there is sufficient genetic variation (as evidenced by large variability in sire relative breeding value) for genetic improvement through direct selection in the long term. Standardization of hoof health data collection should be encouraged.

### **INTRODUCTION**

Maintaining hoof health is a challenge in modern dairy herds. Recent studies in North America and in Europe have reported prevalences of 40 to 70% of the cows with at least 1 hoof lesion (Manske et al., 2002; Sogstad et al., 2005; Buch et al., 2011). Hoof lesions compromise the welfare of animals (Whay et al., 2003) and can result in reduced milk yield (Warnick et al., 2001; Amory et al., 2008), reduced fertility (Hernández et al., 2001; Meléndez et al., 2003), and increased risk of premature culling (Rajala-Schultz and Gröhn, 1999; Booth et al., 2004). Many factors affect the risk of hoof lesions, including environmental factors such as the design of the facilities and management practices (Barker et al., 2009; Cook and Nordlund, 2009; Cramer et al., 2009) and genetics (van der Waaij et al., 2005; van der Linde et al., 2010; Buch et al., 2011).

Therefore, the incidence of hoof lesions can be reduced by improved management practices and genetic selection.

Hoof health is heritable and, although estimated heritability estimates are low (ranging from 0.01 to 0.20; Koenig et al., 2005; van der Waaij et al., 2005; van der Linde et al., 2010), long-term improvement of hoof health traits can be achieved by direct genetic selection for hoof lesion resistance. Several studies showed only low to moderate correlations between hoof health traits and feet and leg conformation traits (Swalve et al., 2008; Onyiro et al., 2008; van der Linde et al., 2010). As a result, indirect genetic selection for hoof lesion resistance has not been very effective. However, genetic correlations differ depending on the definition of the conformation traits and the population studied. Most of the studies on genetic parameters for hoof health have been conducted in Europe (van der Waaij et al., 2005; van der Linde et al., 2010; Buch et al., 2011). Thus, the potential of hoof health data for direct genetic selection for hoof health, and of conformation data to predict, and indirectly select for, hoof health in North America has to be investigated.

Hoof lesions have traditionally been classified according to their etiology and pathogenesis (Greenough, 2007) into infectious/partly infectious lesions (e.g. digital and interdigital dermatitis, foot rot, and heel erosion), mostly related to environmental hygiene, and horn lesions (e.g. sole and toe ulcer, sole hemorrhage, and white line disease) caused by metabolic/mechanical factors. Previous studies reported strong genetic correlations between specific lesions within each category (van der Waaij et al., 2005; van der Linde et al., 2010; Buch et al., 2011), but to our best knowledge, genetic parameters for these 2 categories have not been estimated. On the other hand, there is a dearth of literature on genetic parameters in North America, whereas standardized data routinely collected by hoof trimmers is readily available. Thus, the objectives of the current study were to estimate the co(variance) components of hoof infectious and horn lesions using data collected by hoof trimmers in Canada, and the genetic correlations of hoof health traits with feet and legs conformation traits (including locomotion score) recorded by breed classifiers.

#### **Data Collection**

#### **MATERIAL AND METHODS**

Hoof lesions were recorded by hoof trimmers from March 2004 to July 2005 in Ontario, from June 2009 to October 2011 in Alberta, and from October 2010 to October 2011 in British Columbia, Canada. The data recorded in Ontario were part of an observational study (Cramer et al., 2008) whereas the data recorded in Alberta and British Columbia were part of the ongoing Alberta Dairy Hoof Health Project. Details about the Alberta Dairy Hoof Health Project can be found at <u>www.hoofhealth.ca</u>. In brief, 7 hoof trimmers in Alberta and 6 hoof trimmers in British Columbia were trained to use of a rugged touch-screen computerized lesion recording system (Hoof Supervisor®, Dresser, Wisconsin). These systems facilitate the routine and consistent collection of hoof health data, based on lesion descriptions proposed by the International Lameness Committee, a global collaboration of researchers, veterinarians, academics and hoof trimming professionals.

Only the first hoof trimming session was included in the analyses if cows were hoof trimmed more than once during the same lactation. Records taken more than 500 d after calving were discarded. Four seasons of hoof trimming were defined from January to March, April to June, July to September and October to December, and the variable herd-year-season was created. The herd-year-season categories with less than 5 hoof trimming records were discarded.

