STUDIES ON DISEASE RESISTANCE BASED ON PRODUCER-RECORDED DATA IN CANADIAN HOLSTEIN CATTLE

A Thesis

Presented to

The Faculty of Graduate Studies

of

The University of Guelph

by

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In partial fulfilment of requirements

for the degree of

Doctor of Philosophy

December 2009

Your file Votre référence
ISBN: 978-0-494-64498-0
Our file Notre référence
ISBN: 978-0-494-64498-0

ABSTRACT

STUDIES ON DISEASE RESISTANCE BASED ON PRODUCER-RECORDED DATA IN CANADIAN HOLSTEIN CATTLE

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University of Guelph, 2010

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Health traits are some of the most important cost factors in dairy cattle production. Eight important health traits were chosen for data collection in Canada. They were mastitis, lameness, cystic ovarian disease, left displaced abomasum, ketosis, metritis, milk fever, and retained placenta. Data collected by producers on these 8 diseases were stored in a central database. These recordings were the basis to prepare genetic evaluations for health in Canada.

Effect of the quality of the data was analyzed by using 2 different sampling frames for the inclusion of herds in the analysis: a stringent sampling frame requiring all herds to have collected at least one case of the disease analyzed and a second sampling frame requiring herds to have collected one case of any disease. Variance components were estimated with a linear model. Heritability estimates of all health traits were lower than 0.03. The second sampling frame gave lower estimates than the first one. Correlations between predicted transmitted abilities (**PTA**) calculated with both sampling frames were higher than 0.9.

A second analysis compared the effects of using a threshold model instead of a linear model. Health traits were also grouped according to biological aspects. Heritability estimates calculated with the threshold model were higher than those of the linear model, but when they were transformed to the observable scale, results from both modelling approaches were similar.

Use of indicator traits was investigated in analyzing body condition score (**BCS**) and health traits simultaneously. A longitudinal and a multiple-trait approach were used. BCS was positively correlated with resistance to disease, except for lameness, where a negative correlation was found. Heritability of BCS was moderate and selection for this trait would improve disease resistance.

Finally, a survey was sent to producers to assess data collection practice. Most of the producers collecting health data were collecting data on mastitis. On the other hand, only 50% of producers collected data on lameness, cystic ovarian disease, ketosis or metritis. Awareness for health data collection should be raised through extension work.

Acknowledgements

How to summarize in a few words, three years in Canada, three years of work in a graduate program? There would be so many people to thank, that I will limit myself to those directly involved in the thesis. But first I must say that what attracts me in animal breeding are the animals themselves and more specifically the "Canadian Holsteins". After three years in Canada they haven't disappointed me. I still love this breed and I still love these cows... so much that I have decided to own two of them and leave them in that great cow country.

For the actual work on the thesis, I would like to thank my advisor, Larry Schaeffer. He provided the opportunity to come to Guelph, in the heart of Canada's dairy counties; he also brought the idea of the thesis and his wealth of knowledge to this work.

I'm also very thankful to the other members of my advisory committee: Filippo Miglior, for often bringing new directions of research, for encouraging me to continue researching and for making the link to the industry; Janusz Jamrozik for going so often through the details of the thesis, working through equations and computer programs and helping me find the aspects needing improvement; Dave Kelton and Olaf Berke for giving me the "other perspectives" – the veterinarian and statistician perspectives. I know that it was not always easy to deal with the animal breeders and some of their "inbred thoughts" about statistical models.

For her thorough reading of the manuscript, I would like to thank Jayne Redekop. Besides helping with the thesis, she also provided, along with her husband Jim and their children, a family for my time in Canada. I also want to mention Ed and John Bos, from Bosdale Farms Inc. and Marc-André and Stéphanie Raynaud, from La Ferme Raynaud Ltée, for giving me the connection to the reality of Holstein breeding... far away from the numbers and the statistical models!

And finally, I would like to thank my Lord and Saviour Jesus Christ. So much of what happened in these three years in Canada depended on him. But foremost, I would like to thank him for giving a direction to my life. Without him, it wouldn't be worth much.

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Chapter 1

General Introduction

T. F.-O. Neuenschwander

Most of the genetic improvement in dairy cattle in the past decades has been focused on production and conformation traits. Since the 1990s, a greater emphasis of selection has been put on functional traits. Udder health was one of the first traits selected for in the form of somatic cell count. Herd life has been used as a measure of the ability of the cow to remain in the herd. Fertility and reproduction have recently been added in selection for many dairy populations. Traits that are still missing from formalized breeding programmes in most countries are traits for disease resistance. The reason for this is usually a lack of data. Only the 4 Nordic countries, Denmark, Norway, Sweden and Finland, have health data recording since 1975 on a national scale. By law, all veterinary treatments have been recorded in the national database of these countries. Other countries have also shown interest in having a national dairy cattle health database, but very few have made progress in this direction.

In Canada, a project started in 2005 to put in place a national health database for dairy cattle. Recording of diseases started in Canada in April 2007. Recording in this database is done by producers, except in Quebec where it is partly done by veterinarians. A total of 8 diseases were selected for recording. They were mastitis, lameness, cystic ovarian disease, left displaced abomasum, ketosis, metritis / uterine disease, milk fever and retained placenta. A description of each of the 8 diseases was given to producers, as a guideline to identify diseased cows. The goal of recording health events in a central database in Canada is twofold, national incidence of diseases and trends might be monitored based on these data. As well, health data can be used for genetic evaluation purposes. As the central database raises awareness for health recording, it can encourage

more producers to collect data and therefore improve their management practices on a farm level.

The present project was to perform preliminary research to pave the way towards genetic evaluations for sires for health traits in Canada. The first part is a review of the 8 health traits collected in the Canadian dataset. Aetiology of these diseases, relationships to other diseases and genetic research already completed in other countries were reviewed.

Because producers record diseases on a voluntary basis without incentive for accurate recording, the quality of recording might vary from producer to producer. Not all data may be usable and sampling might have an effect on the estimates of genetic parameters and genetic evaluations. One part of the study was aimed at measuring the effect of sampling on inferences.

Health records are binary traits. Genetic analysis of binary traits is often performed using threshold models to describe more appropriately the specifics of this kind of traits. Health data from Canada were therefore analysed with both a linear model and a threshold model to compare the advantages and disadvantages of the 2 methods.

As the amount of health data gathered since the outset of the national disease recording program was limited, use of another correlated trait with more observations or more precise definition could be helpful. Body condition score (**BCS**) is a trait describing the amount of fat reserves in a cow and can be used as an indicator of metabolic balance. The correlation between BCS and health traits was analyzed in one part of this study.

Finally, recording practices of dairy producers in their herds were analysed based on a survey. The survey was intended to quantify the knowledge of the breeders

about health traits and to determine their specific areas of concern as to accurate recording.

All of these aspects were studied to improve our knowledge of dairy cattle health traits in Canada in preparation for the genetic evaluations of health traits.

Chapter 2

A Review of Eight Production-Related Diseases: Aetiology, Pathogenesis, Epidemiology and Genetic Evaluations

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INTRODUCTION

As milk production of dairy cows continues to increase, health and fertility of these animals have become a major concern of the dairy industry. Many countries have already shifted their selection goals to put more emphasis on "health and reproduction" (Miglior et al., 2005). However, this group of traits rarely includes direct disease information. Traits selecected generally include calving ease, fertility and udder health traits, where udder health is generally somatic cell score (**SCS**). Notable exceptions are the Nordic countries, where treatment data have been recorded for up to 30 years and are included in the breeding goal (Osteras et al., 2007; Steine et al., 2008). A precise definition of disease and accurate recording are 2 important prerequisites for the inclusion of disease resistance in breeding programmes. In Canada, 8 diseases were defined by Kelton et al. (1998) according to the following criteria: the traits had to be recorded in farm management systems, they had an economic significance, their median frequency of occurence was above 5 percent and the disease was clinically manifested, so that the trait can be identified clearly by simple observation. Since April 2007, these traits are being recorded on a voluntary basis in a central national database for use in management decisions and in the near future, for genetic evaluations. The data recording is done by the producers. The data are transmitted to the central database, by the Dairy Herd Improvement representative or by a veterinarian (in Quebec).

The objective of the present review is to describe all 8 diseases recorded in the Canadian National Health Project (**CNHP**) and to present the causality relationships with other traits as well as some preliminary results from a genetic analysis. This review will identify the way these diseases could be included in genetic evaluations rather than

review each disease in-depth. Most of these 8 diseases occur during the peripartum period (Ingvartsen, 2006). The increase in milk yield per day is highest after calving and is closely correlated to the general disease incidence. The median time of first occurrence of diseases is generally during the first 2 months after parturition (Bigras-Poulin et al., 1990); therefore, disease incidence has to be considered in relation to the time of occurrence as will be done in this review.

Genetic analyses have already been undertaken for many traits using many different datasets. A list of studies is presented in Tables 1 and 2, with the result of the evaluations in Table 3. Details about each trait will be presented in the corresponding section. The analyses were generally made on relatively small datasets (less than 50,000 animals). Models used were generally simple sire models. Applications to routine evaluations have only been done in the Nordic countries. In Norway, where the breeding program is for the Norwegian Red Cattle, mastitis (**MAST**) accounts for 22% of the total merit index, and "other diseases", which includes ketosis (**KET**), milk fever (**MF**) and retained placenta (**RP**) accounts for 3% (Steine et al., 2008). In Denmark, the weight is 14% on MAST and 2% on "other diseases" for Holsteins. Both countries use sire models.

MASTITIS

Definition of the Disease. Mastitis is an inflammation of the mammary gland and is one of the most costly diseases in the dairy industry by reason of its incidence and loss of milk production per case (Kelton et al., 1998). Mastitis can be caused by a large number of pathogens. A recent study in Canada showed that the main pathogens present in milk from mastitic udders were *Staphylococcus aureus*, *Escherichia coli* and

Streptococcus uberis (Olde Riekerink et al., 2008). The 1st pathogen is highly contagious, whereas the other 2 are transmitted through the environment (e.g. manure, bedding). Responses to pathogen contamination are the production of abnormal milk (clots, flakes or watery), swelling or pain in the udder as well as systemic signs like fever or anorexia (Harmon, 1994). Production of abnormal milk is the clinical sign used for the definition of MAST (Kelton et al., 1998). Pathogens are transmitted from cow to cow during milking, post milking disinfection (Oura et al., 2002) or through the environment (bedding, manure, etc.); they enter into the mammary gland through the teat canal. The magnitude of the inflammatory response is controlled by many factors including genetics (Harmon, 1994). One of the main elements of the inflammatory response is the entrance into the mammary gland of polymorphonuclear neutrophil (PMN) leukocytes. This is measured as the somatic cell count (**SCC**) and is one of the main indicator traits for clinical mastitis (Harmon, 1994). For genetic analyses, SCC is log-transformed to obtain a normal distribution. This new value is called somatic cell score (**SCS**). The function of the leukocytes is to digest bacteria. Leukocytes stay in the mammary gland until it is healed; this process varies in length, depending on the pathogen, and can take from a few days to many weeks (Harmon, 1994).

Incidence and Prevalence. Mastitis is a problem that occurs throughout the lactation, although the risk is higher during the early postpartum period (Bigras-Poulin et al., 1990). In a review of 62 citations, Kelton et al. (1998) reported a median lactation incidence of 14.2%. Incidence of MAST was reported at 9.8, 12.9 and 14.6% in 1st, 2nd and 3rd lactation respectively (Lin et al., 1989). Bradley et al. (2007) reported a very high herd incidence of 65 cases per 100 cows and per year. Bigras-Poulin et al. (1990) also

found a high herd incidence (36.9%) with a lactation incidence of 24.2%. Moreover 1 out of 4 cows having a case of MAST during lactation had a 2nd one during the same lactation. An analysis in Sweden showed that the incidence of MAST was highest between 51 and 250 DIM, a little lower between 1 and 50 DIM and still lower at the end of the lactation (Hagnestam et al., 2007).

Relationships to Other Traits. There is a positive genetic correlation between MAST and SCS (Heringstad et al., 2000; Zwald et al., 2004b). As the second trait is routinely recorded by DHI programs, many countries use SCS in their breeding program to improve MAST resistance through correlated response (Miglior et al., 2005). Positive genetic correlations were also reported between MAST and dystocia, RP and metritis (**MET**; Lin et al., 1989). Zwald et al. (2004b) found no genetic correlation between MET and MAST. A moderate positive genetic correlation was reported between lameness and MAST (Kadarmideen et al., 2000; Zwald et al., 2004b). Heringstad et al. (2005) reported a moderate positive correlation between KET and MAST in 1st lactation. There is a moderate positive genetic correlation between production traits and MAST (Uribe et al., 1995; Kadarmideen et al., 2000). The correlation between MAST and somatic cell (SCC or somatic cell score) varied from .23 to .8 (Heringstad et al., 2000; Zwald et al., 2004b). There is a negative genetic correlation between udder depth and MAST (Zwald et al., 2004b). All these correlations clearly point to a general genetic predisposition to disease resistance.

Models and Parameters for Genetic Analyses. In a review of the estimates of heritability of MAST in the Nordic countries Heringstad et al. (2000) reported values between 0.001 and 0.06 when based on linear models, and between 0.06 and 0.12 when

based on a threshold model. Using designed field studies, higher heritabilities were found by Lin et al. (1989; 0.19, 0.31 and 0.18 for MAST in 1st, 2nd and 3rd lactation, respectively) and by Uribe et al. (1995; 0.15 for MAST in 1st lactation cows). Uribe et al. (1995) reported that the heritability dropped to 0 when all cows were included in the evaluation. Similar results were found by Nash et al. (2000). Van Dorp et al. (1998) reported a heritability of 0.04; they used an animal model. Analysing data with both threshold sire and linear animal models, Kadarmideen et al. (2000) found heritabilities of 0.13 and 0.04 respectively. Heritabilities in field studies were reported as high as 0.16 (and even 0.31 by Lin et al., 1989; but heritabilities in this study were always very high compared to other health traits studies). In large dataset and routine genetic evaluations, values were lower. In a longitudinal model, Heringstad et al. (2003a) calculated heritabilities for 11 30-days periods of the lactation ranging between 0.04 and 0.09, but for the last 30-days period the heritability was as high as 0.41.

LAMENESS

Definition of the Disease. Lameness (**LAME**) was defined as an abnormal gait due to either a leg or a foot problem (Kelton et al., 1998). Lameness is generally related to problems of the foot rather than the leg (Clarkson et al., 1996). Most cases of lameness are in the hind limbs, where the highest incidence of lesions is also found (Murray et al., 1996; Manske et al., 2002, Cramer et al., 2008). Murray et al. (1996) found that lesions associated with LAME were mostly in the outer claw (65.4%), followed by lesions in the skin (20.2%) and lesions in the inner claw (14.4%). This is due to the greater weight borne by that claw (van der Tol et al., 2002). In Ontario tie-stall herds, the main lesions of

the claw leading to LAME were digital dermatitis, heel horn erosion and hemorrhage (Cramer et al., 2008). Besides these 3 lesions, sole ulcers were also important in free-stall herds. Sole ulcer usually includes heel ulcer (caudal side of the hoof) and actual sole ulcer (central part of the hoof; Blowey et al., 2000). There is a genetic relationship between high milk yield and lameness (Kadarmideen et al., 2000) and the fact of having to stand longer to eat and the need for higher energetic rations may have an effect. The most prevalent diseases in Ontario Holsteins are infectious lesions, in both free-stall and tie-stall housing systems (Cramer et al., 2008). Digital dermatitis, an infectious lesion, was the most common lesion overall. Its prevalence increases when animals are kept in moisture and manure. Besides infections, another important cause of lameness is related to feeding. If the ration has a high proportion of concentrates, the ruminal pH decreases, systematic mediators are released in the blood causing a pathological response in the vessels. This causes oedema and increases the pressure in the corium. As the corium is confined between the hoof and the bone of the 3rd phalanx, it causes pain for the animal. As the irrigation of the corium is not optimal anymore, the horn and the dermal-epidermal junction deteriorate. This deterioration causes the foot to sink in the horn capsule and is called "subclinical laminitis". This situation and the pressure put on the corium lead to haemorrhages and ultimately lesions in the horn. Time-lag between the start of laminitis and the formation of lesions is at least 6 weeks. The sole ulcer is a perforation of the horn layer, caused by a hindrance in horn production following the tissue necrosis in the corium. The location of the sole ulcer depends on the place where the corium is compressed. White line lesion is a widening of the laminar zone following an accumulation of blood or cell debris in the dermal-epidermal junction (Ossent and

Lischer, 1998; Collard et al., 2000). Sole ulcers are also the cause of LAME that leads to the biggest drop in production. Warnick et al. (2001) reported a drop of up to 2.8 kg milk per day, 3 weeks after LAME was diagnosed. Green et al. (2002) found that the effective yield of lame cows is 357 kg per lactation below their potential; however their actual milk yield is higher than cows that are not lame, showing the relationship between high production and LAME.

Incidence and Prevalence. In a review of 39 studies made between 1972 and 1995, Kelton et al. (1998) found a median incidence of 7.0%. The incidence among farms is significantly different (Barkema et al., 1994; Clarkson et al., 1996). Cramer et al. (2008) reported a LAME prevalence of 25.7% in 142 tie-stall herds in Ontario, and 46.8% in 38 free-stall herds of the same province. Half of the cows with a case of LAME will have a 2nd one in the following 6 months (Clarkson et al., 1996). The incidence of LAME related lesions is higher when animals are housed instead of being let out to pasture (Murray et al., 1996), this incidence is also higher in free-stall barns than in tie-stalls (Cramer et al., 2008). Lesions do not always lead to LAME. A study reported LAME and lesion median prevalence of 4% and 70% respectively (Manske et al., 2002); nevertheless, the prevalence of lesions was higher in lame cows than in healthy cows. They also reported that the lesion causing the highest risk of LAME is sole ulcer (OR = 6.02). Prevalence of lesions in primiparous cows is at the highest, between the 1st and the 9th week after calving for the white line and between the 9th and the 20th week for sole lesions (Barkema et al., 1994; Leach et al., 1997; Green et al., 2002).

Relationships to Other Traits. Hirst et al. (2002) reported an effect of LAME in first lactation on that same disease in later lactations. They used a sire model to test the

data; therefore part of the variance explained by first lactation incidence might be confounded by the animal genetic effect. Cows with a case of LAME in the first 30 days of lactation have been reported 2.63 times more likely to develop cystic ovarian disease (**COD**; Melendez et al., 2003). A hypothesis for this relationship is based on the fact that most LAME cases are caused by laminitis. Acidosis in dairy cattle has an effect on laminitis through the release of histamine and endotoxin caused by this disease (Nocek, 1997). Endotoxins also have an effect on luteal activity and cystic follicle formation. A second hypothesis is that the pain induced by LAME results in an increase of the cortisol and progesterone levels altering the normal follicle activity. Finally, lame cows are often in negative energy balance. This is known to have an inhibitory effect on ovarian follicular growth and development (Melendez et al., 2003). There was also a moderate positive genetic correlation of LAME with protein and fat yield as well as MAST (Kadarmideen et al., 2000). Cramer et al. (2009) also reported a positive relationship between lesions and culling rate of different cows.

Models and Parameters for Genetic Analyses. Analyses for lameness reported low heritabilities. Generally, large producer-recorded disease datasets, gave very low estimates of heritability (<0.08; Kadarmideen et al., 2000; Zwald et al., 2004a, 2004b). This is caused by the difficulty of observing and reporting LAME accurately. This general problem of health traits is more acute when, as for LAME, observation is based on a scoring system dependent on subjectivity of the scorer. Not all studies presented in Table 2, used the same definition for LAME. Lyons et al. (1991) reported heritabilities of 0.08 and 0.11 for leg problems and foot problems respectively. Uribe et al. (1995)

reported a 0.15 heritability for "culling for leg problems". Heritability of LAME was reported by van Dorp et al. (1998) at 0.16.

CYSTIC OVARIAN DISEASE

Definition of the Disease. Ovarian cysts are defined as follicular structures with a diameter of at least 2.5 cm on an ovary in the absence of a corpus luteum and uterine tone (Kelton et al., 1998; Peter, 2004). This pathological state is often called cystic ovarian disease. An anovulatory cystic structure develops when ovulation does not occur after maturation of a dominant follicle and the follicle does not regress afterwards (Peter, 2004; Vanholder et al., 2006). Pathological cysts can be divided into 2 types: follicular and luteal cysts. They differ in the thickness of their wall, as follicular cysts have thinner walls (< 3 mm; Kesler and Garverick, 1982; Vanholder et al., 2006). Luteal cysts secrete progesterone, whereas follicular cysts rarely do. Ovarian cysts develop mainly in early lactation (Vanholder et al., 2006). Aetiology is still poorly understood, but it is believed that a neuroendocrine imbalance causes it. The most widely accepted hypothesis is a deficiency in LH release or a dysfunction in the gonadotropin releasing hormone (GnRH) – LH axis (Kesler and Garverick, 1982; Peter, 2004; Vanholder et al., 2006). The pre-ovulatory LH-surge does not occur appropriately. This seems to be caused by a deficient feedback mechanism of oestrogens. Progesterone must reach the hypothalamus to reset that mechanism (Vanholder et al., 2006).

Incidence and Prevalence. The incidence of ovarian cysts was reported between 1.0 and 18.8% (Kesler and Garverick, 1982; Kelton et al., 1998) with a median incidence of 8.0%. This value is underestimated as spontaneaous recovery rate was reported as high

as 40% (López-Gatius et al., 2002). Although animals recover, the cystic condition renders the period open longer. Kesler et al. (1979) reported a delay in the 1st ovulation of 18 days for cystic cows. There is an increased incidence with parity (Laporte et al., 1994) however, as COD does not occur at every lactation or at every cycle, promoters must be responsible for providing the conditions necessary to start a new case.

Relationships to Other Traits. Early postpartum problems such as RP and MET play a role in the development of COD (Bigras-Poulin et al., 1990; López-Gatius et al., 2002). Cows with RP had twice the risk of developing ovarian cysts as cows with normal placenta expulsion. However, Lin et al. (1989) found a negative genetic correlation between COD and retained placenta for animals in 2nd lactation. Odds ratio of change of Body Condition Score during the dry period on COD is very high (López-Gatius et al., 2002). This result signifies that cows suffering a loss in body condition score (**BCS**) during the period prepartum are at a higher risk of COD in early lactation. A high milk production is also related to a higher incidence of COD (van Dorp et al., 1998). High daily milk production is a cause of COD (Laporte et al., 1994). The actual cause of COD might be a negative energy balance, which is often found together with a high milk yield (Vanholder et al., 2006). On the other hand COD increases lactation milk production (Hooijer et al., 2001) as the non-pregnant cow has a longer lactation and therefore a better persistency. There is no difference in daily milk production for the whole lactation between cystic and non-cystic cows.

Models and Parameters for Genetic Analyses. All estimates of heritability for COD were 0.1 or lower, with the exception of the result from Lin et al. (1989). Hooijer et al. (2001) reported one of the highest estimates, using a threshold animal model. The

dataset used in this study was from the recordings of a veterinary practice in a small area during a 10 year period; the dataset was therefore very accurate. Using a threshold model, Hooijer et al. (2001) found a heritability of 0.10 on the underlying scale; when an incidence of 7.7% was assumed, a heritability of 0.09 was reported on the observable scale. The same data analyzed with a linear model, resulted in a heritability of 0.03. The difference in heritability is usual for data with low incidence. Several studies reported a higher heritability for COD in 1st lactation compared to later ones (Lin et al., 1989; Uribe et al., 1995; Zwald et al., 2004a).

LEFT DISPLACED ABOMASUM

Definition of the Disease. The abomasum is normally located under the rumen and across the ventral midline, and is attached to the omasum and to the reticulum (Baird and Harrison, 2001). Left displaced abomasum (**LDA**) is a dislocation of the abomasum under the rumen and to the left along the body wall. This dislocation partly blocks the passage of abomasum outflow to the duodenum (Baird and Harrison, 2001; Ingvartsen, 2006). The space available in the abdominal cavity is one of the factors involved in the disease. After parturition, rumen might not immediately recover its position on the left abdominal floor; moreover, feed intake in the 1st weeks postpartum is often reduced. As a result rumen is less filled and leaves room in the abdominal cavity allowing the abomasum to slide to the left (van Winden et al., 2003). Another factor is gas in the abomasum. Postpartum cows have a higher pH in the abomasum; it allows the rumen flora to continue fermentation and to produce gas leading to LDA (van Winden et al., 2003). A 3rd factor is the motility. A low calcium concentration in the plasma leads to

hypomotility of the abomasum (van Winden and Kuiper, 2003). Right displaced abomasum occurs at a lower rate, 80 to 90% of the cases of displaced abomasum are on the left side (Shaver, 1997), and is therefore not included in the CNHP. Diagnosis of LDA is a decreased appetite and an audible, high pitched tympanic resonance produced by percussion of the left abdominal wall between the 9th and the 12th ribs (Kelton et al., 1998).

Incidence and Prevalence. More than 50% of the LDA cases occur during the first 2 weeks postpartum (Stengärde and Pehrson, 2002) and close to 90% in the 1st month of lactation (Shaver, 1997). Reported incidence was between 0.2 and 10.0% with a median incidence of 1.7% (Kelton et al., 1998; Ingvartsen et al., 2003). A median herd lactation incidence risk of 3.1% was reported in Ontario herds (McLaren et al., 2006). A lower incidence was reported for primiparous cows (Stengärde and Pehrson, 2002). Individual herd incidences can be higher than 20% (Shaver 1997).

Relationships to Other Traits. Low feed intake is an important factor for LDA occurrence. Parturition related problems (MF, dystocia, twin births) have a positive effect on LDA incidence (Odds ratios of 2.3 after exposure to one of the 2 diseases, Correa et al., 1993). Metritis and RP also showed an effect on LDA (LeBlanc et al., 2005). Hypocalcaemia reduces the tonus of the abomasum, rendering it more susceptible to displacement (Stengärde and Pehrson, 2002; van Winden and Kuiper, 2003); but LeBlanc et al. (2005) report that it might only be due to the confounding effect of inadequate feed intake prepartum. Reduced feed intake prepartum is responsible for a small rumen size; after parturition, the lack of pressure of the uterus against the rumen predisposes the cow to displacement of the abomasum (Stengärde and Pehrson, 2002). Cows with KET had at

least 10 times more probability of having LDA than cows without KET (Correa et al., 1993; Stengärde and Pehrson, 2002). Corroborating this point, LeBlanc et al. (2005) found an effect of high plasma non-esterified fatty acids (**NEFA**) on LDA incidence. The higher plasma NEFA is already present in the cow prepartum. Therefore a condition where fat tissues are already mobilised leads to a LDA after parturition. Other authors mentioned that the reduced appetite of cows with LDA brings secondary KET and therefore wouldn't be related to KET as such (Stengärde and Pehrson, 2002). Genetic correlation between LDA and KET is moderate and positive (Zwald et al., 2004b). There is a moderate positive genetic correlation between fat percentage and LDA (Uribe et al., 1995). There is also an effect of parity on LDA; cows in later parities tend to have more cases of LDA, than primiparous cows (Abdel-Azim et al., 2005) although LeBlanc et al (2005) reported a higher incidence of LDA in 1st lactation compared to 2nd lactation. The incidence then increased starting with the 3rd lactation. Abdel-Azim et al. (2005) also found a positive correlation between LDA and MAST.

Models and Parameters for Genetic Analyses. Few studies have estimated the variance components of LDA. Those who did generally found a heritability around 0.1 to 0.2, when a sire model was used. Uribe et al. (1995) reported a higher heritability but their dataset was relatively small. No genetic effect was found by van Dorp et al. (1998) for LDA. The incidence rate in this study was very low and a linear model was used. A study using both left and right displaced abomasum found a heritability of 0.05 for LDA but heritability for right displaced abomasum was not different from 0 (Hamann et al., 2004).

KETOSIS

Definition of the Disease. After parturitions, dairy cows have to adapt to a new physiological state (homeorhesis); this is characterized by endocrine and metabolic adaptations. Milk synthesis requires more energy and nutrients (particularly calcium and glucose) than pregnancy. Glucose demand is twice as high for cows 3 weeks postpartum than for cows at the end of pregnancy (Drackley et al., 2001). As cows in early lactation do not adapt immediately to the increased demand of energy by an increased dry matter intake, they often show a negative energy balance. The increase in nutrient available for milk synthesis is only provided by an increase in metabolic activity and mobilisation of body resources. During this period NEFA, a source of energy for the cow (Ingvartsen, 2006), are mobilised from the adipose tissues. As a result, fat is transported to the liver, where it is oxidised or re-esterified. Several syndromes result from inability to cope with homeorhetical changes (Hayirli and Grummer, 2004). The Fatty Liver Syndrome is an exaggerated fat mobilisation; there is a high fat content in the liver. The symptoms are a loss of appetite and a rapid loss of fat reserves. Fatty liver is a reversible condition, but when the disease continues it becomes subclinical or clinical ketosis. This disease is characterized by high blood concentrations of ketone bodies. A low glucose level (hypoglycaemia) is normally present for the clinical form of the disease. Ketosis occurs when the gluconeogenesis is not enough to answer the glucose demand. The low level of glucose will activate the gluconeogenesis, but as glucogenic substances (propionate, glucogenic amino acids, lactate and glycerol) are limited, ketogenesis will happen instead leading to increase of ketone body concentration. A loss of appetite is observed, ruminal movements are less frequent and there is an acetone smell to the breath (Andrews, 1998).

As similar signs occur after many periparturient diseases, KET is only diagnosed when no other disease is recorded simultaneously (Andrews, 1998; Kelton et al., 1998).

Incidence and Prevalence. Ketosis has an incidence reported between 1.3 and 20.0%, with a median of 4.8% (Bigras-Poulin et al., 1990; Emanuelson et al., 1993; Kelton et al., 1998; Gillund et al., 2001). McLaren et al. (2006) reported a lower median incidence of 1.0%, but these authors reported a high incidence (>50%) of sub-clinical mastitis in the first two weeks of lactation. Rasmussen et al. (1999) showed that the incidence is very low in 1st lactation (0.6%), increases rapidly in 2nd lactation (4.1%) and then increases slowly until the 4th lactation. The same was reported by Gillund et al. (2001) and Ingvartsen (2006). Ketosis in a previous lactation has an important impact on KET in following lactations (2.4 times higher probability of having it). The median time to 1st occurrence is 18 days (Bigras-Poulin et al., 1990); KET rarely occurs after the 1st month of lactation (Ingvartsen, 2006).

Relationships to Other Traits. High BCS at parturition is also a risk factor for KET (Rasmussen et al., 1999; Gillund et al., 2001). When BCS is above 3.5, the risk of KET is doubled. A positive genetic correlation was found between KET and milk production (Simianer et al., 1991; Uribe et al., 1995) and a negative one between KET and milk component deviation (-0.38 with fat, -0.65 with protein; Simianer et al., 1991). Uribe et al. (1995) found a similar correlation with protein but reported a moderate positive genetic correlation with fat percentage. When comparing KET with protein deviation in the previous lactation, Rasmussen et al. (1999) also found a negative effect. There is a detrimental effect of KET on milk components as nutrients are lacking for the synthesis of these substances. A cow with an average protein content of 3.0% during a

given lactation had a 2.5 times higher risk of having KET in the following lactation; protein content is related to the energy available to the cow. Cows with low protein content have a deficit in energy. Based on the findings of Rasmusssen et al. (1999), there seems to be a genetic or at least a permanent environmental effect relating KET to protein deviation. Odds ratio for the effect of MF on KET was reported by Correa et al. (1993) at 2.4 (CI: 1.5 – 4.0); the genetic correlation between these 2 traits was positive and moderate (Heringstad et al., 2005). Moderate positive correlations between KET and MAST in 1st lactation were also reported in this study.

Models and Parameters for Genetic Analyses. Many studies have analyzed KET. Sire models were used in the majority of the cases; Kadarmideen et al. (2000) used a linear animal model and found a very low heritability, but the incidence of the disease was very low (0.2%). Analysing the same dataset with a single trait threshold sire model, they found a slightly higher heritability. Other studies, using threshold or linear models, found values in the range 0.06 to 0.16. Zwald et al. (2004b) found a positive genetic correlation between KET and LDA, as well as COD. All 3 diseases are influenced by a negative energy balance, showing that this problem might be a common cause of many diseases. Simianer et al. (1991) found a heritability of 0.08 with a threshold model, but concluded that these values were probably overestimated. A multivariate threshold model calculated values for 4 different diseases and considering occurrence in each of the first 3 lactations as distinct traits (giving a total of 12 traits) found values for the heritability of KET of 0.14, 0.16, and 0.15 for 1st, 2nd and 3rd lactation respectively (Heringstad et al., 2005). Genetic correlations among lactations were comprised between 0.77 and 0.86; KET seems to be partly controlled by different genetic effect among lactations.

Heritability for mean plasma acetoacetate level was estimated at 0.11 (Tveit et al., 1992), whereas heritability for acetone level in the milk was very low (<0.01; Wood et al., 2004).

