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Investigating the potential for genetic selection of dairy calf disease traits using management data

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ABSTRACT

Genetic selection could be a tool to help improve the health and welfare of calves, however, to date, there is limited research on the genetics of calfhood diseases. This study aimed to understand the current impact of calf diseases, by investigating incidence rates, estimating genetic parameters, and providing industry recommendations to improve calf disease recording practices on farms. Available calf disease data comprised of 69,695 Holstein calf disease records for respiratory problems (**RESP**) and diarrhea (**DIAR**), from 62,361 calves collected on 1,617 Canadian dairy herds from 2006 to 2021. Single and multiple trait analysis using both a threshold and linear animal model for each trait were evaluated. Furthermore, each trait was analyzed using 2 scenarios with respect to minimum disease incidence threshold criterion (herd-year incidence of at least 1% and 5%) to highlight the impact of different filtering thresholds on selection potential. Observed scale heritability estimates for RESP and DIAR ranged from 0.02 to 0.07 across analyses, while estimated genetic correlations between the traits ranged from 0.50 to 0.62. Sires were compared based on their estimated breeding value and their diseased daughter incidence rates. On average, calves born to the bottom 10% of sires were 1.8 times more likely to develop RESP and 1.9 times to develop DIAR compared with daughters born to the top 10% of sires. Results from the current study are promising for the inclusion of both DIAR and RESP in Canadian genetic evaluations. However, for effective genetic evaluation we require standardized approaches on data collection and industry outreach to highlight the importance of collecting and uploading this information to herd management software. In particular, it is important that the herd management software is accessible to the national milk recording system to allow for use in national genetic evaluation.
Key Words: Disease traits, producer recorded data, data quality, genetic parameters

INTRODUCTION

Growing awareness surrounding agricultural production coupled with the effects of disease on a cow's lifetime performance, has led to the dairy industry taking proactive steps to improve animal health. To achieve improved animal health, a multi-faceted approach focusing on the interaction of 3 factors is required, known as the triad of epidemiology. This interaction controls the expression of disease, and includes the pathogen causing the disease, the host's ability to resist the pathogen and the environment in which they both live, including management practices (Leblanc et al., 2006). Improving host resistance has centered on vaccine programs and prophylactic antibiotic treatment. However, antibiotics and vaccines are under increasing scrutiny because they often only reduce clinical signs of the disease or provide short-term immunity and do not prevent the spread of the disease, as is the case in Johne's disease and Leptospirosis in dairy cattle (Phanse et al., 2020; Wilsonwelder et al., 2020). Furthermore, they can contribute to the development of antibiotic resistant bacteria, and vaccine resistant viruses, which makes these interventions less desirable (Gibson and Bishop, 2005).

The host's innate resistance can also be improved through genetic selection, though with the exception of the Nordic countries, this has, until recently, received little attention (Miglior et al., 2017). The reasoning behind this was the low heritability estimates associated with functional traits, such as health, and the opinion that improvements in management could balance out any deterioration in genetic merit of functional traits (Butler and Moore, 2018). Through the adoption of genomic selection, selection on low heritable traits has become more feasible (Miglior et al., 2017; Cole

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et al., 2021). In Canada, national genetic evaluations have been developed for several health related traits including, mastitis (Koeck et al., 2012; Beavers and Van Doormaal, 2013), metabolic diseases (Jamrozik et al., 2016; Miglior et al., 2016), hoof lesions (Malchiodi et al., 2017; Beavers and Van Doormaal, 2018; Jamrozik et al., 2021) and fertility disorders (Fleming and Van Doormaal, 2020). However, limited emphasis pertaining to improving calf health has been applied on a national level in Canada, with the exception of calf survival (Van Doormaal, 2007; van Staaveren et al., 2023).

The 2 most common calf diseases are respiratory problems (**RESP**) and diarrhea (**DIAR**) (Murray, 2011; Windeyer et al., 2014). Average incidence rates for DIAR range from 23% to 44%, while for RESP, incidence rates range between 12% to 22%, however within herds, large variation can be seen around these values (Windeyer et al., 2014; Urie et al., 2018; USDA, 2018). Calfhood diseases have a major impact on the economic viability of cattle operations; heifer's experiencing a disease at least once during rearing have 6% higher rearing costs than healthy heifers (Nor et al., 2013). Sischo et al. (1990) reported that calf disease accounted for 4% of a cow's lifetime cost, with RESP and DIAR responsible for 86% of calf disease costs. These economic consequences are due to costs of treatment, reduced average daily gain (**ADG**), increased age at first calving (**AFC**), increased mortality rates and long-term effects on performance.

The impact of calf disease on ADG and in turn on AFC is because disease reduces growth. Buczinski et al. (2021) reviewed 27 studies and reported that heifers experiencing calfhood RESP had their ADG reduced by 0.067 kg/d. Effects of DIAR on ADG are less conclusive with some studies finding that DIAR decreased ADG by up to 0.03kg/d (Donovan et al., 1998; Stanton, 2011), while others found no effect (Virtala et al., 1996). Potential reductions in ADG have implications for AFC as a heifer's weight is directly associated with sexual maturity (Macdonald et al., 2005), and unfavorable associations between AFC and calf disease incidence have been reported (Heinrichs et al., 2005). Pre-weaning mortality rates in dairy calves range from 5 to 11% (Murray, 2011; Urie et al., 2018; Zhang et al., 2019), and in Canada, DIAR and RESP accounted for 53% and 21% of pre-weaned calf mortality, respectively (Murray, 2011). With respect to production, cows with at least one episode of DIAR as calves produced 344 kg less energy corrected milk than healthy animals in first lactation (Svensson and Hultgren, 2008). Buczinski et al. (2021) also reported that heifers experiencing RESP produced 121.2 kg less milk during their first lactation compared with healthy animals. Together this highlights the impact that calf disease has on animals transitioning from calfhood into the milking herd, and why RESP and DIAR are the main reasons behind involuntary culling or mortality until first calving (Waltner-Toews et al., 1986; Wathes et al., 2008; Zhang et al., 2019). Given the impact of calf disease on farm productivity, efforts to improve dairy calf health are vital.