The final dataset consisted of 34,905 hoof health records from 27,179 cows from 365 herds, collected by 18 different hoof-trimmers.

Hoof health was recorded as presence or absence of specific hoof lesions in each hoof: sole and toe ulcer, sole hemorrhage, white line disease (separation and abscesses), digital and interdigital dermatitis, foot rot, heel erosion, interdigital hyperplasia, fissures, thins soles and corkscrew claw. For the analyses, lesions were classified according to their etiology and pathogenesis (Greenough, 2007) into: a) infectious/partly infectious lesions (digital and interdigital dermatitis, foot rot, and heel erosion), b) horn lesions, caused by metabolic/mechanical factors (sole and toe ulcer, sole hemorrhage, and white line disease), and c) others lesions (interdigital hyperplasia, fissures, thin soles, and corkscrew claw). Nine hoof health traits defined as binary traits (0 = no lesion, 1 = at least 1 lesion) were created at the cow level: a) having at least one lesion in any hoof, b) having at least one lesion in a front hoof, c) having at least one horn lesion in any hoof, f) having at least one infectious lesion in a front hoof, g) having at least one infectious lesion in a rear hoof, h) having at least one horn lesion in a front hoof, hor hor lesion in a front hoof, and i) having at least one horn lesion in a rear hoof.

Conformation data on feet and legs were obtained from the Canadian Dairy Network (Guelph, Ontario). Conformation traits were routinely recorded by professional classifiers from Holstein Canada (Brantford, Ontario). Five conformation traits were considered: a) foot angle, b) heel depth, c) bone quality, d) rear leg side view, and e) rear leg rear view. In addition, locomotion score recorded during conformation evaluation in free-stall barns was available since 2005. All conformation traits were scored on a linear 1-to-9 scale. The optimum score for each trait is shown in Table 1. Conformation scores were transformed to normalize the data by using the procedure of Snell (1964). Only primiparous cows with conformation traits recorded within 365 d after calving were considered. In total, 11,419 primiparous cows had records on conformation traits, and of those, 6,966 cows had a locomotion score.

The pedigree file was generated by tracing the pedigrees of cows 7 generations back, and contained the relationship of 119,484 animals.

#### Models

Data were analyzed with linear animal models using the average information-restricted maximum likelihood (AI-REML) procedure in the derivative-free approach to multivariate analysis (DMU) package (Madsen and Jensen, 2008). Although threshold models are, at least in theory, more appropriate to analyze binary traits, linear models were applied. In a previous study on Canadian health data, Neuenschwander (2010) found that the use of threshold models did not improve the goodness of fit compared to linear models. Besides, genetic correlations are reported to be correct for binary traits using linear models (e.g. Negussie et al., 2008).

*Models for Hoof Lesions.* A first analysis was performed for the hoof health traits, including cows of all parities. The following models were carried out: a) univariate model for occurrence of any lesion, b) bivariate model between any front lesion and any rear lesion, c) bivariate model between any infectious lesion and any horn lesion, and d) 4-variate model between front infectious lesion, front horn lesion, rear infectious lesion, and rear horn lesion. The following linear animal model was applied to all lesion traits:

 $Y_{ijklmno} = \mu + P_i + LT_j + HT_k + HYS_l + a_m + pe_n + e_{ijklmno}$ 

where  $Y_{ijklmno}$  is the observation for one of the lesion traits,  $\mu$  is the overall mean,  $P_i$  is the fixed effect of parity (i = 1 to  $\geq$  6), LT<sub>j</sub> is the fixed effect of stage of lactation at trimming (j = 1 to 16; 1 = 0 to 30 d, 2 = 31 to 60 d, ..., 15 = 421-450 d, and 16 = 451-500 d after calving), HT<sub>k</sub> is the fixed effect of hoof trimmer (k = 1 to 18), HYS<sub>1</sub> is the fixed effect of herd-year-season of hoof trimming (l = 1 to 973), a<sub>m</sub> is the random additive genetic animal effect (m = 1 to 119,484), pe<sub>n</sub> is the random permanent environmental effect (n = 1 to 27,179), and e<sub>ijklmno</sub> is the random error term. Random effects were assumed to be normally distributed with zero means and the covariance structure was:

$$\operatorname{Var}\begin{bmatrix} a \\ pe \\ e \end{bmatrix} = \begin{bmatrix} A\sigma_a^2 & 0 & 0 \\ I\sigma_{pe}^2 & 0 \\ symm. & I\sigma_e^2 \end{bmatrix},$$

where  $\sigma_a^2$ ,  $\sigma_{pe}^2$  and  $\sigma_e^2$  are the additive genetic, permanent environmental, and residual variances, respectively, I is an identity matrix, and A is the additive genetic relationship matrix.

