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Causal relationships between clinical mastitis events, milk yields and lactation persistency in US Holsteins

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ABSTRACT

Complex relationships exist between udder susceptibility to mastitis and milk production traits. Identifying causal association between these traits could help to disentangle these complex relationships. The main objective of the study was to use producer-recorded health data to examine the causal relationship between mastitis events, milk yield and lactation persistency. A total of 48,058 first lactation cows, daughters of 2213 Holstein bulls and raised across 207 herds were analyzed using structural equation models. Traits included in the dataset were mastitis events and average test day milk yields recorded in three different periods: period 1 (5–60 DIM), period 2 (61–120 DIM) and period 3 (121–180 DIM). In addition, lactation persistency was also included. A subset including 28,867 daughters of 1809 Holstein sires having both first and second lactation across 201 herds was further investigated. In these datasets, mastitis events were defined on a lactation basis as binary trait; either a cow was assigned a score of 1 (had a mastitis event in that lactation) or a score of 0 (healthy) for that particular lactation, regardless of the time of occurrence. Total milk yield from first and second lactation were also included in the analyses. We estimated negative structural coefficient (−0.032) between clinical mastitis and test day milk production in early lactation period suggesting that mastitis results in a direct decline in milk production in early lactation. We nonetheless elicited little impact of mastitis on test day milk production of mid and late lactation periods, and on milk yield lactation persistency. Likewise the positive estimate of the structural coefficient (0.123) from mastitis event in first lactation to second lactation suggests an increased risk of mastitis in second lactation if a case of mastitis occurs in the primiparous cow. Heritability estimates obtained from the structural equation models were low for mastitis (ranged 0.04 to 0.07), and negative genetic correlations were found between mastitis events and milk yield. The study illustrates how mastitis events and production are causally linked. Through the use of structural equation models we elicited the causal effect among mastitis and production traits that evolve over the course of cow life.

1. Introduction

Mastitis is a mammary inflammation and is one of the most economically impacting health events in the dairy cattle industry. The losses due to mastitis are costly (Koeck et al., 2012b), mostly due to veterinary and treatment costs (Hinrichs et al., 2005), discarded milk (Shim et al., 2004), and reduced milk production (Bar et al., 2008), but also due to increased risk of culling (Hertl et al., 2011), and increased reproductive problems (Moore et al., 1991). Moreover, replacement costs and increased labor cost due to mastitis directly impact the profitability of dairy enterprises (Huijps et al., 2008). The average cost of a clinical mastitis has been previously estimated at $179 with $115 from milk lost per case, $14 due to increased mortality loss, and $50 from treatment costs (Bar et al., 2008). The antagonistic relationship between health disorders and milk yield in dairy cows is generally accepted (Rauw et al., 1998). In the past 50 years, there has been an intense selection for yield traits. This has resulted in an increased deterioration of dairy health (Miglior et al., 2005). These problems in dairy cattle have pointed towards genetic selection for increased disease resistance and several researchers in the past decade have suggested inclusion of clinical mastitis in the overall breeding goal of Holstein dairy cattle (Kadarmideen and Pryce, 2001; Ødegård et al., 2003). Direct selection for mastitis resistance has been so far fully implemented in Nordic cattle (Heringstad et al., 2003; Philipsson and Lindhé, 2003) and national genetic and genomic

Abbreviations: LP, lactation persistency of milk yield; LMAST, liability to mastitis; MY, milk yield; SEM, structural equation models; TD, test-day; TMY, total milk yield

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evaluations for clinical mastitis has been started in Canada (Jamrozik et al., 2013) and France (Govignon-Gion et al., 2012). Similarly, routine genetic evaluation of mastitis is done in Austria (Fuerst et al., 2011; Koeck et al., 2015).

Several researchers in the past have used mixed models to compute genetic correlations between mastitis and milk yield. Recently, Pfeiffer et al. (2015) described genetic relationships between functional longevity and mastitis as well as other direct health traits. Results from those studies mostly revealed the unfavorable genetic correlations between mastitis events and milk yield traits. Yet correlations do not imply causation and there is still a lack of knowledge about the cause and effect between these traits, which could be addressed using structural equation models (SEM). In the context of animal breeding, Gianola and Sorensen (2004) extended multivariate mixed model theory to infer recursive relationships between phenotypes by accounting for possible feedback situations. Several papers that have been published in the realm of animal breeding over the past few years used structural equation models to infer causal relationships between health traits (Wu et al., 2008; Heringstad et al., 2009; Dhakal et al., 2015). Wu et al. (2008) used a dataset of Norwegian Red cows to study the causal effect between mastitis and milk yield. To the best of our knowledge, no study has been conducted to infer causal relationships between mastitis events and milk yield in the US Holstein cattle population. Similarly, there is a knowledge gap regarding the causal effect of mastitis occurring in first lactation and mastitis events occurring in later lactations. Thus, the objective of the current study was to elicit direct causal phenotypic effects and genetic relationships among mastitis events and production traits (milk yield and lactation persistency of milk yield) in US Holsteins using recursive models.