From a genetic perspective, efforts aimed at breeding for increased disease resistance in both RESP and DIAR offer opportunities. Reported heritability estimates for RESP and DIAR range between 0.04 to 0.10 (Henderson et al., 2011; McCorquodale et al., 2013; Gonzalez-Peña et al., 2019; Haagen et al., 2021; Zhang et al., 2022), indicating the potential for genetic selection. The low heritability for disease traits can be attributed to their complexity and the quality of records (Bishop and Woolliams, 2010). On dairy farms, calf disease data is usually recorded as an event in herd management software. However, factors influencing calf health, such as birth weight, colostrum quality and quantity, duration and severity of disease, and so on, are not routinely measured and recorded (Gonzalez-Peña et al., 2019). Furthermore, in Canada, no standardized approach to recording this information is available, meaning herds likely differ in recording practices and definitions of traits (van Staaveren et al., 2023). For genetic selection, the recording of accurate high quality phenotypes is paramount (Coffey, 2020). With the integration of efficient recording systems, genetic selection for improved health becomes more feasible and effective (Fleming et al., 2018).

Therefore, the objective of this study was to 1) understand the current impact of calf diseases, by estimating incidence rates and level of recording on Canadian dairy farms, 2) to estimate genetic parameters for calf disease traits using data collected via on-farm herd management software (DairyComp, VAS, Tulare, California), and 3) to provide preliminary industry recommendations to improve calf disease recording practices on farms. Specifically, these results can be used to identify gaps in data collection methods which should be addressed in future developments for successful implementation of a national calf health selection strategy.

MATERIALS AND METHODS

Data

Calf disease data recorded by dairy producers with herd management software and uploaded into the national milk recording database were provided by Lactanet Canada (Guelph, Ontario). They were comprised of 69,695 Holstein calf disease records for RESP and

DIAR, from 62,361 calves collected on 1,617 Canadian dairy herds from 2006 to 2021. Within these records, 49,328 were RESP and 20,364 were DIAR. Three animals had multiple records of DIAR cases, which were removed for this analysis. In total, 7,331 animals had records on both traits. A herd inventory file was also supplied by Lactanet, and provided date of birth information for all calves reared in each herd. Any animal present in the herd inventory file that was not found in the disease cases data was assumed healthy.

Both single and multiple trait analysis were conducted. For single trait analysis, RESP and DIAR were treated as binary events, with a value of 0 corresponding to an assumedly healthy animal, and 1 corresponding to a sick animal for each trait. RESP records above 180 d of age were removed, while DIAR records above 60 d of age were removed due to limited records past this point, meaning only early calfhood records were analyzed for both traits. Two thresholds with respect to minimum disease incidence were analyzed, to highlight the impact of filtering on selection potential. The first scenario required each herd-year to have at least 1% incidence (**1%DATA**), while the second scenario required a 5% minimum herd-year incidence (**5%DATA**). This was calculated using the number of disease events in a herd-year divided by the total number of heifer calves raised on the farm in that year. These minimums were chosen because 1% incidence is commonly used in other disease related studies (Koeck et al., 2012; Haagen et al., 2021), and 5% can be seen as the gold standard target for both DIAR and RESP (Dairy Calf & Heifer Association, 2019; RSPCA, 2021). In the case of 1% incidence it may be that incidence are not being recorded in full, or that differences in classification of either disease are used on specific farms.

Records from 2006 and 2021 were removed as recording in those years was incomplete, leaving a useable data set with records collected between 2007 and 2020. All animals with missing sire or dam information were also removed, roughly 16% of total records. Additionally, herds had to have consecutive calf disease records for years leading to and including 2020, or a minimum of 3 consecutive years of data available if there were no 2020 records. This control step was done to ensure only herds with consistent recording practice were included. For the multiple trait analysis between RESP and DIAR, only herds that met the criteria described above for both traits were included. Final data sets are presented in Table 1.

Statistical Analysis.

Due to the discrete nature of the traits, they were evaluated using both a threshold and linear animal model. For the threshold model, a probit link function was applied if the trait was binary, while a logit link function was applied if the trait was categorical. The threshold model is theoretically a better model as it is designed to respect the discrete nature of the trait by fitting a non-Gaussian distribution. However, threshold models are more complex and computationally demanding compared with linear models. Furthermore, studies have shown that with high numbers of records, model fit and animal ranking do not change significantly between threshold and linear animal models (Jamrozik et al., 2005; Negussie et al., 2008). To compare the models, Spearman's rank correlation was calculated between the estimated breeding value (**EBV**) of sires with at least one daughter record.

For the threshold animal model, the 'THRGIBBS1F90' program within the BLUPF90 family of programs was used, which required the disease phenotypes to be recoded to 1 (healthy) and 2 (sick) (Misztal et al., 2014). For each analysis, a chain of 500,000 samples, a burn-in period of 50,000 samples and a thinning interval of 50 samples were used. Convergence of all chains was verified based on the Heidelberger and Welch, and Geweke convergence diagnostic tests within the 'boa' R package (Smith, 2016). For the linear animal model, the

Table 1: Final data sets for single and multiple trait analyses in both 1% and 5% minimum incidence scenarios of diarrhea and respiratory problems in dairy calves on Canadian farms

Analysis		Trait	Diseased	Healthy	Total Records (No. of herds)
Single Trait	1%	Diarrhea	12,662	52,980	65,642 (122)
		Respiratory problems	29,388	128,885	158,273 (288)
	5%	Diarrhea	11,058	28,907	39,965 (61)
		Respiratory problems	26,307	76,131	102,438 (180)
Multiple Trait	1%	Diarrhea only	6,161	33,756	
		Respiratory problems only	8,868		53,683 (96)
		Both diseases	4,898		
	5%	Diarrhea only	4,797		
		Respiratory problems only	5,659	16,164	31,103 (45)
		Both diseases	4,483		

'AIREMLF90' program within the BLUPF90 family of programs was used (Misztal et al., 2014).

For the threshold model analysis of binary traits, heritabilities were originally estimated on the liability scale and then converted to the observed scale using the method described by Alan Robertson in the Appendix of Dempster and Lerner (1950). Conversion from the liability scale to the observed scale was done as follows:

$$h_o^2 = \frac{h_l^2 \times z^2}{p(1-p)},$$

where h_o^2 is heritability on the observed scale, h_l^2 is heritability on the underlying liability scale, p is the proportion of affected individuals in the population of interest, z is the height of the standard normal curve at the truncation threshold for the corresponding value of p.