*Models for Hoof Lesions and Conformation Traits.* A second analysis was performed considering only primiparous cows with hoof health and conformation data (including locomotion). A total of 13 bivariate models were performed between infectious lesions, horn lesions and conformation traits (including locomotion). The following model was used for infectious and horn lesions:

$$Y_{ijklmn} = \mu + AGE_i + LT_j + HT_k + HYS_l + a_m + e_{ijklmn}$$

where  $Y_{ijklmn}$  is the observation for one of the lesion traits,  $\mu$  is the overall mean, AGE<sub>i</sub> is the fixed effect of age at calving (i = 1 to 16; 1 = <22 mo, 2 = 22, ..., 15 = 35 mo, 16 = >35 mo), LT<sub>j</sub> is the fixed effect of stage of lactation at trimming (j = 1 to 16; 1 = 0 to 30 d, 2 = 31 to 60 d, ..., 15 = 421-450 d, and 16 = 451-500 d after calving), HT<sub>k</sub> is the fixed effect of hoof trimmer (k = 1 to 18), HYS<sub>1</sub> is the fixed effect of herd-year-season of hoof trimming (l = 1 to 887), a<sub>m</sub> is the random additive genetic animal effect (m = 1 to 119,484), and e<sub>ijklmn</sub> is the random error term. Random effects were assumed to be normally distributed with zero means and the covariance structure was:

$$\operatorname{Var}\begin{bmatrix} a \\ e \end{bmatrix} = \begin{bmatrix} A\sigma_a^2 & 0 \\ \text{symm.} & I\sigma_e^2 \end{bmatrix},$$

where  $\sigma_a^2$  and  $\sigma_e^2$  are the additive genetic and residual variances, respectively; I is an identity matrix; and A is the additive genetic relationship matrix.

The model for conformation traits was:

 $Y_{ijklm} = \mu + AGE_i + LC_j + HRC_k + a_l + e_{ijklm}$ 

where  $Y_{ijklm}$  is the observation for one of the conformation traits,  $\mu$  is the overall mean, AGE<sub>i</sub> is the fixed effect of age at calving (i = 1 to 16; 1 = <22 mo, 2 = 22, ..., 15 = 35 mo, 16 = >35 mo),

 $LC_j$  is the fixed effect of stage of lactation at classification (j = 1 to 11; 1 = 0 to 30 d, 2 = 31 to 60 d, ..., 10 = 271 to 300 d, and 11 = 301 to 365 d after calving), HRC<sub>k</sub> is the fixed effect of herd-round-classifier (k = 1 to 1,459), a<sub>l</sub> is the random additive genetic animal effect (l = 1 to 119,484), and  $e_{ijklm}$  is the random error term. Random effects were assumed to be normally distributed with zero means and the covariance structure was:

$$\operatorname{Var}\begin{bmatrix} a \\ e \end{bmatrix} = \begin{bmatrix} A\sigma_a^2 & 0 \\ symm. & I\sigma_e^2 \end{bmatrix},$$

where  $\sigma_a^2$  and  $\sigma_e^2$  are the additive genetic and residual variances, respectively, I is an identity matrix, and A is the additive genetic relationship matrix.

## **Estimation of Breeding Values**

Breeding values for resistance to the occurrence of any lesion were obtained from the univariate analysis. Only sires with at least 20 daughters were considered. Estimated breeding values (**EBV**) were standardized to relative breeding values (**RBV**) with a mean of 100 and a standard deviation of 5 and reversed in sign. Thus, higher RBV indicate sires with daughters more resistant to lesions.