UTERINE DISEASE

Definition of the Disease. Uterine disease, often called metritis, is an inflammation of the uterus. Most inflammatory conditions are caused by bacterial contamination of the uterus (Sheldon et al., 2006). The highest period of incidence is the first month postpartum (Bigras-Poulin et al., 1990). Immunosuppressive effects of progesterone are active during the peripartum period which leads to a higher contamination and infection rate during this period; moreover during and following parturition, the uterus is at a higher risk of contamination through the open birth canal. An impaired contractility of the uterus also increases the risk of contamination, as the uterine content (lochia) is not removed (Földi et al., 2006). Involution of the uterus takes approximately 40 to 50 days. Inflammation of the uterus complex includes 3 diseases: puerperal metritis, endometritis and pyometra. Sheldon et al. (2006) proposed a definition for all 3 diseases. Puerperal metritis generally occurs at the end of the 1st week postpartum and is an infection of all layers of the uterus wall (Földi et al., 2006). Clinical signs are a fetid red-brown watery uterine discharge, signs of systemic illness and usually fever. This disease is an acute putrid inflammatory disease caused by a massive bacterial infection of the uterus (Földi et al., 2006), which causes extended damage to the epithelium and can touch the entire thickness of the uterine wall. Puerperal metritis is only diagnosed in cows in the first 21 days postpartum, as uterine discharges have been

normally expelled before that time (Hoedemaker, 1998; Sheldon et al., 2006). Clinical endometritis is defined as a purulent or mucopurulent uterine discharge in the vagina without systemic signs and occurring after the 3rd week after parturition. The cervix is generally still open and has a diameter >7.5 cm. Only the endometrium is inflamed. When endometritis occurs after the closure of the cervix, pyometria ensues (Földi et al., 2006). This disease is characterized by an accumulation of purulent material in the uterus while a corpus luteum is present on the ovaries (Sheldon et al., 2006). The definition for the CNHP includes all 3 diseases.

Incidence and Prevalence. A review of 43 publications reported a median MET incidence at 10.1% without a precise definition of the diseases. The values ranged from 2.2 to 37.3% (Kelton et al., 1998). In a large dairy operation, Benzaquen et al. (2007) found a 21% incidence for puerperal metritis and 24% incidence for clinical endometritis. Sheldon et al. (2006) reported a 16.9% incidence of endometritis. Defining metritis as puerperal metritis, endometritis and pyometra, Lin et al. (1989) found 75% of the cases before 31, 35 and 39 DIM for 1st, 2nd and 3rd respectively.

Relationships to Other Traits. Gilbert et al. (2005) showed that cows with a case of endometritis were open for a longer period. Only 8% of these cows were in calf at 100 days postpartum compared to 43% for cows without endometritis ($P = 0.002$). Retained placenta delays involution of the uterus and is therefore a cause of metritis. Almost half of the cows having RP at the onset of lactation developed metritis later (Bartlett et al., 1986; Bigras-Poulin et al., 1990; Correa et al., 1993). Cows having an abnormal calving (dystocia or retained placenta) are at a higher risk of puerperal metritis and clinical endometritis (Benzaquen et al., 2007). The last 2 relationships were confirmed by the

positive genetic correlations found by Lin et al. (1989), who also reported a positive correlation between metritis in 2nd lactation and COD.

Models and Parameters for Genetic Analyses. Uterine disease has been analysed with linear and threshold models. Most analyses with linear models gave very low heritabilities (0.01). The only exception is again Lin et al. (1989) who have a more precise dataset and often find higher values for genetic variances. Using only data about endometritis, Distl et al. (1991) and Ouweltjes et al. (1996) found even lower heritabilities. Animal model gave slightly higher values. Threshold models were used recently on producer-recorded data giving heritabilities around 0.1. No significant genetic correlations were found by Zwald et al. (2004b) between MET and any other health trait. Using sires' PTA, Abdel-Aziz et al. (2005) found a positive correlation between MET and MAST.

MILK FEVER

Definition of the Disease. Milk fever is diagnosed when the following signs are observed postpartum: a) 1st stage: stiffness, weakness and high temperature; b) 2nd stage: lying down, cold extremities and low temperature; c) 3rd stage: lying down with legs stretched out (Kelton et al., 1998). The disease is caused by a low level of plasma calcium (Tveit et al., 1992). At the onset of lactation demand for Ca increases rapidly; therefore most cows have hypocalcaemia, but only few of them have a large enough Ca-deficiency to develop a case of periparturient paresis (milk fever, Ramberg et al., 1984). Milk fever is therefore a pathological exaggeration of a normal physiological process (Tveit et al., 1992). Calcium (**Ca**) concentration in plasma of ruminants is regulated by 2 hormones:

parathyroid hormone (PTH) and 1,25-dihydroxyvitamin D (1,25-$(OH)_2D$), which is a metabolite of vitamin D produced in the kidney. Sources of Ca for the animal are dietary Ca and bone Ca. The 2 hormones regulate Ca concentration through control of the intestinal Ca absorptive and bone Ca resorptive processes. The rate of absorption (or resorption) from both sources depends on the Ca concentration level in the plasma (Horst, 1986; Horst et al., 1994). A decrease in plasma Ca results in an increase of 1,25-$(OH)_2D$ production which in turn increases the resorption from bone Ca and the general level of Ca in the plasma. As animals grow older this mechanism is less efficient and hypocalcaemia is more acute (Horst et al., 1994). The lowest level in plasma Ca is reached 18 to 30 h after calving (Tveit et al., 1992). In a study where no animal presented a case of milk fever, the level of plasma Ca after 1st parturition dropped 11% when compared to the level before parturition; after the 2nd parturition, the decrease was 16.4%. The distribution of plasma Ca level after 2nd calving had a heavier lower tail (Tveit et al., 1992) showing that relatively more animals have very low plasma Ca level than a high one. Milk fever-prone cows are unable to respond to the increase of plasma PTH and 1,25-$(OH)_2D$ (Horst, 1986) and therefore are not able to keep their Ca level high enough. Moreover, during the 1st week postpartum, the dairy cow is unable to increase the Ca resorption from the bone; being solely dependent on dietary Ca (Horst, 1986) and having a reduced feed intake during this period, the cows are very susceptible to milk fever.

Incidence and Prevalence. The median incidence of 33 studies reported by Kelton et al. (1998) was 6.5%. In a study of herds in South-western Ontario, Bigras-Poulin et al. (1991) found an incidence of 5.6% for MF. A more current study in Ontario, reported a median incidence of 3.4% (McLaren et al., 2006). The lower incidence in this

study might be caused by a better prevention in the herds studied, as 25% of the producers collecting data for the study by McLaren et al. (2006) were administering calcium preventively. A recent random sample of 176 herds in Denmark had an incidence of 3.0% (Hansen et al., 2007). Incidence increases from 1st calving to subsequent ones, Heringstad et al. (2005) reported incidences of 0.1, 1.9 and 7.9% in 1st, 2nd and 3rd lactation, respectively, for Norwegian Red cattle. Tveit et al. (1991) even reported incidences as high as 16.0% in fifth lactation. The median time to 1st occurrence was 0 days, showing clearly that this is a parturition related disease (Lin et al., 1989; Bigras-Poulin et al., 1991).

Relationships to Other Traits. As hypocalcaemia reduce the muscle tone in the uterus, it might account for the higher incidence of RP (Goff and Horst, 1997). Cows with MF also have a reduced feed intake, which increases the risk of KET and LDA (Goff and Horst, 1997).

Models and Parameters for Genetic Analyses. Estimation of breeding values for MF is difficult as the incidence is very low in 1st and 2nd lactation. When a threshold model is used, extreme category problems arise because some classes have only healthy cows. Heringstad et al. (2005) modeled larger lactation classes to deal with this problem. Tveit et al. (1991) made a genetic evaluation for the nadir of postpartum level of plasma Ca. They found a heritability equal to 0.11. Heritability estimates varied greatly; values as low as 0 (Pryce et al., 1999) and as high as 0.42 (Lin et al., 1989) were reported. There does not seem to be a relationship between model used or data quality and value for heritability. However, comparing data obtained with linear multivariate and threshold single trait models, Kadarmideen et al. (2000) found heritabilities of 0.01 and 0.07,

respectively. Uribe et al. (1995) reported a heritability of 0.09 with a univariate model and 0.10 as an average of bivariate model including MF and 1 production trait. Milk fever in each of the first 3 lactations was considered as 3 different traits by Heringstad et al. (2005). They reported posterior means for heritabilities of 0.09, 0.11 and 0.13 liability to milk fever in 1st, 2nd and 3rd lactation, respectively. Genetic correlation between 2nd and 3rd lactation's liability to milk fever was moderately high (0.71); correlation between 1st lactation's liability and 2nd or 3rd lactation was low (0.29 and 0.19, respectively) but had a large standard deviation reflecting the lack of variability for this trait. The rank correlation of sire for liability to MF in 1st, 2nd and 3rd lactation was between 0.57 and 0.89. There is a genetic component to the relationship beween KET and MF as it was shown in a study of Norwegian Red cattle (Heringstad et al., 2005), where liability to KET in 1st lactation was genetically correlated to liability to MF after the 2nd calving (r_g = 0.40); the last trait was also correlated to liability to KET in 2nd and 3rd lactations (r_g = 0.35 and 0.33 respectively). A moderate positive genetic correlation was found between MF and COD (Lin et al., 1989). Milk fever had a negative genetic correlation to milk production estimated at -0.67 by Uribe et al. (1991). Tveit et al. (1991) reported a negative genetic correlation (r_g = -0.46) between the nadir of postpartum level of plasma Ca and milk production. This means that cows with higher milk production will have a lower level of plasma Ca (hypocalcaemia) and therefore are more at risk of milk fever.

RETAINED PLACENTA

Definition of the Disease. Parturient cows expel the foetal membranes in the first 24 hours postpartum. When this does not occur in the given time window the cow is

diagnosed with a case of retained placenta (RP; Kelton et al., 1998). During pregnancy, the placenta has to be tightly attached to the maternal endometrium for the transfer of nutrient from the mother to the foetus. After calving, these bindings must be rapidly broken. The placenta, although being an allograft, is not rejected by the mother during the pregnancy. The causes for this are not completely clear, but a lack of expression of polymorphic major histocompatibility complex (MHC) antigens on the trophoblast cells is a cause (Davies et al., 2004). After calving, when antigens are recognized on trophoblast surface, a destructive but necessary inflammatory process is initiated, leading to the placenta release. During pregnancy, placenta matures; a full maturation is a prerequisite for a normal loosening process (Joosten and Hensen, 1992). When the gestation is too short (e.g. in the case of an abortion) the maturation process is not complete and therefore RP incidence is higher (Han and Kim, 2005). In addition to the maturation, the mechanical process of parturition is also responsible for the detachment of the placenta from the endometrium. Joosten and Hensen (1992) reported that in the case of MHC compatibility between cow and calf, incidence of RP increased. They concluded that part of the mechanism of retention is related with defective foetal-maternal immunogenic signalling. Blood neutrophils from cows with RP are less activated than those from cows with expelled placenta (Kimura et al., 2002). When RP occurs, it generally lasts for 1 to 2 weeks (Kimura et al., 2002).

Incidence and Prevalence. Incidence of RP was reported at values between 4.4 and 7.7% (Joosten et al., 1987; Lin et al., 1989; Bigras-Poulin et al., 1990). In Ontario Holsteins, median lactation incidence rate was reported at 7.2% (McLaren et al., 2006). There is an increase of incidence with parity (Joosten et al., 1987; Lin et al., 1989; Distl

et al., 1991; Heringstad et al., 2005). Moreover Joosten et al. (1987) found a recurrent effect of RP; cows having RP in the 1st parity had a 7.5% chance of having it in 2nd parity compared to 2.8% for cows who did not have it. In 3rd lactation the risk was 25.0, 12.4 and 4.2% for cows having had RP in 1st and 2nd, only 2nd, and never respectively. As RP is a parturition related trait, cases are diagnosed the day of calving (Lin et al., 1989). Osteras et al. (2007) presented results from 30 years of evaluation in Norway. During this period, the incidence of RP went from 3 to 5% at the end of the 1980s to go down to 3.1% in 2005. The dairy population in Norway is mainly Norwegian Red and selection is applied on health traits since 1978.

Relationships to Other Traits. In a study of the Dutch MRY breed (Joosten et al., 1987), calving difficulty, weight of the calf, multiple births, stillbirth and abortion had an effect on the incidence of RP. The incidence for cows having a single live calf was 4.4%. Cows with difficult calving showed an incidence of 13.3% and cows with multiple births 36.8%. Dystocia had a moderate positive genetic correlation with RP (0.32 to 0.41; Lin et al., 1989). The odds ratio of MET and COD after exposure to RP were 4.49 and 2.36 respectively (Bigras-Poulin et al., 1990). The relationship between these 3 reproductive disorders is therefore clearly presented.

Models and Parameters for Genetic Analyses. Most analyses of data for RP have been made with linear sire models. Results from these analyses always gave values below 0.06, except a value of 0.09 found by Lin et al. (1989) for RP in 2nd lactation. As already observed, this study used a smaller dataset and data collection was more precise, which gave a higher heritability. Using a multivariate threshold model, Heringstad et al. (2005) found a slightly higher heritability (0.08) for RP in each of the first 3 lactations. Genetic

correlations between RP at each of the first 3 lactations were between 0.55 and 0.65; showing that although they are positively correlated, they seem to be different traits, determined by different genes. Genetic correlation between RP and metritis was reported at 0.66 (Distl et al., 1991). There is a low positive genetic correlation between RP and MAST (Heringstad et al., 2005); the interaction between these traits is less obvious, but shows that there is a genetic component of "general resistance to diseases".

ACKNOWLEDGMENTS

The author wishes to thank S. Cartwright for the research made on these 8 diseases and for providing documents to prepare this paper.

REFERENCES

Abdel-Azim, G. A., A. E. Freeman, M. E. Kehrli, Jr., S. C. Kelm, J. L. Burton, A. L. Kuck, and S. Schnell. 2005. Genetic basis and risk factors for infectious and noninfectious diseases in US Holsteins. I. Estimation of genetic parameters for single diseases and general health. J. Dairy Sci. 88:1199-1207.

Andrews, T. 1998. Ketosis and fatty liver in cattle. In Practice 20:509-513.

Baird, A. N., and S. Harrison. 2001. Surgical treatment of left displaced abomasum. Comp. Cont. Educ. Pract. S102-S108 Suppl. S. Oct. 2001.

Barkema, H. W., J. D. Westrick, K. A. S. van Keulen, Y. H. Schukken, and A. Brand. 1994. The effects of lameness on reproductive performance, milk production and culling in Dutch dairy farms. Prev. Vet. Med. 20:249-259.

Bartlett, P.C., J. H. Kirk, M. A. Wilke, J. B. Kaneene, and E. C. Mather. 1986. Metritis complex in Michigan Holstein-Frisian cattle: incidence, descriptive epidemiology and estimated economic impact. Prev. Vet. Med. 4:235-248.

Benzaquen, M. E., C. A. Risco, L. F. Archibald, P. Melendez, M.-J. Thatcher, and W. W. Thatcher. 2007. Rectal temperature, calving-related factors, and the incidence of puerperal metritis in postpartum dairy cows. J. Dairy Sci. 90:2804-2814.

Bigras-Poulin, M., A. H. Meek, S. W. Martin, and I. McMillan. 1990. Health problems in selected Ontario Holstein cows: frequency of occurrences, time to first diagnosis and associations. Prev. Vet. Med. 10:79-89.

Blowey, R. D., P. Ossent, C. L. Watson, V. Hedges, L. E. Green, and A. J. Packington. 2000. Possible distinction between sole ulcers and heel ulcers as a cause of bovine lameness. Vet. Rec. 147:110-112.

Booth, C. J., L. D. Warnick, Y. T. Gröhn, D. O. Maizon, C. L. Guard, and D. Janssen. 2004. Effect of lameness on culling in dairy cows. J. Dairy Sci. 87:4115-4122.

Bradley, A. J., K. A. Leach, J. E. Breen, L. E. Green, and M. J. Green. 2007. Survey of the incidence and aetiology of mastitis on dairy farms in England and Wales. Vet. Rec. 160:253-258.

Clarkson, M. J., D. Y. Downham, W. B. Faull, J. W. Hughes, F. J. Manson, J. B. Merritt, R. D. Murray, W. B. Russell, J. E. Sutherst, and W. R. Ward. 1996. Incidence and prevalence of lameness in dairy cattle. Vet. Rec. 138:563-567.

Collard, B. L., P. J. Boettcher, J. C. M. Dekkers, D. Petitclerc, and L. R. Schaeffer. 2000. Relationships between energy balance and health traits of dairy cattle in early lactation. J. Dairy Sci. 83:2683-2690.

Correa, M. T., H. Erb, and J. Scarlett. 1993. Path analysis for seven postpartum disorders of Holstein cows. J. Dairy Sci. 76:1305-1312.

Cramer, G., K. D. Lissemore, C. L. Guard, K. E. Leslie, and D. F. Kelton. 2008. Herd- and cow-level 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. The association between foot lesions and culling risk in Ontario Holstein cows. J. Dairy Sci. 92:2572-2579.

Davies, C. J., J. R. Hill, J. L. Edwards, F. N. Schrick, P. J. Fisher, J. A. Eldridge, and D. H. Schlafer. 2004. Major histocompatibility antigen expression on the bovine placenta: its relationship to abnormal pregnancies and retained placenta. Anim. Reprod. Sci. 82-83:267-280.

Distl, O., M. Ron, G. Francos, E. Mayer, and H. Kraeusslich. 1991. Genetic analysis of reproductive disorders in Israeli Holstein dairy cows. Theriogenology 35:827-836.

Drackley, J. K., T. R. Overton, and G. N. Douglas. 2001. Adaptations of glucose and long-chain fatty acid metabolism in liver of dairy cows during the periparturient period. J. Dairy Sci. 84:E100-E112.

Emanuelson, U., P. A. Oltenacu, and Y. T. Gröhn. 1993. Nonlinear mixed model analyses of five production disorders of dairy cattle. J. Dairy Sci. 76:2765-2772.

Földi, J., M Kulcsár, A. Pécsi, B. Huyghe, C. de Sa, J. A. C. M. Lohuis, P. Cox, and G. Huszenicza. 2006. Bacterial complications of postpartum uterine involution in cattle. Anim. Reprod. Sci. 96:265-281.

Gilbert, R. O., S. T. Shin, C. L. Guard, H. N. Erb, and M. Frajblat. 2005. Prevalence of endometritis and its effects on reproductive performance of dairy cows. Theriogenology 64:1879-1888.

Gillund, P., O Reksen, Y. T. Gröhn, and K. Karlberg. 2001 Body condition related to ketosis and reproductive performance in Norwegian dairy cows. J. Dairy Sci. 84:1390-1396.

Goff, J. P., and R. L. Horst. 1997. Physiolocival changes at parturition and their relationship to metabolic disorders. J. Dairy Sci. 80:1260-1268.

Green, L. E., V. J. Hedges, Y. H. Schukken, R. W. Blowey, and A. J. Packington. 2002. The impact of clinical lameness on the milk yield of dairy cows. J. Dairy Sci. 85:2250-2256.

Hagnestam, C., U. Emanuelson, and B. Berglund. 2007. Yield losses associated with clinical mastitis occurring in different weeks of lactation. J. Dairy Sci. 90:2260-2270.

Hamann, H., V. Wolf, H. Schölz, and O. Distl. 2004. Relationships between lactational incidence of displaced abomasum and milk production traits in German Holstein cows. J. Vet. Med. A 51:203-208.

Han, Y.-K., and I.-H. Kim. 2005. Risk factors for retained placenta and the effect of retained placenta on the occurrence of postpartum diseases and subsequent reproductive performance in dairy cows. J. Vet. Sci. 6:53-59.

Hansen, S. S., A. K. Ersboll, J. Y. Blom, and R. J. Jorgensen. 2007. Preventive strategies and risk factors for milk fever in Danish herds: a questionnaire survey. Prev. Vet. Med. 80:271-286.

Harmon, R. J. 1994. Physiology of mastitis and factors affecting somatic cell counts. J. Dairy Sci. 77:2103-2112.

Hayirli, A., and R. R. Grummer. 2004. Factors affecting dry matter intake prepartum in relationship to etiology of peripartum lipid-related metabolic disorders: a review. Can. J. Anim. Sci. 84:337-347.

Heringstad, B., Y. M. Chang, D. Gianola, and G. Klemetsdal. 2003a. Genetic analysis of longitudinal trajectory of clinical mastitis in first-lactation Norwegian cattle. J. Dairy Sci. 86:2676-2683.

Heringstad, B., Y. M. Chang, D. Gianola, and G. Klemetsdal. 2005. Genetic analysis of clinical mastitis, milk fever, ketosis, and retained placenta in three lactations of Norwegian Red cows. J. Dairy Sci. 88:3273-3281.

Heringstad, B., G. Klemetsdal, and J. Ruane. 2000. Selection for mastitis resistance in dairy cattle: a review with focus on the situation in the Nordic countries. Livest. Prod. Sci. 64:95-106.

Heringstad, B., R. Rekaya, D. Gianola, G. Klemetsdal, and K. A. Weigel. 2001. Bayesian analysis of liability of clinical mastitis in Norwegian cattle with a threshold model: effects of data sampling method and model specification. J. Dairy Sci. 84:2337-2346.

Heringstad, B., R. Rekaya, D. Gianola, G. Klemetsdal, and K. A. Weigel. 2003b. Genetic change for clinical mastitis in Norwegian cattle: a threshold model analysis. J. Dairy Sci. 86:369-375.

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.

Hoedemaker, M. 1998. Postpartal pathological vaginal discharge: to treat or not to treat? Reprod. Dom. Anim. 33:141-146.

Hooijer, G. A., R. B. F. Lubbers, B. J. Ducro, J. A. M. van Arendonk, L. M. T. E. Kaal-Lansbergen, and T. van der Lende. 2001. Genetic parameters for cystic ovarian disease in Dutch black and white dairy cattle. J. Dairy Sci. 84:286-291.

Horst, R. L. 1986. Regulation of calcium and phosphorus homeostasis in the dairy cow. J. Dairy Sci. 69:604-616.

Horst, R. L., J. P. Goff, and T. A. Reinhardt. 1994. Calcium and vitamin D metabolism in the dairy cow. J. Dairy Sci. 77:1936-1951.

Ingvartsen, K. L. 2006. Feeding- and management-related diseases in the transition cow. Physiological adaptations around calving and strategies to reduce feeding-related diseases. Anim. Feed Sci. Tech. 126:175-213.

Ingvartsen, K. L., R. J. Dewhurst, and N. C. Friggens. 2003. On the relationship between lactational performance and health: is it yield or metabolic imbalance that cause production diseases in dairy cattle? A position paper. Livest. Prod. Sci. 83:277-308.

Joosten, I., E. J. Hensen. 1992. Retained placenta: an immunological approach. Anim. Reprod. Sci. 28:451-461.

Joosten, I., P. van Eldik, L. Elving, and G. J. W. van der Mey. 1987. Factors related to the etiology of retained placenta in dairy cattle. Anim. Reprod. Sci. 14:251-262.

Kadarmideen, H. N., R. Thompson, and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71:411-419.

Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. J. Dairy Sci. 81:2502-2509.

Kesler, D. J., and H. A. Garverick. 1982. Ovarian cysts in dairy cattle: a review. J. Anim. Sci. 55:1147-1159.

Kesler, D. J., H. A. Garverick, C. J. Bierschwal, R. G. Elmore, and R. S. Youngquist. 1979. Reproductive hormones associated with normal and abnormal changes in ovarian follicles in postpartum dairy cows. J. Dairy Sci. 62:1290-1296.

Kimura, K., J. P. Goff, M. E. Kehrli Jr., and T. A. Reinhardt. 2002. Decreased neutrophil function as a cause of retained placenta in dairy cattle. J. Dairy Sci. 85:544-550.

Laporte, H. M., H. Hogeveen, Y. H. Schukken, and J. P. T. M. Noordhuizen. 1994. Cystic ovarian disease in Dutch dairy cattle, I. Incidence, risk factors and consequences. Livest. Prod. Sci. 38:191-197.

Leach, K. A., D. N. Logue, S. A. Kempson, J. E. Offer, H. E. Ternent, and J. M. Randall. 1997. Claw lesions in dairy cattle: development of sole and white line haemorrhages during the first lactation. Vet. J. 154:215-225.

LeBlanc, S. J., K. E. Leslie, and T. F. Duffield. 2005. Metabolic predictors of displaced abomasum in dairy cattle. J. Dairy Sci. 88:159-170.

Lin, H. K., P. A. Oltenacu, L. D. Van Vleck, H. N. Erb, and R. D. Smith. 1989. Heritabilities of and genetic correlations among six health problems in Holstein cows. J. Dairy Sci. 72:180-186.

López-Gatius, F., P. Santolaria, J. Yániz, M. Fenech, and M. López-Béjar. 2002. Risk factors for postpartum ovarian cysts and their spontaneous recovery or persistence in lactating dairy cows. Theriogenology 58:1623-1632.

Lyons, D. T., A. E. Freeman, and A. L. Kuck. 1991. Genetics of health traits in Holstein cattle. J. Dairy Sci. 74:1092-1100.

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.

Mäntysaari, E. A., Y. T. Gröhn, and R. L. Quaas. 1993. Repeatability and heritability of lactational occurrence of reproductive disorders in dairy cows. Prev. Vet. Med. 17:111-125.

McLaren, C. J., K. D. Lissemore, T. F. Duffield, K. E. Leslie, D. F. Kelton, and B. Grexton. 2006. The relationship between herd level disease incidence and a return over feed index in Ontario dairy herds. Can. Vet. J. 47:767-773.

Melendez, 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.

Miglior, F., B. L. Muir, and B. J. Van Doormaal. 2005. Selection indices in Holstein cattle of various countries. J. Dairy Sci. 88:1255-1263.

Murray, R. D., D. Y. Downham, M. J. Clarkson, W. B. Faull, J. W. Hughes, F. J. Manson, J. B. Merritt, W. B. Russell, J. E. Sutherst, W. R. Ward. 1996.

Epidemiology of lameness in dairy cattle: description and analysis of foot lesions. Vet. Rec. 138:586-591.

Nash, D. L., G. W. Rogers, J. B. Cooper, G. L. Hargrove, J. F. Keown, and L. B. Hansen. 2000. Heritability of clinical mastitis incidence and relationships with sire transmitting abilities for somatic cell score, udder type traits, productive life, and protein yield. J. Dairy Sci. 83:2350-2360.

Nocek. J. E. 1997. Bovine acidosis: implications on laminitis. J. Dairy Sci. 80:1005-1028.

Olde Riekerink, R. G. M., H. W. Barkema, D. F. Kelton, and D. T. Scholl. 2008. Incidence rate of clinical mastitis on Canadian dairy farms. J. Dairy Sci. 91:1366-1377.

Ossent, P., and C. Lischer. 1998. Bovine laminitis: the lesions and their pathogenesis. In Practice 20:415-427.

Osteras, O., H. Solbu, A. O. Refsdal, T. Roalkvam, O. Filseth, and A. Minsaas. 2007. Results and evaluation of thirty years of health recordings in the Norwegian dairy cattle population. J. Dairy Sci. 90:4483-4497.

Oura, L. Y., L. K. Fox, C. C. Warf, and G. K. Kemp. 2002. Efficacy of two acidified chlorite postmilking teat disinfectants with sodium dodecylbenzene sulfonic acid on prevention of contagious mastitis using an experimental challenge protocol. J. Dairy Sci. 85:252-257.

Ouweltjes, W., E. A. A. Smolders, L. Elving, P. van Eldik, and Y. H. Schukken. 1996. Fertility disorders and subsequent fertility in dairy cattle. Livest. Prod. Sci. 46:213-220.

Peter, A. T. 2004. An update on cystic ovarian degeneration in cattle. Reprod. Dom. Anim. 39:1-7.

Pryce, J. E., B. L. Nielsen, R. F. Veerkamp, and G. Simm. 1999. Genotype and feeding system effects and interactions for health and fertility traits in dairy cattle. Livest. Prod. Sci. 57:193-201.

Ramberg, C. L. Jr., E. K. Johnson, R. D. Fargo, and D. S. Kronfeld. 1984. Calcium homeostasis in cows, with special reference to parturient hypocalcemia. Am. J. Physiol. 246:R698-704.

Rasmussen, L. K., B. L. Nielsen, J. E. Pryce, T. T. Mottram, and R. F. Veerkamp. 1999. Risk factors associated with the incidence of ketosis in dairy cows. Anim. Sci. 68:379-386.

Shaver, R. D. 1997. Nutritional risk factors in the etiology of left displaced abomasum in dairy cows: a review. J. Dairy Sci. 80:2449-2453.

Sheldon, I. M., G. S. Lewis, S. LeBlanc, and R. O. Gilbert. 2006. Defining postpartum uterine disease in cattle. Theriogenology 65:1516-1530.

Simianer, H., H. Solbu, and L. R. Schaeffer. 1991. Estimated genetic correlations between disease and yield traits in dairy cattle. J. Dairy Sci. 74:4358-4365.

Steine, G., D. Kristofersson, and A. G. Guttormsen. 2008. Economic evaluation of the breeding goal for Norwegian Red dairy cattle. J. Dairy Sci. 91:418-426.

Stengärde, L. U., and B. G. Pehrson. 2002. Effects of management, feeding, and treatment on clinical and biochemical variables in cattle with displaced abomasum. Am. J. Vet. Res. 63:137-142.

Tveit, B., F. Lingaas, M. Svendsen, and O. V. Sjaastad. 1992. Etiology of acetonemia in Norwegian cattle. 1. Effect of ketogenic silage, season, energy level, and genetic factors. J. Dairy Sci. 75:2421-2432.

Tveit, B., M. Svendsen, and K. Hove. 1991. Heritability of hypocalcemia at first parturition in Norwegian cattle: genetic correlations with yield and weight. J. Dairy Sci. 74:3561-3567.

Uribe, H. A., B. W. Kennedy, S. W. Martin, and D. F. Kelton. 1995. Genetic parameters for common health disorders of Holstein cows. J. Dairy Sci. 78:421-430.

van der Tol, P. P. J., J. H. M. Metz, E. N. Noordhuizen-Stassen, W Back, C. R. Braam, and W. A. Weijs. 2002. The pressure distribution under the bovine claw during square standing on a flat substrate. J. Dairy Sci. 85:1476-1481.

van Dorp, T. E., J. C. M. Dekkers, S. W. Martin, and J. P. T. M. Noordhuizen. 1998. Genetic parameters of health disorders, and relationships with 305-day milk yield and conformation traits of registered Holstein cows. J. Dairy Sci. 81:2264-2270.

Vanholder, T., G. Opsomer, and A. de Kruif. 2006. Aetiology and pathogenesis of cystic ovarian follicles in dairy cattle: a review. Reprod. Nutr. Dev. 46:105-119.

van Winden, S. C. L., R. Jorritsma, K. E. Müller, and J. P. T. M. Noordhuizen. 2004. Feed intake, milk yield, and metabolic parameters prior to left displaced abomasum in dairy cows. J. Dariy Sci. 86:1465-1471.

van Winden, S. C. L., and R. Kuiper. 2003. Left displacement of the abomasum in dairy cattle: recent development in epidemiological and etiological aspects. Vet. Res. 34:47-56.

Warnick, L. D., D. Janssen, C. L. Guard, and Y. T. Gröhn. 2001. The effect of lameness on milk productin in dairy cows. J. Dairy Sci. 84:1988-1997.

Wood, G. M., P. J. Boettcher, D. F. Kelton, and G. B. Jansen. 2004. Phenotypic and genetic influences on test-day measures of acetone concentration in milk. J. Dairy Sci. 87:1108-1114.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004a. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. J. Dairy Sci. 87:4287-4294.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004b. Genetic selection for health traits using producer-recorded data. II. Genetic correlations, disease probabilities, and relationships with existing traits. J. Dairy Sci. 87:4295-4302.