Models. The general form of the models used in both the linear and threshold analysis are described as:

$$y = Xb + Z_{hys}hys + Z_a a + e,$$

where y in the threshold model is a vector of underlying liabilities corresponding to the binary observation (0= healthy, 1= diseased), and in the linear model is the observed binary phenotype (0= healthy, 1= diseased), or categorical phenotype in the case of the MORB+ trait (0= healthy, 1= one disease, 2= both disease), b is a vector of fixed effects of year born-season [years 2007 to 2020 were considered; 4 birth seasons were defined as: Spring (Apr-Jun), Summer (Jul-Sep), Fall (Oct-Dec), and Winter (Jan-Mar)], hys is a vector of random effects of herd-year-season at birth, a is a vector of random additive genetic effects, e is a vector of random residuals and, X, Z_{hys} and Z_a are corresponding incidence matrices. For the single trait analysis, the covariance matrix ($\mathbf{V}$) was defined as:

$$\mathbf{V} = \text{var}\begin{bmatrix} a \\ hys \\ e \end{bmatrix} = \begin{bmatrix} \mathbf{A}\sigma_a^2 & 0 & 0 \\ 0 & \mathbf{I}\sigma_{hys}^2 & 0 \\ 0 & 0 & \mathbf{I}\sigma_e^2 \end{bmatrix},$$

assuming that $\begin{bmatrix} a \\ hys \\ e \end{bmatrix} \sim \text{N}(0, \text{V})$, where $\mathbf{A}$ is the additive relationship matrix, σ_{hys}^2 is the herd-year-season born variance, σ_a^2 is the additive genetic variance and σ_e^2 is the residual variance. For the multi trait analysis, the same model factors were fit and the covariance matrix ($\mathbf{V}$) was defined as:

$$\mathbf{V} = \text{var}\begin{bmatrix} a \\ hys \\ e \end{bmatrix} = \begin{bmatrix} \mathbf{A} \otimes \mathbf{G} & 0 & 0 \\ 0 & \mathbf{I} \otimes \mathbf{H} & 0 \\ 0 & 0 & \boldsymbol{I} \otimes \boldsymbol{R} \end{bmatrix},$$

assuming that $\begin{bmatrix} a \\ hy \\ e \end{bmatrix} \sim \text{MVN}(0, \text{V})$, where a is a vector of random additive genetic effects, hys is a vector of random effects of herd-year-season at birth, e is a vector of random residuals, with all three vectors sorted by trait, $\mathbf{G}$ is the covariance matrix of random additive genetic effects between traits, $\mathbf{A}$ is the additive relationship matrix, $\mathbf{H}$ is the covariance matrix of random herd-year-season effects between traits, $\mathbf{R}$ is residual covariance matrix between traits, and $\mathbf{I}$ is an identity matrix.

Comparison of Sires

To compare the difference between sire daughters' performances, sires with a minimum of 20 daughter records were subset from each of the single trait and multiple trait analyses. Half of the sire's daughters' records were removed at random, while the other half were used to predict the sire's EBV. Model parameters were fixed to estimates from each respective analysis within this study, and the 'BLUPF90' program within the BLUPF90 family of programs was used to predict EBV (Misztal et al., 2014). Sires were then ranked based on their predicted EBV. The removed records were used to calculate the percentage of diseased daughters for each sire, and in turn acted as an independent sample. The percentage of diseased daughters per sire was compared for RESP and DIAR. This process was completed 5 times, and the reported values were averaged across the 5 iterations.

Given the large environmental effect on disease traits, simply comparing raw phenotypes can be biased, especially if a sire's daughters are not evenly spread across herds. Sires with only daughters in a single well managed herd may have a low diseased daughter percentage mainly due to the environment and not the sire's influence. Therefore, a second analysis was conducted, whereby the raw phenotypes were corrected for the environmental effect of herd-year. These corrected phenotypes provided a better indication of the sires influence on the phenotype. However, by accounting for herd-year effects the binary phenotype is changed to a continuous variable, removing the ability to compare incidence rates between sires. Therefore, the sires' mean daughter corrected phenotype were compared based on standard deviations from the mean. This correction was completed using linear regression of the raw

phenotype on the herd-year effect. The 'lm' function in R (R Core Team, 2021) was used to estimate herd-year effects. The major value in the first analysis is to better understand differences both groups at a phenotypic level, but is unfortunately biased by environmental factors that impact the phenotype. In the second analysis, phenotypes are adjusted for herd-year effects, accounting for environmental differences, and allowing for a better understanding of differences at a genetic level.

RESULTS AND DISCUSSION

Distribution by Age

Each disease occurrence was plotted against the age at recording in the full original data set using R (R Core Team, 2021). Upon removing outliers, RESP occurred from 0 to 184 d of age, while DIAR occurred from 0 to 94 d of age. Distributions of RESP and DIAR by age are given in Figures 1 and 2, respectively. The distribution of RESP peaked around 30 d of age, with a median age of occurrence of 43 d of age. This distribution is largely in agreement with previous studies (Gonzalez-Peña et al., 2019), however, within the current study no peak is seen between d 70 to 110. During this time, calves are generally weaned and moved, which can be stressful for calves and is associated with a spike in RESP cases (Gorden and Plummer, 2010). In Canada, there is a trend toward moving animals to group environments earlier in life, and this transition early in life may better equip calves to deal with the stress of changing environments later in life (Costa et al., 2016). However, occurrences might not be recorded as strictly past weaning, due to reduced animal viewing time. In this study, 4% of herds did not collect records past 70 d of age, with a further 15% of herds which had less than 20% of their total records past d 70.

The distribution of DIAR peaked around 10 d of age, which was the same as the median, and was similar to other studies (Windeyer et al., 2014; Gonzalez-Peña et al., 2019). This peak was expected as the majority of DIAR cases occur within the first 2 weeks of life (Blanchard, 2012). However, though the distribution matched expectations, 15% of herds did not collect records after 21 d of age. Both distributions within this study highlight that many producers are limiting recording at a certain age, which is an issue for genetic selection, as animals are likely being assumed healthy when they may have been diseased.