## **RESULTS AND DISCUSSION**

#### **Descriptive** Analysis

At least 1 lesion was found in nearly 40% of the hoof trimming records (Table 2), suggesting that modern dairy herds in North America could benefit from genetic selection for hoof health. Prevalence of hoof lesions (at least one lesion in one of the 4 hooves) in modern dairy herds varies across studies, ranging mostly from 40 to 70% (Manske et al., 2002; Sogstad et al., 2005; Buch et al., 2011). Lesions were more frequent in the rear than in the front hooves in agreement with the literature (Manske et al., 2002; Sogstad et al., 2005). This is likely because rear hooves are more exposed to manure and urine, and because of differences in the anatomy/mechanical function of the front and rear limbs that results in more strain exerted in the rear hooves (Phillips, 2002). Infectious lesions were the most common hoof disorder, in agreement with other studies that classified lesions similarly (Somers et al., 2003; Holzhauer et al., 2006). In the rear hooves, horn lesions increased with parity, whereas infectious lesions decreased. The prevalence of lesions in a herd and its relationship with parity is influenced by culling practices as well as age-related factors. Repeated damage in the corium of the hoof horn might be irreversible, resulting in an increasing prevalence of horn lesions with age (Offer et al., 2000; Koenig et al., 2005). Moreover, horn lesions developed in early life are likely to be recurrent in subsequent lactations (Hirst et al., 2002). On the contrary, the risk of infectious lesions decreases with parity (Rodrigues-Lainz et al., 1999; Somers et al., 2005; Holzhauer et al., 2006), suggesting an increase in local immunity with age or culling of older affected cows. Figure 1 shows the percentage of records at each stage of lactation. The percentage of records with lesions was consistent over the productive cycle (Figure 2), as expected since lameness is a long-term condition likely to persist over the lactation.

In agreement with other studies that used similar 1-to-9 scores (van der Waaij et al., 2005; Onyiro and Brotherstone, 2008; van der Linde et al., 2010), most of the mean scores for conformation traits and locomotion were below the optima (Table 1). The exception was the rear leg side view, trait for which the optimum is the middle score. These results suggest an

opportunity for improvement in conformation through genetic selection. For most of the conformation traits, the optimum scores were associated with lower frequencies of lesions (Figure 3). Specifically, low foot angle (score = 1 to 3) was associated with a higher percentage of horn lesions than average, and very low (score = 1 and 2) or very steep foot angle (score = 8 and 9) with a higher percentage of infectious lesions (Figure 3A) than average. Additionally, extremely shallow heel depth (score = 1) was associated with a much higher percentage of horn lesions than average (Figure 3B). Regarding rear leg side view, curved legs were associated with higher percentage of horn lesions (Figure 3D). Higher percentage of horn lesions and infectious lesions (Figure 3F).

## Genetic Parameter Estimation for Hoof Lesions

The heritability estimates for hoof health traits ranged from 0.01 to 0.09 (Table 3). These estimates are in line with those reported in the literature, which range from 0.01 to 0.17 for different individual hoof lesions based on linear models (Swalve et al., 2008; van der Linde et al., 2010; Buch et al., 2011). van der Linde et al. (2010) also estimated a heritability of 0.07 for having at least one hoof lesion. To our knowledge, this is the first study to estimate genetic parameters of hoof lesions separately for the front and rear hooves (most of the studies focus on the rear hooves or make no distinction), and to categorize lesions according to their etiology and pathogenesis into infectious and horn lesions. The heritability was higher for lesions in the rear than in the front hooves, and for infectious than for horn lesions. These differences could be partly explained by the higher prevalence of lesions in the rear than in the front hooves, and the higher prevalence of lesions, as heritability estimates are frequency-dependent when applying linear models to binary data (Gianola, 1982).

The bivariate models showed there was a positive genetic correlation  $(0.551\pm0.136)$  between the occurrence of any lesion in the front hooves and any lesion in the rear hooves, but not between the occurrence of any infectious lesion and any horn lesions in any hoof  $(0.077\pm0.110)$ . Similarly, the 4-variate model (Table 3) showed there were positive genetic correlations between the occurrence of front and rear infectious lesions, and front and rear horn lesions, but not between infectious and horn lesions. Other studies have reported positive genetic correlations between different lesions, particularly within horn lesions and within infectious lesions, when considering all hooves together (Buch et al., 2011) or just the rear hooves (van der Waaij et al., 2005; van der Linde et al., 2010). Most of these studies reported low or non-significant genetic correlations between different infectious and horn lesions and horn lesions. These results confirm that infectious and horn lesions are different traits, and that within each hoof lesion category, susceptibility is shared between front and rear hornes. This is in accordance with the different etiology and pathogenesis of infectious and horn lesions (Greenough, 2007).

