

**Abstracts of the
2017 American Dairy Science Association®
Annual Meeting**

**June 25–28, 2017
Pittsburgh, PA**

***Journal of Dairy Science*®
Volume 100, Supplement 2**

Table 1 (abstract 376). Estimates of heritability (in bold), genetic correlations (above diagonal), and phenotypic correlations (below diagonal) for the treatment costs of 5 health categories and total health treatment cost for first parity

	MAST	REPRO	LAME	META	MISC	THC
MAST	0.13*	0.85*	0.34	0.52	0.66	0.92*
REPRO	0.00	0.04	0.41	0.73*	0.59	0.91*
LAME	0.03	-0.01	0.10*	0.56*	0.21	0.65*
META	0.02	0.14*	0.02	0.12*	0.40	0.85*
MISC	0.04*	0.02	-0.05*	0.16*	0.04	0.72*
THC	0.34*	0.66*	0.27*	0.63*	0.39*	0.27*

*Estimate significantly different from zero based on 95% CI.

Key Words: health treatment cost, Holstein

377 Development of genomic evaluations for direct measures of health in US Holsteins and their correlations with fitness traits.

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The objectives of this research were to estimate variance components for 6 common health events recorded by producers on US dairy farms, as well as investigate correlations with fitness traits currently used for selection. Producer-recorded health event data were available from Dairy Records Management Systems (NCSU, Raleigh, NC) for 6 common health events occurring in US dairy herds: hypocalcemia (CALC), displaced abomasum (DSAB), ketosis (KETO), mastitis (MAST), metritis (METR), and retained placenta (RETP). Standardization and editing constraints were applied to ensure data validity. After editing, the number of phenotypic records ranged from 1.2 million for CALC up to 2.5 million for MAST. Traditional predicted transmitting abilities (PTA) were calculated for 63.1 million Holsteins using a linear animal model accounting for year-season, age-parity, herd-year, and permanent environmental effects, as well as a regression on inbreeding. Heritability estimates on the observed scale were 0.6%, 1.1%, 1.2%, 3.1%, 1.4%, and 1.0% for CALC, DSAB, KETO, MAST, METR, and RETP, respectively. Genomic PTA were calculated using 60,671 markers for 1.36 million Holsteins. For bulls with >90% reliability (>75% for CALC and RETP), health trait PTA had low correlations with PTA protein (-0.03 to 0.23) but much higher correlations with official PTA for several fitness traits included in net merit. Largest correlations for each health trait were -0.68 for MAST with somatic cell score (SCS), 0.59 for KETO with daughter pregnancy rate (DPR), 0.47 for DSAB with livability, 0.46 for METR with DPR, -0.29 for CALC with SCS, and 0.17 for RETP with productive life (PL). An economically weighted sum of all 6 health trait PTA was correlated by 0.56 with PL, 0.55 with livability, 0.50 with DPR, and -0.45 with SCS, using estimated costs per case of \$72 for MAST, \$178 for DSAB, \$105 for METR, \$64 for RETP, \$38 for CALC, and \$28 for KETO. Young animal reliabilities averaged 11–18% from the pedigree model vs. 40–49% from genomic predictions. The standard deviation of lifetime net merit is \$193 compared with \$8 for the sum of health trait PTA that could be included in the near future.

Key Words: genetic evaluation, health, fitness

378 Genomic analysis of ketosis susceptibility in Jersey cattle.

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The objectives of this research included estimating variance components for ketosis susceptibility and identification of genomic regions associated with ketosis in Jersey cattle. Ketosis is one of the most commonly reported metabolic diseases in dairy herds. Genetic analyses of ketosis have been conducted previously, but few focus specifically on Jersey cattle. Voluntary producer-recorded health event data related to ketosis were available from Dairy Records Management Systems (NCSU, Raleigh, NC). Standardization was implemented to account for the various acronyms used by producers to designate an incidence of ketosis. Ketosis events were restricted to the first reported incidence within 60 d of calving in first through fifth parity. After editing, there were a total of 42,233 records from 23,865 cows. A total of 1,750 genotyped animals were used for analyses using 60,671 markers. Given the binary nature of the trait, a threshold animal model was fitted using THRG-IBBS1F90 (version 2.110) using only pedigree information and then also incorporating genomic information using a single-step genomic BLUP approach. postGSf90 (version 1.38) was used to calculate SNP effects as well as variance explained by 10-SNP windows. Heritability of ketosis susceptibility was 0.083 (SD = 0.021) and 0.078 (SD = 0.018) in pedigree-based and genomic analyses, respectively. The marker with the largest estimated SNP effect was located on chromosome 10 at 66.3 Mbp. Additional peaks were identified on chromosomes 11, 14, and 23. The region explaining the largest proportion of variance (0.70%) was located on chromosome 6 at 56.1 Mbp. Additional regions explaining large proportions of variance were located on chromosomes 11 (0.51%), 3 (0.45%), and 25 (0.40%). Genes located in these regions were investigated for having a role in ketosis susceptibility. Results indicate that ketosis susceptibility in Jerseys has a significant genetic component, making feasible the selection for animals more resistant to ketosis. Associated genomic regions could be incorporated into genetic evaluations in the future, as well as used to further understand the underlying biology of this disease.

Key Words: genomic analysis, Jersey, ketosis

379 Genome-wide association study for clinical mastitis, metritis, and ketosis in US Holstein cattle.

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Health traits impact the profitability of dairy production and affect animal welfare. The objective of this study was to perform a genome-wide association study (GWAS) to identify genomic regions, and preferably candidate genes associated with Clinical Mastitis (CM), Metritis (MET) and Ketosis (KET) in US Holstein cattle. Data consisted of 28,000 producer-recorded health event records from 14,000 cows in one large commercial dairy farm. Whole-genome single nucleotide polymorphism (SNP) data were available for 7,500 animals. The association analyses were performed using the single-step genomic BLUP approach combining all the available phenotypic, pedigree and genotypic data into threshold models. These models included year-season and parity as fixed effects, and animal and permanent environmental as random effects. Candidate regions were identified based on the amount of genetic variance explained by 2-Mb SNP windows. Several genomic regions were associated with these 3 relevant postpartum diseases. For instance, 4 regions located on BTA5, BTA6, BTA8 and BTA 29 explained together more than 3.0% of the genetic variance for CM. These regions harbor many candidate genes, such as *CXCL13*, *SPTLC1*, and *FADD* that are involved in mammary gland inflammation. Similarly, different regions on BTA9, BTA13, BTA14 and BTA29 explained between 0.75% and 1.0% of the genetic variance for MET. These regions harbor several interesting genes, including *GSDMC* and *CCR6* that are directly