Table 1 Description of data used for the different analyses

Study	Description of the data	Size of dataset
Lin et al., 1991	Result of a field study; accurately described data. 30 months of data. US Holstein.	7,712 lactations 33 herds
Simianer et al., 1991	Veterinarian treatment data. Only 1st lactation. 5 years of data. Norwegian cattle.	208,693 records 71,406 HYS[1]
Lyons et al., 1991	Producer recorded data for a study. US data. Breed unknown (probably Holstein)	9,187 records
Distl et al., 1991	Veterinarian routinely recorded data. 3 years of data. Israeli Holsteins.	76,170 lactations 102 herds
Mäntysaari et al., 1993	Veterinarian treatment data, 3 years of data. Finnish Ayrshire	28,277 cows 13,285 records
Uribe et al., 1995	Producer/veterinarian recorded data in a field study. 3 years of data. Southern Ontario Holsteins.	7,416 cows 98 herds
Ouweltjes et al., 1996	Producer/veterinarian in a field study. 5 years of data. Dutch cows, breed unknown	10,426 records 33 herds
Van Dorp et al., 1998	Producer recorded data, voluntary basis. 3 years of data. Canadian (BC) Holsteins	7,542 cows 32 herds
Pryce et al., 1999	Data from 1 research herd. 16 years of data. UK Holsteins.	935 cows 1 herd
Kadarmideen et al., 2000	Producer routinely recorded data. 5 years of data. UK Holsteins.	43,193 cows 960 herds
Hoijer et al., 2001	Veterinarian routinely recorded data. 10 years of data. Dutch Holsteins (Friesland).	15,562 lactations 32 herds
Heringstad et al., 2001	Veterinary treatment data. Data from 1st lactation. 2 years of study. Norwegian cattle.	13,070 cows 1,868 herds
Heringstad et al., 2003b	Veterinary treatment data. Data from 1st lactation. 11 years of study. Norwegian cattle.	1.6 million cows 28,491 herds
Zwald et al., 2004a	Producer recorded data for management purpose. 3 years of data. US Holsteins.	For MAST[2] 105,029 cows 724 herds
Zwald et al., 2004b	Producer recorded data for management purpose. 3 years of data. US Holsteins.	161,622 cows 646 herds
Heringstad et al., 2005	Veterinary treatment data. 11 years of data. Norwegian cattle.	372,227 cows
Abdel-Aziz et al., 2005	Producer recorded data for study purpose. 4 years of data. US Holstein.	14,473 cows 177 herds

[1]HYS: Herd-year-season

[2]MAST: Mastitis

Table 2. Models used for genetic evaluation of health traits in different studies

	Health Trait							
Study	MAST	LAME	COD	LDA	KET	MET	MF	RP
Lin et al., 1989	SM[1] ST[2] LM[3]		SM ST LM			SM ST LM	SM ST LM	SM ST LM
Simianer et al., 1991	SM MT[4] TM[5]				SM MT[4] TM[5]			
Lyons et al., 1991	SM MT LM	SM MT LM	SM MT LM	SM MT LM	SM MT LM	SM MT LM	SM MT LM	SM MT LM
Distl et al., 1991			SM MT LM			SM MT LM		SM MT LM
Mäntysaari et al., 1993			SM MT LM TM			SM MT LM TM		
Uribe et al., 1995	SM MT TM	SM MT TM	SM MT TM	SM MT TM	SM MT TM		SM MT TM	
Ouweltjes et al., 1996						SM MT LM		SM MT LM
Van Dorp et al., 1998	AM[6] MT LM	AM[6] MT LM	AM[6] MT LM	AM[6] MT LM		AM[6] MT LM	AM[6] MT LM	AM[6] MT LM
Pryce et al., 1999	MGS[7] MT LM	MGS MT LM			MGS MT LM	MGS MT LM	MGS MT LM	MGS MT LM
Kadarmideen et al., 2000	AM MTLM SM STTM	AM MTLM SM STTM			AM MTLM SM STTM		AM MTLM SM STTM	
Hooijer et al., 2001			AM ST TM					
Heringstad et al., 2001	SM ST TM							
Heringstad et al., 2003b	SM ST TM							
Zwald et al.,	SM	SM	SM	SM	SM	SM		

2004a	ST TM	ST TM	ST TM	ST TM	ST TM	ST TM		
Zwald et al., 2004b	SM MT TM	SM MT TM	SM MT TM	SM MT TM	SM MT TM	SM MT TM		
Heringstad et al., 2005	SM MT TM				SM MT TM		SM MT TM	SM MT TM
Abdel-Azim et al., 2005	MGS ST TM		MGS ST TM	MGS ST TM		MGS ST TM	MGS ST TM	

[1]Sire model

[2]Single trait

[3]Linear model

[4]Multiple trait

[5]Threshold model

[6]Animal model

[7]Sire-maternal grandsire model

Table 3. Heritabilities calculated for health traits in selected studies.

	Health Trait							
Study	MAST		LAME		COD		LDA	
Lin et al., 1989	1st lact[1] 2nd lact 3rd lact	0.18 0.31 0.18			1st lact 2nd lact 3rd lact	0.12 0.08 0.02		
Simianer et al., 1991		0.06 – 0.09						
Lyons et al., 1991		0.14		0.11		0.05		0.09
Distl et al., 1991					1st lact 2nd lact 3rd lact	0.006 0.005 0.002		
Mäntysaari et al., 1993					LM 1st lact 2nd lact TM 1st lact 2nd lact	 0.01 0.02 0.03 0.03		
Uribe et al., 1995	1st lact All lact	0.15 0.00		0.15	1st lact All lact	0.13 0.08		0.28
Ouweltjes et al., 1996								
Van Dorp et al., 1998		0.04		0.16		0.02		0.00
Pryce et al., 1999		0.04		0.08				
Kadarmideen et al., 2000	LM[2] TM[2]	0.04 0.13	LM TM	0.02 0.08				
Hooijer et al., 2001						0.10		
Heringstad et al., 2001	1st lact	0.06 – 0.07						
Heringstad et al., 2003b	1st lact	0.07						
Zwald et al., 2004a	1st lact All lact	0.10 0.09	1st lact All lact	0.07 0.06	1st lact All lact	0.08 0.05	1st lact. All	0.18 0.15
Zwald et al., 2004b	1st lact	0.09	1st lact	0.04	1st lact	0.04	1st lact	0.14
Heringstad et al., 2005	1st lact 2nd lact 3rd lact	0.08 0.07 0.07						
Abdel-Aziz et al., 2005		0.16				0.03		0.09

[1]Cows included in the analysis: 1st lact = cows in 1st lactation; 2nd lact = cows in 2nd lactation; 3rd lact = cows in 3rd lactations and more; All lact = cows in all lactations

[2]LM = linear model; TM = threshold model

Table 4. Heritabilities calculated for health traits in selected studies.

	Health Trait							
Study	KET		MET		MF		RP	
Lin et al., 1989			1st lact[1] 2nd lact	0.19 0.26	 2nd lact 3rd lact	 0.30 0.42	1st lact 2nd lact	0.05 0.09
Simianer et al., 1991		0.08 – 0.11						
Lyons et al., 1991		0.08		0.06		0.40		0.05
Distl et al., 1991			1st lact 2nd lact 3rd lact	0.01 0.01 0.002			1st lact 2nd lact 3rd lact	0.01 0.01 0.02
Mäntysaari et al., 1993			LM[2] 1st lact 2nd lact TM[2] 1st lact 2nd lact	 0.01 0.01 0.04 0.02				
Uribe et al., 1995		0.08				0.09		
Ouweltjes et al., 1996				0.008				0.006
Van Dorp et al., 1998				0.02		0.04		0.01
Pryce et al., 1999		0.01		0.01		0.00		0.02
Kadarmideen et al., 2000	LM TM	0.01 0.02			LM TM	0.01 0.07		
Hooijer et al., 2001								
Heringstad et al., 2001								
Heringstad et al., 2003b								
Zwald et al., 2004a	1st lact All lact	0.11 0.06	1st lact All lact	0.08 0.07				
Zwald et al., 2004b	1st lact	0.06	1st lact	0.06				
Heringstad et al., 2005	1st lact 2nd lact 3rd lact	0.14 0.16 0.15			1st lact 2nd lact 3rd lact	0.09 0.11 0.13	1st lact 2nd lact 3rd lact	0.08 0.08 0.08
Abdel-Aziz et al., 2005				0.14				

[1]Cows included in the analysis: 1st lact = cows in 1st lactation; 2nd lact = cows in 2nd lactation; 3rd lact = cows in 3rd lactations and more; All lact = cows in all lactations

[2]LM = linear model; TM = threshold model

Chapter 3

Comparison of Two Different Sampling Methods on Heritabilities of Producer-Recorded Health Events.

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ABSTRACT

Producer-recorded health events of Canadian Holstein cows were sampled for use in variance component analysis. Diseases analysed were mastitis, lameness, cystic ovarian disease, left displaced abomasum, ketosis, metritis, milk fever and retained placenta. Binary coding was used to record presence of the trait (0 = sick, 1 = healthy). One case per lactation per animal was kept and contemporary groups were made of all cows having a test-day record in the same herd during the same year. Single trait linear sire model analyses were conducted for each of the 8 health traits. Two sampling frames were used. The first kept only herds with at least 1 occurrence of the disease. The second kept all herds with at least 1 occurrence of any of the 8 diseases.

Lactational incidence of all diseases was lower than values previously reported in many studies. Using the first sampling frame, heritability estimates were below 0.025 for all traits except for left displaced abomasum (0.029). Variance component estimations based on the second sampling frame gave heritability estimates below 0.01, except for left displaced abomasum (0.013). All heritability estimates were lower with the second sampling frame than with the first due to the quadratic relationship between dispersion and location parameters for binary traits. Correlations between predicted transmitting abilities (**PTA**) calculated with data from the two sampling frames were generally high, but were lower for metritis, the trait with the lowest estimate of heritability and the lowest lactational incidence.

These results show the importance of appropriate sampling frames and the importance of removing underreporting of diseases to provide reasonable heritability estimates.

INTRODUCTION

Reduction of production costs is an important aspect of dairy farming and production-related diseases are a significant cause of costs. Treatments, culling, loss of production and non-usable milk are all sources of economic loss (Bar et al., 2008). Besides management changes, an improvement of the genetics involved in disease resistance would be beneficial to the entire dairy industry. An essential aspect of selection for disease resistance is the availability of good quality data.

In 2006, national organizations in Canada worked together to develop a system for the recording of diseases. Since April 2007, data for 8 health traits have been recorded by producers and collected by dairy herd improvement (**DHI**). Use of these data can be made for genetic evaluations. However, one important limitation is the quality of data recording. Producers are involved in this project on a voluntary basis. Therefore, data quality varies among farms and even for a given farm over time. Underreporting can be present for certain diseases or certain periods, e.g. during the summer when the producer spends more time in the fields than in the barn.

Only a few genetic analyses were conducted, based on producer-recorded traits for a large number of herds (e. g. Kadarmideen et al., 2001; Zwald et al., 2004; Abdel-Azim et al., 2005). Other studies focused on health events used smaller groups of herds taking part in research projects, or used datasets compiled by practicing veterinarians

(e.g. Van Dorp et al., 1998). Inclusion criteria for health data is a very important aspect when data quality is not known. As part of that process, there is a need to assess the trade-off of keeping more data, but of lesser quality, to have as large a population size as possible, or to remove herds with poor data quality with the risk of losing many animals but also the risk of keeping only a selected (biased) group of animals. The last 2 types of sources generate more consistent data than producer-recorded data.

In the present study, 2 different methods are presented to sample data for estimating variance components. Other methods than the 2 presented would be possible, such as the definition of a period of recording, but they were not considered in the present study as the amount of data available and the total period of health event recordings were still relatively small. The results are compared to obtain a better understanding of the effects of different sampling frames.

MATERIALS AND METHODS

Data

Data from the Canadian Dairy Network, Guelph, ON, were used for this study. The dataset contained 89,107 disease events collected between April 2007 and September 2008. The starting date corresponds to the beginning of data collection for the Canadian National Health Project (**CNHP**). Data were collected by producers and transmitted to their respective DHI company (CanWestDHI, Guelph, ON, for the Western provinces and Ontario; Valacta, Sainte-Anne-de-Bellevue, QC, for Quebec and the Maritime provinces) or to their veterinarian (only in Quebec with a program called "Dossier santé animale – animal health records" – DS@HR or **DSA**). The distribution of records by

month is presented in Table 1. Eight diseases were reported: mastitis (**MAST**), lameness (**LAME**), cystic ovarian disease (**COD**), displaced abomasum (**LDA**), ketosis (**KET**), metritis / uterine disease (**MET**), milk fever (**MF**) and retained placenta (**RP**); as defined by Kelton et al. (1998). For every record the date of observation, the herd and the disease (from the list of 8 diseases above) were reported. The decrease in number of records at the end of the periods was caused by a 2 month delay in data delivery from the DSA program, in Quebec. In total, 2,979 herds reported disease cases in 2007 representing 29% of all the herds that were on DHI recording during that year.

Test-day (**TD**) records were obtained from this period and contained production results and days in milk (**DIM**) at test date. Age at calving was also reported. Records were used to calculate calving date and to build contemporary groups based on all cows having at least one TD record in the same herd as a cow with a reported disease event.

Data sampling was done separately for each disease. Two different data sampling frames were used. In the first sampling frame (**DATA1**), all herds with at least 1 record of the disease being analyzed, were kept. Herds with at least 1 recording of a given disease were assumed to record this disease event consistently whenever it occurred throughout the study period. This assumption might not be true for all herds and may in fact have biased our study to exclude well managed herds with low disease incidence, as some disease events might have not occurred during the period of data recording and these herds would have been excluded from further analysis. Ultimately, it was decided to retain this approach in order to remove herds not recording this disease. Then, all cows (sick and healthy) that had a TD record in those herds and which calved in April 2007 or later were included in the dataset. Only 1 occurrence of that disease event per parity was

included. Time of disease occurrence (measured as days in milk between the last calving preceding the disease occurrence and the day of the disease occurrence) for all health events were calculated. Lactation days in milk (DIM) measured as days between the calving immediately preceding the disease event and the day of the disease event was calculated for each occurrence of the disease. Given the severely right skewed distribution of these DIM measures, and the biological implausibility of the DIM for disease events at the high end of the DIM distribution, only the earliest 95% of the disease cases were retained in the dataset. Therefore the 95^{th} percentile DIM for each event was set as the upper limit for inclusion of each disease event. The time limits for each disease are presented in Table 2. Further restrictions were made after this step to include only herds with at least 1% lactational disease incidence and at least 5 cows. Lactational incidence was calculated as the ratio of sick animals to the total number of animals included in the contemporary group defined above (sick and healthy animals).

For the second sampling frame (**DATA2**), all herds with recording of at least 1 case of any of these diseases during the study period were kept in the analysis. For example, if a herd had 1 case of MAST, it was included in the analysis of all disease traits. The assumption was that herds collecting data for one disease were collecting data for all diseases. More herds were included in this relaxed sampling frame. Data preparation was the same as the first sampling frame except that the minimum incidence for the trait was not applied, as herds with no occurrence of the disease analyzed were kept in the dataset based on the occurrence of 1 of the other 8 diseases (Table 3).

All measures of incidence were lactational incidence risk (**LIR**). This the number of lactations with at least one case of a disease divided by the total number of lactations

(Bigras-Poulin et al., 1990). Calculation of lactational incidence, showed that incidence for MF in first lactation was not different from zero. Therefore, first lactation animals (sick and healthy) were removed from the analyses for MF.

Models

For both datasets, single trait analyses of each of the 8 diseases were performed. Use of a linear model was made as it is generally used in genetic evaluations of Canadian Holsteins, even with binary traits (Jamrozik et al., 2005). The following linear model was applied for all traits:

$$y_{ijklm} = H_i + M_j + P_k + s_l + e_{ijklm},$$

where, y_{ijklm} is the observation (1 for healthy animals, 0 for animals with the disease), H_i is the fixed effect of the i^{th} herd, M_j is the fixed effect of the j^{th} month of calving preceding the disease or the lactation considered as part of the contemporary group, P_k is the fixed effect of the k^{th} parity of the cow, s_l is the random effect of the l^{th} sire of the cow and e_{ijklm} is the residual effect. No cow within sire effect (permanent environment and genetics of the cow, without relationship matrix) was included in the analyses as a significant proportion of the cows had only 1 parity with an observation. Dispersion properties of the random effects were:

$$\begin{pmatrix} \mathbf{s} \\ \mathbf{e} \end{pmatrix} \sim N\left(\mathbf{0}, \begin{pmatrix} \mathbf{A}\sigma_s^2 & \mathbf{0} \\ \mathbf{0} & \mathbf{I}\sigma_e^2 \end{pmatrix}\right),$$

where, **A** is the additive relationship matrix of the sires of animals with records and their ancestors. Pedigree data were built starting with sire of animals with records and including their sires and maternal grand-sires for three generations. **I** is an identity matrix

of size m x m where m is the number of observations, σ^2_s is the sire variance and σ^2_e is the residual variance.

Comparison of the results of the 2 models was made by the correlation between predicted transmitting abilities (**PTA**) of sires from both samplings. Only bulls with at least 50 daughters in DATA1 were included in the comparison.

Number of records, contemporaries and herds are presented in Tables 2 and 3 for DATA1 and DATA2, respectively. Out of the 3,710 herds collecting health data (Table 3), 2,401 were collecting data on MAST, as can be determined by the data provided. The diseases with the lowest number of herds participating were KET and MET; about 700 herds were included in the analysis of these traits. Parities 5 and higher were grouped together to have enough records to estimate this effect.

Variance components were estimated with the VCE 6 software (Groeneveld et al., 2008). This software estimates variance components by REML using analytical gradient methods (Neumaier and Groeneveld, 1998).

RESULTS

Estimated lactational incidences in DATA1 are presented in Table 4. All incidences were between 3% and 8%. The highest incidence was for MAST and the lowest for LDA. Infectious diseases (MAST and MET) had relatively high incidences. Results from DATA2 are presented in Table 5. All lactational incidences were lower than for DATA1 as the number of herds with no recording for each of the traits increased. The only trait with a lactational incidence remaining above 4% in the analysis with DATA2 was MAST. This was also the trait collected in the largest number of herds. Diseases with

a more complicated diagnosis (KET and MET) had a particularly sharp drop of incidence between the 2 datasets, being collected in only a small amount of farms.

Variance components estimates for DATA1 are presented in Table 4. Heritability was highest for LDA at 0.029. Heritability was 0.02 for MAST, MF, RP and COD. Heritabilities for LAME and MET were very low. All heritability values for DATA2 were smaller than those estimated with DATA1. Phenotypic variances for all traits in DATA2, except MAST were lower than for DATA1 by a factor of at least 2. Only LDA and MAST had heritability over 0.01 in DATA2.

Correlations between PTA of sires with at least 50 daughters in DATA1 are presented in Table 6. The correlation was generally above 0.9 showing that evaluations for most traits stay similar independently of the sampling frame. However for MET, the correlation was 0.87. Of all health traits, MET was the trait with the lowest heritability.

DISCUSSION

Compared with the incidences previously reported by Kelton et al. (1998), lactational incidence for DATA1 was relatively low for MET (5.3% vs. 10.1%). McLaren et al. (2006) reported higher incidence than in the present study for MAST (7.7% vs. 21.8%) and RP (4.4% vs. 9.1%). There seems to be underreporting for these diseases; MAST is a very common disease in dairy populations and is often treated without requiring the intervention of a veterinarian. This might be a reason for not recording all occurrences of this disease. Difficulty of diagnosis might be the reason for the lower incidence of MET, as the diagnostics for this disease before 20 DIM requires an assessment of the uterus. On the other hand, lactational incidence for the metabolic

disease LDA was similar to the values in McLaren et al. (2006; 3.1% vs. 4.1%), but higher than the median reported by Kelton et al. (1998; 1.7%). Kelton et al. (1998) included some Finnish herds which have different types of cows which are not representative of Canadian conditions. In DATA2, where the conditions for inclusion of data were less stringent, incidences decreased for all traits. However, for LDA, the incidence seemed to be closer to the value reported in previous studies (1.4% vs. 1.7%; Kelton et al., 1998). Based on these observations and on the fact that LDA necessitates surgery and, therefore, the intervention of a veterinarian, reporting for this trait is likely accurate.

Animals with a case of another disease often eat less and, as a consequence, develop a ketotic condition with symptoms similar to KET, but with different causes. This likely leads to an uncertainty of diagnosis and a lower reporting of the disease. This is one of the reasons of the low lactational incidence for KET in DATA2.

Comparable studies using producer-recorded data are few. Data sampling reported by Zwald et al. (2004) was a minimum lactation incidence rate per herd for the herd to be included in the analysis. These authors also applied a maximum lactation incidence rate to remove herds recording preventive treatment of some disease (e.g. LAME). More recently Appuhamy et al. (2009) kept only herd-years with health records for at least 2 diseases. In the present study a system similar to the one presented by Appuhamy et al. (2009) was proposed.

Underreporting has been shown to decrease the accuracy of genetic evaluations (Schaeffer, 2009) and therefore to decrease the rate of genetic progress. In the present study, underreporting was also present, as the minimum requirement for inclusion of a

farm in the analyses had a massive influence on the incidence of the traits. Based on the results from 2 different sampling frames, many producers do not record all 8 traits, but only a few of them. At the most a quarter of the herds collecting data on MAST, were recording LDA events. The most probable reasons are that only certain diseases are of concern in those dairy operations and producers tend to record only those diseases. Some producers record only diseases where follow up is necessary, for reasons such as milk removal or reproduction treatments. Another reason might be that some diseases are not well known to the producers. These 2 data sampling frames show that the current quality of the raw data is not sufficient to estimate accurate incidences and that stringent data sampling is needed to improve the quality of the data analyzed, by picking only herds that are collecting health data. On the other hand, sampling frames have to be carefully employed as they can remove important data and create artificially high disease incidences, as was seen for LDA and KET.

Heritability estimates for all traits using DATA1 were low. Results of the analysis with DATA2 gave even lower results. For binary traits, mean and variance are not independent. Therefore, when the mean decreases, as was the case between DATA1 and DATA2, the variance also decreases. In the present study, the phenotypic variance of DATA2 was lower than in DATA1. But sire variance decreased even more than the phenotypic variance. With so many cows without disease observation in DATA2, and therefore considered "healthy" by the model, most of the variance between sires was removed. When analysed with a linear model, variance components will therefore also depend on the incidence of the trait. This effect was found here when both datasets were compared. The lower incidence in DATA2 compared to DATA1 resulted in lower

estimates of heritability. The nature of the model used (linear model for a binary trait) is one of the reasons for such results, as location and dispersion parameters have a quadratic relationship, in a linear model. Models taking into account the binary nature of health traits (threshold models or logistic regression models) could be used to remove this problem, but a linear model was used here enabling comparison with other studies using similar models (e.g. Van Dorp et al., 1998; Kadarmideen et al., 2000).

In the following section only results from DATA1 will be discussed, when not specifically stated that DATA2 results are meant. The estimate for MAST was in the range of published heritability estimates (0.001 – 0.06; Heringstad et al., 2000), but at the lower end. Estimates of heritability for LAME and for MET were extremely low. Some studies have found heritability estimates for these traits above 0.05 (Zwald et al., 2004) and 0.04 (Ouweltjes et al., 1996) for LAME and MET, respectively. Diagnosis of these diseases by the farmer is difficult; moreover, LAME as defined by Kelton et al. (1998) regroups a number of diseases with different causes and therefore, likely different genetic pathways of resistance to these diseases. Because of the heterogeneity of the trait LAME, it is difficult to devise a correct genetic model to describe it, resulting in very low heritability estimates. Three types of uterine diseases are included in the trait MET. The same difficulty as for LAME in modelling the trait might be applied here.

Metabolic diseases (LDA and MF) had heritability estimates between 0.02 and 0.03. Other studies have reported heritability estimates as high as 0.14 for LDA (Heringstad et al., 2005). The low estimate calculated in our study might be caused by a selected dataset, as mentioned above. Instead of removing herds not collecting data, the sampling frame might have removed herds collecting data but without case of the disease

during the period studied, thus removing much of the variation occurring in the population. However, the heritability estimate of LDA from DATA2, a dataset with less selected data, was not higher than the one from DATA1. This result seems to negate the assumption that the stringent data sampling for DATA1 removed some of the genetic variance in the data. DATA2 has probably many herds with no LDA recording at all, besides having the herds with LDA recording that were removed from DATA1. This aspect probably removed the advantage of having more herds. As presented earlier in this discussion, DATA1 is probably a highly selected population for LDA, where the lactational incidence is higher than the "true incidence" of the population, and estimates for this trait might be biased. Using a dataset covering a longer period of time should remove this problem as most herds collecting the disease will eventually have at least 1 case of LDA. For MF, the estimate was lower than the estimate of heritability of 0.04 reported by Van Dorp et al. (1998). These authors used only 1st lactation animals. Other studies found estimates not different from 0 (Pryce et al., 1999). As MF has a very low incidence in 1st lactation, variance is very low and can give results for heritability that are not different from 0. In the present analysis, 1st lactation animals were removed to avoid this problem, but this removal might have made some selection in the animals included in the dataset. The almost complete absence of MF in first lactation rendered selection of animals in 1st lactation based on MF occurrence unlikely. However, animals with low production, which are less prone to MF, are at the same time more likely to be voluntarily culled during the 1st lactation.

Heritability estimates for COD was the highest of all the estimates, but it was still lower than estimates reported in other studies (Zwald et al., 2004). Diagnosis of RP, the

other reproduction trait, is generally simple, as the disease is readily observed. This disease is also commonly found in the dairy cattle population. Consequently, data sampling does not seem to have removed many herds which were collecting data on RP, but did not have any case of disease during the 17 months of the study. Although the lactational incidence found in the present study was lower than the values reported by Kelton et al. (1998), it was still substantial. Similar heritability estimates for RP had been published previously (Van Dorp et al., 1998; Pryce et al., 1999).

CONCLUSIONS

Data sampling is a crucial aspect of analysis of producer-recorded health data. Using stringent data sampling, lactational incidences slightly below the range of expected results based on other studies can be obtained. Less stringent data sampling gave very low lactational incidence estimates for all traits analysed. Based on the analyses performed in this study, careful data sampling needs to be made on the health dataset, before it can be used for genetic evaluation. Variance components depend heavily on the data sampling frame. Both requirements, too stringent or too relaxed, can cause bias in the estimates of heritability either by selecting the data or by having a low incidence, and thus having a low variance as dispersion parameters depend on location parameters. Results of the present analyses must be viewed with regard to the very short time period of data collection. Some effects of data sampling might not be the same when data are collected over a longer period. Variance components estimated with such data might describe the data sampling frame rather than the actual population. However, low heritability might be partly caused by the model used and a different model choice could

improve the results. For example, a model accounting for the binary nature of the traits (such as a threshold model; Harville and Mee, 1984) might be applied to the data. Moreover, datasets over a longer period would be beneficial to analyze health traits as permanent environment effects might be included and data collected over a few generations of cows might help separate genetic effects from environmental effects.

REFERENCES

Abdel-Azim, G. A., A. E. Freeman, M. E. Kehrli, Jr., S. C. Kelm, J. L. Burton, A. L. Kuck, and S. Schnell. 2005. Genetic basis and risk factors for infectious and non-infectious diseases in US Holsteins. I. Estimation of genetic parameters for single diseases and general health. J. Dairy Sci. 88:1199-1207.

Appuhamy, J. A. D. R. N., B. G. Cassell, and J. B. Cole. 2009. Phenotypic and genetic relationships of common health disorders with milk and fat yield persistencies from producer-recorded health data and test-day yields. J. Dairy Sci. 92:1785-1795.

Bar, D., L. W. Tauer, G. Bennett, R. N. González, J. A. Hertl, Y. H. Schukken, H. F. Schulte, F. L. Welcome, and Y. T. Gröhn. 2008. The cost of generic clinical mastitis in dairy cows as estimated by using dynamic programming. J. Dairy Sci. 91:2205-2214.

Bigras-Poulin, M., A. H. Meek, S. W. Martin, and I. McMillan. 1990. Health problems in selected Ontario Holstein cows: frequency of occurrences, time to first diagnosis and associations. Prev. Vet. Med. 10:79-89.

Groeneveld, E., M. Kovac, and N. Mielenz. 2008. VCE User's guide and reference manual. Version 6.0.

Harville, D. A., and R. W. Mee. 1984. A mixed model procedure for analyzing ordered categorical data. Biometrics 40:393-408.

Heringstad, B, Y. M. Chang, D. Gianola, and G. Klemetsdal. 2005. Genetic analysis of clinical mastitis, milk fever, ketosis, and retained placenta in three lactations of Norwegian red cows. J. Dairy Sci. 88:3273-3281.

Heringstad, B., G. Klemetsdal, and J. Ruane. 2000. Selection for mastitis resistance in dairy cattle: a review with focus on the situation in the Nordic countries. Livest. Prod. Sci. 64:95-106.

Jamrozik, J., J. Fatehi, G. J. Kistemaker, and L. R. Schaeffer. 2005. Estimates of genetic parameters for Canadian Holstein female reproduction traits. J. Dairy Sci. 88:2199-2208.

Kadarmideen, H. N., R. Thompson, and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71:411-419.

Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. J. Dairy Sci. 81:2502-2509.

McLaren, C. J., K. D. Lissemore, T. F. Duffield, K. E. Leslie, D. F. Kelton, and B. Grexton. 2006. The relationship between herd level disease incidence and a return over feed index in Ontario dairy herds. Can. Vet. J. 47:767-773.

Neumaier, A., and E. Groeneveld. 1998. Restricted maximum likelihood estimation of covariances in sparse linear models. Genet. Sel. Evol. 30:3-26.

Ouweltjes, W., E. A. A. Smolders, L. Elving, P. van Eldik, and Y. H. Shukken. 1996. Fertility disorders and subsequent fertility in dairy cattle. Livest. Prod. Sci. 46:213-220.

Pryce, J. E., B. L. Nielsen, R. F. Veerkamp, and G. Simm. 1999. Genotype and feeding system effects and interactions for health and fertility traits in dairy cattle. Livest. Prod. Sci. 57:193-201.

Schaeffer, L. R. 2009. Effects of under-reporting of health events on genetic evaluation and genetic change. Report to the Dairy Cattle Breeding and Genetics Committee. http://cgil.uoguelph.ca/dcbgc/Agenda0902/health.pdf accessed June 30, 2009

Van Dorp, T. E., J. C. M. Dekkers, S. W. Martin, and J. P. T. M. Noordhuizen. 1998. Genetic parameters of health disorders, and relationships with 305-day milk yield and conformation traits of registered Holstein cows. J. Dairy Sci. 81:2264-2270.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. J. Dairy Sci. 87:4287-4294.

Table 1. Number of health events reported for all the diseases in the dataset and number of herds reporting at least 1 record per month.

Month	Number of records	Number of herds
4-2007	2,921	794
5-2007	3,513	934
6-2007	4,301	1,110
7-2007	5,234	1,331
8-2007	5,985	1,475
9-2007	5,385	1,440
10-2007	5,987	1,527
11-2007	5,860	1,524
12-2007	5,572	1,519
1-2008	6,711	1,758
2-2008	6,423	1,660
3-2008	6,526	1,726
4-2008	6,414	1,669
5-2008	5,846	1,588
6-2008	5,186	1,394
7-2008	4,879	1,239
8-2008	2,320	762
9-2008	44	33

Table 2. Number of cows with disease (Records), total number of cows with data (sick animals and healthy contemporaries), total number of sires and total number of herds per disease when only herds with at least 1 case of the given disease were kept (DATA1). DIM is days in milk until which the data were kept.

Disease	Records	Cows	Sires	Herds	DIM
Mastitis	15,523	201,671	8,278	2,401	210
Lameness	4,550	89,041	5,741	969	210
Cystic ovarian disease	6,935	108,298	5,881	1,194	180
Displaced abomasum	4,252	137,991	6,563	1,587	60
Ketosis	2,209	61,812	4,427	722	40
Metritis / uterine disease	3,863	72,476	5,292	711	90
Milk fever	2,618	62,543	4,607	1,078	5
Retained placenta	6,286	141,638	6,923	1,544	10

Table 3. Number of cows with disease (Records), total number of cows with data (sick animals and healthy contemporaries), total number of sires and total number of herds per disease when herds with at least 1 case of any of the 8 diseases, were kept (DATA2).

Disease	Records	Cows	Sires	Herds
Mastitis	15,661	314,786	10,420	3,710
Lameness	4,684	314,786	10,420	3,710
Cystic ovarian disease	7,035	314,786	10,420	3,710
Displaced abomasum	4,483	314,786	10,420	3,710
Ketosis	2,339	314,786	10,420	3,710
Metritis / uterine disease	3,995	314,786	10,420	3,710
Milk fever	2,680	206,284	8,766	3,693
Retained placenta	6,412	314,786	10,420	3,710

Table 4. Estimated lactational incidence, sire (σ^2_s) and phenotypic (σ^2_y) variances, and heritabilities (h^2) for 8 disease traits when only data from herds with at least 1 case of the disease analyzed are kept in the dataset (DATA1).

Disease	Incidence	$\sigma^2_s \times 10^4$	$SE(\sigma^2_s) \times 10^4$	σ^2_y	h^2
Mastitis	7.7%	3.049	0.667	0.067	0.018
Lameness	5.1%	0.501	0.285	0.045	0.004
Cystic ovarian disease	6.4%	2.618	0.647	0.057	0.018
Displaced abomasum	3.1%	2.154	0.337	0.030	0.029
Ketosis	3.6%	0.772	0.289	0.033	0.009
Metritis / uterine disease	5.3%	0.247	0.207	0.046	0.002
Milk fever	4.2%	2.024	0.568	0.038	0.021
Retained placenta	4.4%	1.655	0.400	0.042	0.016

Table 5. Estimated lactational incidence, sire (σ^2_s) and phenotypic (σ^2_y) variances, and heritabilities (h^2) for 8 disease traits when data from all herds with at least 1 case of any of the 8 diseases are in the dataset (DATA2).

Disease	Incidence	$\sigma^2_s \times 10^4$	SE(σ^2_s) $\times 10^4$	σ^2_y	h^2
Mastitis	5.0%	1.191	0.260	0.043	0.011
Lameness	1.5%	0.062	0.033	0.013	0.002
Cystic ovarian disease	2.2%	0.346	0.083	0.020	0.007
Displaced abomasum	1.4%	0.453	0.066	0.014	0.013
Ketosis	0.7%	0.038	0.012	0.007	0.002
Metritis / uterine disease	1.3%	0.017	0.015	0.011	0.001
Milk fever	1.3%	0.252	0.069	0.012	0.008
Retained placenta	2.0%	0.360	0.085	0.019	0.007

Table 6. Correlations between predicted transmitting ability (**PTA**) of sire with at least 50 daughters in DATA1 with PTA from DATA2.

Disease	Number of sires in DATA1	Correlation
Mastitis	325	0.994
Lameness	172	0.957
Cystic ovarian disease	187	0.986
Displaced abomasum	244	0.988
Ketosis	145	0.934
Metritis / uterine disease	163	0.874
Milk fever	122	0.943
Retained placenta	252	0.993

Chapter 4

Use of Linear and Threshold Models for Analysis of Producer-Recorded Health Data in Canadian Holstein Cattle.