## Estimation of Breeding Values for the Occurrence of Any Lesion

Despite the low heritability of occurrence of any lesion, there was a large variability in sire RBV (Figure 4). The average percentage of healthy daughters (no hoof lesion recorded at the first hoof trimming of the lactation) was 42% for the 10 sires with the worst RBV and 76% for the 10 sires with the best RBV for resistance to any lesion.

## Genetic Parameter Estimation for Hoof Lesions and Conformation Traits

Heritabilities and genetic correlations for infectious and horn lesions in primiparous cows (Table 5) were in agreement with our previous analyses. The heritability estimates of the conformation traits ranged from 0.03 for locomotion to 0.22 for bone quality. Compared with the heritability estimates used in the routine genetic evaluation in Canada (Interbull, 2011), estimates

in this study were slightly lower for most traits. This could be due to the smaller dataset used in the present study and differences in data editing procedures.

The genetic correlations between the hoof health and the conformation traits were low to moderate, yet most of the estimates were associated with high SE (Table 5). Therefore, a larger dataset is necessary to get more accurate estimates. Swalve et al. (2008) and Uggla et al. (2008) found most of the genetic correlations between hoof health and conformation traits were low or not significant, whereas Onyiro et al. (2008) and van der Linde et al. (2010) obtained higher correlations. Onyiro et al. (2008) found a negative genetic correlation between digital dermatitis and locomotion (-0.67) and bone quality (-0.21). Similarly, van der Linde et al. (2010) found that locomotion was genetically correlated with all the investigated hoof health traits, except for sole hemorrhage and white line disease, with estimates ranging from -0.24 to -0.58. In addition, van der Linde et al. (2010) found significant genetic correlations between several lesion traits and rear leg rear view, rear leg side view and foot angle, ranging from -0.32 to 0.25.

#### CONCLUSIONS

Infectious and horn hoof lesions are prevalent in Canada, and thus, genetic selection for hoof health should be incorporated in breeding programs. Although hoof lesions are low heritable traits, there is sufficient genetic variation (as evidenced by large variability in sire relative breeding value) for genetic improvement through direct selection in the long term. Infectious and hoof lesions were not genetically correlated, whereas front and rear hoof lesions within each lesion category were moderately correlated. Genetic correlations between hoof lesions and conformation traits and locomotion were low to moderate (albeit associated with high SE) which might explain why indirect selection for improving hoof health by using conformation traits has not been effective so far. Overall, these results show that hoof health, and therefore, standardization of hoof health data collection should be encouraged. Moreover, standardization of hoof health data collection is fundamental to monitor the incidence of hoof lesions and its association with management practices.

### ACKNOLEDGEMENTS

The authors are grateful to the hoof trimmers who participated in this study. This project was funded by DairyGen council of Canadian Dairy Network (Guelph, Ontario, Canada), Natural Sciences and Engineering Research Council of Canada (Ottawa, ON, Canada), and Alberta Milk (Edmonton, AB, Canada). Núria Chapinal was supported by a Beatriu de Pinós postdoctoral grant from the Generalitat de Catalunya.

#### REFERENCES

- Amory, J. R., Z. E. Barker, J. L. Wright, S. A. Mason, R. W. Blowey, and L. E. Green. 2008. Associations between sole ulcer, white line disease and digital dermatitis and the milk yield of 1824 dairy cows on 30 dairy cow farms in England and Wales from February 2003-November 2004. Prev. Vet. Med. 83:381-391.
- Barker, Z. E., J. R. Amory, J. L. Wright, S. A. Mason, R. W. Blowey, and L. E. Green. 2009. Risk factors for increased rates of sole ulcers, white line disease, and digital dermatitis in dairy cattle from twenty-seven farms in England and Wales. J. Dairy Sci. 92:1971–1978.
- Booth, C. J., L. D. Warnick, Y. T. Gro<sup>--</sup>hn, D. O. Maizon, C. L. Guard, and D. Janssen. 2004. Effect of lameness on culling in dairy cows. J. Dairy Sci. 87:4115–4122.

