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Inferring relationships between clinical mastitis, productivity and fertility: A recursive model application including genetics, farm associated herd management, and cow-specific antibiotic treatments

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ABSTRACT

A dataset of test-day records, fertility traits, and one health trait including 1275 Brown Swiss cows kept in 46 small-scale organic farms was used to infer relationships among these traits based on recursive Gaussian-threshold models. Test-day records included milk yield (MY), protein percentage (PROT-%), fat percentage (FAT-%), somatic cell score (SCS), the ratio of FAT-% to PROT-% (FPR), lactose percentage (LAC-%), and milk urea nitrogen (MUN). Female fertility traits were defined as the interval from calving to first insemination (CTFS) and success of a first insemination (SFI), and the health trait was clinical mastitis (CM). First, a tri-trait model was used which postulated the recursive effect of a test-day observation in the early period of lactation on liability to CM (LCM), and further the recursive effect of LCM on the following test-day observation. For CM and female fertility traits, a bi-trait recursive Gaussian-threshold model was employed to estimate the effects from CM to CTFS and from CM on SFI. The recursive effects from CTFS and SFI onto CM were not relevant, because CM was recorded prior to the measurements for CTFS and SFI. Results show that the posterior heritability for LCM was 0.05, and for all other traits, heritability estimates were in reasonable ranges, each with a small posterior SD. Lowest heritability estimates were obtained for female reproduction traits, i.e. $h^2 = 0.02$ for SFI, and $h^2 \approx 0$ for CTFS. Posterior estimates of genetic correlations between LCM and production traits (MY and MUN), and between LCM and somatic cell score (SCS), were large and positive (0.56–0.68). Results confirm the genetic antagonism between MY and LCM, and the suitability of SCS as an indicator trait for CM. Structural equation coefficients describe the impact of one trait on a second trait on the phenotypic pathway. Higher values for FAT-% and FPR were associated with a higher LCM. The rate of change in FAT-% and in FPR in the ongoing lactation with respect to the previous LCM was close to zero. Estimated recursive effects between SCS and CM were positive, implying strong phenotypic impacts between both traits. Structural equation coefficients explained a detrimental impact of CM on female fertility traits CTFS and SFI. The cow-specific CM treatment had no significant impact on performance traits in the ongoing lactation. For most treatments, beta-lactam-antibiotics were used, but test-day SCS and production traits after the beta-lactam-treatment were comparable to those after other antibiotic as well as homeopathic treatments.

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1. Introduction

Clinical mastitis (CM) is a multi-factorial disease with substantial impact on farm economy. The financial loss of CM is mainly due to costs for treatments, discarded milk after antibiotic treatments, a decline in test-day milk yield, and increased labor. A detailed summary based on updated cost components per cow in an international context, underlining the economic importance of CM was provided by Sadeghi-Sefidmazgi et al. (2011). Multiple factors with impact on CM can be decomposed into "cow specific" and "general herd associated" factors. Environmental, herd-associated risk factors include feeding, husbandry, and farm management, which might be identical for cows in the same parity with a comparable lactation stage and a comparable calving season, kept in the same herd. Cow-specific factors include an environmental and a genetic component. This environmental component can be interpreted as preferential treatment, which is a specific, individual treatment that is only relevant for a single cow, or for a certain group of cows. The relationships between the cow-specific antibiotic treatment and incidences of CM and bacteriological cure rates in the dry period and in early lactation were shown by Gundelach et al. (2011). As a consequence, the applied antibiotic therapy not only affects incidences of CM and somatic cell score in the ongoing lactation, but also has effects on related traits describing productivity and milk quality (Milner et al., 1997). The fact that different treatments can cause differences in physiological reactions was shown by Van Eenennaam et al. (1995).

The most sustainable method for preventing CM is the consolidation of the genetic background for CM resistance. In the past, breeding strategies in dairy cattle focused on productivity by neglecting functionality (e.g. Miglior et al., 2005). Both direct selection on clinical mastitis (CM) and inclusion of CM into an overall breeding goal for Holstein dairy cattle have been suggested by several authors in the past decade (e.g. Kadarmideen and Pryce, 2001; Ødegard et al., 2003). A prerequisite when considering CM in an overall breeding goal is the availability of genetic (co)variance components between CM and other traits of interest. The traditional and mostly used method to infer relationships among CM and production traits (or other functional traits) is the application of standard mixed model theory and restricted maximum likelihood (e.g. Emanuelson et al., 1988). More recent studies applied threshold methodology in a Bayesian framework on binary health disorders to estimate genetic parameters for CM on the underlying liability scale (e.g. Heringstad et al., 2005). Based on estimates of genetic correlations, most of these studies supported antagonistic relationships between CM and production traits, either on the observed scale or on the underlying liability scale. A wider range of correlations is reported in the literature between CM and fertility, or between CM and other health disorders (e.g. Gernand et al., 2011).

However, correlations have no implication on the cause-effect relationships in a multi-trait system. For example, when analyzing associations between 305-d lactation records for milk or protein yield with health disorders, variation of the production trait of interest can be a result, at least partially, from the previous or from the current lactation (Fleischer et al., 2001). Hence, this leads to the need to quantifying the relationship between the cause and the effect. For studying production-related diseases, Gianola and Sorensen (2004) proposed recursive and simultaneous models to infer the relationships between phenotypes in the context of animal breeding and genetics. Their models, as a special category of the so-called 'structural equation models' (SEM), extended multivariate mixed model theory by accounting for possible feedback situations and recursive relationships among phenotypes. Furthermore, Wu et al. (2008) extended the models of Gianola and Sorensen (2004) to allow joint analysis of Gaussian and categorical traits, leading to so called "recursive and simultaneous Gaussian-threshold models". Originally, the idea for describing simultaneous and recursive relationships between phenotypes was developed for biological systems in humans by Haldane and Priestley (1905). In the past five years, structural equation models have been applied to infer genetic parameters in dairy cattle. In particular, this type of novel models was used to explore recursive and simultaneous effects in a limited number of studies (Table 1), with a strong focus on the estimation of genetic parameters. When modeling the environmental component, studies summarized in Table 1 did not distinguish between "cow-specific" and "general herd effects". Janssens and van Duijn (2008) focused on a broader application and further prospects of SEM or pathway model applications in human genetics by combining information of genetic variants with environmental risk factors, e.g. for an accurate disease prediction.

The aim of the present study was to infer genetic parameters and structural equation coefficients on the phenotypic pathway between CM and test-day records for production traits, and between CM and fertility traits. Unlike previous studies analyzing the genetic background of CM, detailed information on treatments of CM was collected and considered in the statistical modeling to distinguish in detail between general herd effects and cow-specific risk factors.

2. Materials and methods

2.1. Data

The final dataset comprised test-day records, the health disorder CM, and fertility traits from 1275 Brown Swiss cows kept in 46 small-scale organic farms located in the alpine region of Switzerland. The data pool includes over 80% of the organic and low input herds registered with the official Brown Swiss herdbook. Cows were daughters of 333 sires. Up to now, an independent organic breeding program does not exist, and consequently, the same sires are used in organic and conventional Brown Swiss herds. Test-day records included milk yield (MY), protein percentage (PROT-%), fat percentage (FAT-%), somatic cell score (SCS), the ratio of FAT-% to PROT-% (FPR), lactose percentage (LAC-%), and milk urea nitrogen (MUN). Organic and low input farming entails limitations in feeding of concentrates. Despite some similarities, also the different organic herds are characterized by differences in feeding