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ABSTRACT

Health traits are of paramount importance for economic dairy production. Improvement for these traits has been made with better management practices, but genetic aspects of health traits have received less attention. Dairy producers in Canada have recorded 8 health traits (mastitis, lameness, cystic ovarian disease, left displaced abomasum, ketosis, metritis, milk fever and retained placenta) since April 2007. Genetic analyses of these traits were performed in this study for the Holstein breed. Traits were analysed either individually or grouped according to biological similarities. A minimum number of diseases recorded per herd was applied to ensure a sufficient quality of disease recording in herds included in the analysis. Variance components estimation of health traits was made using 8 different models fitted for each trait; 4 sire linear models and 4 sire threshold models. The differences between models resulted from the inclusion of days at risk with or without cow effects in addition to herd, parity and sire effects. Data included 46,104 cases of any of the above diseases. Incidence ranged from 2.6% for ketosis to 9.7% for mastitis. Metritis and milk fever had an incidence below 4.0%. Heritability for all traits with any of the linear models was below 0.04. The highest heritability was for left displaced abomasum at 0.03; lameness and metritis' heritabilities were below 0.01. Heritabilities on the liability scale calculated with threshold models were between 0.02 (metritis) and 0.21 (left displaced abomasum). Converted to the observable scale, these values were close to those estimated with a linear model. There was a moderate, positive genetic correlation between left displaced abomasum and ketosis as well as between metritis and retained placenta. The effect of days at risk was

not always significant and it was negative for some traits. This was likely due to the highest risk of culling of sick animals.

INTRODUCTION

Health problems generate high costs to the dairy producers. Much attention has been given to animal health from a management perspective, in order to reduce the incidence of disease on dairy farms. A genetic component exists for most diseases (e.g. Zwald et al., 2004a), but this aspect has not been given much attention in the dairy industry. Apart from the use of somatic cell score as an indicator trait to improve resistance to mastitis, selection for health has been limited to Nordic countries (Osteras et al., 2007; Steine et al., 2008). The reason for the absence of direct selection for health traits is often the result of a lack of information suitable for analysis. Health data recording is difficult and expensive as exact diagnoses should be made and follow up of a large population is necessary. Health traits have generally a low heritability; information from a relatively large number of cows and herds is needed to obtain reliable estimates of breeding values. Given the low heritability, results of selection are not as readily observable as results of improved management practices; for this reason producers are often not too interested in making health data available for genetic analysis .

In Canada, a project was launched in 2005 to collect health data for the dairy cattle population. The 1st phase was to select the most important diseases from an economic perspective and to define clear and simple diagnoses for health disorders. The list and diagnostics of diseases of main interest were taken from Kelton et al. (1998). For the 2nd phase a national database to store producer-recorded health information was set up in 2007 and recording started in April of that year. Data recording is performed by

producers or veterinarians (in the province of Quebec); data are transmitted to the DHI association for the region (CanWestDHI, Guelph, ON for Western Canada and Ontario; Valacta, Sainte-Anne-de-Bellevue, QC for Atlantic Canada and Quebec) and loaded into the national database at the Canadian Dairy Network (**CDN**), Guelph, ON. The 3rd phase is the use of this data in programs for the dairy industry. One aspect is the preparation of management tools based on the occurrence of diseases. The other aspect is a genetic analysis of health traits, with a goal to provide genetic evaluations of bulls for the 8 traits. The present study contributes to the last aspect.

Dealing with producer-recorded data requires a good sampling system, as the recording accuracy might vary from herd to herd (Zwald et al., 2004a). Many models have been used for genetic analysis of health data. In some studies, linear models were used (e.g. Van Dorp et al., 1998; Kadarmideen et al., 2000). Linear models assume that location and dispersion parameters are independent of each other. Health events are recorded as binary data (present or absent) and with such data the assumption of independence of location and dispersion parameters is violated. Threshold models (Harville and Mee, 1984) have been proposed to deal with binary data. Some health data analyses have already used threshold models (e.g. Uribe et al., 1995; Zwald et al., 2004a, b). One of the goals of the present study is to compare different models for the analysis of health data.

The most critical time in a cow's life is the peripartum period. During this time, some of the most costly diseases occur. Many diseases are dependent on each other and the occurrence of 1 disease can have an impact on culling decisions. The same can be said of 2 diseases caused by metabolic imbalance: left displaced abomasum (**LDA**) and

ketosis (**KET**). Often, when 1 of the diseases is observed and recorded, other diseases, which might be present, are not recorded. Traits correlated to each other can be analyzed using a multiple-trait methodology. The 2 main advantages of this methodology are the increase in the accuracy of estimation of genetic effects and the reduction in the bias caused by selection before the measurement of some of the traits included (Mrode, 2005).

Animals which live longer have more opportunities to contract a disease. In addition, not all diseases have the same duration of risk. Some diseases are intimately related to parturition and have a period of occurrence limited to a few weeks (or days), while others can occur over the entire lactation period. The effect of the length of the period at risk has been accounted for in previous analyses (de Haas et al., 2002; Abdel-Azim et al., 2005) and is often called days at risk (**DAR**).

The objectives of this study were 1) to estimate variance components for 8 diseases recorded in Canadian Holsteins using single-trait or multiple-trait analyses, 2) to compare linear and threshold models, and 3) to estimate the effect of days at risk on estimates of variance components.

MATERIALS AND METHODS

Data

Data were obtained from the Canadian Dairy Network. The dataset contained animal herd book registration, sire and dam, test dates, test-day (**TD**) milk records, DIM at TD, as well as health events and dates of health events. All health events reported from the beginning of data recording (April 2007) until the end of August 2008, were available. The 8 diseases recorded were clinical mastitis (**MAST**), lameness (**LAME**),

cystic ovarian disease (**COD**), left displaced abomasum (**LDA**), ketosis (**KET**), uterine disease / metritis (**MET**), milk fever (**MF**) and retained placenta (**RP**). Definitions of these diseases were given by Kelton et al. (1998).

The number of cases of disease reported in the database is presented in Table 1. Cases of disease per month increased from less than 3,000 during the 1st month of recording to 6,711 in January 2008. The decrease in number of records during the last 3 months is due to a lag of about 2 months for the transfer of data recorded by veterinarians from the province of Quebec. This group does a high percentage of the recording and therefore an important part of the information is missing. The number of herds reporting each month was as high as 1,758; the total number of herds with at least 1 event reported over the whole period was 3,891. Table 1 also presents the number of herds having reported diseases in the previous 6 months and are reporting at least 1 case of any disease in the month of interest. The result is given as a ratio to the total number of herds having reported diseases in the previous 6 months. This value is an indication of the proportion of herds recording at least 1 case per month. About 50% of the herds record at least 1 disease event each month. Of the 3,891 herds having recorded data since 2007, 2,979 did so in 2007 only and 3,309 in 2008 only. Thus 912 new herds started recording data in 2008, but 582 herds that recorded data in 2007 stopped doing so in 2008.

Data were analysed with single-trait (MAST, LAME and COD) and multiple-trait (LDA-KET, MET-MF-RP) models. Multiple-trait analyses were conducted for traits presenting biological similarities. One group was formed by the diseases occurring immediately after parturition or being a direct consequence of calving. This group included MET, MF and RP. Another group was formed by the metabolic diseases of the

production peak, namely KET and LDA (Stengärde and Pehrson, 2002). The 3 other diseases (MAST, LAME and COD) have no obvious relationship to each other or to either of the groups and were, therefore, analysed separately with single-trait models.

The study population needed to be defined in an attempt to remove herds which underreported disease events; moreover, contemporary groups needed to be built for the analyses. Data sampling made to ensure that reporting was correctly done, was as follows: 1st, a minimum of 2 events of the same disease, or one of the diseases included for multiple-trait analyses, in a given herd were required to include that herd in the analysis. Additionally, the 1st and last event recorded in this herd had to be at least 30 days apart, for the herd to be included in the analysis. This sampling was made to remove herds in which recording of diseases was not done and herds in which diseases had only been recorded once or during a very short period of time. Herds included in the analysis were assumed to be recorded for the disease only during the time between the events recorded at the earliest and latest date. Although this assumption is not exact, as these herds were likely observed for a certain period before the first observation and after the last observation or might not have been observed for some time in-between, the interval gave a rough estimate of the period of recording (**POR**).

For each disease case, DIM was calculated. Limits in DIM were assigned to each disease. These limits were calculated as the time from parturition until 95% of the disease cases occurred. Health events happening after the DIM limit, were removed from the dataset. The removal of health events marking the start or the end of the POR of a herd did not change the POR of this herd nor the decision about the inclusion of the herd in the

dataset. The limits of DIM were 210 days for MAST and LAME, 180 for COD, 90 for MET, 60 for LDA and KET, 10 for RP and 5 for MF.

Contemporary groups were made of all animals that had a TD record in a herd kept in the analysis, and that were alive during the POR of this herd. Animals from the contemporary group that were not in the stage of lactation given by the DIM limits during the POR of the herd were removed from the dataset. When animals moved to another herd, only data from the 1st herd was used.

The covariate DAR was calculated for each animal and disease as the difference between the end date and the starting date of risk (de Haas et al., 2002). The starting date was the latest of the animal's calving date and the beginning of the herd's POR. The end date was the earliest of the date of the cow's last TD record in the herd, the date of the DIM limit for the disease or the end of the herd's POR. Animals with DAR smaller than 1 day were removed from the dataset. Only the earliest case of a disease was kept per lactation and animal.

The sizes of the edited datasets for each analysis are presented in Table 2. Over 160,000 cows were included in the MAST analysis. Mastitis was the disease with the largest number of animals in contemporary groups and the largest number of herds recording it (1,937). The dataset for LAME included 80,178 cows (4,792 of them with a case of LAME) in 792 herds. This was the disease with the vaguest diagnosis and the disease recorded in the fewest herds. Despite having a lower incidence than LAME, the dataset for metabolic diseases (LDA-KET) was larger as these diseases were recorded in more herds.

Incidences of the 8 diseases were all below 10%. They varied from 2.3% (MF) to 9.7% (MAST). Both metabolic diseases had relatively low incidences (4.0% for LDA and 2.6% for KET).

Models

There were 8 different models applied to the 5 analyses (2 multiple-trait and 3 single-trait analyses): 4 of the models were linear models and 4 of them were threshold models (Harville and Mee, 1984). Use of a linear model was made as it is generally used in genetic evaluations of Canadian Holsteins, even with binary traits (Jamrozik et al., 2005). Threshold models account for the categorical nature of the traits. The assumption is that the observable trait is the result of an unobservable, underlying variable, called liability, which is normally distributed. When the liability is below the threshold, the observation has a certain phenotype (e.g. diseased) and when it is above the threshold, the observation has another phenotype (e.g. healthy). Inclusion of DAR in the model was made to estimate the effect of this covariate on the disease. Cow within sire effect needs to be included in such an analysis, but given the short period of data included, many cows had only 1 record and the effect was partly confounded with the residual for this record.

For each model class, there was 1 model with all effects and 3 models with some effects removed. The single trait linear model (**LDCS**) was:

$$y_{ijklm} = H_i + M_j + L_k + b \cdot d_{kl} + c_l + s_m + e_{ijklm}$$

where, y_{ijklm} is a health event record in a lactation coded as 0 when a disease is present and 1 when no disease is present. For cows with many cases of diseases during the lactation, only the 1st one was kept. H_i is a fixed herd of calving effect, M_j is a fixed

month of calving effect, L_k is a fixed parity effect (with 4 classes, later lactation were included in the 4th lactation effect), d_{kl} are the DAR of the cow, b is the regression of DAR on the observation, c_l is the random cow within sire effect of the cow, s_m is a random sire effect of the sire of the cow e_{ijklm} is a residual effect for each observation. The decision to use herd instead of herd-year effect was based on the fact that more than 60% of the herds had less than 12 months of data recording. The other 40% had at the most 17 months of recording. Random effects followed a normal distribution with location parameters equal to 0 and dispersion parameters as:

$$\mathbf{V}\begin{pmatrix} c \\ s \\ e \end{pmatrix} = \begin{pmatrix} \mathbf{I}\sigma_c^2 & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{A}\sigma_s^2 & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma_e^2 \end{pmatrix}$$

where, $\mathbf{I}$ is an identity matrix, $\mathbf{A}$ is an additive relationship matrix, σ_c^2 is the cow within sire variance, σ_s^2 is the additive sire variance and σ_e^2 is the residual variance.

The other linear models had no DAR (**LCS**), no cow effect (**LDS**) or neither DAR nor cow effect (**LS**). All linear analyses were run with VCE6 (Groeneveld et al., 2008). This software estimates variance components by REML using analytical gradient methods (Neumaier and Groeneveld, 1998).

The single trait threshold model (**TDCS**) was given as follows. The binary observation y is the expression of an underlying variable l, which is given as:

$$(l_i|\boldsymbol{\theta}) \sim N(\mathbf{w}'\boldsymbol{\theta}, 1)$$

where, $\boldsymbol{\theta}$ is a vector of parameters for fixed and random effects and $\mathbf{w}$ is a row incidence vector linking $\boldsymbol{\theta}$ to the i^{th} observation (Sorensen et al., 1995).

The observation y is 1 when l is above a certain threshold (set to 0) and 0 when l is below that threshold. The threshold was set to 0 in order to obtain an identifiable likelihood. Conditionally on $\boldsymbol{\theta}$ and y, l follows a truncated normal distribution, given as:

$$p(l_i|\boldsymbol{\theta}, y_i = 1) = \frac{\phi(\mathbf{w}'\boldsymbol{\theta}, 1)}{\Phi(\mathbf{w}'\boldsymbol{\theta})} 1(l_i > 0)$$

where, $\phi(.)$ is the normal density $\Phi(.)$ is the cumulative density function of the standard normal distribution and 1(.) is the indicator function that takes the value 1 if the random variable l is larger than 0, and 0 otherwise (Sorensen et al., 1995).

A linear model of $\boldsymbol{\theta}$ on the liability scale is given as:

$$l_{ijklm} = H_i + M_j + L_k + b \cdot d_{kl} + c_l + s_m + e_{ijklm},$$

where l_{ijklm} is the liability to a disease for a lactation, and the other elements are as in the linear model. The distribution assumption is the same as for the linear model, except that the residual variance is set to 1.

Threshold model parameters were estimated by Gibbs sampling (Geman and Geman, 1984) with the software THRGIBB1F90 and post-gibbs analyses were run with POSTGIBBSF90 (Misztal et al., 2002). Flat priors were assumed for all model parameters; 100,000 samples were drawn and the first 10,000 were discarded as burn-in. Posterior means were used as estimates of the parameters and posterior standard deviations were calculated.

For the multiple-trait linear analyses, the model was:

$$\mathbf{y} = \mathbf{Xf} + \mathbf{Z}_1\mathbf{c} + \mathbf{Z}_2\mathbf{s} + \mathbf{e},$$

where, $\mathbf{y}$ is the vector of observation (traits within parities within animal), $\mathbf{f}$ is the vector of fixed effects (herds, month of calving, parity effect and regression on DAR), $\mathbf{c}$

is a vector of cow within sire effects, **s** is a vector of sire effects and **e** is the vector of residual effects, **X**, $\mathbf{Z_1}$ and $\mathbf{Z_2}$ are incidence matrices relating effects to the observations. Random effects followed multivariate normal distribution, with location parameters equal to 0 and dispersion parameters as:

$$\mathbf{V}\begin{pmatrix}\mathbf{c}\\ \mathbf{s}\\ \mathbf{e}\end{pmatrix}=\begin{pmatrix}\mathbf{I}\otimes\mathbf{P} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{A}\otimes\mathbf{G} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{I}\otimes\mathbf{R}\end{pmatrix}$$

where, **P** is a covariance matrix for cow effects, **G** is a covariance matrix for sire effects, **R** is a residual covariance matrix and $\otimes$ is the Kronecker product.

The multiple-trait model for the threshold analyses was similar to the linear model except that the observation was replaced by the liability of the observation and the **R** matrix was an identity matrix.

Estimates of heritability from the threshold models were transformed from the liability scale to the observable scale with the formula by Robertson in Dempster and Lerner (1950). The formula is:

$$h_O^2 = \frac{h_L^2 z^2}{p(1-p)},$$

where, h_O^2 is the heritability on the observable scale, h_L^2 is the heritability on the liability scale, p is the incidence of the trait and z^2 is the square of the quantile of the standard normal density function at the threshold.

Model comparison

Two methods were used to compare models. Linear models were compared using the Aikaike's information criterion (**AIC**; Akaike, 1973). The AIC is defined by

$$AIC = -2\ln(ML) + 2k,$$

where, *ML* is the maximum likelihood and k is the number of independently adjusted parameters. The model with the minimum AIC is deemed to be the best.

The 2nd method was the goodness of fit of the models. The mean squared error statistic (**MSE**) was used to estimate it. For the linear models, MSE was calculated as

$$MSE = \sum_{i=1}^{n} \frac{(\hat{y}_i - y_i)^2}{n},$$

where, $\hat{y}_i$ is the predicted value of the observation, y_i is the observed value and n is the number of observations. Solutions were estimated with PEST 4.2 (Groeneveld, 2006).

For the threshold model, MSE was calculated as

$$MSE = \frac{\sum_{k=1}^{m}\sum_{j=1}^{s}\left(y_{jk} - \sum_{k=1}^{m} a_k \hat{P}_{jk}\right)^2}{\sum_{k=1}^{m}\sum_{j=1}^{s} n_{jk}},$$

where, a_k is the frequency of event in each of the k classes (2 classes in the present study: healthy and sick); n_{jk} is the number of observations, and $\hat{P}_{jk}$ is the predicted probability, calculated as

$$\hat{P}_j = \Phi\left(\hat{t} - \mathbf{w}'_j \hat{\theta}_t\right)$$

where, $\hat{t}$ is the estimated threshold, $\mathbf{w}_j$ is a vector of the matrix **W** of incidences of fixed and random effects and $\hat{\theta}_t$ is a vector of solutions (Matos et al., 1997). In the present analysis, $\hat{t}$ was set to 0. Posterior means of solutions from each sample after the burn-in were used as solutions.

RESULTS

Estimates of heritabilities from the linear and threshold models are presented in Table 3. Within the linear models, the heritabilities were all below 0.04. There were problems of convergence with the full model for multiple-trait analyses. The binary nature of the traits, the low incidences of the diseases and the small number of animals contributing to the estimation of the cow effect were all reasons for the lack of convergence. Three traits (LAME, KET and MET) had heritability estimates below 0.01. The traits with the highest heritability estimates were LDA and MF (both at 0.03).

Threshold models generally gave higher heritabilities on the liability scale. With these analyses, the highest heritability was again for LDA (0.21 – 0.22) and MF (0.15 – 0.19). The 6 other traits had heritabilities ranging between 0.02 and 0.09. Visual analysis of Gibbs samples for COD with TDCS and TCS models showed a worse mixing for cow effects than for sire or residual effects. This problem was likely due to the small number of animals with more than 1 observation. Transformation of threshold results on the observable scale, gave slightly higher results than those from linear models, except for MF, where the heritability was lower (0.02, Table 3).

Variance components of the LCS and TCS models are presented in Table 4. In the 2 multiple-trait analyses (LDA-KET and MET-MF-RP), the genetic correlations between traits were estimated. The results from the analyses with cow effects but without DAR (LCS and TCS) are presented in Table 5. Two genetic correlations (LDA-KET, MET-RP) were moderately positive, 1 genetic correlation (MET-MF) was not different from 0 and 1 (MF-RP) had a moderate negative value with the threshold model and was not different from 0 with the linear model. All cow effects correlations were highly negative within

threshold models. For the linear analyses these correlations were moderately positive except for the correlation between MF and RP.

Results from different models among the general groups (linear or threshold) gave similar results. The largest difference was found for MF, where the removal of the cow effect increased the heritability, in both the linear and threshold models.

The solutions for DAR are presented in Table 6. They were all very close to 0. All solutions for the traits calculated with multiple-trait analyses had slightly negative values for linear models and positive values for threshold models; the opposite was found for traits analysed with single-trait models.

Linear models comparison, as given by AIC, is presented in Table 7. For all traits, the best model was LS, followed by LCS. The 2 models without DAR effects had similar AIC, and the 2 models with this effect had similar AIC. Mean squared errors of all models are given in Table 8. Threshold and linear models give similar MSE, with a tendency towards lower MSE for single-trait linear models. For multiple-trait analyses, threshold models with cow effects showed the lowest MSE. Among each type of model, the model with cow effect (LDCS, LCS and TDCS, TCS) had the lowest MSE.

DISCUSSION

The descriptive statistics showed that herd enrolment in the dairy health recording system has increased since the beginning of data recording, but less than half of all herds collecting health data have disease cases in any given month. Although in small herds, no disease may occur during a given month; most of the herds recording all 8 diseases should have at least 1 case of a disease. The results show that for some herds, data

recording is not complete. The health data recording system is very new in Canada. A look at Norwegian data shows that during the 1st few years of data recording, reporting and incidences were low (Osteras et al., 2007). Producers need time to become familiar with new management and selection tools. The reporting of health data needs to be monitored and encouraged to ensure accurate recording and consequently quality data for genetic evaluations.

Linear and threshold models Variance components estimated with linear and threshold models are not directly comparable as the former are expressed on the observable scale and the latter on the underlying scale. To deal with that a transformation of the results from the threshold model on the observable scale can be done. Heritabilities resulting from this transformation were comparable between the 2 models for all traits. For MF, heritability was slightly lower with a threshold model; the difference might be caused by difficulty of estimating variance parameters with the threshold model as a result of the extreme category problem (**ECP**, Harville and Mee, 1984), where all animals in a fixed factor occur in the category (e.g. all healthy). For MF, very few animals had occurrences of the disease in the 1st lactation.

The ranking of the models given by AIC showed that models without DAR fit the data better. This ranking also showed a better fit for models without cow effect. The latter point is caused by the fact that data were recorded during a period of less than 2 years. The cow within sire effect was difficult to estimate as a majority of animals had only 1 observation. For cows with only 1 observation, the model will confound cow effect (including permanent environmental effect and genetic effect of the cow) and residual of

the observation. This effect might still be important in a dataset spanning a longer period, but in the present data, the model with the least parameters was the one with the best fit.

Results from the MSE showed that the inclusion of cow effect tends to improve the goodness of fit. As mentioned above, many cows have only 1 observation; for these animals, cow effect has only 1 observation to fit. The goodness of fit of the model for these animals will be greatly improved by including cow effect.

Given the dependency between mean and variance in binary traits, the variance components calculated with a linear model depend on the incidence of the trait. This is not the case with threshold models. Meijering and Gianola (1985) showed that threshold models gave a better accuracy of estimated breeding values (**EBV**) than linear models, when the incidence and the heritability were low. Health traits present both of these characteristics. On the other hand, linear models give correct estimates of genetic correlations (Mäntysaari et al., 1991), do not have difficulties related to ECP and are easier to implement. The difference of standard error (or posterior standard deviation) for the heritability were generally minimal among either model (linear or threshold).

Engel et al. (1995) showed that generalized linear mixed models with a given distribution were equivalent to threshold models with the same distribution. They also mentioned that results of analyses with a probit or logit link are usually virtually equivalent. A model frequently used for non-genetic health analyses is a general linear model with a logit link. This method has rarely been used in genetic studies. However Vazquez et al. (2009) used it to compare logit and linear models for the genetic analysis of MAST. Correlation between the 2 models for estimates of sire effects was 0.94. Correlations for estimates of herd effects and for genetic effects of the animals were

larger than 0.99. The MSE for healthy animals was lower with the logit model than with the linear model, but it was higher for sick animals. Sun and Su (2009) reported that the correlation of EBV for fertility traits calculated with threshold models using a probit or a logit link was essentially 1. In the present study, no significant changes would be expected from the use of a logistic model compared to the threshold model.

Mastitis A recent publication (Olde Riekerink et al., 2008) reported an incidence rate of 23.0% in Canada, which is more than 3 times higher than the rate reported in this study. However, their incidence was calculated as number of cases per 100 cow-years at risk (36,500 DAR), whereas the incidence in the present study was calculated as the percentage of lactations with disease, and the average DAR per lactation was only 113 days (Table 9). Only 1 case per lactation was kept in the present study; therefore, the incidence was lower than in Olde Riekerink et al. (2008). The magnitude of the difference is probably a result of the inaccuracy of reporting of MAST, in the present study. Although the incidence of MAST in the present dataset was lower than in most studies (e.g. Kelton et al., 1998), the difference was not large compared to data recorded on a large scale (Zwald et al., 2004a).

Heritability of MAST estimated with a linear model was in the range of observed data (Heringstad et al., 2000), but at the lower end. A recent study reported a similar result (Negussie et al., 2008). Heringstad et al. (2000) reported that the majority of studies found heritabilities between 0.02 and 0.03, when analysis was made with a linear model.

The higher heritability estimates from the threshold model for MAST is consistent with some studies (Heringstad et al., 2000; Kadarmideen et al., 2000), but was lower than

the one found in most studies. Using data from Ontario, Uribe et al. (1995) found higher heritabilities for resistance to mastitis in 1st lactation ($h^2 = 0.15$), but when data from all cows were included, these authors found that the heritability was not different from 0.

Lameness The incidence for lameness was slightly lower than the value of 7.0% reported by Kelton et al. (1998) as the median from 39 studies. More recent studies showed a higher incidence (Zwald et al., 2004a), but the incidence rate in the large Norwegian dataset was much lower (Osteras et al., 2007). Comparison to the latter must be taken with caution as it deals with another breed of cattle and a population that has had a health recording program for many years with genetic evaluations for selection of animals for disease resistance.

Linear analysis of LAME gave a very low heritability (0.007). The phenotypic variance of this trait was also lower than in the other 2 traits evaluated with single-trait analysis. There is often a large discrepancy between observers' scoring for this trait, even after training (Thomsen et al., 2008). The only training that most producers had for the present study is a written description of LAME. The whole herd is rarely scored for LAME and only obvious cases are noticed. Only extreme cases are recorded by producers. These cases do not have a high genetic component, as injuries and management practices often play a major role in their occurrences. Moreover, a few different diseases are responsible for lameness and resistance to these diseases is likely controlled by different genes. Other studies found higher heritability (Van Dorp et al., 1998; Pryce et al., 1999), but these studies used data from smaller groups of herds with a better follow-up of herd health. Kadarmideen et al. (2000), using producer-recorded data, found slightly higher results ($h^2 = 0.02$).

Threshold models gave heritabilities that were comparable to other results reported (Zwald et al., 2004a, b).

Cystic ovarian disease Incidence reported in this study, was similar to the value published by Kelton et al. (1998) and similar to the one reported by Zwald et al. (2004a). The reporting for COD in the present dataset seems to be consistent with other studies. Cystic ovarian disease is often recorded following a veterinarian visit. Therefore the accuracy of diagnostic is higher than for other traits.

Heritability for COD with a linear model (0.015) was consistent with results based on veterinarian reports (Distl et al., 1991; Mäntysaari et al., 1993). More recent estimates with threshold models were close to 0.05 (Hooijer et al., 2001; Zwald et al., 2004a, b). Similar results were also found in the present data when using a threshold model. The low heritability for COD seems to be caused by the large influence of the producer on the observation of the trait. As long as the animal does not need to be bred, the producer will not check the ovaries for presence of cysts. Therefore, the incidence in the 1st weeks of lactation is very low and increases sharply after 70 DIM, a time when producers realise that the animal did not cycle again since calving. Of all the traits analysed, COD is the one showing the largest positive effect of DAR on risk of disease. There is a higher risk of COD in a longer lactation, but this estimate might be biased by the fact that collecting of COD data is rarely done at the beginning of lactation. Cows with short lactations, and therefore short DAR, are often not checked for COD. This situation shows the risk of confounding healthy cows and cows not observed for the disease (missing value). This problem is general for all health traits, but more acute with COD.

Left displaced abomasum / Ketosis The bivariate analysis of LDA and KET gave very low heritabilities for KET (<0.01) for linear models, while the heritabilities for LDA were from 0.03 to 0.04 and from 0.21 to 0.22 for linear and threshold models, respectively. LDA is a trait with a low incidence, but it is generally accurately recorded as its treatment always requires the intervention of a veterinarian. This shows the importance of data with a good quality for the estimation of effects. The large difference of estimates between linear and threshold models also show the impact of low incidence on estimates made with linear models.

The heritability estimates from the threshold model for LDA were similar to those reported by Uribe et al. (1995). Heritabilities for KET were similar to those reported by Pryce et al. (1999) and Kadarmideen et al. (2000), but were lower than more recent estimates of Heringstad et al. (2005; $h^2 = 0.14$). This trait does not always seem to be recorded when LDA is present, as the latter causes more economic losses. Another reason for the low heritability of KET is that it is often a result of any other disease which reduces the feed intake of cows. In that case, KET has a completely different genetic origin than when it occurs independently.

The genetic correlations between the 2 traits were moderate and positive. Zwald et al. (2004b) found a similar result. Cows with KET have a high probability of getting LDA (Stengärde and Pehrson, 2002), therefore the phenotypic correlation should be high. Results from the present dataset show no phenotypic correlation between the traits (r_p = -0.03 with LCS model). As mentioned before, for a cow with both diseases, KET is not recorded, being less costly and visible than LDA. Therefore KET is often not recorded when LDA is present. With complete recording, the correlations (phenotypic and genetic)

might be even higher. This observation is corroborated by the cow effects correlation between the 2 traits with the threshold model. The value of -0.95 for this correlation shows that animals having 1 disease are not susceptible to the other.

An aspect worth mentioning is that LDA can happen only once in an animal's life. When a case of LDA is detected, a surgery is generally performed and it ensures that the animal will not have a 2nd incidence of the disease later in life. This might have an impact on the cow effects correlation estimates as only 1 case of LDA is possible for a cow during her entire life; reducing the incidence of LDA in later lactations, for animals susceptible to LDA in 1st lactation.

Metritis / Milk fever / Retained placenta The 3 traits closely linked to calving were analysed with a multiple-trait model. Heritabilities were low for all 3 traits, but especially for MET (0.001 and 0.03 with linear and threshold models, respectively). This is in agreement with the result reported by Ouweltjes et al. (1996). Estimates of heritability for MF were from 0.03 to 0.04. This is in agreement with Van Dorp et al. (1998) and similar to Kadarmideen et al. (2000) and Zwald et al. (2004b). Some studies found higher estimates for the heritability of MF (up to 0.4; Lin et al., 1989), but these studies generally used data from later lactations for variance components estimation. As the incidence is changed with this inclusion criterion, it has an effect on the variance components calculated with linear models. Heritability for RP was 0.02 with a linear model, corresponding to results published in previous studies (Distl et al., 1991; Van Dorp et al., 1998; Pryce et al., 1999). The estimate based on the threshold model was higher (0.09). Heringstad et al. (2005) found the same result for each of the first 3 lactations.

Genetic correlations between MET and MF were not significantly different from 0. This was expected as the biology of these diseases is different (infectious vs. metabolic causes) although both diseases are influenced by events at parturition. There is a moderate to high positive genetic correlation between MET and RP. The longer period during which the birth canal is open in case of RP, leave the uterus exposed to infection and therefore to MET (Benzaquen et al., 2007). Other studies found similar correlations (Lin et al., 1989). The genetic correlation between RP and MF was moderate and negative. This result was surprising as hypocalcaemia (and therefore MF) render the animal more susceptible to RP (Goff and Horst, 1997). Moreover, Heringstad et al. (2005) found a low positive correlation between MF in 2nd and 3rd lactations and RP in any of the first 3 lactations. On the other hand, these authors reported that MF in the 1st lactation had a weak negative genetic correlation with RP, but these results were not significant. The inclusion, in the present study, of MF from all lactations might have caused a different genetic correlation with RP.

Use of DAR Regression coefficients for DAR did not show positive relationships to all diseases. Moreover, the sign of the effect was changed between linear and threshold models. For the 3 diseases with the longest period at risk (MAST, LAME and COD), the regression coefficient was positive with linear models. Abdel-Azim et al. (2005) reported a value of 0.005 for udder health and 0.004 for COD, with a threshold model. The analyses made with a threshold model in the present study gave contradictory results (-0.02 for MAST and -0.005 for COD).

For the 5 other diseases (LDA, KET, MET, MF and RP), the regression coefficients either had a negative relationship with the occurrence of disease or did not

have any effect with linear models. All 5 diseases had short period at risk (5 – 60 days). For MF, 96% of the animals were at risk for the maximum DAR possible; for RP 94% were at risk for 10 days (maximum DAR). Figure 1 shows the distribution of the animals according to DAR for RP. Animals with 10 DAR have been left out of the figure: they represent 79% and 94% of the sick and healthy animals, respectively. This data structure removed most of the variance in that trait for the analysis; consequently the estimates are based on a few animals. Averages of DAR for all traits are presented in Table 9. If animals are grouped according to health status, there is a difference in the average between sick and healthy animals of 0.7 days for RP (9.1 days vs. 9.8 days for sick vs. healthy animals), 0.3 days for MF (4.6 days vs. 4.9 days) and 5.4 days for LDA (42.4 days vs. 47.8 days). These results corroborate the effects calculated in the genetic analysis with linear models. Abdel-Azim et al. (2005) using the whole length of lactation for all diseases found a slightly positive value for the regression coefficients on DAR for LDA, MET and MF. This result was also obtained in the present study, when using threshold models.