- Buch, L. H., A. C. Sørensen, J. Lassen, P. Berg, J. Å. Eriksson, J. H. Jakobsen, and M. K. Sørensen MK. 2011. Hygiene-related and feed-related hoof diseases show different patterns of genetic correlations to clinical mastitis and female fertility. J. Dairy Sci. 94:1540–1551.
- Cook, N. B., and K. V. Nordlund. 2009. The influence of the environment on dairy cow behaviour, claw health and herd lameness dynamics. Vet. J. 179:360-369.
- Cramer, G., K. D. Lissemore, C. L. Guard, K. E. Leslie, and D. F. Kelton. 2008. Herd- and cowlevel prevalence of foot lesions in Ontario dairy cattle. J. Dairy Sci. 91:3888–3895.
- Cramer, G., K. D. Lissemore, C. L. Guard, K. E. Leslie, and D. F. Kelton. 2009. Herd-level risk factors for seven different foot lesions in Ontario Holstein cattle housed in tie stalls or free stalls. J. Dairy Sci. 92:1404–1411.
- Gianola, D., 1982. Theory and analysis of threshold characters. J. Anim. Sci. 54:1079-1096.
- Greenough, P. R. 2007. Bovine Laminitis and Lameness. A Hands-On Approach. W. B. Saunders Company, Edinburgh, UK
- Hernández, J., J. K. Shearer, and D. W. Webb. 2001. Effect of lameness on the calving-toconception interval in dairy cows. JAVMA 218:1611–1614.
- Hirst, W. M., R. D. Murray, W. R. Ward, and N. P. French. 2002. A mixed-effects time-to-event analysis of the relationship between first-lactation lameness and subsequent lameness in dairy cows in the UK. Prev. Vet. Med. 54:191-201.
- Holzhauer, M., C. Hardenberg, C. J. Bartels, and K. Frankena. 2006. Herd- and cow-level prevalence of digital dermatitis in the Netherlands and associated risk factors. J. Dairy Sci. 89:580-588.
- Interbull. 2011. Description of national genetic evaluation systems (Canada). http://www-interbull.slu.se/national\_ges\_info2/framesida-ges.htm. Accessed July 31, 2012.
- Koenig, S., A. R. Sharifi, H. Wentrot, D. Landmann, M. Eise, and H. Simianer. 2005. Genetic parameters of claw and foot disorders estimated with logistic models. J. Dairy Sci. 88:3316 -3325.
- Madsen, P., and J. Jensen. 2008. An User's Guide to DMU. A package for analyzing multivariate mixed models. Version 6, release 4.7. Danish Institute of Agricultural Sciences, Tjele, Denmark.
- Manske, T., J. Hultgren, and C. Bergsten. 2002. Prevalence and interrelationships of hoof lesions and lameness in Swedish dairy cows. Prev. Vet. Med. 54:247-263.
- Meléndez, P., J. Bartolome, L. F. Archbald, and A. Donovan. 2003. The association between lameness, ovarian cysts and fertility in lactating dairy cows. Theriogenology 59:927–937.
- Negussie, E., Strandén, I. and Mäntysaari, E. A. 2008. Genetic analysis of liability to clinical mastitis, with somatic cell score and production traits using bivariate threshold-linear and linear-linear models. Livest. Sci. 117:52-59.
- Neuenschwander, T. F. O. 2010. Studies on disease resistance based on producer-recorded data in Canadian Holsteins. PhD thesis. University of Guelph, Guelph, Canada.
- Offer, J. E., D. McNulty, and D. N. Logue. 2000. Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. Vet. Rec. 147:105-109.
- Onyiro, O. M., L. J. Andrews, and S. Brotherstone. 2008. Genetic parameters for digital dermatitis and correlations with locomotion, production, fertility traits, and longevity in Holstein-Friesian dairy cows. J. Dairy Sci. 91:4037-4046.