2. Materials and methods

2.1. Data

Health information records were made available from Dairy Records Management Systems (Raleigh, NC) from US dairy farms from 1996 through June 2013. Holstein cows with mastitis records in first and second parity were retained for the analyses which included calving records from 1996 to 2012. Health data quality edits were applied as described in detail in Parker Gaddis et al. (2012) with some slight modifications for herd edits. In order to avoid herds that over or under reported mastitis events, maximum and minimum constraints were applied to the dataset. The maximum constraint was imposed by excluding records when reporting frequency of herds were greater than two standard deviations above the mean reporting frequency of mastitis event. Similarly, minimum constraint was imposed by selecting records from herd with at least one reported incidence of the mastitis event and herds consisting of at least 5 cows. In addition, only records of cows having lactation length less than or equal to 400 days in milk (DIM) were included. After applying data quality edits, a dataset was formed to identify causal effects between mastitis and production measures (test-day milk yields and lactation persistency) in first parity US Holsteins. This dataset included 48,058 first parity daughters of 2213 Holstein sires across 207 herds and will be referred to as First-Lactation dataset.

The First-Lactation dataset included test-day (TD) records for milk yield (MY) and lactation persistency of MY in addition to mastitis events. Test Day records from 5 to 180 days after calving were included and cows with missing TD records were removed from the original dataset in the process of forming the First-Lactation dataset. Days in milk up to 180 days after calving were divided into 3 lactation periods such that period 1 included 5–60 days, period 2 included 61–120 days, and period 3 included 121–180 days similarly to the procedure adopted by Wu et al. (2008). Single MY TD records were assigned to each period as the closest in time to the midpoint of each segment and will be hereafter referred to as MY1, MY2, and MY3, respectively. Cows were assigned a value of 0 (healthy) or 1 (mastitis) in each period. Only mastitis records that were prior and temporally closer to the assigned TD for each period were considered. This definition implies that pre-existing mastitis events would affect the MY of the following TD. Lactation persistency of MY (LP), a measure describing the shape of the lactation curve after peak milk yield, was calculated for each cow using BESTPRED software (Cole and VanRaden, 2007).

A subset (First & Second-Lactation) dataset was formed to identify causal effects from mastitis in first lactation to second lactation and from mastitis to total milk yield for first and second lactation. The First & Second-Lactation dataset included 28,867 daughters from 1809 sires having first and second lactation across 201 herds. Only cows showing records for both lactations were included in the dataset. To reduce complexity and improve the interpretation of the results mastitis events were in this case defined on a lactation basis as binary trait; a cow was assigned either a score of 1 (had a mastitis event in that lactation) or a 0 (healthy) for that particular lactation, regardless of the time of occurrence. Total milk yield (305-day milk yield) from first and second lactation were also included in the analysis as calculated from the BESTPRED software (Cole and VanRaden, 2007).

2.2. Statistical analysis

Recursive Gaussian-threshold sire models were used for the statistical analyses. The threshold model assumed an underlying continuous variable, liability (l_i), for binary mastitis events that defines the observed binary variable into a value of 1 if liability is larger than a fixed threshold and 0 otherwise. Two different series of analysis (Lactation first (LAC1) and Lactation first and second (LAC12)) were defined for the purpose of identifying causal relationships between mastitis events and production measures. The LAC1 series of analyses employed a SEM to find recursive relationships between mastitis events, TD milk yields, and LP in first lactation. Four analyses were performed, which are as follows:

- LAC1. A: This analysis included two traits: liability to mastitis in the first period (LMAST1) and MY1. The direct recursive effect was assumed from LMAST1 to MY1.
- LAC1. B: This analysis included MY1, liability to mastitis in the second period (LMAST2) and MY2. The direct recursive effect was assumed from MY1 to LMAST2 and from LMAST2 to MY2.
- LAC1. C: This analysis included MY2, liability to mastitis in the third period (LMAST3) and MY3. The direct recursive effect was assumed from MY2 to LMAST3 and from LMAST3 to MY3.
- LAC1. D: Liability to mastitis of each period and LP were included in this analysis. The direct recursive effects were assumed from mastitis of each period (LMAST1, LMAST2, and LMAST3) to lactation persistency of milk yield.

Lastly, the LAC12 analysis (using First & Second-Lactation dataset) assumed direct recursive effects from liability to first lactation mastitis (LM1) to liability to second lactation mastitis (LM2). Direct recursive effects were also considered from LM1 to total milk yield of first parity (TMY1) and also to total milk yield of second parity (TMY2). An indirect recursive effect was also assumed from LM1 to LM2 and TMY2. A direct recursive effect measures how much TMY1, LM2, and TMY2 would be affected by changes in LM1. An indirect recursive effect measures how much LM2, and TMY2 would be affected by changes in LM1 through the