CONCLUSIONS

Producer-recorded data were used for genetic analyses of health traits. Incidence of diseases can be low with this kind of data, due to underreporting. Definition of the study population is therefore important for obtaining reliable data.

Variance components for 8 health disorders were calculated and 3 of them (LAME, KET and MET) showed a heritability below 0.01. Genetic improvement for these traits would be very slow. Analyses with linear models showed an advantage in

using models without DAR effect. The inclusion of cow effect improved the goodness of fit of the models. The use of threshold models did not improve the goodness of fit of the single-trait models. Given the increased complexity of the implementation of threshold models and their lack of improvement of goodness of fit over the linear models, it might be better to use linear models for genetic evaluation of health traits in Canada.

Inclusion of DAR did not influence estimates of parameters. Regression coefficient for DAR showed contradictory relationship with diseases between linear and threshold models, and for some diseases these effects were due to the short period at risk considered in the analysis.

Health data recorded by producers can be used for genetic evaluations. There is variance in disease resistance explained by genetic effects enabling selection for this resistance. But the very low heritability estimates calculated based on the present dataset imply that no genetic progress will be made unless health traits are heavily weighted in the selection programme and the selection index. Moreover, in the present progeny testing structure, the number of daughters will likely be too small for bulls with new proof to have an accurate prediction. Stability of proofs for health traits will likely be lacking. A close monitoring of the completeness of data recording and stringent data validation would be needed to ensure good data quality and accurate evaluations.

REFERENCES

Abdel-Azim, G. A., A. E. Freeman, M. E. Kehrli, Jr., S. C. Kelm, J. L. Burton, A. L. Kuck, and S. Schnell. 2005. Genetic basis and risk factors for infectious and non-

infectious diseases in US Holsteins. I. Estimation of genetic parameters for single diseases and general health. J. Dairy Sci. 88:1199-1207.

Akaike, H. 1973. Information theory and an extension of the maximum likelihood principle. 2nd Int. Symp. Information Theory. B.N. Petrov and F Csaki, ed. Akademiai Kiado, Budapest, Hungary.

Bar, D., L. W. Tauer, G. Bennett, R. N. González, J. A. Hertl, Y. H. Schukken, H. F. Schulte, F. L. Welcome, and Y. T. Gröhn. 2008. The cost of generic clinical mastitis in dairy cows as estimated by using dynamic programming. J. Dairy Sci. 91:2205-2214.

Benzaquen, M. E., C. A. Risco, L. F. Archibald, P. Melendez, M.-J. Thatcher, and W. W. Thatcher. 2007. Rectal temperature, calving-related factors, and the incidence of puerperal metritis in postpartum cows. J. Dairy Sci. 90:2804-2814.

de Haas, Y., H. W. Barkema, and R. F. Veerkamp. 2002. Genetic parameters of pathogen-specific incidence of clinical mastitis in dairy cows. Anim. Sci. 74:233-242.

Dempster, E. R., and I. M. Lerner. 1950. Heritability of threshold characters. Genetics 35:212-236.

Distl, O., M. Ron, G. Francos, E. Mayer, and H. Kraeusslich. 1991. Genetic analysis of reproductive disorders in Israeli Holstein dairy cows. Theriogenology 35:827-836.

Engel, B., W. Buist, and A. Visscher. 1995. Inference for threshold models with variance components from the generalized linear mixed model perspective. Genet. Sel. Evol. 27:15-32.

Geman, S., and D. Geman. 1984. Stochastic relaxation, Gibbs distributions , and the Bayesian restoration of images. IEEE T. Pattern Anal. 6:721-741.

Goff, J. P., and R. L. Horst. 1997. Physiological changes at parturition and their relationship to metabolic disorders. J. Dairy Sci. 80:1260-1268.

Groeneveld, E. 2006. PEST User's Manual.

Groeneveld, E., M. Kovac, and N. Mielenz. 2008. VCE User's guide and reference manual. Version 6.0.

Harville, D. A., and R. W. Mee. 1984. A mixed model procedure for analyzing ordered categorical data. Biometrics 40:393-408.

Heringstad, B, Y. M. Chang, D. Gianola, and G. Klemetsdal. 2005. Genetic analysis of clinical mastitis, milk fever, ketosis, and retained placenta in three lactations of Norwegian red cows. J. Dairy Sci. 88:3273-3281.

Heringstad, B., G. Klemetsdal, and J. Ruane. 2000. Selection for mastitis resistance in dairy cattle: a review with focus on the situation in the Nordic countries. Livest. Prod. Sci. 64:95-106.

Hooijer, G. A., R. B. F. Lubbers, B. J. Ducro, J. A. M. van Arendonk, L.M. T. E. Kaal-Lansbergen, and T. van der Lende. 2001. Genetic parameters for cystic ovarian disease in Dutch black and white dairy cattle. J. Dairy Sci. 84:286-291.

Jamrozik, J., J. Fatehi, G. J. Kistemaker, and L. R. Schaeffer. 2005. Estimates of genetic parameters for Canadian Holstein female reproduction traits. J. Dairy Sci. 88:2199-2208.

Kadarmideen, H. N., R. Thompson, and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71:411-419.

Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. J. Dairy Sci. 81:2502-2509.

Lin, H. K., P. A. Oltenacu, L. D. Van Vleck, H. N. Erb, and R. D. Smith. 1989. Heritabilites of and genetic correlations among six health problems in Holstein cows. J. Dairy Sci. 72:180-186.

Mäntysaari, E. A., Y. T. Gröhn, and R. L. Quaas. 1993. Repeatability and heritability of lactational occurrence of reproductive disorders in dairy cows. Prev. Vet. Med. 17:111-125.

Matos, C. A., D. L., Thomas, D. Gianola, M. Perez-Enciso, and L. D. Young. 1997. Genetic analysis of discrete reproductive traits in sheep using linear and nonlinear models: II. Goodness of fit and predictive ability. J. Anim. Sci. 75:88-94.

Meijering, A., and D. Gianola. 1985. Linear versus nonlinear methods of sire evaluations for categorical traits: a simulation study. Gen. Sel. Evol. 17:115-132.

Misztal, I., S. Tsuruta, T. Strabel, B. Auvray, T. Druet, and D. H. Lee. 2002. BLUPF90 and related programs (BGF90). In: 7th World Congress on Genetics Applied to Livestock Production, Montpelier, 19-23 August 2002.

Mrode, R. A. 2005. Linear models for the prediction of animal breeding values. 2nd edition. CABI Publishing, Wallingford, UK

Negussie, E., I. Strandén, and E. A. Mäntysaari. 2008. Genetic association of clinical mastitis with test-day somatic cell score and milk yield during first lactation of Finnish Ayrshire cows. J. Dairy Sci. 91:1189-1197.

Neumaier, A., and E. Groeneveld. 1998. Restricted maximum likelihood estimation of covariances in sparse linear models. Genet. Sel. Evol. 30:3-26.

Olde Riekerink, R. G. M., H. W. Barkema, D. F. Kelton, and D. T. Scholl. 2008. Incidence rate of clinical mastitis on Canadian dairy farms. J. Dairy Sci. 91:1366-1377.

Osteras, O., H. Solbu, A. O. Refsdal, T. Roalkvam, O. Filseth, and A. Minsaas. 2007. Results and evaluation of thirty years of health recordings in the Norwegian dairy cattle population. J. Dairy Sci. 90:4483-4497.

Ouweltjes, W., E. A. A. Smolders, L. Elving, P. van Eldik, and Y. H. Shukken. 1996. Fertility disorders and subsequent fertility in dairy cattle. Livest. Prod. Sci. 46:213-220.

Pryce, J. E., B. L. Nielsen, R. F. Veerkamp, and G. Simm. 1999. Genotype and feeding system effects and interactions for health and fertility traits in dairy cattle. Livest. Prod. Sci. 57:193-201.

Sorensen, D. A., S. Andersen, D. Gianola, and I. Korsgaard. 1995. Bayesian inference in threshold models using Gibbs sampling. Genet. Sel. Evol. 27:229-249.

Steine, G., D. Kristofersson, and A. G. Guttormsen. 2008. Economic evaluation of the breeding goal for Norwegian Red dairy cattle. J. Dairy Sci. 91:418-426.

Stengärde, L. U., and B. G. Pehrson. 2002. Effects of management, feeding, and treatment on clinical and biochemical variables in cattle with displaced abomasum. Am. J. Vet. Res. 63:137-142.

Sun, C., and G. Su. 2009. Comparison on models for genetic evaluations of non-return rate and success in first insemination of the Danish Holstein cows. Livest. Sci. doi:10.1016/j.livsci.2009.09.015

Thomsen, P. T., L. Munksgaard, and F. A. Togersen. 2008. Evaluation of a lameness scoring system for dairy cows. J. Dairy Sci. 91:119-126.

Uribe, H. A., B. W. Kennedy, S. W. Martin, and D. F. Kelton. 1995. Genetic parameters for common health disorders of Holstein cows. J. Dairy Sci. 78:421-430.

Van Dorp, T. E., J. C. M. Dekkers, S. W. Martin, and J. P. T. M. Noordhuizen. 1998. Genetic parameters of health disorders, and relationships with 305-day milk yield and conformation traits of registered Holstein cows. J. Dairy Sci. 81:2264-2270.

Vazqzez, A. I., D. Gianola, D. Bates, K. A. Weigel, and B. Heringstad. 2009. Assessment of Poisson, logit, and linear models for genetic analysis of clinical mastitis in Norwegian Red cows. J. Dairy Sci. 92:739-748.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004a. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. J. Dairy Sci. 87:4287-4294.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004b. Genetic selection for health traits using producer-recorded data. II. Genetic correlations, disease probabilities and relationships with existing traits. J. Dairy Sci. 87:4295-4302.

Table 1. Data characteristics: proportion of herds reporting is the ratio of herds which recorded data in the month of interest to the herds having a record in the previous 6 months.

Month	Number of records	Number of herds	Proportion of herds reporting – 6 months
4-2007	2,921	794	
5-2007	3,513	934	
6-2007	4,301	1,110	
7-2007	5,234	1,331	
8-2007	5,985	1,475	
9-2007	5,385	1,440	
10-2007	5,987	1,527	54%
11-2007	5,860	1,524	52%
12-2007	5,572	1,519	50%
1-2008	6,711	1,758	56%
2-2008	6,423	1,660	52%
3-2008	6,526	1,726	55%
4-2008	6,414	1,669	52%
5-2008	5,846	1,588	49%
6-2008	5,186	1,394	41%
7-2008	4,879	1,239	35%
8-2008	2,320	762	22%

Table 2. Description of edited data. Number of disease records, total number of animals with data (sick animals and healthy contemporaries), total number of herds per disease.

Disease	Records	Total	Herds	Incidence
Mastitis	16,095	165,535	1,937	9.7%
Lameness	4,792	80,178	792	6.0%
Cystic ovarian disease	7,631	96,523	1,007	7.9%
Displaced abomasum	3,362	84,749	1,231	4.0%
Ketosis	2,191	84,749	1,231	2.6%
Metritis / uterine disease	3,113	97,058	1,604	3.2%
Milk fever	2,241	97,058	1,604	2.3%
Retained placenta	5,679	97,058	1,604	5.9%

Table 3. Estimates of heritabilities for 8 diseases (MAST = clinical mastitis; LAME = lameness; COD = cystic ovarian disease; LDA = left displaced abomasum; KET = ketosis; MET = metritis/uterine disease; MF = milk fever; RP = retained placenta) from 8 different models: linear (L) or threshold (T), including days at risk (D) and cow within sire (P) effects. Standard errors for linear models (SE) and standard deviations of posterior standard deviation for threshold models (PSD) are given as an average of the 4 models. Results from the threshold model converted to the observable scale are also given (TCSo)

Disease	LDCS	LDS	LCS	LS	SE	TDCS	TDS	TCS	TS	PSD	TCSo
MAST	0.016	0.017	0.017	0.017	0.004	0.047	0.048	0.048	0.050	0.010	0.016
LAME	0.006	0.007	0.007	0.007	0.003	0.043	0.044	0.045	0.046	0.017	0.011
COD	0.015	0.015	0.014	0.014	0.004	0.052	0.052	0.046	0.047	0.012	0.014
LDA	NC[1]	0.034	0.034	0.034	0.005	0.210	0.214	0.214	0.213	0.034	0.041
KET	NC	0.007	0.009	0.007	0.002	0.088	0.088	0.090	0.092	0.026	0.013
MET	NC	0.001	0.001	0.001	0.001	0.032	0.033	0.032	0.022	0.014	0.005
MF	NC	0.032	0.029	0.031	0.006	0.152	0.186	0.157	0.181	0.038	0.021
RP	NC	0.017	0.016	0.016	0.004	0.067	0.081	0.066	0.076	0.016	0.016

[1]NC = result not converged

Table 4. Variance components estimates for 8 diseases (MAST = clinical mastitis; LAME = lameness; COD = cystic ovarian disease; LDA = left displaced abomasum; KET = ketosis; MET = metritis/uterine disease; MF = milk fever; RP = retained placenta) obtained with a linear or threshold models including cow within sire and sire effects (LCS and TCS). Standard errors (for linear model) and standard deviations of posterior standard deviation (for threshold models) are given in brackets.

Disease	Linear model (LCS)			Threshold model (TCS)	
	$10^3\ \sigma^2_s$	$10^3\ \sigma^2_{pe}$	$10^3\ \sigma^2_e$	σ^2_s	σ^2_{pe}
MAST	0.35	1.57	81.3	0.01	0.09
	(0.08)	(0.58)	(0.63)	(0.003)	(0.014)
LAME	0.09	0.88	51.9	0.01	0.06
	(0.04)	(0.58)	(0.63)	(0.005)	(0.033)
COD	0.25	8.48×10^{-8}	69.5	0.01	0.06
	(0.06)	(0.0004)	(0.32)	(0.003)	(0.024)
LDA	0.31	1.06	36.0	0.08	0.48
	(0.03)	(0.28)	(0.33)	(0.015)	(0.078)
KET	0.05	0.47	23.2	0.04	0.57
	(0.01)	(0.22)	(0.22)	(0.009)	(0.085)
MET	0.01	0.17	27.2	0.01	0.85
	(0.01)	(0.21)	(0.24)	(0.007)	(0.120)
MF	0.15	2.65	18.0	0.11	1.78
	(0.04)	(0.32)	(0.33)	(0.029)	(0.279)
RP	0.21	3.46	49.6	0.03	0.98
	(0.05)	(0.69)	(0.70)	(0.007)	(0.098)

[1]NC = result not converged

Table 5. Estimates of genetic correlations for 5 diseases (LDA = left displaced abomasum; KET = ketosis; MET = metritis/uterine disease; MF = milk fever; RP = retained placenta) from a linear and a threshold model including cow within sire and sire effects (LCS and TCS). Standard error (for linear model) and standard deviations of posterior means (for threshold model) are given in brackets.

Disease	Linear model (LCS)			Threshold model (TCS)		
	KET	MF	RP	KET	MF	RP
LDA	0.53			0.58		
	(0.14)			(0.13)		
MET		-0.05	0.50		0.08	0.79
		(0.27)	(0.26)		(0.31)	(0.32)
MF			-0.39			-0.02
			(0.15)			(0.10)

Table 6. Estimates of the regression of day at risk (DAR) on 8 diseases (MAST = clinical mastitis; LAME = lameness; COD = cystic ovarian disease; LDA = left displaced abomasum; KET = ketosis; MET = metritis/uterine disease; MF = milk fever; RP = retained placenta) from 4 different models: linear (L) or threshold (T), including cow within sire (P) and sire effects (S).

Disease	LDCS	LDS	TDCS	TDS
MAST	0.0003	0.0003	-0.021	-0.020
LAME	0.0003	0.0003	-0.025	-0.025
COD	0.0006	0.0006	-0.005	-0.005
LDA	NC[1]	-0.0004	0.006	0.005
KET	NC	-0.0001	0.003	0.002
MET	NC	0	0.002	0.001
MF	NC	-0.0223	0.261	0.267
RP	NC	-0.0250	0.177	0.143

[1]NC = result not converged

Table 7. Akaike's Information Criterion (AIC) for 4 linear models including days at risk (D) and cow within sire effect (C). Diseases included in the 5 different runs are MAST = clinical mastitis; LAME = lameness; COD = cystic ovarian disease; LDA = left displaced abomasum and KET = ketosis; MET = metritis/uterine disease, MF = milk fever and RP = retained placenta.

Disease	LDCS	LDS	LCS	LS
MAST	-330,723	-330,746	-332,219	-332,237
LAME	-161,678	-161,689	-162,490	-162,497
COD	-194,298	-194,300	-197,406	-197,408
LDA – KET	NC[1]	-352,874	-353,040	-353,076
MET – MF – RP	NC	-580,265	-584,700	-584,889

[1]NA = results not converged

Table 8. Mean squared error statistic for 8 diseases (MAST = clinical mastitis; LAME = lameness; COD = cystic ovarian disease; LDA = left displaced abomasum; KET = ketosis; MET = metritis/uterine disease; MF = milk fever; RP = retained placenta) from 8 different models: linear (L) or threshold (T), including days at risk (D) and cow within sire (P) effects.

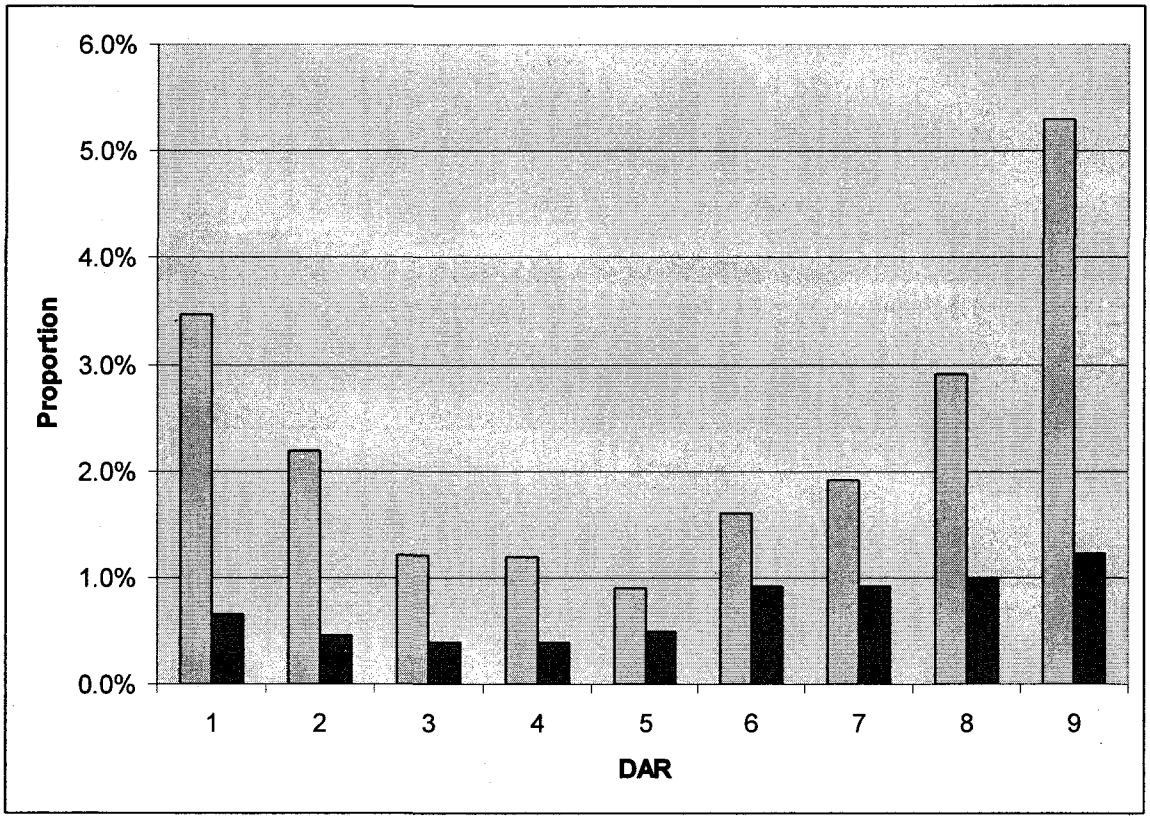
Disease	LDCS	LDS	LCS	LS	TDCS	TDS	TCS	TS
MAST	0.080	0.081	0.079	0.082	0.083	0.087	0.083	0.088
LAME	0.050	0.052	0.050	0.052	0.051	0.054	0.053	0.055
COD	0.067	0.067	0.069	0.069	0.069	0.071	0.070	0.072
LDA-KET	NC[1]	0.030	0.028	0.030	0.024	0.030	0.024	0.030
MMR	NC	0.033	0.029	0.033	0.021	0.033	0.021	0.037

[1]NA = results not converged

Table 9. Mean days at risk (DAR) for 8 diseases (MAST = clinical mastitis; LAME = lameness; COD = cystic ovarian disease; LDA = left displaced abomasum; KET = ketosis; MET = metritis/uterine disease; MF = milk fever; RP = retained placenta) according to the status of the animal (sick or healthy) and limit of DIM for inclusion of diseases.

Disease	Overall mean DAR	Mean DAR among sick animals	Mean DAR among healthy animals	Limit of DIM
MAST	113	131	112	210
LAME	109	131	108	210
COD	105	133	102	180
LDA	48	42	48	60
KET	48	46	48	60
MET	73	74	73	90
MF	4.9	4.6	4.9	5
RP	9.7	9.1	9.8	10

Figure 1 Distribution (in %) of animals by days at risk (DAR) for retained placenta (RP) according to health status: left columns are animals with a case of RP and right columns are healthy animals.



Chapter 5

Multivariate Analyses of Producer-Recorded Health Events and BCS in Canadian Holstein Cattle.

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ABSTRACT

Improvement of genetics of health is sought after by dairy producers, but it is at the same time difficult to achieve as heritability of health traits is low and amount of data collected is limited. Selection for health traits could be enhanced by using data on correlated traits. One such trait is body condition score (**BCS**), a measure of the fat deposition of a cow, that is used as an indicator of metabolic balance. Health records for 8 traits (mastitis, lameness, cystic ovarian diseases, left displaced abomasum, ketosis, metritis, milk fever and retained placenta) and multiple BCS records per lactation were available for Canadian Holsteins from the province of Quebec. Diseases were recorded by producers and checked for plausibility by veterinarians before being included in the national database. Six of the 8 health traits were grouped into: reproductive disease (cystic ovarian disease, metritis and retained placenta) and metabolic disease (left displaced abomasum, ketosis and milk fever).

The relationship between BCS and disease resistance was analyzed by 2 different modelling approaches. The first was a multiple-trait model in which BCS recorded at 5 different time periods in the lactation were considered as different traits. Mastitis and reproductive disease before and after 50 days in milk were also considered as different traits. The second modelling approach was a bivariate (BCS and 1 health trait) longitudinal model.

Overall, there was a positive additive genetic correlation between BCS and resistance for mastitis, lameness and metabolic disease ($r_g \leq 0.59$). Genetic correlations were negative between BCS and resistance to reproductive disease in the middle of lactation using the longitudinal model, and with resistance to lameness across the

lactation using the multivariate model. Overall genetic correlations between BCS and resistance to diseases were similar using both models, with the exception of lameness were the sign of the correlation was reversed. The highest positive correlations were generally at the beginning of the lactation for the multivariate analysis and around the 3rd month of lactation for the longitudinal analysis. All BCS traits had highly positive genetic correlations with each other and heritability estimates were in the range of values reported by other studies. Heritability estimates for health traits were all low and below 0.02.

Use of BCS as an indicator trait could improve selection for most of the health traits. Selection index combining disease resistance traits with BCS or multiple-trait analyses using BCS as a correlated trait to disease resistance traits would increase accuracy of selection against diseases.

INTRODUCTION

In Canada diseases of dairy cows are reported by producers and recorded in a national database since 2007. As diseases have a profound impact on the economic outcomes of dairy farms, they need to be closely monitored in order to take all measures required to reduce their incidence. Several European countries, Norway (Osteras et al., 2007), Denmark, Finland and Sweden (Johansson et al., 2008), have central databases to record health data. In these countries cows' resistance to diseases has improved since data collecting began (Osteras et al., 2007). In the Nordic countries, veterinarian initiated treatments are recorded (Heringstad et al., 2003), as opposed to producer diagnosed and recorded health information in Canada. However, in one of Canada's provinces, Quebec,

some of the recording is either made or validated by veterinarians. Collection of health data in Canada had 2 main objectives. Health data are used as a herd management tool, to detect problematic diseases in herds. Data are also used for genetic evaluations. Many authors reported existence of genetic variance for health traits (e.g. Kadarmideen et al., 2000; Zwald et al., 2004a). Some authors also found that many health traits were inherited together (e. g. Zwald et al., 2004b).

Body Condition Score (**BCS**) is a subjective measure used to assess the amount of fat and muscle in specific body parts of dairy cows (Wildman et al., 1982; Edmonson et al., 1989). It has been successfully used to assess the energy balance of dairy cows, which is an indication of the metabolic state of the cow. An increasing number of countries such as Belgium, Germany, the Netherlands, the Nordic countries, Switzerland and the UK already compute estimated breeding values (**EBV**) for BCS (Interbull, 2009). This trait is generally considered a good predictor of the cow's energy balance and therefore has been used as an indicator trait for disease resistance (particularly metabolic diseases) and fertility (Lawlor and Klei, 2008). Some of the diseases recorded in Canada have a metabolic cause, such as left displaced abomasum (**LDA**), ketosis (**KET**) and milk fever (**MF**; Tveit et al., 1992; van Winden et al., 2004; Ingvartsen, 2006) and could therefore be correlated to BCS. Relationships between BCS and health have already been reported at a phenotypic level. Gearhart et al. (1990) reported a higher risk of over-conditioned cows (high BCS) for metritis (**METR**) and cystic ovarian disease (**COD**) occurrences. Hoedemaker et al. (2009) reported a better resistance to endometritis and lameness for cows with a high BCS at calving; these cows were also at lower risk of not being cycling. Cows with a low BCS at 10 weeks postpartum were also at a higher risk of retained

placenta (**RP**) in the subsequent lactation. On a genetic level, a study by Lassen et al. (2003) reported negative (favourable) genetic correlation between BCS and mastitis (**MAST**) occurrence, and between BCS and occurrence of diseases other than MAST for Danish Holsteins. Dechow et al. (2004) carried similar analysis on US Holsteins, they estimated positive (unfavourable) genetic correlations between BCS and reproductive diseases, but reported negative correlations with other diseases (among which was MAST).

Multiple-trait analysis is a common approach for correlated traits in genetic evaluations. This method is used to analyze multiple functional traits such as reproduction (e. g. Jamrozik et al., 2005). Multivariate analyses increase the reliability of EBV and allow the estimation of BV for all traits for animals having records for only a few of them. Multivariate analysis of health traits has already been carried out (Zwald et al., 2004b; Neuenschwander et al., 2009) and positive genetic correlations were found between some of them. Joint analysis of mastitis with an indicator trait (somatic cell score) has also been made to improve the accuracy of evaluations (Johansson et al., 2006).

Analysis of traits with repeated measurement at different time intervals (longitudinal traits) has been proposed with random regression models. This method accounts for the covariance structure of repeated records during a specific period of time (Jamrozik et al., 1997).

The objectives of this study were: 1) to examine the relationships between BCS and disease resistance in Canadian Holstein using multiple-trait approach; and 2) to apply

a random regression model and a multiple-trait model to analyze producer recorded health data.

MATERIALS AND METHODS

Data

Data were obtained from the Canadian Dairy Network (**CDN**, Guelph, ON, Canada). The "health" data contained records from the Canadian National Health Database received through the DS@HR (**DSA** dossier santé animale – animal health records), the computer software used by veterinarians from Quebec. This dataset contained only health events validated by veterinarians. Producers use DSA mainly for reproduction management. Health data collection is done in many of these herds with the help of a veterinarian (Émile Bouchard, Université de Montréal, Saint-Hyacinthe, QC, personal communication). Data in the "health" dataset were collected from April 2007 (the beginning of health recording on a national scale) to December 2008. There were 53,219 health events recorded in this dataset. Only health data from the first three lactations were kept. The eight diseases recorded were clinical mastitis (**MAST**), lameness (**LAME**), cystic ovarian disease (**COD**), left displaced abomasum (**LDA**), ketosis (**KET**), uterine disease / metritis (**METR**), milk fever (**MF**) and retained placenta (**RP**). Definitions of these diseases were given by Kelton et al. (1998).

The "BCS" dataset had information about BCS for Quebec dairy herds. Valacta (Quebec and Atlantic Canada DHI association) offers to its member herds the recording of BCS by their staff on a voluntary basis. This information is primarily used for management purposes like herd grouping for feeding (Robert Moore, Valacta, Sainte-

Anne-de-Bellevue, QC, personal communication). The "BCS" dataset included data from January 2000 until September 2008. Scores were available for cows in the first three parities and were taken on a scale from 1 (thin) to 5 (fat) (at increments of 0.25) (Edmonson et al., 1989). A BCS could be recorded several times during lactation and during the dry period. Herds with less than 5 cows recorded across the data set were deleted. Across the data set, herds had to have a BCS standard deviation higher than 0.25 units. Then, BCS records for a given herd × test-day were not included in the analyses, if less than five records were taken at that herd × test-day. Finally, BCS records taken after 335 days in milk (**DIM**) were deleted and cows with a drying period greater than 80 days were eliminated. Body condition score was available for 179,821 cows with 666,201 records. There was an average of 2.5 BCS records per cow-lactation. Distribution of the number of BCS records according to the DIM and evolution of BCS along the lactation are presented in Figures 1 and 2.

The "test-day" dataset contained test-day milk records for the period of January 2007 to February 2009. It totalled over 5 million records. As the "health" dataset included only cows with a case of a disease, the "test-day" dataset was used to add all the healthy cows present in herds in which cows with a case of disease were.

Contemporary groups for health traits were built according to the method described in Neuenschwander et al. (2009). In short, only herds having at least 2 cases of the health trait analyzed were kept. Moreover, the first and last case of the disease had to be at least 30 days distant from each other. This editing removed herds having done health recording for only a short period of time. Finally, all herds without data on BCS were removed from the analysis. The number of herds for each trait is given in Table 1.

The time between the first and last cases of a disease was called the period of recording (**POR**) for a particular herd. Cases of diseases occurring after 335 days in milk were not used. All animals without cases of disease and having a test-day record during a period 30 days before the start of the POR and 30 days after the end of the POR of the herd were kept to form contemporary groups. The inclusion of 30 days before and after the POR was made to include animals that might have been culled shortly before the start of the POR and 30 days were chosen as the average interval between two milk tests. The number of cows with records on health and BCS was from 13,146 to 24,812 depending on the disease considered (Table 1).

Health traits were coded as the presence (0) or absence (1) of disease diagnostic per lactation. Disease resistence, as defined in this study, is the absence of occurrence of the disease or, for a sire, a higher proportion of daughters without occurrences of the disease. Therefore only the first case of a disease for a given cow-lactation was kept in the data. Health traits, other than MAST and LAME, were also grouped according to their etiology, following the grouping used in the Nordic countries (Johansson et al., 2006, 2008). The groups were:

- Metabolic diseases (**METAB**): LDA, KET and MF
- Reproductive diseases (**REPR**): COD, METR and RP

Mastitis and LAME are traits which have a unique etiology among the 8 health traits and they were analyzed separately. All 3 metabolic diseases are caused by imbalances in the metabolism of the cow: MF is caused by hypocalcaemia, LDA and KET, 2 closely related diseases (Stengärde and Pehrson, 2002), are caused by a high mobilization of fat reserve. All three events occur during early lactation when production

is at its peak and were therefore grouped. The 3 reproductive diseases, although having very different causes, are all related to the reproductive system of the cow. COD is a disease of the ovaries and seems to be caused by hormonal problems; METR and RP are disorders of the uterus which are generally caused by events related to calving. Two 2 diseases, MAST and REPR, were further split into 2 categories: (1) early cases occurring in the first 50 DIM (**EMAST** and **EREPR**) and (2) late cases occurring after 50 DIM (**LMAST** and **LREPR**). The other 2 diseases were kept as 1 trait, given that the etiology does not change during the lactation (LAME) or the disease occurs only at the beginning of the lactation (METAB).

Models

Data on BCS and health were analyzed with multiple-trait and longitudinal models. The multiple-trait model included BCS recorded at different times of the lactation as different traits. There were 5 "BCS traits" defined: (1) BCS at 0-10 DIM, (2) BCS at 11-40 DIM, (3) BCS at 41-100 DIM, (4) BCS at 101-200 DIM and (5) BCS at 201-335 DIM. The number of cows with BCS records in each of the 5 traits, when data were edited for MAST and BCS were: 1,114, 3,423, 5,119, 6,215 and 5,202. When a cow had more than 1 record during a given period, only the 1st BCS record was kept. Health traits (MAST, LAME, METAB and REPR) were also included in the analysis. Therefore, the models were a 6-trait analyses when LAME and METAB were included. The models were 7-trait analysis when MAST and REPR were used, as these two traits were split in 2 (EMAST and LMAST; EREPR and LREPR).