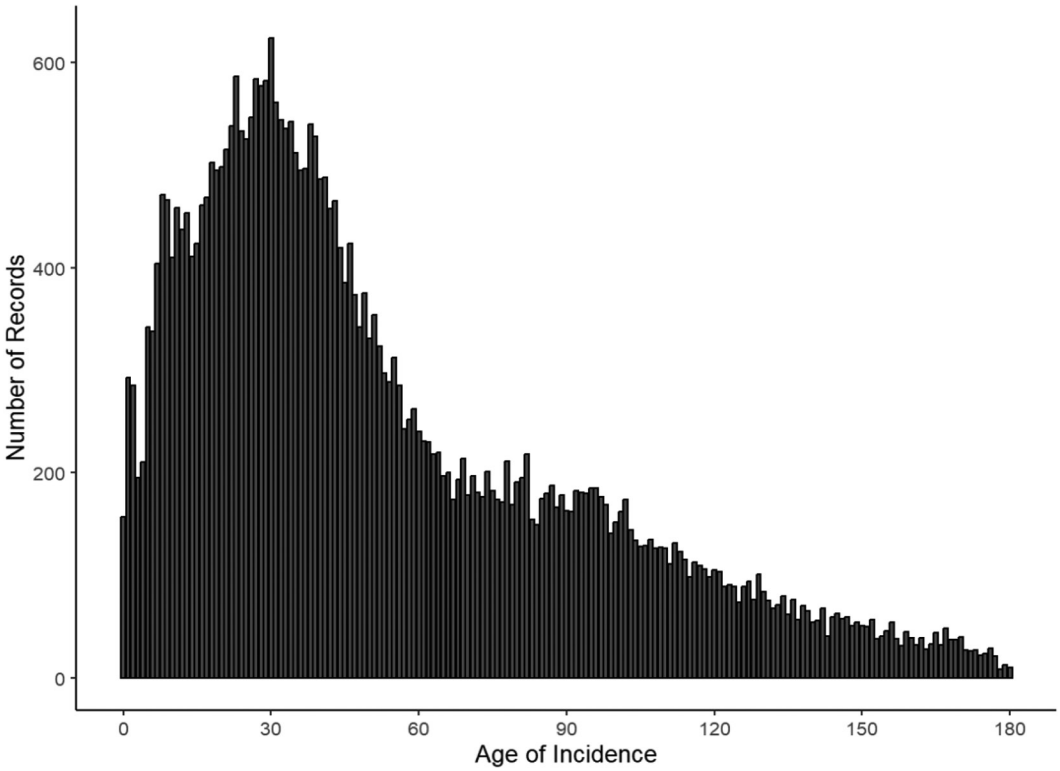

Figure 1. Distribution of respiratory problems in Canadian dairy calves by age of incidence (in days).

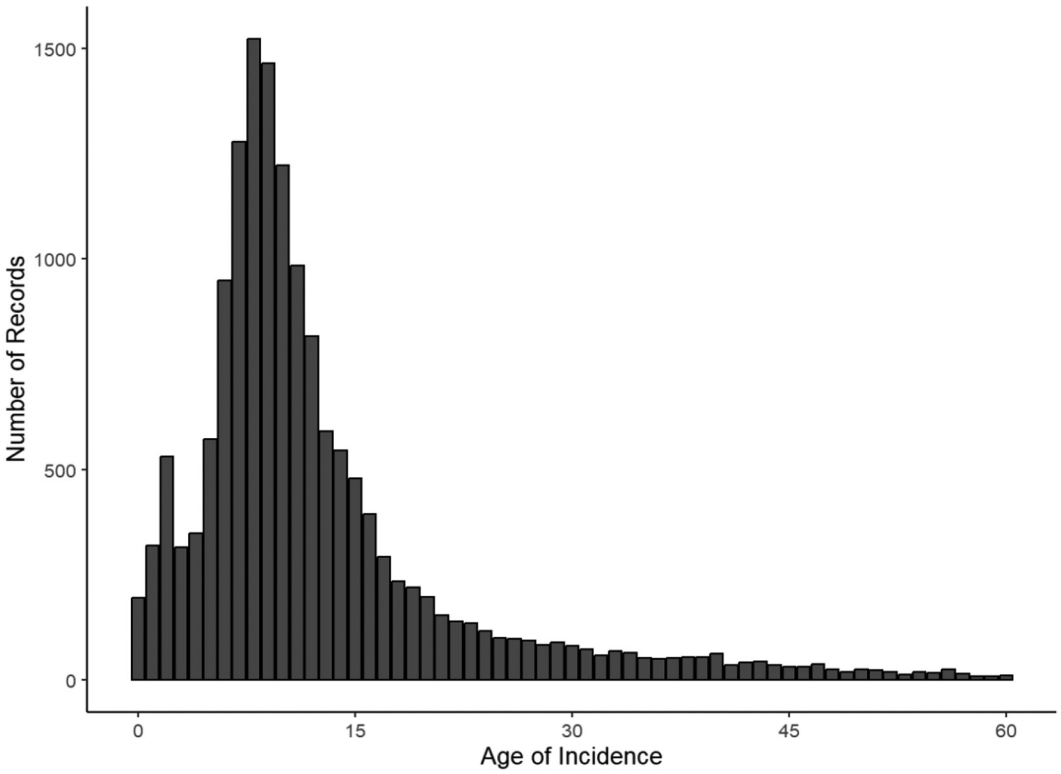

Figure 2. Distribution of diarrhea in Canadian dairy calves by age of incidence (in days)

Incidence Rates

Incidence rates in the first 6 mo of life for RESP and in the first 2 mo of life for DIAR were calculated by year in R (R Core Team, 2021) for the full, 1%DATA and 5%DATA data sets (Table 2). The median incidence rate was reported as it provides a better representation of a typical incidence rate, due to a small number of herds with high incidence rates inflating the mean value. For RESP, the median incidence rate ranged from 6% to 20%, and increased across years and with the higher thresholds (Table 2). A similar trend was observed for DIAR (median incidence range: 5% to 21%). Increased median incidence rates for RESP and DIAR were expected as the thresholds increased (1%DATA, 5%DATA) due to the low incidence herds being removed. The main point of interest, however, is the number of herds in each data set across years. As mentioned, 5% is the gold standard target for incidence of both diseases, however the majority of herds were removed when filtering for the 5%DATA. For example in 2020, the 5%DATA contained 54% fewer herds for RESP and 61% fewer herds for DIAR than the full data set. This highlights that though RESP and DIAR are being recorded on farms, they are likely being under recorded.