- Onyiro, O. M., and S. Brotherstone. 2008. Genetic analysis of locomotion and associated conformation traits of Holstein-Friesian dairy cows managed in different housing systems. J. Dairy Sci. 91:322-328.
- Phillips, C. J. C. 2002. Cattle Behaviour and Welfare. 2nd ed. Blackwell Science Ltd., Oxford, UK.
- Rajala-Schultz, P. J., and Y. T. Gro"hn. 1999. Culling of dairy cows. I. Effects of diseases on culling in Finnish Ayrshire cows. Prev. Vet. Med. 41:195–208.
- Rodrigues-Lainz, A., P. Melendez-Rentamal, D. W. Hird, D. H. Read, and R. L. Walker. 1999. Farm- and host-level risk factors for papillomatous digital dermatitis in Chilean dairy cattle. Prev. Vet. Med. 42:87-97.
- Snell, E. J. 1964. A scaling procedure for ordered categorical data. Biometrics 20:592-607.
- Sogstad, A. M., T. Fjeldaas, O. Osteras, and K. P. Forshell. 2005. Prevalence of claw lesions in Norwegian dairy cattle housed in tie stalls and free stalls. Prev Vet Med 70:191-209.
- Somers, J. G., K. Frankena, E. N. Noordhuizen-Stassen, and J. H. Metz. 2003. Prevalence of claw disorders in Dutch dairy cows exposed to several floor systems. J. Dairy Sci. 86:2082-2093.
- Somers, J. G., K. Frankena, E. N. Noordhuizen-Stassen, and J. H. M. Metz. 2005. Risk factors for interdigital dermatitis and heel erosion in dairy cows kept in cubicle housing in The Netherlands. Prev. Vet. Med. 71:23–34.
- Swalve, H. H., H. Alkhoder, and R. Pijl. 2008. Estimates of breeding values for sires based on diagnoses recorded at hoof trimming: Relationships with EBV for conformation traits. Interbull Bull. 38:87-90.
- Uggla, E., J. H. Jakobsen, C. Bergsten, J.-A. Eriksson, and E. Strandberg. 2008. Genetic correlations between claw health and feet and leg conformation traits in Swedish dairy cows. Interbull Bull. 38:91-95.
- van der Linde, C., de Jong, G., Koenen, E.P.C., and Eding, H. 2010. Claw health index for Dutch dairy cattle based on claw trimming and conformation data. J. Dairy Sci. 93: 4883–4891.
- van der Waaij, E. H., M. Holzhauer, E. Ellen, C. Kamphuis, and G. de Jong. 2005. Genetic parameters for claw disorders in Dutch dairy cattle and correlations with conformation traits. J. Dairy Sci. 88:3672–3678.
- Whay, H. R., D. C. Main, L. E. Green, and A. J. Webster. 2003. Assessment of the welfare of dairy cattle using animal-based measurements: direct observations and investigation of farm records. Vet. Rec. 153:197-202.
- Warnick, L. D., D. Janssen, C. L. Guard, and Y. T. Grohn. 2001. The effect of lameness on milk production in dairy cows. J. Dairy Sci. 84:1988–1997.

Trait	Description of trait	Optimum	Records, n	Mean	SD
Foot angle	Angle of toe $(1 = \text{extremely low}, 9 = \text{extremely steep})$	7	11,419	5.5	1.3
Heel depth	Depth of heel on outside claw $(1 = \text{extremely shallow}, 9 = \text{extremely deep})$	7/8	11,419	5.5	1.1
Bone quality	Flatness of bone $(1 = \text{extremely coarse}, 9 = \text{extremely flat})$	8	11,419	6.0	1.4
Rear leg side	Degree of curvature viewed from side $(1 = \text{extremely straight}, 9 = \text{extremely})$	5	11 / 10	5 2	1 1
view	curved)	5	11,419	5.5	1.1
Rear leg rear	Turn of hock when viewed from rear $(1 = \text{extremely hocked- in}, 9 =$	Q	11 / 10	53	1 /
view	extremely straight)	)	11,417	5.5	1.7
Locomotion	1 = lame, $9 =$ even gait, long strides	9	6,966	5.3	1.4

Table 1. Description of conformation and locomotion traits in primiparous cows

Table 2. Percentage of hoof trimming records with hoof lesions including all records, and stratified by parity (n = 27,179 cows).

	All	Parity					
		1	2	3	4	5	$\geq 6$
Records (n)	34,905	11,793	9,772	6,510	3,693	1,774	1,363
				%			
Front lesion	7.0	7.2	6.1	6.4	8.1	7.7	9.9
Front infectious lesion <sup>1</sup>	2.1	2.7	1.8	1.8	1.9	2.0	2.1
Front horn lesion <sup>2</sup>	4.4	4.1	3.8	4.2	5.5	5.3	7.5
Front other lesion <sup>3</sup>	0.9	0.8	0.9	0.7	1.2	0.6	1.2
Rear lesion	34.5	31.0	35.4	35.5	36.6	41.3	38.2
Rear infectious lesion	22.6	23.8	25.3	22.2	18.4	16.6	14.2
Rear horn lesion	13.1	8.1	11.2	14.6	19.7	27.7	26.3
Rear other lesion	2.9	1.9	3.0	3.4	3.7	3.6	4.0
Any lesion	38.3	34.8	38.8	39.2	41.0	45.4	43.1