The linear model used was as follows:

$$y_{ijklmop} = S_{ij} + A_{ik} + h_{ijln} + s_{ijlno} + c_{ijlmn} + e_{ijklmnop}$$

where $y_{ijklmop}$ is the p^{th} record of the m^{th} cow, daughter of the o^{th} sire for trait i (BCS or health), S_{ij} is the j^{th} season x 2 years effect for BCS and season effect for health; A_{ik} is the k^{th} random herd-2 years-season and herd-season effect for BCS and health, respectively; this is a deviation from the fixed effect S_{ij} for a given herd; h_{ijln} is the n^{th} fixed regression coefficient for a trait i specific to the l^{th} age – parity class for BCS and parity for health; s_{ijlno} is the n^{th} random regression coefficient for a trait i specific to the sire o of cow m; c_{ijlmn} is the n^{th} random regression coefficient for a trait i specific to cow m; and $e_{ijklmop}$ is the residual of a specific record for a trait of interest.

In matrix form, it was:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{h} + \mathbf{Z}_2\mathbf{c} + \mathbf{Z}_3\mathbf{s} + \mathbf{e}$$

where, **y** was the vector of observations for BCS and one of the health traits; **b** was the vector of fixed effects S and A; **h** was the vector of random effects of herd × class of two years of calving × season for BCS and the vector of random effect of herd × season of calving for health traits; **c** was the vector of cow effects for BCS and health; **s** was the vector of the random additive sire effects for BCS and health; **e** was a vector of residuals; **X** and $\mathbf{Z_i}$ (i=1, 3) were incidence matrices assigning observations to effects.

Variance-covariance structure of the effects was:

$$\mathbf{V}\begin{pmatrix} h \\ c \\ s \\ e \end{pmatrix} = \begin{pmatrix} \mathbf{H} \otimes \mathbf{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{C} \otimes \mathbf{I} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{S} \otimes \mathbf{A} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{R} \otimes \mathbf{I} \end{pmatrix}$$

where, **H**, **C**, **S** and **R** are the covariance matrices for herd-season, cow, sire and residual effects, respectively. All 4 matrices are (6 x 6) matrices for the models with LAME and METAB and (7 x 7) matrices for the models with MAST and REPR. **A** is the

additive genetic relationship matrix, a square matrix of the dimension of the number of animals in the pedigree files of each and **I** is an identity matrix.

The 2nd modelling approach was based on a random regression model with Legendre polynomials (Jamrozik et al., 1997). Traits were defined as BCS or health (MAST, LAME, METAB or REPR) in a given interval of lactation. The first interval was defined as DIM 5-30; the following intervals all included a period of 30 days and the 11th interval included 34 days and was from DIM 301 to 335. When multiple records of BCS were available on a cow during the same interval, only the first score was kept. Health events were defined as presence (0) or absence (1) of the disease during the interval of interest. Therefore the health trait represents a resistance to disease. Cows' health status was assumed to be monitored constantly and all health events were recorded: in each interval of the lactation, when the cow was not recorded as sick, it was assumed that she was healthy. Observations were assigned to intervals instead of DIM as in Averill et al. (2006). This was driven by the fact that the daily incidence of the diseases was very low. This would cause the variance to be extremely low.

The linear random regression model was:

$$y_{ijklmop} = SA_{ij} + AG_{ik} + \sum_{n=0}^{3} \lambda_{ijln} z_n(t) + \sum_{n=0}^{2} \alpha_{ijlno} z_n(t) + \sum_{n=0}^{2} \gamma_{ijlmn} z_n(t) + e_{ijklmop}$$

where $y_{ijklmop}$ is the p^{th} record of the m^{th} cow, daughter of the o^{th} sire for trait i (BCS or health), SA_{ij} is the j^{th} season x 2 years effect for BCS and season effect for health; AG_{ik} is the k^{th} random herd-2 years-season and herd-season effect for BCS and health, respectively; this is a deviation from the fixed effect SA_{ij} for a given herd; λ_{ijln} is the n^{th} fixed regression coefficient for a trait i specific to the l^{th} age – parity class for BCS

and parity for health; $z_n(t)$ is the n^{th} covariate to describe the shape of the curve of fixed and random effects; α_{ijlno} is the n^{th} random regression coefficient for a trait i specific to the sire o of cow m; γ_{ijlmn} is the n^{th} random regression coefficient for a trait i specific to cow m; and $e_{ijklmop}$ is the residual of a specific record for a trait of interest. The z covariates were 3rd order Legendre polynomials for the fixed regression and 2nd order Legendre polynomials for the random regressions (Bastin et al., 2009). They were:

$$z_0(t) = 1$$

$$z_1(t) = \sqrt{3}x$$

$$z_2(t) = \sqrt{5}\left(\frac{3}{2}x^2 - \frac{1}{2}\right)$$

$$z_3(t) = \sqrt{7}\left(\frac{5}{2}x^3 - \frac{3}{2}x\right)$$

where x is

In matrix notation, the model can be described as:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_1h} + \mathbf{Z_2s} + \mathbf{Z_3c} + \mathbf{e}$$

with

$$V\begin{pmatrix} h \\ c \\ s \\ e \end{pmatrix} = \begin{pmatrix} \mathbf{H} \otimes \mathbf{I} & \mathbf{0} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{C} \otimes \mathbf{A} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{S} \otimes \mathbf{I} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{R} \otimes \mathbf{I} \end{pmatrix}$$

where, **H**, **C**, **S** and **R** are the covariance matrices for herd-season, cow, sire and residual effects, respectively. All 4 matrices are (6 x 6) matrices for the models with LAME and METAB and (7 x 7) matrices for the models with MAST and REPR. **A** is the additive genetic relationship matrix, a square matrix of the dimension of the number of animals in the pedigree files of each and **I** is an identity matrix.

All models were fitted with the software REMLF90 (Misztal et al., 2002). This software estimates variance components based on the EM REML algorithm with acceleration (Misztal et al., 2002).

RESULTS

Estimates of genetic parameters from the multivariate analysis of BCS and health are given in Tables 2 and 3. Heritability estimates of BCS are reported as the average of the estimates calculated with each of the 4 bivariate analyses; 1 analysis was made for each disease or disease group (MAST, LAME, REPR and METAB).

Heritability of BCS ranged between 0.09 and 0.21, and it was highest at the beginning of lactation (DIM 0-10) and the lowest during the 3rd and 4th months of lactation. Heritability estimates were again higher toward the end of the lactation.

Genetic correlations between estimates of BCS at different times in lactation were high and positive. The highest correlation was between the 2 periods around the production peak. Correlations were generally higher between BCS at neighbouring time periods.

Heritability estimates for health traits were all low (Table 3). The highest estimate (0.02) was for METAB. Heritability estimate for LAME was extremely low (0.002). Genetic correlations between BCS and health were from moderately positive to moderately negative. Correlations between BCS and MAST in late lactation (>50 DIM) were always positive (0.07 – 0.59), meaning that a cow with a high BCS had a better resistance to MAST. For early MAST (<50 DIM), the correlation to the first BCS trait (0-11 DIM) was similar to the one found with late MAST but the correlation was not or

tentatively negatively correlated for the other BCS traits. It must be stressed that BCS after 50 DIM (last 3 BCS traits) was measured after the occurrence (or non-occurrence) of the disease and should therefore be considered as a result rather than a cause of the MAST recording.

Cows with a genetic potential for high BCS tended to have a poorer LAME resistance throughout the lactation (r_g from -0.13 to -0.54), whereas metabolic problems were positively correlated to BCS (more resistance as BCS increases). Early reproductive diseases also had a positive genetic correlation with BCS. Cows with a higher BCS had less genetic risk for these diseases. On the other hand, correlation between BCS at 11-40 DIM and EREPR was 0.02. This period is the time of the highest incidence for METR. Finally, LREPR had low genetic correlations with BCS.

Heritability estimates of BCS, from the longitudinal analyses are reported as averages of 4 bivariate analyses with health traits (Figure 3). Estimates ranged from 0.11 to 0.22. The lowest heritability estimates were for the period from the 2nd to the 3rd month of lactation. This corresponds to the lactation peak period. Heritability increased to its highest value towards the end of the lactation. Heritability for the 1st month of lactation (0.12) was only slightly higher than the minimum estimate calculated for the lactation peak period.

Figure 4 summarizes heritability estimates for all health traits from the longitudinal model. All estimates were below 0.04 for each month of lactation. For all traits, the heritability was highest during the first 2 months of lactation and it decreased below 0.005 sometime later during the lactation. Estimates for lameness were very low and stayed below 0.01 for the whole lactation. Metabolic diseases also had a low

heritability which was unlike the result found with multivariate analysis. Estimate for MAST was at 0.02 during the 1st month but fell to the level of LAME and METAB after the 4th month. Finally REPR had a different pattern of heritability: relatively high (0.04) at the beginning of the lactation, dropping to 0 at the 5th month, before increasing again to the level of other traits later in lactation.

Genetic correlations between BCS with MAST, LAME and METAB were moderate and positive along the whole lactation period. Correlations were highest at the 4th month for MAST and METAB (0.34 and 0.33, respectively). For LAME, the highest correlation was at the 6th month (0.37). Estimate of genetic correlation between BCS and REPR showed larger variation during the lactation, changing signs 3 times along the lactation.

DISCUSSION

The incidence estimates as reported in this study are lower than those reported by Kelton et al. (1998) and Zwald et al. (2004a). Although health data was verified by veterinarians, it was still originally recorded by producers. Verification by veterinarians was a simple assessment at the time of the herd visit, if the disease was likely to have happened, based on time of calving and surgical interventions. This means that under-reporting was possible as producers might not have reported some occurrences of a disease.

Variation of BCS scores over the lactation was more extreme than reported by de Haas et al. (2007). Average BCS at the beginning of lactation was also 2.9, but these authors reported a minimum BCS of 2.7 around the production peak, which was higher

than the value of 2.4 reported in the present study. The difference might be caused by the scoring method. BCS in our study were recorded at different times in the lactation, including periods when some cows are very thin, whereas the study by de Haas et al. (2007) used classification results.

Heritabilities for BCS were in the range of other reported values, but at the lower end (e.g. Lassen et al., 2003; Interbull, 2009). Using different models, but the same dataset, Bastin et al. (2009) found similar range of heritabilities for BCS, but these authors reported the highest heritability at the middle of lactation and the lowest one at both end.

Estimate of heritability for MAST was similar for early and late lactation, and slightly lower value reported by Neuenschwander et al. (2009) based on the whole Canadian population. The lower value was partly due to the low incidence of the trait, as location and dispersion parameters are not independent for binary traits. Use of a threshold model would remove this problem, but extreme category problems arise with the small dataset used in this study. Rekaya et al. (2003) reported a higher heritability (0.21-0.57) at the beginning of lactation using a threshold random regression model. The shape of the heritability along the lactation reported by these authors was similar to the one found in the present study. Converting heritability estimates from the observable scale to an underlying scale (Dempster and Lerner, 1950) gives heritability of 0.06 at the start of lactation and 0.006 in the middle of the lactation, which are still lower than the heritability estimates reported by Rekaya et al. (2003) on the underlying scale. Heritability estimates for MAST with the longitudinal model decreased with lactation stage. The highest heritability was reported during the 1st month of lactation. This is

consistent with results published by Carlén et al. (2009) who used a similar model to analyse mastitis in Swedish Holsteins. There seems to be a higher genetic component to MAST resistance at the beginning of the lactation. More environmental factors are responsible for MAST occurrence as the lactation continues because exposure to bacterial source of MAST is continuous.

Generally, lameness had a very low heritability. This disease was defined as "any difficulty with mobility" (Kelton et al., 1998). The causes of this disorder are multiple and they are very likely controlled by different genes. In a study of Nordic countries' Holsteins, a low heritability (0.01) was also reported for LAME (Johansson et al., 2008). Lameness needs a more precise definition (at least to the level of separating infectious causes from metabolic causes), in order to separate the genetic and environmental components. Other countries have used the specific lesions responsible for lameness as traits observed and have done a genetic analysis of these traits finding a higher heritability (e.g. Koenig et al., 2005). Use of a threshold model would probably give a higher estimate of heritability on the underlying scale (Zwald et al., 2004a, Neuenschwander et al., 2009), but not necessarily on the observable scale, and phenotypic improvement needs to be done on the observable scale.

Reproductive traits had a slightly higher heritability (0.008 for EREPR and 0.014 for LREPR). Johansson et al. (2008) reported similar estimates (0.02 and 0.01 for EREPR and LREPR, respectively). Similar to LAME, reproductive disease is a group of different diseases that are not all highly correlated to each other (Zwald et al., 2004b). The advantage of grouping diseases is to have a higher incidence of the "disease" as well as a larger amount of data available, as some producers might only record 1 of the diseases

included in reproductive diseases. The estimate for EREPR is close to the median of the heritability estimates of the traits composing EREPR, when comparing the heritability for reproductive disease in the present analysis with those reported previously for each disease separately (Neuenschwander et al., 2009). With a longitudinal analysis, heritability estimates for reproductive diseases are higher at the beginning of the lactation (0.04). At the beginning of lactation, the trait "reproductive disease" describes mainly RP, as the other diseases are nonexistent at this time of lactation and therefore do not contribute to the variance of the trait. From the end of the 2nd month, COD starts to be recorded, as it is the time when producers start breeding their cows again. Changes in heritability during the lactation might be due to the diseases included (COD vs. RP). On the other hand, some changes, especially at the beginning and at the end of the lactation, might be an effect of the Legendre parameters.

The last trait (METAB) has the highest heritability of all at 0.02. This value was higher than reported by Johansson et al. (2008). Two of the diseases included (LDA and MF) have even higher heritability when analysed as single traits and the 3rd (KET) is moderately correlated to LDA (Neuenschwander et al., 2009). Using a longitudinal model, heritability was lower during the whole lactation. Some producers treat all cows prophylactically for MF and record it as a treatment. Taking this aspect into consideration in the model could increase the accuracy of the heritability estimate for MF.

Genetic correlations

The high positive genetic correlations between BCS traits were expected. Genetic predisposition for high body condition is independent of the stage of lactation. The only correlation below 0.8 was between BCS at 11-40 DIM and BCS at 201-335 DIM. This is

BCS at peak yield and BCS at the end of lactation, 2 time periods where environment and management have a large influence on body condition.

Genetic correlations between BCS and both MAST traits estimated with the multivariate model showed that a high BCS at the beginning of lactation is better for resistance to MAST. Lassen et al. (2003) reported an unfavourable correlation between BCS and disease occurrence. These authors did not differentiate the time of BCS recording and they kept only MAST cases before 50 DIM in their analysis.

The negative correlation between BCS from 41-100 DIM to the end of lactation and MAST < 50 DIM has to be interpreted differently than other correlations. As BCS is recorded after the observation for health, this result might show the effect of health on BCS rather than the reverse, but the exact causality should be further investigated. During that period, the correlations were closer to 0, indicating that the effect is less strong and that these traits are becoming more independent of each other. The slight negative correlation indicates that healthy cows have a lower BCS. The reason for this might be the ability of healthy cows to produce more milk than a mastitic cow and therefore to keep a lower body condition. Longitudinal analysis gave higher positive correlation for the latter part of the lactation. The interpretation is not exactly the same for this model. Here the genetic correlation is between BCS and health at the same month in milk, whereas in the multiple-trait model, it was the correlation between BCS at a given time in the lactation and MAST at any time before or after 50 DIM.

Estimates for LAME gave contradictory results between the longitudinal and the multiple-trait models. The longitudinal model indicates a positive correlation, in agreement with Collard et al. (2000) who compared energy balance and health. The

multiple-trait model gave negative correlations, with a larger magnitude in the 2nd part of the lactation. Some LAME problems, such as laminitis, are caused mainly by feeding plan. Some producers adapt their feeding plan (or build feeding groups) according to the BCS of their cow. A more significant correlation would be between the change in BCS during the lactation and LAME, as the change in BCS is also a result of feeding. A decrease could mean lower feed intake or higher production, depending on the level of BCS. The effect of these 2 events on LAME will be different.

There is a major disagreement between the multiple-trait and the longitudinal models as regards genetic correlation between LAME and BCS. The reason for this should be looked for in the extremely low heritability estimate for LAME. The daily disease incidence of this disease remains constant through the lactation (results not shown) and is constantly at a low level. Gearhart et al. (1990) reported that BCS and foot problems do not have a linear relationship. Both low BCS cows and high BCS cows were at higher risk of foot problems. This non-linear relationship is probably applicable to most health traits.

The positive correlation between EREPR and BCS at calving indicates that cows with a higher BCS will be less susceptible to RP as this is the main trait of EREPR. The correlation is closer to 0 later in lactation, as the time of recording of BCS and EREPR is not the same. The large positive genetic correlation between BCS at the end of the lactation and EREPR seems to indicate that cows without reproduction problems store more reserves at the end of the lactation. During this period there might be a confounding effect with pregnancy. Cows without reproductive problems will get pregnant earlier than cows with RP or COD and will therefore be close to calving at the end of lactation (335

DIM). The more advanced pregnancy will induce a lower production and a higher fat deposition.

All correlations were closer to 0 among LREPR and BCS. The traits in LREPR are COD and METR, 2 traits which are mainly caused by hormonal and infectious factors. The longitudinal analysis showed the same pattern as the multiple-trait analysis for the beginning of lactation, but correlation becomes negative further along. The incidence of diseases changes drastically between the 1st month and later months, as RP is only present during the 1st week of lactation. This change makes it difficult to model reproductive diseases after the 1st month of lactation and is probably the cause for the irregular correlations in the 2nd half of lactation.

Finally, resistance to metabolic diseases is positively correlated to BCS. This was expected as BCS is also a measure of the metabolic state of dairy cows. The correlation was only moderate and showed a similar pattern for both models: higher at both ends of the lactation and lower in the middle. BCS is a trait with an intermediate optimal value. A very high BCS can be as detrimental to metabolic health as a low BCS. Therefore, the correlation cannot be extremely high. However, as the Holstein population average is low for BCS, there is still a small positive correlation between BCS and METAB. If the Holstein cow attained the ideal BCS for metabolic health, there should be a zero genetic correlation between BCS and METAB, as both an increase as well as a decrease of BCS would be connected to more metabolic disorders.

CONCLUSIONS

There was a positive genetic correlation between BCS and health traits in Canadian Holsteins. As accurate BCS recording is easier to do than accurate health recording, BCS could be used as an indicator trait in selection for more health. For some health traits, genetic correlation to BCS is low, and selection for BCS would only improve health marginally. The optimal approach would be to combine BCS and health traits in a selection index or to use BCS as a correlated trait in the genetic evaluations of health traits.

The non-linear relationship between BCS and health must be kept in mind, when applying BCS as an indicator trait for selection. In the present population, a higher BCS is desired for a better disease resistance, but this relationship might be inversed in a population with a high average BCS.

Use of multiple-trait analysis and longitudinal analysis gave similar genetic correlations between BCS and the different health traits, except LAME. However, longitudinal analyses gave even lower heritability for health traits that multiple traits and this could be a hindrance for selection for health traits. Therefore, multiple-trait analysis could be more appropriate for a practical use of health and BCS in breeding programmes.

REFERENCES

Averill, T., R. Rekaya, and K. Weigel. 2006. Random regression models for male and female fertility evaluation using longitudinal binary data. J. Dairy Sci. 89:3681-3689.

Bastin, C., S. Loker, N. Gengler, and F. Miglior. 2009. Genetic relationships between body condition score and reproduction traits for Canadian first-parity cows. J. Dairy Sci. (submitted).

Carlén, E., K. Grandinson, U. Emanuelson, and E. Strandberg. 2009. Random regression models for genetic evaluations of clinical mastitis in dairy cattle. Animal 3:1-9.

Collard, B. L., P. J. Boettcher, J. C. M. Dekkers, D. Petitclerc, and L. R. Schaeffer. 2000. Relationships between energy balance and health traits of dairy cattle in early lactation. J. Dairy Sci. 83:2683-2690.

de Haas, Y., L. L. G. Janss, and H. N. Kadarmideen. 2007. Genetic correlations between body condition scores and fertility in dairy cattle using bivariate random regression models. J. Anim. Breed. Genet. 124:277-285.

Dechow, C. D., G. W. Rogers, U. Sander-Nielsen, L. Klei, T. J. Lawlor, J. S. Clay, A. E. Freeman, G. Abdel-Azim, A. Kuck, and S. Schnell. 2004 Correlations among body condition scores from various sources, dairy form, and cow health from the United States and Denmark. J. Dairy Sci. 87:3526-3533.

Dempster, E. R., and I. M. Lerner. 1950. Heritability of threshold characters. Genetics 35:212-236.

Edmonson, A. J., I. J. Lean, L. D. Weaver, T. Farver, and G. Webster. 1989. A body condition scoring chart for Holstein dairy cows. J. Dairy Sci. 72:68-78.

Gearhart, M. A., C. R. Curtis, H. N. Erb, R. D. Smith, C. J. Sniffen, L. E. Chase, and M. D. Cooper. 1990. Relationship of changes in condition score to cow health in Holsteins. J. Dairy Sci. 73:3132-3140.

Heringstad, B., R. Rekaya, D. Gianola, G. Klemetsdal, and K. A. Weigel. 2003. Genetic change for clinical mastitis in Norwegian Cattle: a threshold model analysis.

Hoedemaker, M., D. Prange, and Y. Gundelach. 2009. Body condition change ante- and postpartum, health and reproductive performance in German Holstein cows. Reprod. Dom. Anim.44:167-173.

Ingvartsen, K. L. 2006. Feeding- and management-related diseases in the transition cow. Physiological adaptations around calving and strategies to reduce feeding-related diseases. Anim. Feed Sci. Tech. 126:175-213.

Interbull. 2009. Description of national genetic evaluation systems for dairy cattle traits as applied in different Interbull member countries. International Bull Evaluation Service, Uppsala, Sweden. www.interbull.org. Accessed August 30, 2009.

Jamrozik, J., J. Fatehi, G. J. Kistemaker, and L. R. Schaeffer. 2005. Estimates of genetic parameters for Canadian Holstein female reproduction traits. J. Dairy Sci. 88:2199-2208.

Jamrozik, J. G. J. Kistemaker, J. C. M. Dekkers, and L. R. Schaeffer. 1997. Comparison of possible covariates for use in a random regression model for analyses of test day yields. J. Dairy Sci. 80:2550-2556.

Johansson, K., S. Eriksson, J. Pösö, M. Toivonen, U. S. Nielsen, J.-A. Eriksson, and G. P. Aamand. 2006. Genetic evaluation of udder health traits for Denmark, Finland and Sweden. Interbull Bull. 35:92-96.

Johansson, K., J. Pösö, U. S. Nielsen, J.-A. Eriksson, and G. P. Aamand. 2008. Joint genetic evaluation of other disease traits in Denmark, Finland and Sweden. www.nordicebv.info, Accessed on April 30, 2009.

Kadarmideen, H. N., R. Thompson, and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71:411-419.

Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. J. Dairy Sci. 81:2502-2509.

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.

Lassen, J., M. Hansen, M. K. Sørensen, G. P. Aamand, L. G. Christensen, and P. Madsen. 2003. Genetic relationship between body condition score, dairy character, mastitis, and diseases other than mastitis in first-parity Danish Holstein cows. J. Dairy Sci. 86:3730-3735.

Lawlor, T. J., and B. Klei. 2008. Results of the pilot study for Body Condition Score and locomotion. Interbull Bull. 38:96-100.

Misztal, I., S. Tsuruta, T. Strabel, B. Auvray, T. Druet, and D. H. Lee. 2002. BLUPF90 and related programs (BGF90). In: 7th World Congress on Genetics Applied to Livestock Production, Montpelier, 19-23 August 2002.

Neuenschwander, T. F.-O., F. Miglior, J. Jamrozik, and L. R. Schaeffer. 2009. Use of linear and threshold models for analysis of producer-recorded health data in Canadian Holstein cattle. Report Dairy Cattle Breeding and Genetics Committee, February 3, 2009. http://cgil.uoguelph.ca/dcbgc/Agenda0902/agenda0902.htm

Osteras, O., H. Solbu, A. O. Refsdal, T. Roalkvam, O. Filseth, and A. Minsaas. 2007. Results and evaluation of thirty years of health recordings in the Norwegian dairy cattle population. J. Dairy Sci. 90:4483-4497.

Rekaya, R., D. Gianola, K. Weigel, and G. Shook. 2003. Longitudinal random effects models for genetic analysis of binary data with application to mastitis in dairy cattle. Genet. Sel. Evol. 35:457-468.

Schaeffer, L. R., and J. C. M. Dekkers. 1994. Random regression in animal models for test-day production in dairy cattle. Proc. 5th World Congress on Genetics Applied to Livestock Production, Guelph, Canada, 18:443-446.

Tveit, B., F. Lingaas, M. Svendsen, and O. V. Sjaastad. 1992. Etiology of acetonemia in Norwegian cattle. 1. Effect of ketogenic silage, season, energy level, and genetic factors. J. Dairy Sci. 75:2421-2432.

van Winden, S. C. L., R. Jorritsma, K. E. Müller, and J. P. T. M. Noordhuizen. 2004. Feed intake, milk yield, and metabolic parameters prior to left displaced abomasum in dairy cows. J. Dariy Sci. 86:1465-1471.

Wildman, E. E., G. M. Jones, P. E. Wagner, R. L. Boman, H. F. Troutt, Jr., and T. N. Lesch. 1982. A dairy cow body condition scoring system and its relationship to selected production characteristics. J. Dairy Sci. 65:495-501.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004a. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. J. Dairy Sci. 87:4287-4294.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004b. Genetic selection for health traits using producer-recorded data. II. Genetic correlations,

disease probabilities and relationships with existing traits. J. Dairy Sci. 87:4295-4302.

Table 1. Number of herds, sires, cows, and health event lactation incidence.

Disease	Herds	Cows	Sires	Incidence
Mastitis	293	17,766	1,587	9.7%
Lameness	221	13,146	1,457	8.7%
Reproductive diseases	412	24,812	1,773	11.2%
Metabolic diseases	305	18,577	1,616	5.2%

Table 2. Heritabilities (on the diagonal) and genetic correlations (above diagonal) for multiple-trait analysis of BCS.

Trait	BCS 0-10 DIM	BCS 11-40 DIM	BCS 41-100 DIM	BCS 101-200 DIM	BCS 201-335 DIM
BCS 0-10 DIM	**0.21**	0.83	0.83	0.82	0.86
BCS 11-40 DIM		**0.11**	0.97	0.87	0.76
BCS 41-100 DIM			**0.09**	0.91	0.81
BCS 101-200 DIM				**0.13**	0.96
BCS 201-335 DIM					**0.13**

Table 3. Heritabilities (top line) and genetic correlations between BCS and health traits.with a multiple-trait analysis.

Trait	MAST < 50 DIM	MAST > 50 DIM	LAME	EREPR	LREPR	METAB
Heritability	0.013	0.012	0.002	0.008	0.014	0.020
BCS 0-10 DIM	0.39	0.59	-0.13	0.44	-0.03	0.29
BCS 11-40 DIM	0.01	0.34	-0.54	0.02	0.15	0.21
BCS 41-100 DIM	-0.16	0.21	-0.39	0.15	0.16	0.16
BCS 101-200 DIM	0.00	0.07	-0.42	0.28	0.12	0.25
BCS 201-335 DIM	0.15	0.09	-0.22	0.54	0.10	0.32

Mastitis (MAST), separated into early mastitis (<50 DIM) and late mastitis (≥ 50 DIM),

Lameness (LAME),

Early reproductive diseases (EREPR),

Late reproductive diseases (LREPR) and

Metabolic diseases (METAB).

Figure 1 Distribution of body condition score (BCS) records according to days in milk (DIM) of the lactation.

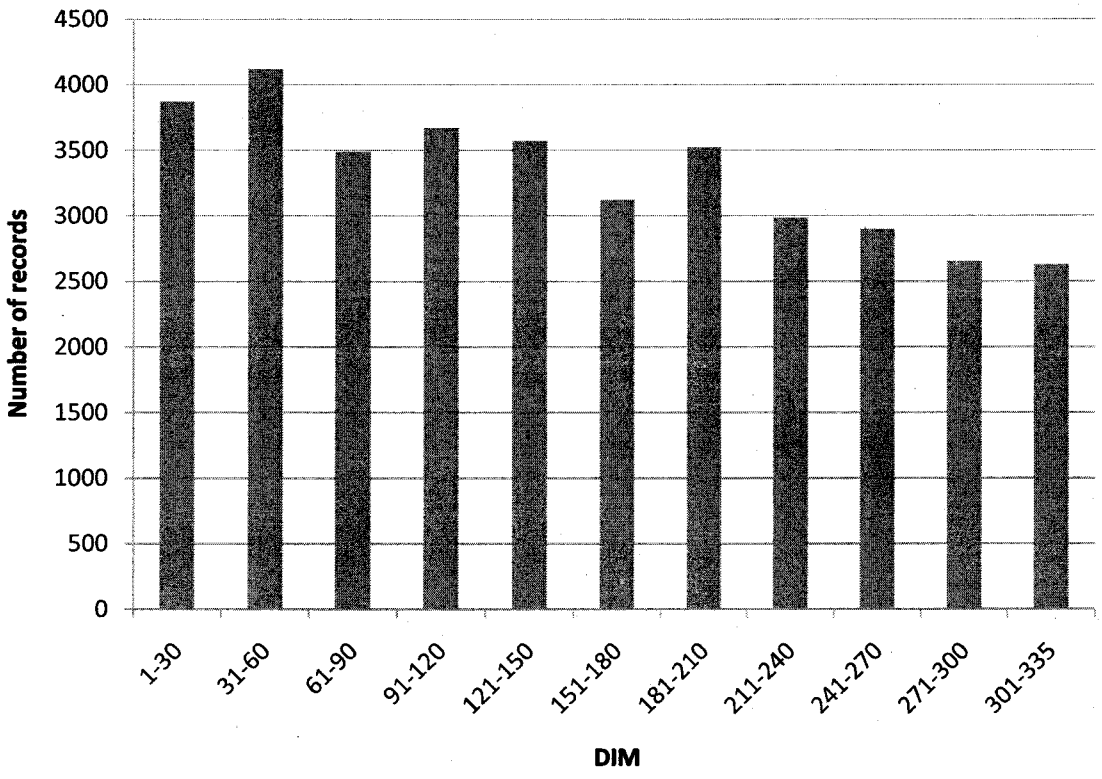


Figure 2 Average body condition score (BCS) by stage of lactation.

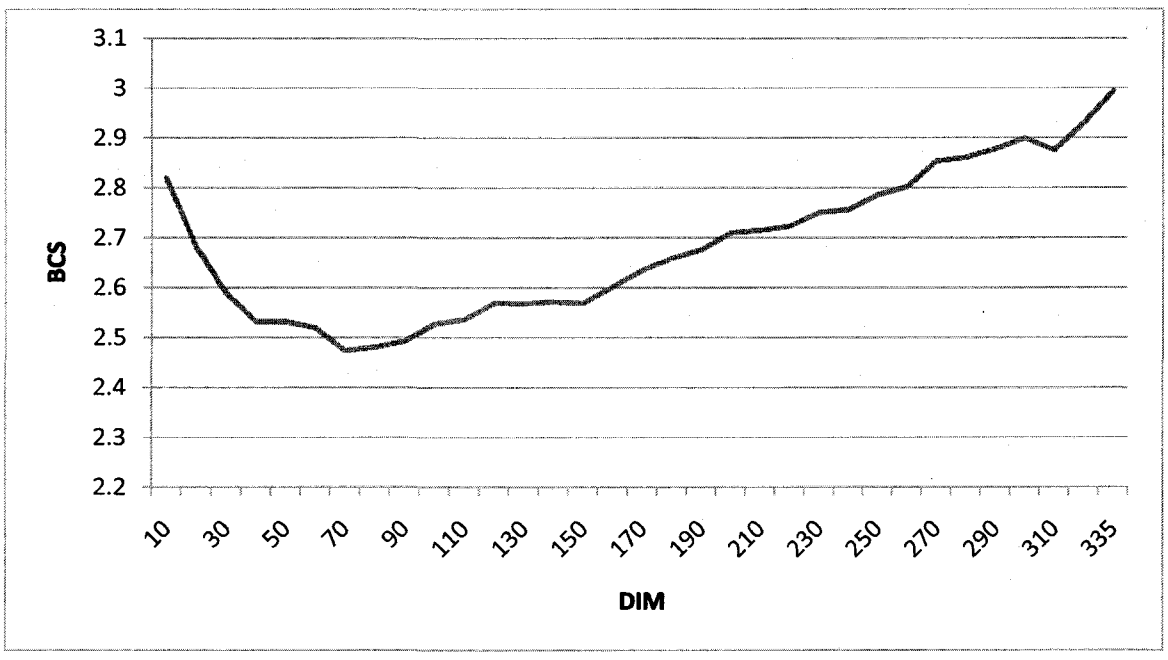


Figure 3 Heritability of BCS from the longitudinal model.