Reported incidence rates for RESP were similar to incidence rates previously reported in literature in both the 1%DATA and 5%DATA, while the rates for DIAR were only similar in the 5%DATA (Urie et al., 2018; USDA, 2018; Haagen et al., 2021). The under-recording across both traits may be due to several reasons, including the lack of standardisation of data collection practices and perceived value in uploading calf disease information to herd management software. The issue with under-recording is that it will impact genetic evaluations going forward, as animals may be assumed healthy when they were actually sick, ultimately reducing the reliability of EBVs. However, it is important to note that the average number of herds reaching the 5% threshold gradually increased in both traits across years in the data set. This means that improvements in calf disease data collection have occurred, and provides promise for standardized large-scale data collection on Canadian farms.

Variance Components

Variance components for RESP and DIAR were estimated in single and multiple trait analysis, using both linear and threshold models, and are presented in Table 3 and Table 4. On average, computing time for the

Table 2: Disease incidence rates for respiratory problems and diarrhea in Canadian dairy calves across years in the full data set, 1% and 5% minimum herd year incidence scenarios

	Median DIAR % (No. of herds)			Median RESP % (No. of herds)		
Year	Full Data	1% Data	5% Data	Full Data	1% Data	5% Data
2007	5 (55)	18 (15)	21 (8)	6 (149)	10 (61)	20 (33)
2008	5 (84)	8 (27)	14 (13)	7 (189)	11 (89)	14 (56)
2009	5 (77)	8 (33)	14 (16)	7 (192)	9 (120)	20 (74)
2010	6 (110)	11 (43)	18 (21)	6 (237)	9 (141)	17 (77)
2011	6 (115)	12 (49)	15 (28)	7(297)	10 (172)	20 (96)
2012	6 (129)	12 (57)	19 (31)	7 (300)	9 (185)	18 (108)
2013	6 (145)	10 (60)	18 (37)	7 (355)	11 (212)	16 (124)
2014	6 (132)	11 (62)	20 (39)	7 (408)	11 (224)	18 (134)
2015	6 (148)	13 (65)	21 (39)	9 (371)	9 (230)	18 (141)
2016	6 (109)	7 (63)	13 (32)	7 (320)	10 (214)	14 (128)
2017	5 (122)	6 (79)	17 (35)	7 (375)	11 (234)	16 (148)
2018	7 (163)	10 (90)	16 (49)	9 (469)	12 (300)	18 (188)
2019	7 (213)	10 (127)	15 (75)	9 (499)	12 (341)	19 (226)
2020	9 (176)	12 (112)	20 (68)	9 (404)	13 (275)	18 (184)

DIAR = Diarrhea; RESP = Respiratory Problems.

linear model was under 2 h, while the threshold model was roughly 37 h. In each analysis, the Spearman rank correlation between the linear and threshold models for sires with daughter records was above 0.98. This means that sires ranked similarly in both models, and that the linear model is sufficient to use for EBV prediction. Heritability estimates from the threshold model were estimated on the liability scale and therefore, needed to be converted to the observed scale. It was considered most appropriate to discuss parameter estimates on the observed scale because linear models are used in Canadian national genetic evaluations for other discrete traits and therefore, it would be more meaningful for the dairy industry. Furthermore, methods based on Gibbs Sampling are not usually recommended for official or national evaluations because of their fluctuations in EBV/Genomic EBV in subsequent evaluations.

Observed scale heritability estimates for RESP ranged from 0.03 to 0.07 in the linear model and from 0.02 to 0.06 in the threshold model across analyses in this study. For DIAR, observed scale heritability estimates ranged from 0.04 to 0.07 in the linear model and from 0.03 to 0.06 in the threshold model. These heritability estimates were similar to those reported in the literature (Mahmoud et al., 2017; Haagen et al., 2021; Zhang et al., 2022), however, higher estimates have also been reported. Henderson et al. (2011) reported observed scale heritability estimates of 0.10 for RESP. This higher estimate may be due to their highly accurate phenotyping and the incorporation of specific calf information into their model, such as calf weight and serum total protein information (as an indicator for colostrum intake). Coupled together, this added information further accounts for environmental variation

Table 3: Variance components of single trait linear models for disease traits with a minimum herd year incidence of 1% and 5% in Canadian dairy calves

		1% Minimum Incidence		5% Minimum Incidence	
Model	Parameters	Diarrhea	Respiratory Problems	Diarrhea	Respiratory Problems
Linear Model	σ^2_e	0.103 (0.001)	0.106 (0.001)	0.137 (0.001)	0.139 (0.001)
	σ^2_{HYS}	0.036 (0.001)	0.036 (0.001)	0.043 (0.002)	0.041 (0.001)
	σ^2_a	0.006 (0.001)	0.004 (0.0004)	0.012 (0.001)	0.007 (0.001)
	h^2	0.043 (0.004)	0.027 (0.003)	0.060 (0.007)	0.037 (0.003)
Threshold Model	σ^2_e	1.000	1.000	1.000	1.000
	σ^2_{HYS}	1.257 (0.002)	1.048 (0.001)	0.869 (0.001)	0.758 (0.001)
	σ^2_a	0.162 (0.003)	0.103 (0.001)	0.198 (0.002)	0.115 (0.001)
	h^2_l	0.067 (0.001)	0.048 (0.001)	0.096 (0.001)	0.061(0.001)
	h^2	0.032	0.023	0.054	0.033

σ^2_e= Residual Variance; σ^2_{HYS}= Herd-Year-Season Variance; σ^2_a= Genetic Variance; h^2 = Heritability on observed scale; h^2_l= Heritability on the liability scale.