<sup>1</sup> Included digital and interdigital dermatitis, foot rot, and heel erosion <sup>2</sup> Included sole and toe ulcer, sole hemorrhage, and white line disease (separation and abscesses) <sup>3</sup> Included interdigital hyperplasia, fissures, thin soles and corkscrew claw

Table 3. Estimates of variances [additive genetic variance ( $\sigma_a^2$ ), permanent environmental variance ( $\sigma_{pe}^2$ ), residual variance ( $\sigma_e^2$ ), total variance ( $\sigma_p^2$ ); values were multiplied by 100] and heritability (h<sup>2</sup>; SE in parentheses) for hoof lesions (n = 34,905 records from 27,179 cows).

Model	Trait	$\sigma_a^2$	$\sigma_{pe}^2$	$\sigma_e^2$	$\sigma_p^2$	h²
Uni-variate model	Any lesion	1.487	2.14	16.128	19.755	0.075 (0.010)
Bi-variate model	Front lesion	0.085	1.218	4.887	6.19	0.015 (0.004)
	Rear lesion	1.509	2.406	15.246	19.161	0.079 (0.010)
Bi-variate model	Infectious lesion	1.393	1.765	11.926	15.084	0.092 (0.011)
	Horn lesion	0.555	2.205	9.536	12.296	0.045 (0.008)
Four-variate model	Front infectious lesion	0.039	0.394	1.556	1.989	0.020 (0.005)
	Front horn lesion	0.046	0.766	3.218	4.03	0.012 (0.004)
	Rear infectious lesion	1.308	1.878	11.61	14.796	0.089 (0.010)
	Rear horn lesion	0.470	2.029	7.875	10.374	0.045 (0.008)

Table 4. Estimates of genetic correlations between front infectious lesion, front horn lesion, rear infectious lesion and rear horn lesion from the 4-variate model (SE in parentheses) (n = 34,905 records from 27,179 cows).

Trait	Front horn lesion	Rear infectious	Rear horn lesion
Front infectious	0.113 (0.211)	0.771 (0.090)	-0.270 (0.143)
Front horn lesion		0.060 (0.162)	0.612 (0.158)
Rear infectious			0.078 (0.107)

Table 5. Estimates of genetic parameters for infectious lesion, horn lesion, conformation traits and locomotion from bivariate analyses: heritability ( $h^2$ ; SE in parentheses) and genetic correlation ( $r_g$ ; SE in parentheses) (n = 11,793 records for lesion traits, n = 11,419 records for conformation traits; n = 6,966 records for locomotion).

Trait	$h^2$	r <sub>g</sub>			
ITali	11	Infectious lesion	Horn lesion		
Infectious lesion	0.076 (0.017)				
Horn lesion	0.028 (0.011)	0.153 (0.225)			
Foot angle	0.050 (0.015)	-0.182 (0.198)	0.217 (0.245)		
Heel depth	0.070 (0.018)	-0.051 (0.186)	-0.124 (0.240)		
Bone quality	0.223 (0.026)	0.222 (0.137)	-0.131 (0.182)		
Rear leg side view	0.117 (0.022)	0.267 (0.162)	-0.077 (0.215)		
Rear leg rear view	0.041 (0.014)	-0.394 (0.198)	-0.372 (0.255)		
Locomotion	0.029 (0.015)	-0.464 (0.242)	-0.352 (0.329)		



Figure 1. Percentage of hoof trimming records recorded at each stage of lactation (n = 34,905 records from 27,179 cows).

Figure 2. Percentage of hoof trimming records with any lesion (dashed line), infectious lesions (black line) and horn lesions (grey line) in at least one hoof at each stage of lactation (n = 34,905 records from 27,179 cows).



Stage of lactation (days after calving)

Figure 3. Distribution of infectious (black line) and horn lesions (grey line) by score of conformation traits and locomotion in primiparous cows (n = 11,793 records for lesion traits, n = 6,966 records for locomotion, n = 11,419 records for all other conformation traits). Black squares in the x- axis indicate the optimal score.



14









F)



16

Figure 4. Relationship between sire relative breeding value (RBV) for resistance to the occurrence of any lesion and percentage of healthy daughters (no hoof lesion recorded at the first hoof trimming of the lactation) (n=297 sires with at least 20 daughters).