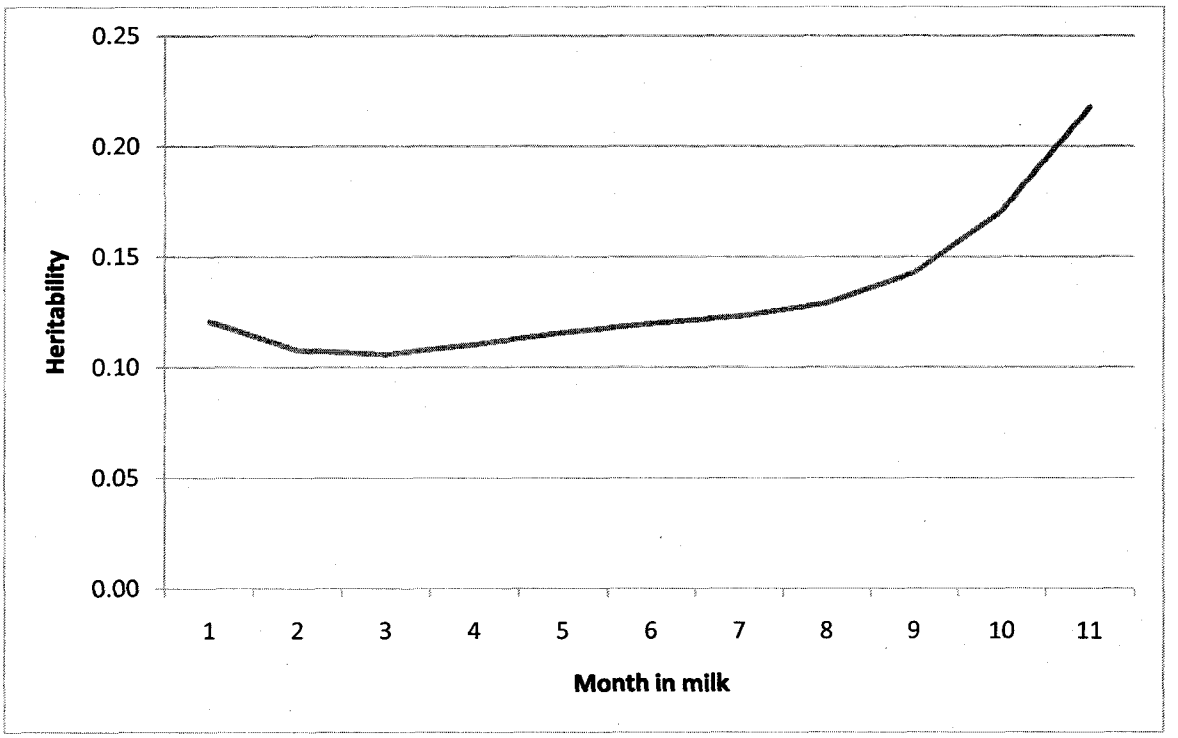
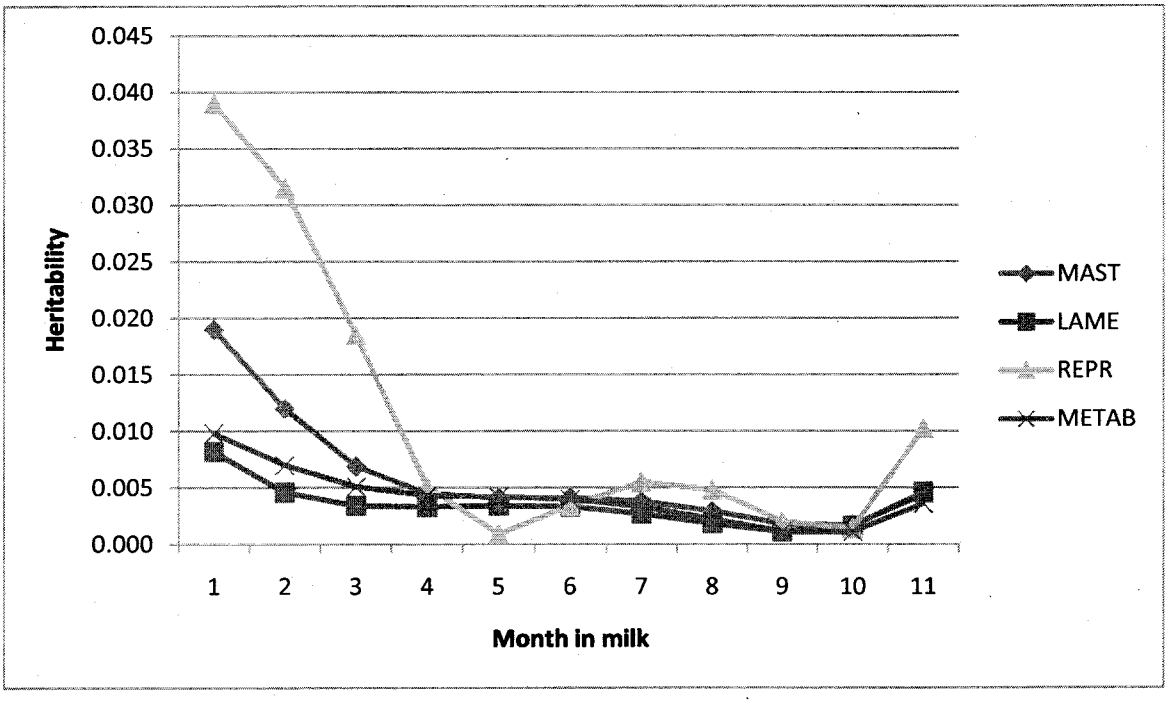


Figure 4 Heritability estimates of health traits calculated with a logitudinal model.



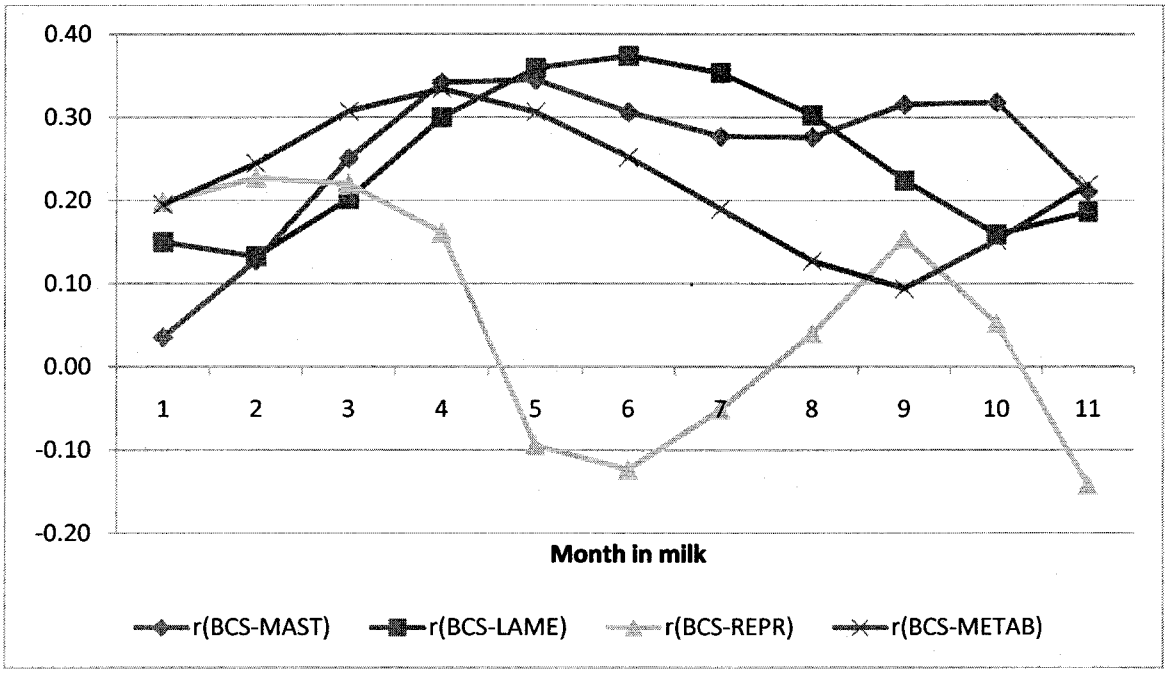
Mastitis (MAST),

Lameness (LAME),

Reproduction diseases (REPR, including cystic ovarian disease, metritis / uterine disease and retained placenta) and

Metabolic diseases (METAB, including ketosis, left displaced abomasum and milk fever).

Figure 5 Genetic correlations between BCS and health traits from the longitudinal model.



Mastitis (MAST),

Lameness (LAME),

Reproductive traits (REPR, including cystic ovarian disease, metritis/uterine disease and retained placenta) and

Metabolic diseases (METAB, including ketosis, left displaced abomasum and milk fever).

Chapter 6

Health Recording Practices of Canadian Dairy Producers.

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ABSTRACT

A survey was sent to dairy producers from Ontario and the Western provinces of Canada to assess the recording of the 8 health traits of interest in the National Health Project. The survey consisted of 16 multiple choice questions related to the number of health traits recorded, the herd's participation in the National Health Database Project and the frequency of data collecting.

The survey was sent to 1,716 dairy producers and answered by 459 producers; a participation rate of about 25% was achieved. Of the herds collecting health data, more than 70% were transmitting health data to the central database and 80% of the herds collecting data were in a herd management program with a veterinarian. Producers recorded event data for 1 to 8 health traits. The traits collected in the largest number of herds were mastitis, left displaced abomasum and retained placenta. These three diseases have clear diagnostic criteria and have a large impact on the economic performance of dairy production. The traits with the lowest herd participation in recording were lameness, ketosis, metritis, and cystic ovarian disease. The last 3 traits are more difficult to diagnose as they need a veterinary or technician intervention. In the case of lameness, the trait has many different causes and the location of the inciting cause is not always recorded accurately. For milk fever, some producers recorded prophylactic treatments whereas others recorded only clinical disease or treatments. This wide variation in recording methods showed the need for extension work directed towards Canadian dairy producers to improve the quality of health data recording to be used in genetic evaluations.

INTRODUCTION

Health traits of dairy cattle are recorded by producers in Canada. In the past few years, this group of traits has received much attention in many countries. At first, the focus of attention on health traits was put on better treatment and prevention practices. Improvement has been made on these traits, but expenses due to diseases are still high. Some research was conducted to estimate genetic components of these traits. Uribe et al. (1995), Kadarmideen et al. (2000) and Zwald et al. (2004) reported low heritability estimates for various health traits.

Many of the studies were based on relatively small datasets. The need for larger datasets, including more farms and more animals, became evident as the incidences of the main diseases were low. First nationwide health data collection systems were used in the Nordic Countries (Osteras et al., 2007). In Norway, veterinarians record high quality and complete dairy cow health data since 1975. This database formed the basis for a national genetic evaluation for health in Norway, resulting in a substantial genetic improvement for health resistance as documented by Heringstad et al. (2007).

In 2007, a database for recording health traits was developed in Canada. Contrary to the Nordic countries, health traits have been recorded by Canadian producers on a voluntary basis. In the Nordic countries, any treatment of an animal must be made by a veterinarian or under his supervision (Osteras et al., 2007); moreover, veterinarians are required to record treatments on each cows' health card and register it in the central database. No such regulation exists in Canada. Canadian producers do not need to ask a veterinarian before treating an animal. Treatments do not need to be reported to the central database neither is there any government incentive for health data recording. This

fact gives a different significance to the traits recorded in Canada as opposed to those in the Nordic countries. In Canada, disease cases are recorded, whereas in the Nordic countries, a mandatory system to report all health treatments is in place. The knowledge of the person doing the actual recording also results in differences between the 2 databases. As veterinarians have extended training in disease detection, a large number of different health disorders (67 in Norway; Osteras et al., 2007) are defined for recording. Some of these diseases need a detailed diagnostic or even laboratory analysis. In Canada, the goal was to have diseases that are simple to define and which can be diagnosed without laboratory analysis (Kelton et al., 1998). This has led to the choice of 8 diseases: clinical mastitis (**MAST**), lameness (**LAME**), cystic ovarian disease (**COD**), left displaced abomasum (**LDA**), ketosis (**KET**), uterine disease/metritis (**MET**), milk fever (**MF**) and retained placenta (**RP**). The definition for each of these diseases has been provided to the producers in the form of a memo with the principal characteristics needed to diagnose the disease.

Presently, there is no monitoring in Canada of the quality of disease event recording or of the precision of the diagnoses made by the producers. As recording is on a voluntary basis, producers have the choice to record all, some or none of the 8 diseases included in the health database. To assess the recording practices in Canada, a study was undertaken which included a survey being sent to producers.

The aims of this study were 1) to have an overview of the recording practices in Canada and 2) to compare the recording practices with the data actually collected in the central database.

MATERIALS AND METHODS

Questionnaire

A survey (Appendix 1) was prepared with 16 multiple choice questions referring to the number of diseases recorded and the way each disease was recorded. Some questions were about the transfer of data from the farm computer or herd book to the central database and the participation in a herd management program with local veterinarians. Questions specific to diseases were designed 1) to give information about the knowledge of the producers as to the disease diagnostics recommended by the Dairy Industry in Canada and 2) to determine if treatment, disease occurrence or, for a few diseases, prevention measures were recorded.

CanWestDHI (Guelph, Ontario), the dairy herd improvement (**DHI**) organization responsible for the herds in Ontario and the Western Provinces of Canada sent the survey to 1,716 producers who were coded by CanWestDHI, as recording diseases in their cow herds. This coding was based on the response of the producers to a question asked by CanWestDHI when health recording started in Canada. It represents the intention of the producer, but does not necessarily mean that the producer is actually recording health data. Surveys were sent to the producers together with results from a DHI test day (**TD**) and the shipping of these was therefore spread over a period of slightly more than a month (the average TD interval at CanWestDHI is 34 days). The first surveys were sent to the producers at the end of February 2009. A self-addressed stamped envelope was sent with the survey to encourage response to the questionnaire. No other incentive or reminder was made to increase the response rate. A total of 459 surveys have been returned; this represents 27% of the surveys sent.

Data

Results from the questionnaire were compared with data recorded in the Canadian Health Database by the same 459 herds to estimate daily incidences of the 8 diseases for August 2008 to January 2009, as the questionnaire referred to the situation in the 6 months preceding the reception of the survey. Using milk recording data, contemporary groups for animals with disease cases were constructed. Detailed description can be found in Neuenschwander et al. (2009). In short, the sampling frame included all cows with a TD record on farms with at least 2 separate disease cases recorded. The TD record had to be made during a time period going from 30 days before the first disease occurrence in this farm until 30 days after the last disease occurrence. Restrictions were also set with regard to minimum overall disease incidence in the herd. The minimum was set to 5%. A minimum overall incidence was set because some producers, although they declared they were recording diseases, did not have any case recorded in the database. Although, this restriction will bias the results upward, the impact on relative distribution of the daily incidence will likely not be influenced.

Days at risk (**DAR**) were defined for each cow-lactation, as starting at calving or 30 days before the first disease case on the farm, whichever was later. The end of the period at risk was the 335th DIM, the last TD record or 30 days after the last disease case in the herd, whichever was earliest. The dates of the first and last DAR were converted from a date to DIM.

Daily incidences were calculated as the ratio of animals diseased on a given DIM to the total number of animals at risk on that particular day. Restrictions were set for multiple cases of a disease for a single cow. A MAST case was considered if there were

at least 8 days between the previous case and the case considered. For LAME, COD, KET and MET, 30-day intervals were required to include the new case of the disease.

All significance tests used a test for equality of two proportions under the normal distribution assumption to the 5% level.

RESULTS

The number of surveys received from each province is presented in Table 1. The participation rate reflects the provincial distribution of dairy operations, with about 3 quarters of the herds in Ontario. The average size of herds participating in the survey was 87 cows. The largest herds were found in the Western Provinces (BC, AB, SK and MB). Of the producers answering the survey, 246 (55%) kept their cows in tie-stall barns. This proportion was much lower when only Western Provinces were considered. The proportion of herds reporting diseases to DHI was the lowest in ON (68%), but significantly higher in AB (90%, $p < 0.01$) and the other Western provinces also had a high proportion of herds reporting. Health management programs under the supervision of a veterinarian were conducted in about 80% of the herds. This result was relatively constant across the provinces.

The proportion of herds recording each of the 8 diseases in relation to the total number of herds recording any of these diseases is presented in Figure 1. Only 3 diseases (MAST, LDA and RP) are recorded in at least 80% of the herds. Some diseases (LAME, COD, KET and MET) are recorded in only about half of the herds. A higher proportion of Western provinces herds than Ontario herds are recording LAME (74% vs. 47%; $p < 0.01$), KET (57% vs. 44%; $p < 0.05$), MET (73% vs. 46%; $p < 0.01$) and MF (82 vs. 69%;

$p < 0.05$). For the other 4 traits, proportion of herds recording was similar. In British Columbia, participation for recording of reproductive traits (COD, MET and RP) was very high.

Comparison between herds reporting disease events into the central database compared to herds keeping records on the farm shows that a higher proportion of larger herds (96 vs. 78 cows; $p < 0.05$) in free-stalls (52% vs. 39% in free stalls; $p < 0.05$) were reporting diseases. Herds that did not report diseases to the central database were recording more of COD (68% vs. 54%; $p < 0.05$) than herds reporting to the central database. On the other hand, a significantly smaller proportion of herds without reporting to the central database recorded data on LAME (42% vs. 59%; $p < 0.01$), LDA (78% vs. 91%; $p < 0.01$), KET (35% vs. 53%; $p < 0.01$), MF (60% vs. 77%; $p < 0.01$) and RP (74% vs. 85%; $p < 0.05$).

All producers at CanWestDHI had received an information sheet for the accurate recording of diseases with a short description of what was considered a positive case of the disease. A question in the survey was used to assess the knowledge of the producers as to this information sheet and more specifically as to the diagnostic of lameness. Only 35% of the producers recording lameness were doing it according to the guidelines presented in the information sheet.

Two questions were asked to assess the regularity of recording. One concerned LAME (frequency of observation) and the other COD (time elapsed from calving until first checks for ovarian cysts). For LAME, 57% of the producers observed cows at least once a day and 33% of the producers only recorded obvious cases, without making regular observations. In general, cows are checked for ovarian cysts only when they are

not cycling, but producers might have different expectations as to how long after calving the cows are supposed to start cycling again. Most of the producers in the study, started to check for ovarian cysts before 6 weeks postpartum (63%), but a significant number (18%) did not start examining cows before 2 months postpartum.

Of the 8 diseases, 2 needed a veterinary intervention or at least an AI technician examination. The first is LDA which requires a surgical intervention. The second is COD, which needs an ovary palpation generally conducted by a veterinarian. For these diseases the question asked was whether the producers recorded all veterinarian findings or only some of them: 96% recorded all LDA cases; but only 86% recorded COD cases. For the other 6 diseases, the questions asked were if all occurrences and cases treated by producers were recorded or if only veterinary treatments were recorded. The results are presented in Table 2. For MAST, most of the producers (97%) reported all cases, whereas for METR, 29% reported all cases or cases they reported themselves and 66% reported only veterinary treatments. A majority of the producers reported all cases or cases they treated themselves for the 4 other diseases.

Daily disease incidence is presented for all diseases in Figures 2 to 9. The daily incidence never exceeded 1.2% for any of the 8 diseases. As expected, distribution of daily incidence over the lactation varied largely between diseases. For MF and RP, which are calving related diseases, most of the cases were in the first week of lactation. Given the nature of these diseases, it is very likely that records later in lactation are misclassifications. Metabolic diseases (LDA and KET) and MET happened almost exclusively in the first 2 months of lactation. A peak in the first 10 days was observed for MAST, but incidence remained substantial during the whole lactation. Incidence for

LAME slightly increased during the lactation, but always remained extremely low (< 0.12%). COD was not observed before 20 DIM as cows are rarely bred before that time and therefore are not checked for this disorder. The highest incidence for COD was between 25 and 80 DIM.

DISCUSSION

Distribution of responses to the survey was similar to the distribution of all herds in the provinces (CanWestDHI, 2009). This survey did not represent a random sample of the herds in Ontario and Western Canada. First, only herds coded as "recording diseases" were included. Coding of herds was made based on answers from producers when the health recording program was initiated and it was not based on actual recording of health data. Producers with this code are either recording disease events or they have an interest in breed improvement for health, but have not yet started recording diseases. Only a quarter of the producers receiving a survey answered it. This group is a "highly selected" group. Most of the producers who do not record diseases probably did not answer. A lack of interest in collecting health data is certainly linked to a lack of interest to participate in the survey. In the following discussion, this survey is not considered to be a representative sample of the "population" of producers.

The average size of herds included in the survey was the same as the average size of all herds registered at CanWestDHI (CanWestDHI, 2009). Similar herd sizes were also reported in ON, BC and AB. Herds answering the survey were larger than the provincial average for MB and SK. Type of barn was on average different between the survey and all the herds and this changed from province to province. In ON and MB, a higher

proportion of producers with free-stall herds answered. This was expected as producers having free-stalls are often looking for more cost efficient methods of farming and health is an important component of costs. Producers with free-stalls are also looking for a reduction of work per unit of livestock kept. This can be achieved when cows are healthy, because sick cows generally take more time in a free-stall operation (fetching the cow, bringing her to the nursery, milking her separately in the milking parlour, etc.) than in tie-stall barns.

Slightly less than a third of the producers do not report the health recording to the central database. Many data remain on the farm and cannot be used for genetic evaluations. The most obvious reason is the lack of knowledge that it is possible to send data to the central database or as to the procedures for doing it. Many producers answered that they were reporting data to the central database, but actually did not as they had no records in the database. This shows that there is still a lack of understanding as to the procedure needed to transfer data to the central database. Another reason is the fact that many producers record diseases only for quality control, for which treatments with medications needing milk removal have to be registered. These producers have no direct interest in genetic improvement of the trait and therefore keep the information on the farm. In comments written on the returned survey, some producers mentioned that all diseases were registered in a "farm herd book", but that only severe cases were written on the DHI document used to transfer data. Comparing herds with and without transfer of data to the central database, there is a clear difference in the amount or type of diseases recorded. Herds without transfer of data, recorded more reproduction diseases than other herds. This fact indicates that herds without data transfer, record diseases mostly for

management purposes. Recording COD will help producers to know which cows to treat and which cows have recurrent occurrences of the disease. There is also a high proportion of reporting herds for MAST and MET – recurrent diseases like COD – which can be followed for better management throughout the life of each cow. Finally, another reason might be the desire to keep these data private. There is a need for more extension work to increase the awareness of the benefit of transmitting data to the central database and to encourage producers to do so.

The proportion of all herds recording diseases shows the importance of the definition of health traits. The 3 most reported are all easy to diagnose and have a large economic influence. LDA requires a veterinary intervention and is therefore difficult to misclassify. For RP, it is simple to observe that foetal membranes are not expelled 24 hours after calving. MAST is probably the health trait best known by producers of all health traits studied here as well as the one influencing the economic performance of dairy cows the most.

On the other hand, the 4 traits that were recorded in about 50% of the herds were traits that are more difficult to diagnose or whose causes were so variable that a single treatment was not possible. For COD diagnosis, ovarian palpation is necessary in order to determine if the animal has the disease. An ovarian check must be made for the disease to be detected. Therefore COD often remains undetected. This is also clearly seen in the distribution of the disease over the lactation. No case is present before 20 DIM; this is actually an absence of disease detection as it is rare that the ovaries are checked during that time. Answers to the survey explain that fact, as only 16% of the producers started checking their cows for ovarian cysts before the third week of lactation. Therefore the

reported incidence of COD will largely depend on the recording practices. Moreover, many producers record COD only for management of the reproduction techniques, and therefore do not report all cases to the central database. Disease events in cows which the producers are not planning to breed will probably be ignored and will therefore create a bias in the data, as only selected data will be analysed.

Another disease needing more detailed diagnostics is KET, normally detected by an on-farm urine test. Not all producers are doing this test on fresh cows and the detection of the disease might therefore be flawed. The practices of checking for KET are probably different from one producer to another. Some test all cows, whereas others might only test cows with obvious reduced feed intake.

The third disease with a low participation of herd recording is LAME. This disease has a very complex aetiology. Moreover, it is not necessarily simple to decide if the level of intensity of a case of LAME is high enough to declare the animal as "sick". Moreover, as causes of disease are varied (feeding regime, infection, accident, floor type), recording LAME might not even really help, for a better prevention of LAME. The fact that a majority of producers do not know or do not use the definition of LAME shows the need to increase the extension work for this disease. The awareness of an accurate recording of diseases needs to be raised, particularly for traits with low herd participation.

Metritis, the last disease with low participation in health recording, is a trait that has 2 different definitions based on the time of lactation. The disease before 20 DIM is defined as an abnormally enlarged uterus containing fetid watery red-brown fluid (Kelton et al., 1998). This diagnostic can only be made with a rectal palpation and hinders the

widespread recording of the disease. After 20 DIM, the disease is simply defined as a abnormal vaginal discharge, which can be more easily diagnosed.

For MF, an additional difficulty arises. Many producers (30%) record all cases of calcium treatment as a case, although some of these treatments were only a preventive treatment. Normally, the treatment is only made for cows susceptible to the disease, but some of them might not have been diseased even without calcium. Here again, it is an aspect requiring more attention. Treating cows preventatively is good from an animal welfare and economic perspective, but this brings inaccuracies in health recording and hinders selection for healthier cows. A consistent way of recording needs to be defined.

The goal of the Canadian National Health Project was to record cases of diseases as well as treatments by veterinarians as in Nordic countries. For some traits, such as MAST, this seems to have been achieved. However, for other traits, a substantial number of producers record only cases treated by veterinarians. This aspect should also be addressed, either by extension work to encourage the producers to record all occurrences of the diseases or in reviewing the best way to record diseases.

CONCLUSIONS

The results from a mail survey among dairy producers from 5 Canadian provinces gave interesting insights into recording practices in Canadian dairy herds. The survey showed which aspects are satisfactory for consistent data collection, but also highlighted challenges to be met. As well as encouraging health recording in all herds, the main challenge is to encourage producers to send their data to the central database. More precise descriptions and herd reports based on health results might help raise awareness

for health traits and encourage more consistent recording in participating herds. Some traits are only recorded when veterinary treatments are made. This was not the original goal of the Canadian National Health Project as the intention was to have recording for all traits. This must also be addressed to ensure a consistent recording in the future.

The survey showed that a clear and simple description is essential to ensure participation of herds. Traits with a complicated diagnostic will generally not be recorded by producers. Extension work is needed to explain the reasons for recording, the way to diagnose and report diseases, and the procedure to transfer data to the central database.

The present study also showed that for the majority of the diseases, the highest incidence was found at the beginning of lactation. Although disease occurrences later in lactation are important from an economic point of view, they might not be important from a genetic point of view as most of the variance in disease incidence will be found at the beginning of the lactation. This aspect should be considered for the design of genetic evaluations for health traits.

Based on the results of this survey, it seems that MAST, LDA and RP are already recorded accurately enough for use in genetic evaluations. Recording for these traits is also made in the majority of herds with health recording. Further extension work should be made for other traits, in order to increase the accuracy of recording. Without more accurate recording for LAME, COD, KET and MET, estimated breeding values (**EBV**) for these traits will not be accurate enough for use in breeding programs.

REFERENCES

CanWestDHI, 2009, Progress Report 2008, CanWestDHI, Guelph, ON.

Heringstad, B., G. Klemetsdal, and T. Steine. 2007. Selection response for disease resistance in two selection experiments with Norwegian Red cows. J. Dairy Sci. 90:2419-2426.

Kadarmideen, H. N., R. Thompson, and G. Simm. 2000. Linear and threshold model genetic parameters for disease, fertility and milk production in dairy cattle. Anim. Sci. 71:411-419.

Kelton, D. F., K. D. Lissemore, and R. E. Martin. 1998. Recommendations for recording and calculating the incidence of selected clinical diseases of dairy cattle. J. Dairy Sci. 81:2502-2509.

Neuenschwander, T. F.-O., F. Miglior, J. Jamrozik, and L. R. Schaeffer. 2009. Use of linear and threshold models for analysis of producer-recorded health data in Canadian Holstein Cattle. Report to the Dairy Cattle Breeding and Genetics Committee, February 3, 2009; Guelph, ON, Canada.

Uribe, H. A., B. W. Kennedy, S. W. Martin, and D. F. Kelton. 1995. Genetic parameters for common health disorders of Holstein cows. J. Dairy Sci. 78:421-430.

Zwald, N. R., K. A. Weigel, Y. M. Chang, R. D. Welper, and J. S. Clay. 2004. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. J. Dairy Sci. 87:4287-4294.

Table 1 Distribution of herds by province (Ontario = ON, Manitoba = MB, Saskatchewan = SK, Alberta = AB and British Columbia = BC) and by recording type (DHI = records uploaded in the DHI database; Farm = records kept at the farm). "Tie-stall" is the percentage of tie-stall herds; "Recording" is the percentage of herds recording diseases; "DHI" is the percentage of herds transmitting data to DHI, "Management" is the percentage of herds participating in health management programs with a veterinarian. The diseases are: mastitis (MAST), lameness (LAME), cystic ovarian disease (COD), left displaced abomasum (LDA), ketosis (KET), metritis (MET), milk fever (MF) and retained placenta (RP). Values are absolute numbers for herds and herd size; all the other values are percentage of the total number of herds.

	Total	ON	MB	SK	AB	BC	DHI	Farm
Number of Herds	459	359	19	8	43	27	271	108
Herd Size	87	75	158	194	116	122	96	78
Tie-stall	55	64	28	22	26	11	48	61
Recording	89	88	95	100	95	93	100	100
DHI	72	68	83	88	90	78	100	0
Management	81	80	94	88	80	84	82	78
MAST	91	90	100	88	90	100	93	91
LAME	53	47	72	100	76	64	59	42
COD	57	55	67	75	59	68	54	68
LDA	86	84	94	100	93	84	91	78
KET	47	44	39	75	61	56	53	35
MET	52	46	61	75	71	84	55	48
MF	72	69	78	100	88	68	77	60
RP	81	79	89	100	90	80	85	74

Table 2 Proportion (%) of herds recording all diseases or all treatments done by the producer, and proportion of herds recording only veterinarian treatments based on the total of herds recording each of the diseases[1].

	Mastitis	Lameness	Ketosis	Metritis	Milk fever	Retained placenta
Disease /Treatment by producers	97%	76%	66%	29%	74%	73%
Treatment by veterinarians	2%	16%	32%	66%	25%	23%

[1]Difference to 100% is made by herds not answering the specific question.

Figure 1 Proportion (%) of herds recording each of the 8 traits. Diseases are mastitis (MAST), lameness (LAME), cystic ovarian disease (COD), left displaced abomasum (LDA), ketosis (KET), metritis (MET), milk fever (MF) and retained placenta (RP).

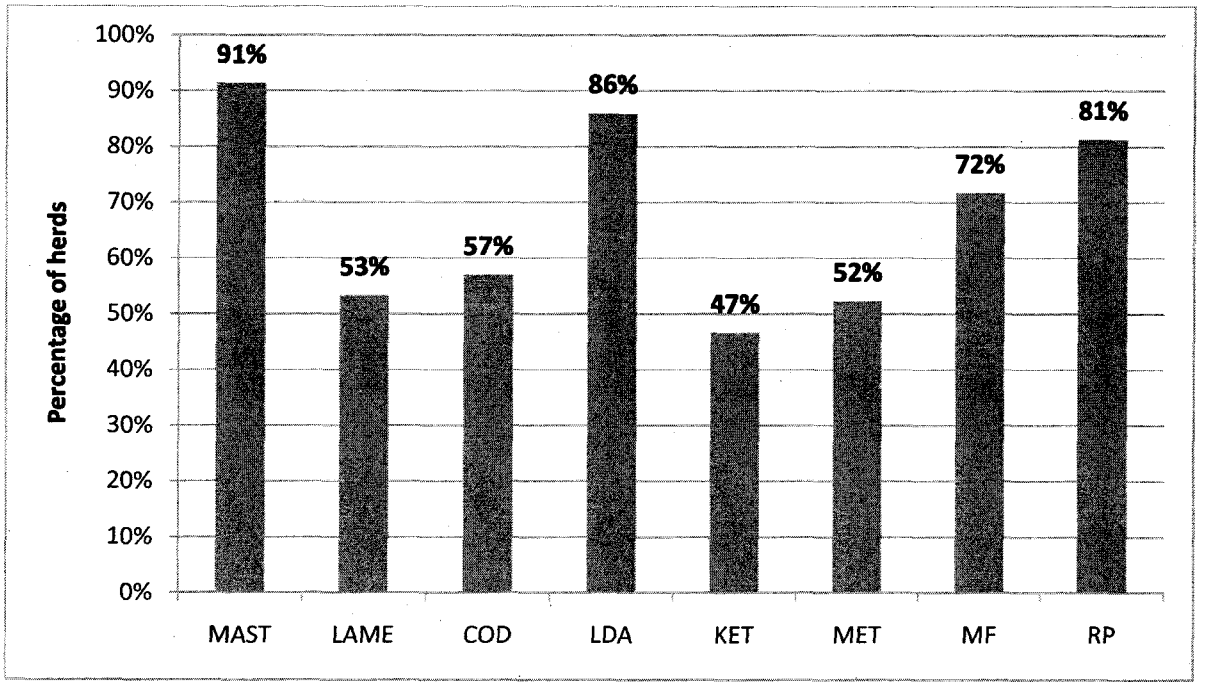


Figure 2 Daily incidence of mastitis from day in milk (DIM) 1 to DIM 305 for animals in herds with recording for mastitis.