Table 4: Variance components of multiple trait model for disease traits with a minimum herd year incidence of 1% and 5% in Canadian dairy calves

		1% Minimum Incidence		5% Minimum Incidence	
Model	Parameters	Diarrhea	Respiratory Problems	Diarrhea	Respiratory Problems
Linear Model	σ^2_e	0.107 (0.001)	0.132 (0.001)	0.142 (0.002)	0.157 (0.002)
	σ^2_{HYS}	0.041 (0.002)	0.046 (0.002)	0.045 (0.003)	0.045 (0.003)
	σ^2_a	0.007 (0.001)	0.008 (0.001)	0.013 (0.002)	0.015 (0.002)
	h^2	0.044 (0.005)	0.043 (0.005)	0.066 (0.007)	0.071 (0.007)
	r_g	0.528 (0.071)		0.616 (0.067)	
Threshold Model	σ^2_e	1.000	1.000	1.000	1.000
	σ^2_{HYS}	1.284 (0.001)	0.971 (0.001)	0.821 (0.001)	0.680 (0.001)
	σ^2_a	0.165 (0.002)	0.135 (0.002)	0.210 (0.002)	0.198 (0.002)
	h^2_l	0.067 (0.001)	0.064 (0.001)	0.103 (0.001)	0.105 (0.001)
	h^2	0.034	0.035	0.059	0.062
	r_g	0.499 (0.014)		0.585 (0.013)	

σ^2_e= Residual Variance; σ^2_{HYS}= Herd-Year-Season Variance; σ^2_a= Genetic Variance; h^2 = Heritability on observed scale; h^2_l= Heritability on the liability scale; r_g = Genetic correlation.

associated with calf diseases, increasing the heritability estimate of the trait. It is important to note that only the liability scale genetic parameters were presented in several studies. To ensure fair comparison of estimates, it is important to know which model approach was implemented, as liability scale estimates will tend to be higher compared with those estimated on the observed scale. Heritability estimates on the observed scale depend on the prevalence of the trait, with maximum heritability observed when prevalence is 0.5, and heritability approaches 0 when prevalence are close to 0 or 1 (Dempster and Lerner, 1950; Bijma et al., 2022).

The effect of prevalence rate on observed scale heritability partly explains the differences seen between the 1%DATA and 5%DATA when we compared parameter estimates. For the 1%DATA, prevalence for RESP and DIAR was 19%, while for the 5%DATA, prevalence for RESP and DIAR was 26% and 28%, respectively. As prevalence approaches 0.5 the respective observed scale heritability will increase. Furthermore, genetic variance in binary traits is determined by heritability and thus prevalence (Bijma et al., 2022), meaning that with limited prevalence, there will be limited ability to differentiate between top and low preforming animals genetically. Given this, it is important that data used in genetic analysis of disease traits is consistently and accurately recorded to maximize the potential response to selection of disease traits (Bishop and Woolliams, 2010). In the case where prevalence of disease is actually very low, conventional quantitative genetic modeling may not provide the best solution for eradication of a disease, as heritability estimates will approach zero (Hulst et al., 2021). Through the incorporation of indirect genetic effects (i.e., positive feedback effects), such as infectivity rates, and the use of epidemiological modeling of transmission, it is possible to achieve herd immunity and eradicate disease (Hulst et al., 2021). Furthermore, greater genetic variation likely exists in infectivity increasing potential response to selection. This is because an individual's susceptibility is linked to their fitness, and therefore, natural selection across generations has worked to reduce genetic variation in susceptibility (Lipschutz-Powell and Woolliams, 2012). The impact of an individual's infectivity on herd mates is not a component of its own fitness, and therefore, may have greater genetic variation as a result (Boddicker et al., 2012). Therefore, future studies should look to investigate the potential of incorporating this information and apply methods (e.g., as outlined by Bijma et al., 2022), to understand the necessary data infrastructure required to maximize response to selection of calf disease on dairy farms.

Genetic correlation estimates between RESP and DIAR vary widely in the literature, with values ranging from 0.15 to 0.56 (Mahmoud et al., 2017; Gonzalez-Peña et al., 2019; Haagen et al., 2021; Zhang et al., 2022). Genetic correlation estimates within this study were on the high end of these estimates with both models in the 5%DATA being slightly higher (Linear = 0.62; Threshold = 0.59). Zhang et al. (2022) reported large differences in genetic correlations estimated by linear (0.15) and threshold models (0.50), which were not seen in the current study, possibly due to differences in disease incidence rates within Zhang et al. (2022). The positive genetic correlation between RESP and DIAR makes sense from a biological point of view. Dairy calves experiencing a disease have increased chances of developing a second illness later in life (McCorquodale

et al., 2013). DIAR specifically has been shown to cause dehydration, anorexia, and reduced immune function (Schinwald et al., 2022), which are all risk factors for the development of RESP (Gorden and Plummer, 2010). When looking at the age of disease occurrence, calves exhibiting both diseases had DIAR occur first in 81% of cases, meaning DIAR could be a potential precursor to RESP. Furthermore, when looking at the differences in heritability estimates between the single and multiple trait analysis, the RESP estimates in the multiple trait analysis were in most cases higher than those for DIAR, while for the single trait analysis the opposite was true. This means that the RESP estimates are benefiting from the inclusion of DIAR records in the model, enabling the model to better determine the genetic component behind RESP expression.

Comparison of Sires

To compare sire performance, the top and bottom 10% of sires based on their predicted EBV were compared on their percentage of diseased daughters (Table 5), and their mean daughter corrected phenotype (Figure 3). In both analyses, differences between sire performances were clearly identifiable. In Table 5, on average across analyses, calves born to the bottom 10% of sires were 1.8 times more likely to develop RESP, and 1.9 times more likely to develop DIAR compared with daughters born to the top 10% of sires. When comparing analyses within Table 5 for RESP, the differences between data sets (1% vs 5%) were similar but there was a considerable difference between the single and multiple trait analyses. In Figure 3, this is further confirmed, whereby differences in standard deviations between groups for RESP doubled when comparing the single and multiple trait analyses. This stems from the moderate positive genetic correlation between the traits, and the fact that the heritability of DIAR is substantially higher than RESP. Biologically, DIAR could act as a precursor for RESP due to its impact on the immune system. For DIAR in Table 5, differences across analyses are more consistent. However, small improvements between data sets (1% vs 5%) in terms of percentage difference between groups can be seen, with a 4% and 6% greater difference between top and bottom bulls in the 5%DATA compared with the 1%DATA for the single and multiple traits analyses, respectively. In Figure 3, differences are more apparent, with a 47% and 71% increase in standard deviation difference between groups when comparing data sets (1% vs 5%) in the single and multiple trait analyses, respectively. These differences could be attributed to the higher phenotypic variation within the 5%DATA allowing for clearer distinction between sires and further emphasizes the value in improved data collection.