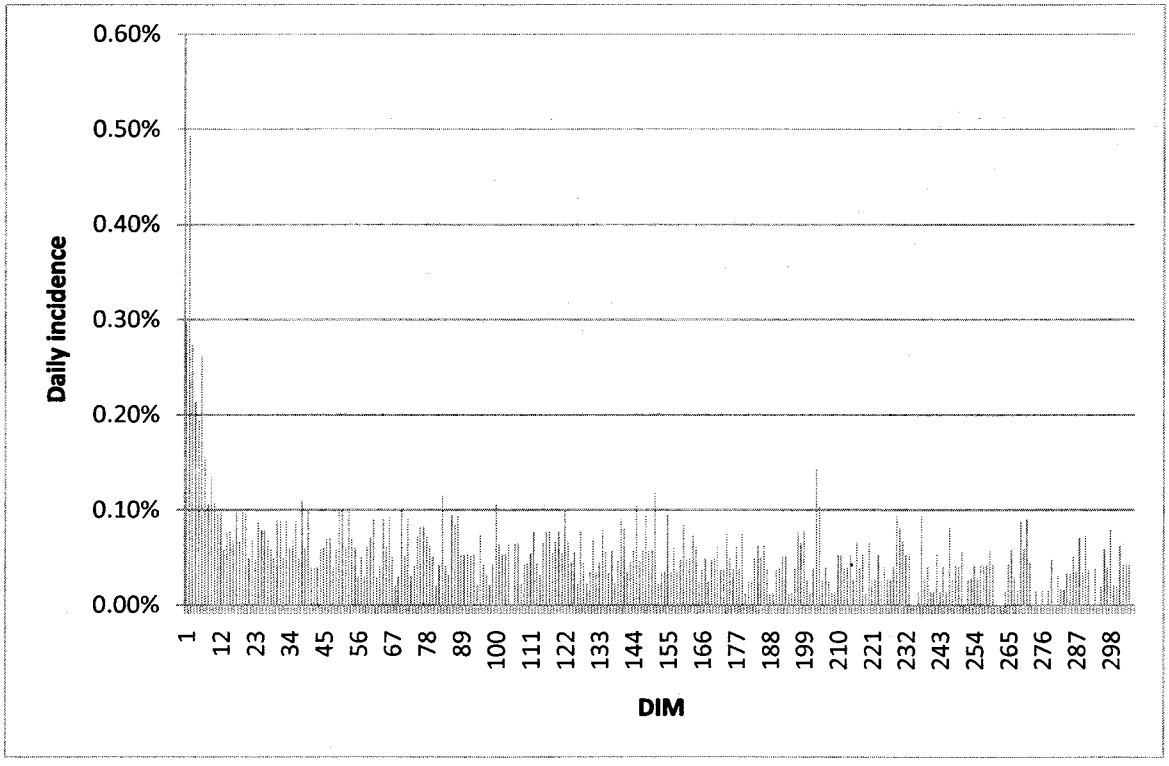


Figure 3 Daily incidence of lameness from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for lameness.

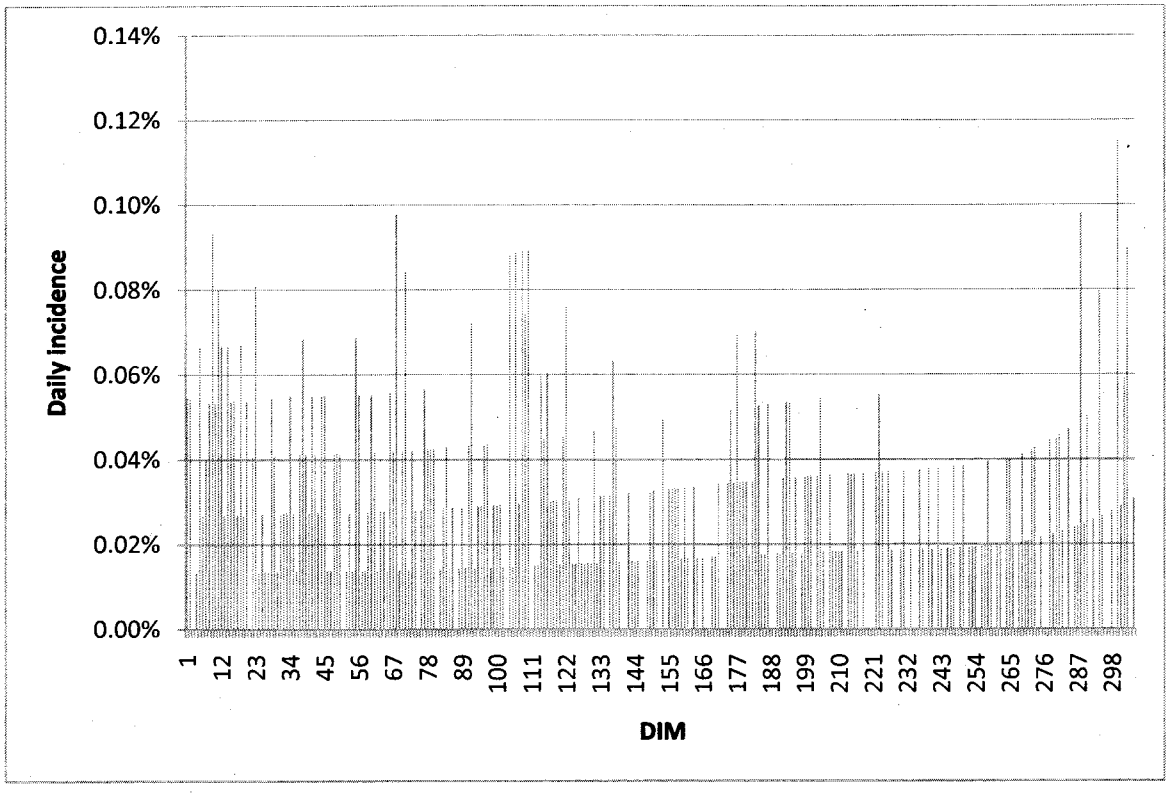


Figure 4 Daily incidence of cystic ovarian disease from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for cystic ovarian disease.

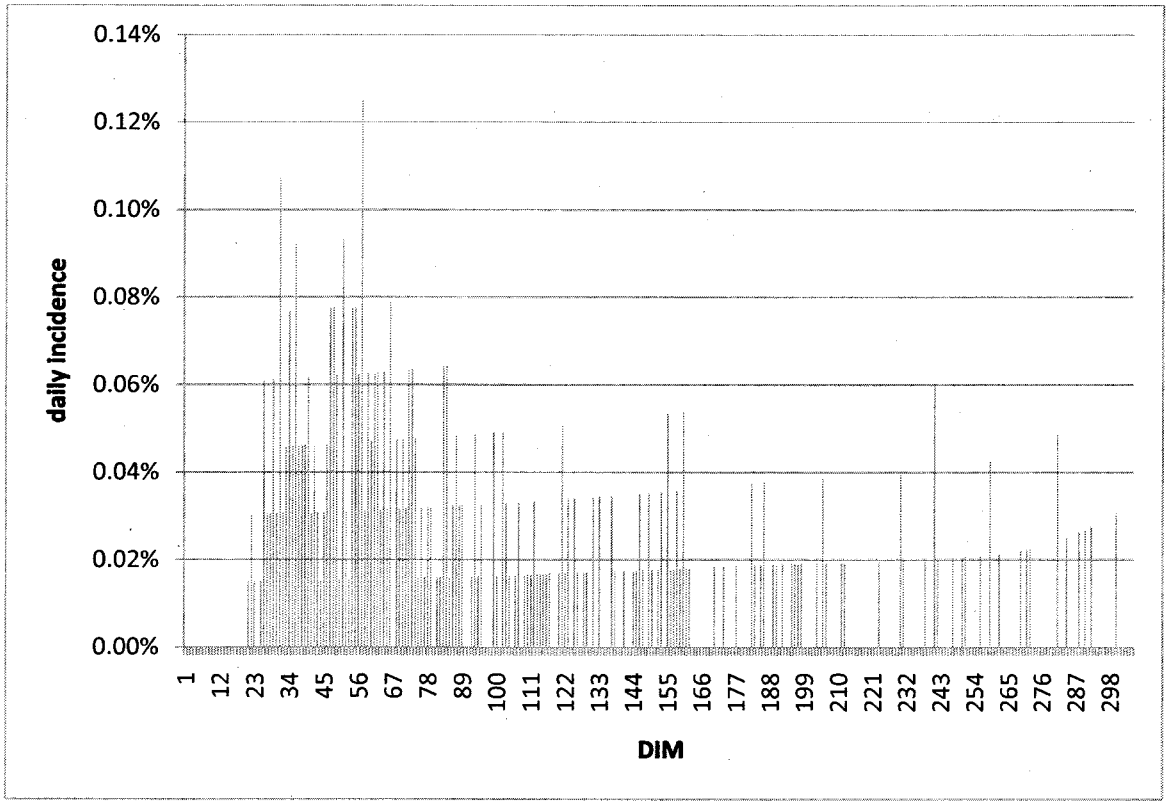


Figure 5 Daily incidence of left displaced abomasum from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for left displaced abomasum.

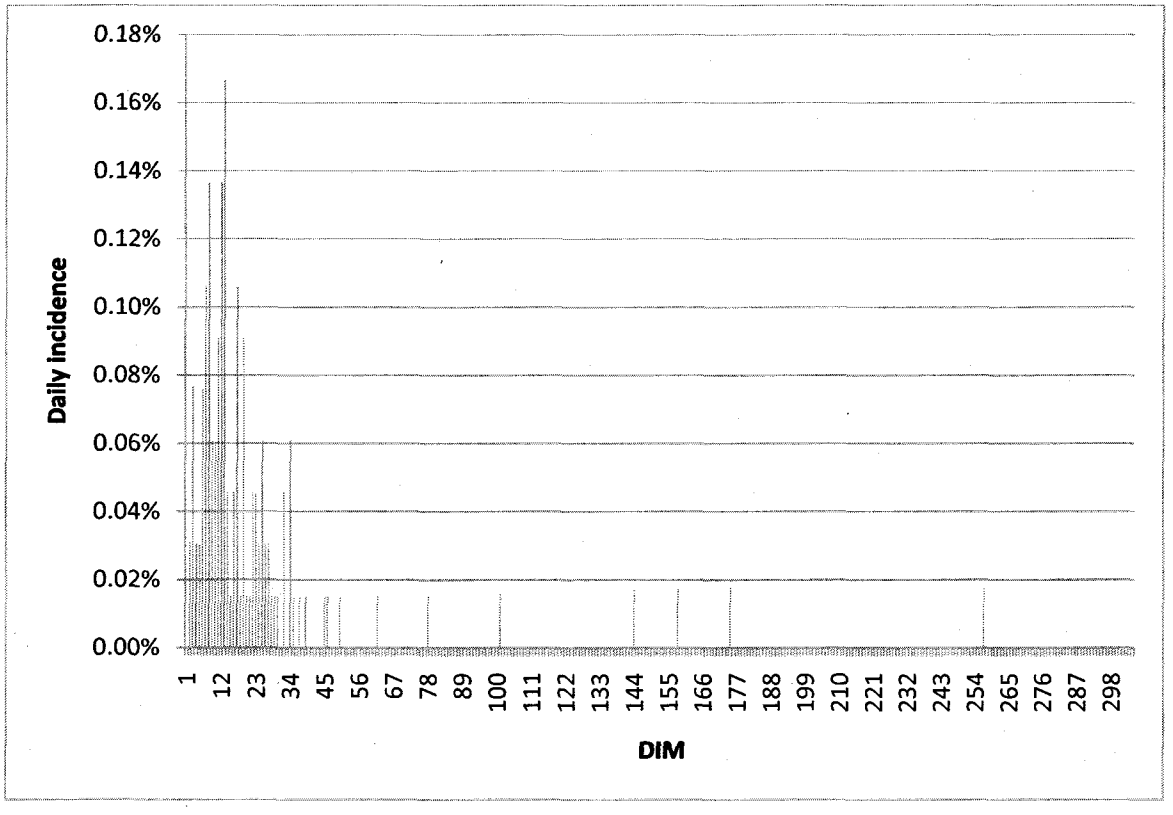


Figure 6 Daily incidence of ketosis from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for ketosis.

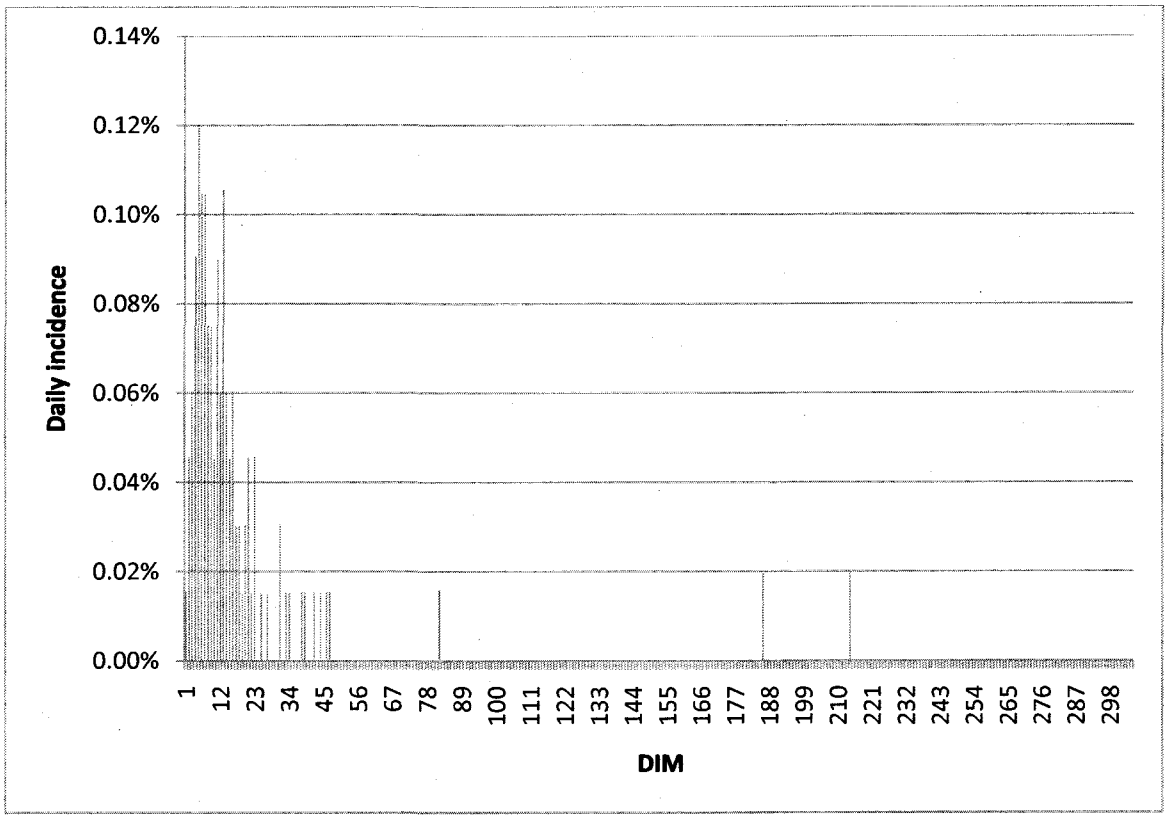


Figure 7 Daily incidence of metritis from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for metritis.

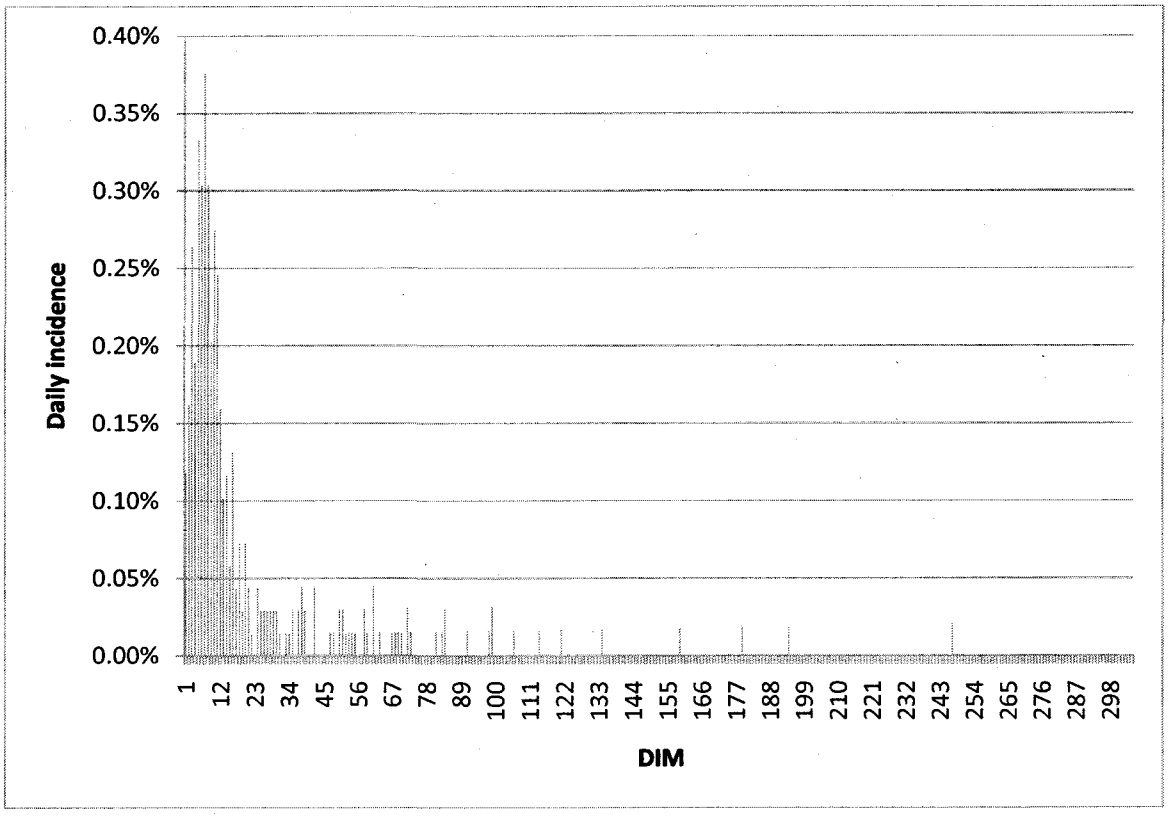


Figure 8 Daily incidence of milk fever from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for milk fever.

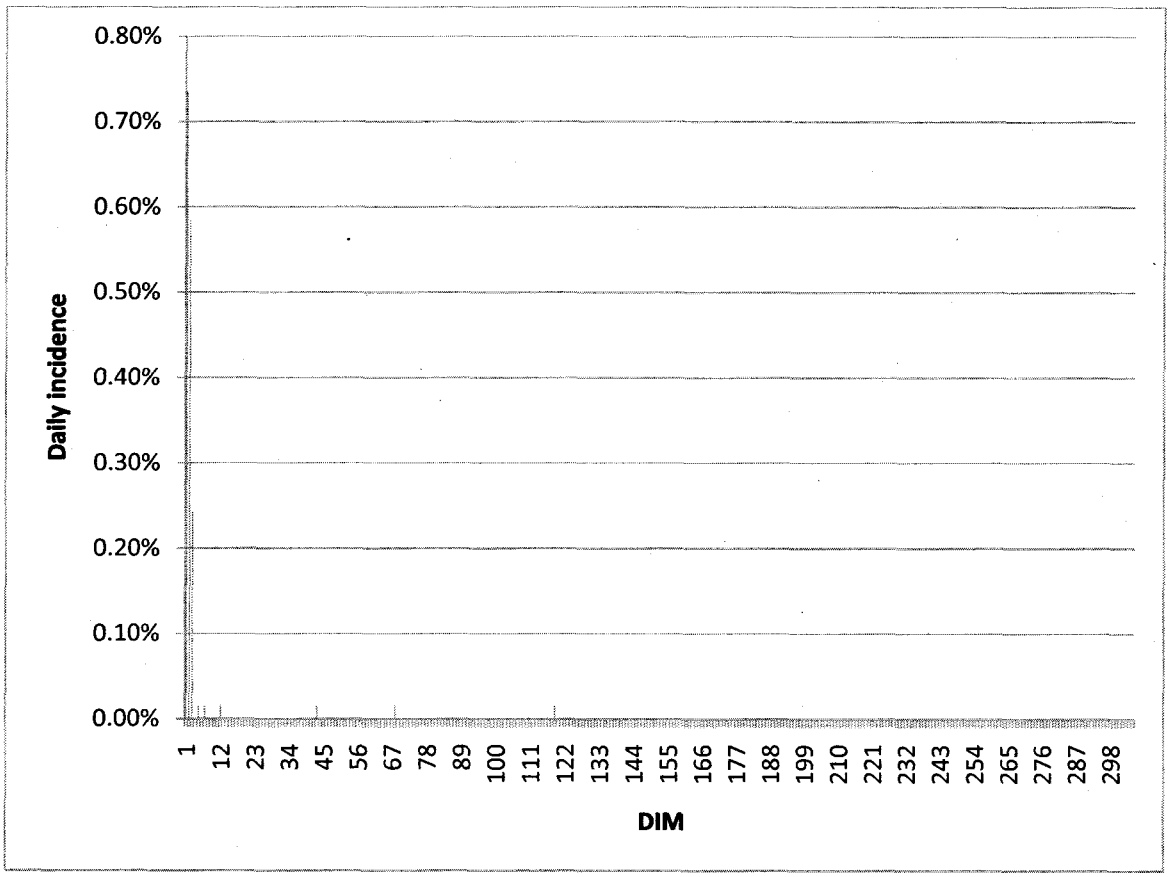
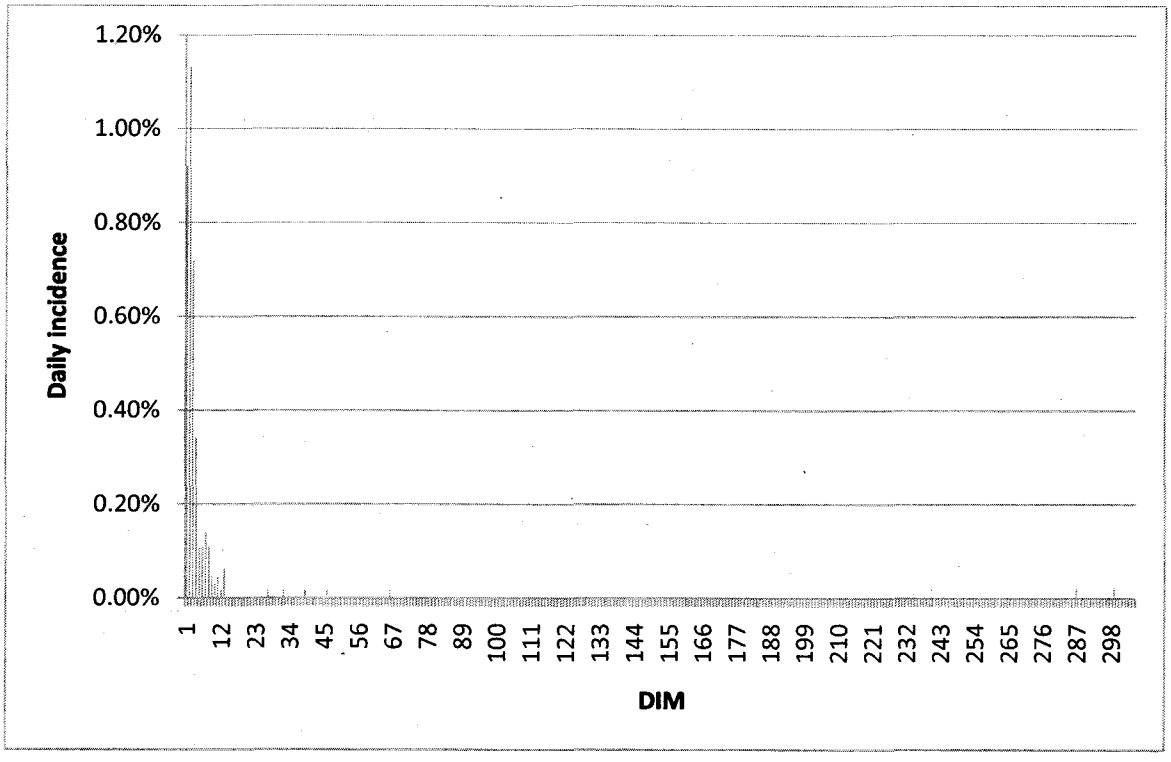


Figure 9 Daily incidence of retained placenta from day in milk (DIM) 1 to DIM 305 for animals in herds with health recording for retained placenta.



Chapter 7

General discussion

T. F.-O. Neuenschwander

The goal of this study was to gain deeper understanding of the genetic aspects of 8 producer-recorded health traits in Canadian dairy cows. First, the effect of different sampling frames was appraised (Chapter 3) and then the effect of linear and threshold models on variance components estimates was evaluated (Chapter 4). Chapter 5 dealt with the relationship between selected health traits and an indicator trait, namely body condition score (**BCS**). Finally, a survey of the recording practices of Canadian producers was conducted (Chapter 6).

In this general discussion, the focus will be on the aspects related to the implementation of genetic evaluation for health traits in Canadian Holsteins.

Data sampling

Data sampling had a profound impact on results of genetic analyses, both the variance components estimates and the ranking of bulls. A more stringent sampling, keeping only herds with a certain incidence for the diseases, increased the heritability of most traits and the variance (phenotypic and genetic) of the traits. This showed that a minimum level of health event recording is needed to obtain higher heritabilities which are needed if any substantial genetic progress is to be made for health traits.

For binary variables, location and dispersion parameters are not independent of each other. A less stringent sampling frame causes the incidence (and therefore the mean) of the traits to be lower and as a result caused a lower variance. This was observed in the dataset analysed. Using data from all herds having at least one case of any disease, instead of using only herds collecting data on the specific disease, reduced the phenotypic variance, the sire variance and the heritability. Decrease was the sharpest for traits collected only in a small number of herds. This result showed the importance of using

only high quality data; but the sampling frame to use to obtain good quality data is difficult to define as there is both a risk to include herds not collecting data as to remove herds with good health management and therefore less cases of disease. Both problems would bias the data. The problem lessens as a longer period of data recording is included as all herds collecting data will eventually have a case of each of the 8 diseases collected. At this point, a stringent sampling frame will not remove herds collecting data, but remove mostly herds not participating in data collection.

Comparison of models

The threshold model was designed to model binary traits using an underlying normal distribution to describe the observable binary traits. The dependency between location and dispersion parameters is removed. Higher estimates of heritabilities were found on the underlying scale from the threshold model than from the linear model, but when heritabilities from the binary model were converted to an observable scale, they were similar to the heritabilities estimated with the linear model. The threshold model does not have an obvious advantage over the linear model, as the heritability important for selection (heritability on the observable scale) is similar with both models. The mean squared error of prediction slightly favoured linear models over threshold models.

The effect of including days at risk in the model was also assessed in this analysis. There did not seem to be an improvement of the modeling in using this covariate. The reason was not obvious and might be due to the opposite effects of 2 elements. Cows with disease tend to be culled earlier in the lactation, but on the other hand cows culled earlier have less time to develop a case of a disease and therefore have less disease events

recorded. The low effect of days at risk on health seemed to support the non-inclusion of this effect in a genetic evaluation model for health traits.

Correlated trait

Many herds in Canada do not record health events. Many years could pass before a larger proportion of Canadian herds can be included in genetic evaluation. Use of correlated traits could be of interest in the meantime. A trait that is often cited in relation to health resistance is body condition score (**BCS**). This trait has a moderate heritability (0.20), is easy to measure and is collected in more herds than health data. A moderate positive correlation between BCS and disease resistance was found. This result supported the fact that cows with higher BCS have a better health, but BCS has an intermediate optimum. Cows with a very high (fat) or very low (thin) BCS are undesirable. This also reduced the strength of the correlation between disease resistance and BCS. The Canadian Holstein population had an average BCS slightly below the optimum.

Recording practices

Recording practices of producers are of paramount importance for quality of health data. Survey of herds from Ontario and Western Canada showed that many producers do not report health events to the central database, although they do record them on the farm. A large quantity of health events is, therefore, not available for genetic evaluations. This problem should be addressed and ways to encourage producers to make data available to the central database need to be found.

The survey also revealed large discrepancies in the definition of the disease from one producer to another, or at least in the definition of the cases needing to be reported. Extension work is needed in this area and collaboration with veterinarians to help

standardize health data recording. For some diseases (MAST and LAME) very different pathogens can cause the disease. A further differentiation of the diseases into sub-classes might be needed to have an accurate description. This differentiation would, on the other hand, incur more errors in recording. Possible lower herd participation could also be expected as the recording system becomes more complicated.

Final implications and recommendations

Many issues should be addressed before a genetic evaluation system for health traits can be implemented in Canada. The 1st issue is to ensure a high participation in health recording and an accurate diagnosis of the disease events. Some diseases are defined differently by different breeders. Effects of inaccurate recording on the estimation of genetic effects can be significant. One way of ensuring good recording would be to have an incentive system in place. Secondly, a good data sampling system needs to be put in place. Data sampling also has an important effect on the genetic evaluation, especially in the case of binary traits, and for which the difference between "healthy" records and "missing values" are not always clear. If sampling is not made properly, many cows with missing records could be included in the genetic analysis as healthy animals and, therefore, bias the results.

Different models result in similar variance component estimates. Changes between estimates seem to be more influenced by sampling method than by differences in models. The theoretical advantages of threshold models over linear models are mostly removed when results are seen on the observable scale.

Based on the results from the present study, but also on the current practice in Nordic countries, the following recommendations are made for a routine genetic evaluation system.

- Some health traits should be grouped to obtain a higher incidence and a larger group of herds with recording. Mastitis and lameness should be kept as single traits. The other 6 diseases should be grouped into reproductive (COD, MET, RP) and metabolic traits (LDA, KET, MF) traits.
- Only cases at the beginning of lactation should be used as it is the period of greatest risk and also showing the greatest genetic variation. Incidence later in lactation is often influenced by culling policy and treatment of the herds. Depending on the disease, inclusion of cases should only be made in the first 50 to 100 days of lactation.
- Validation of the data before use in variance components estimation and genetic evaluations should be made based on minimum incidence per herd. Herds should also be removed when no regular recording is made. A maximum time period between two occurrences of a disease should be set. This value should depend on the size of the herd. Validation should be made separately per disease or disease group.
- As linear models require less computing and the results are similar to those found with a threshold model, linear models might be favoured for application in routine genetic evaluations.
- A sire model should be used.

- Use of BCS as a correlated trait should be made for metabolic and reproductive diseases. For MAST, it would be possible to use SCS as a correlated trait; but this trait is presently analysed in a test-day model. More analyses need to be done with the Canadian population for the use of SCS in prediction of MAST.

The present thesis has shown that producer-recorded health events have a genetic component in their expression. However, data from this source need to be sampled to ensure that producers involved in the process are actually collecting health data. A survey has shown that producers collect more data on diseases that are easy to detect or that require in any case the intervention of a veterinarian. It has also been shown that the use of different models does not have a large impact on the heritability, when it is measured on the observable scale. Finally, it was shown that body condition score can be used as correlated traits and have a predictive ability for disease resistance.

Appendix

Questionnaire concerning recording of health traits

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Herd name	
Herd number DHI	
Size of herd (number of cows)	

Free-stall	
Tie-stall	

All questions refer to the situation in your herd in the last 6 months. Please check the answer corresponding to the situation in you herd.

		yes	no
1.	Do you record any disease of cows?		

If your answer is "no", you have already finished answering the questionnaire. Please send it back in the pre-printed envelope. Thank you.

If your answer to the first question is "yes", please answer the following questions.

2.	Do you transfer these records to the CanWestDHI database?		
3.	Do you participate in a herd management program with your veterinarian?		

4. Which diseases do you record:

4.1	Mastitis	
4.2	Lameness	
4.3	Cystic ovarian disease (cysts)	
4.4	Displaced abomasum (twisted stomach)	
4.5	Ketosis	
4.6	Metritis	
4.7	Milk fever	
4.8	Retained placenta	

Please answer to the questions corresponding to the diseases you announced in question 4.

Questionnaire concerning recording of health traits

yes no

5. MASTITIS

Do you record

5.1 all cases of mastitis? ☐

5.2 only cases which needed treatment? ☐

5.3 only cases for which you had to call a veterinarian? ☐

6. LAMENESS

Do you record lameness for all cows having a symptom 3 or 4 according to the Dairy Cattle Health Definitions? ☐ ☐

7. How often do you check your herd for cases of lameness:

7.1 every milking? ☐

7.2 daily? ☐

7.3 weekly? ☐

7.4 only when a case is obvious? ☐

8. Do you record lameness only when you have to call the veterinarian for it? ☐ ☐

9. CYSTIC OVARIAN DISEASE

After how many weeks following calving do you start checking cows for ovarian cysts?

9.1 less than 3 weeks ☐

9.2 less than 6 weeks ☐

9.3 less than 2 months ☐

9.4 after more than 2 months ☐

10. Do you record all cases of cysts that the veterinarian diagnosed? ☐ ☐

11. DIPLACED ABOMASUM

Do you record all cases of displaced abomasum that were treated by your veterinarian? ☐ ☐

Questionnaire concerning recording of health traits

yes noyes no

12. KETOSIS

Do you record ketosis for

12.1 all cows that are treated for it? ☐

12.2 only cases treated by a veterinarian? ☐

13. Do you record cases of ketosis, when a case of left displaced abomasum is treated at the same time? ☐ ☐

14. METRITIS

Do you record metritis for

14.1 all cows with a vaginal discharge after 20 DIM? ☐

14.2 only cases treated by a veterinarian? ☐

15. MILK FEVER

Do you record milk fever for

15.1 all downer cows after calving, without sign of injury? ☐

15.2 all cows that received calcium post-calving? ☐

15.3 only cases that were treated by a veterinarian? ☐

16. RETAINED PLACENTA

Do you record

16.1 all cases of retained placenta? ☐

16.2 only cases that were treated by a veterinarian? ☐

Thank you for your answers! Please send back the questionnaire in the pre-printed envelope.