Across both tables it is clear that the top sires were considerably closer to the mean than the bottom sires. This means that the majority of sires were performing similarly, with a minority performing considerably worse. This is beneficial from a genetic selection point of view as problematic sires can be identified. This means improvements in calf health can be achieved without having a major impact on genetic diversity across generations.

Overall, these differences suggest that even though both traits have a low heritability, and given the aforementioned issues surrounding data collection practices, it is still possible to effectively identify sires who sired healthier daughters. Therefore, the inclusion of both traits in Canadian national selection indices could help improve the overall health of calves born on Canadian farms.

Industry Recommendations

Results reported in this study are promising for the incorporation of calf disease traits within Canadian national genetic evaluations. It is recommended that

Table 5: Difference in the mean sire diseased daughter's percentage between top and bottom 10% sires based on their estimated breeding value from the linear model in both the 1% and 5% minimum incidence analysis, with a minimum of 20 daughters

				Percentage of diseased daughters			
Analysis		Trait	No. of Sires	Mean	Top 10%	Bottom 10%	Difference
Single Trait	1%	Diarrhea	737	18%	15%	29%	1.6x
		Respiratory Problems	1,607	18%	15%	26%	1.7x
	5%	Diarrhea	466	27%	20%	39%	2.0x
		Respiratory Problems	1,083	25%	22%	36%	1.6x
Multiple Trait	1%	Diarrhea	629	19%	16%	32%	2.0x
		Respiratory Problems	629	24%	21%	40%	1.9x
	5%	Diarrhea	363	30%	22%	44%	2.0x
		Respiratory Problems	363	32%	25%	50%	2.0x

a multiple trait analysis for DIAR and RESP be used given the moderate/strong positive genetic correlation between traits and the clear improvement in RESP estimates. For genetic evaluation to be feasible long-term, several issues need to be addressed. The recording of accurate and high-quality phenotypes is the backbone of any successful selection program (Coffey, 2020), while the development of a cost-effective data pipeline is a requirement for including novel traits in routine genetic evaluations (Miglior et al., 2016). In Canada, there are currently no definitive requirements or standardized criteria in place for recording calf disease (van Staaveren et al., 2023). Data for this study were recorded through DHI herd management software, but many farms may not be uploading information to DHI. The Dairy Farmers of Canada reported that between 2019 and 2020, 83% of 2,447 Canadian farms recorded disease events for cows and calves (Dairy Farmers of Canada, 2021). In 2020, within the current study, 4.5% of herds on DHI collected information on RESP, while 2.7% collected information on DIAR, meaning that the majority of DHI herds in Canada were not collecting or not uploading this information to herd management software. Furthermore, after data editing, the percentages dropped to 4.3% and 1.8% for RESP and DIAR, respectively. Bauman et al. (2016) reported that Canadian producers ranked RESP and DIAR as the fifth and third most important management consideration on farms, respectively. This indicates an interest in calf health, but uploading this information to herd management software has not been encouraged and this needs to change. Furthermore, of the recorded data, there is

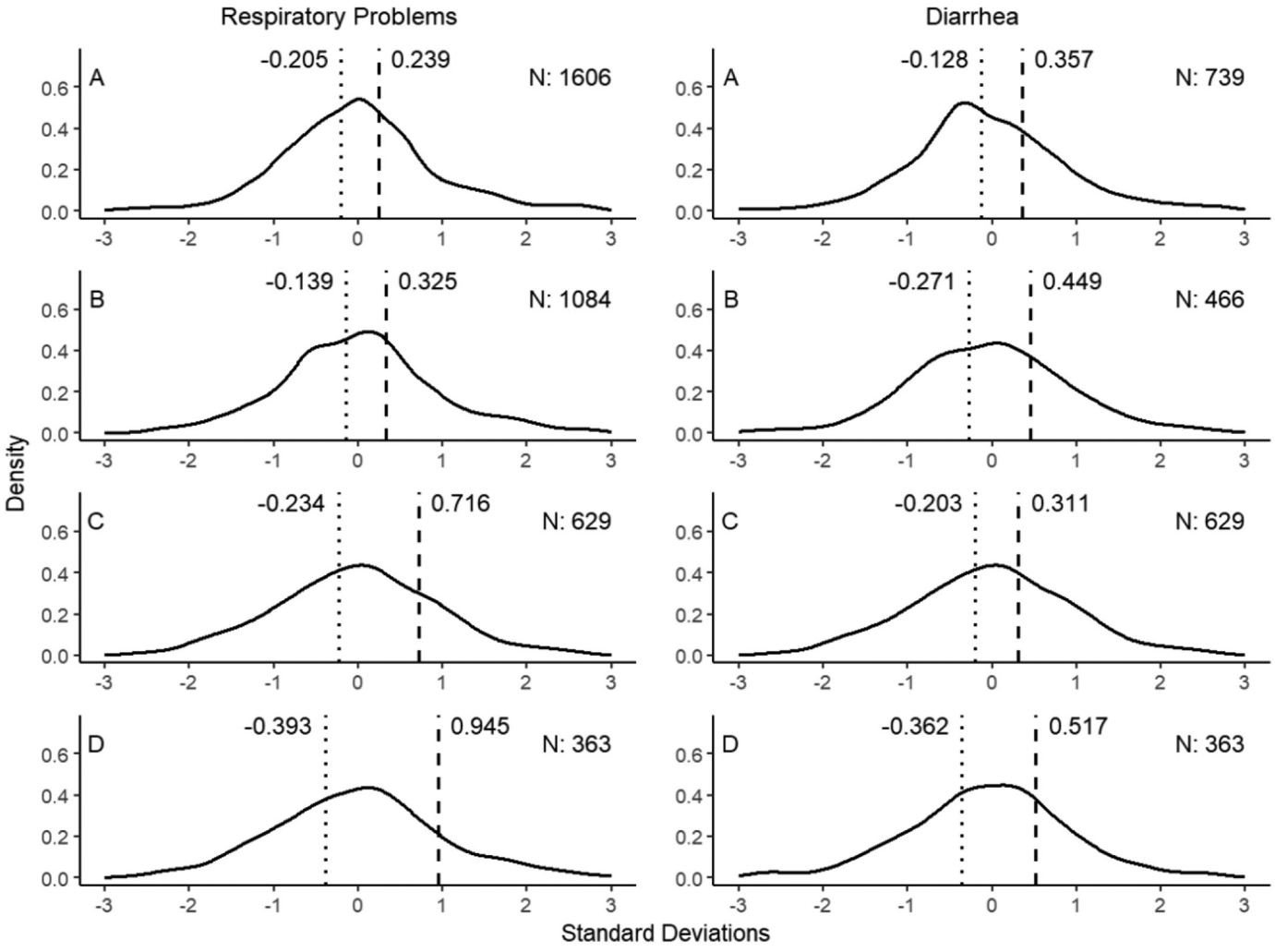

Figure 3. Difference in standard deviations of a sires mean daughter corrected phenotype between top 10% (dotted lines) and bottom 10% (dashed lines) sires based on their estimated breeding value from the linear model in both the 1% and 5% minimum incidence analysis, with a minimum of 20 daughters. n = Number of sires in analysis; A = 1% DATA single trait analysis; B = 5% DATA single trait analysis; C = 1% DATA multiple trait analysis; D = 5% DATA multiple trait analysis.

variability in disease diagnosis factors between producers (van Staaveren et al., 2023), meaning there were also differences in trait definitions across herds. Ensuring recording practices are consistent and accurate will make genetic selection for improving calf health feasible.

To maximize the selection potential, specific calf and disease information will need to be incorporated into evaluations. Calf information, such as birth weights and colostrum intake information have been incorporated in other studies (Henderson et al., 2011), and several studies have shown the connection between colostrum intake and disease expression in dairy calves (Godden et al., 2019; Hammon et al., 2020). Disease information such as duration of disease could be used to determine the severity of the disease and potentially enable for multiple classes, further increasing selection potential through more specific phenotypes. Overall, to maximize the response to selection of calf disease traits in Canada, a collaborative effort between producers, industry, academia and veterinarians is required to provide clear recommendations around recording practices and development of a data pipeline.

CONCLUSIONS

Genetic selection for improving calf health on Canadian dairy farms is possible and the results from the current study were promising for the implementation of both DIAR and RESP into Canadian national genetic evaluations. However, for this to be feasible, standardized approaches on collection practices, and industry outreach to highlight the importance of collecting and uploading this information to herd management software is required to make continuous genetic evaluation possible. Despite the issues surrounding data collection, the genetic parameters within this study were similar to those reported in the literature and clear differences between daughters' of sires were identified, which highlights that the potential for selecting on calf disease traits is potentially even greater than reported here. Overall, the importance of health and welfare in dairy animals is becoming more important, and this study provides the foundations for incorporating calf disease traits into national genetic evaluations within Canada.

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REFERENCES

Bauman, C. A., H. W. Barkema, J. Dubuc, G. P. Keefe, and D. F. Kelton. 2016. Identifying management and disease priorities of Canadian dairy industry stakeholders. J. Dairy Sci. 99:10194–10203. https://doi.org/10.3168/jds.2016-11057.

Beavers, L., and B. Van Doormaal. 2013. Official Genetic Evaluation for Mastitis Resistance. Accessed. https://www.cdn.ca/document.php?id=333.

Beavers, L., and B. Van Doormaal. 2018. Genetic Selection for Improved Hoof Health Is Now Possible! Accessed. https://www.cdn.ca/document.php?id=513.

Bijma, P., A. D. Hulst, and M. C. M. de Jong. 2022. The quantitative genetics of the prevalence of infectious diseases: hidden genetic variation due to indirect genetic effects dominates heritable variation and response to selection. Genetics 220:iyab141. https://doi.org/10.1093/genetics/iyab141.

Bishop, S. C., and J. A. Woolliams. 2010. On the Genetic Interpretation of Disease Data. PLoS One 5:1–6. https://doi.org/10.1371/journal.pone.0008940.

Blanchard, P. C. 2012. Diagnostics of Dairy and Beef Cattle Diarrhea. Vet. Clin. North Am. Food Anim. Pract. 28:443–464. https://doi.org/10.1016/j.cvfa.2012.07.002.

Boddicker, N., E. H. Waide, R. R. R. Rowland, J. K. Lunney, D. J. Garrick, J. M. Reecy, and J. C. M. Dekkers. 2012. Evidence for a major QTL associated with host response to Porcine Reproductive and Respiratory Syndrome Virus challenge1. J. Anim. Sci. 90:1733–1746. https://doi.org/10.2527/jas.2011-4464.

Buczinski, S., D. Achard, E. Timsit, S. Cliniques, F. D. M. Vétérinaire, U. De Montréal, and C. Js. 2021. Effects of calfhood respiratory disease on health and performance of dairy cattle : A systematic review and meta-analysis. J. Dairy Sci. 104:8214–8227. https://doi.org/10.3168/jds.2020-19941.

Butler, S. T., and S. G. Moore. 2018. Genetic control of reproduction in dairy cows under grazing conditions. Anim. Reprod. 15(Suppl. 1):933–939. https://doi.org/10.21451/1984-3143-AR2018-0054.

Coffey, M. 2020. Dairy cows: in the age of the genotype, #phenotypeisking. Anim. Front. 10:19–22. https://doi.org/10.1093/af/vfaa004.

Cole, J. B., J. W. Dürr, and E. L. Nicolazzi. 2021. Invited review: The future of selection decisions and breeding programs: What are we breeding for, and who decides? J. Dairy Sci. 104:5111–5124. https://doi.org/10.3168/jds.2020-19777.

Costa, J. H. C., M. A. G. Von Keyserlingk, and D. M. Weary. 2016. Invited review : Effects of group housing of dairy calves on behavior, cognition, performance, and health. J. Dairy Sci. 99:2453–2467. https://doi.org/10.3168/jds.2015-10144.

Dairy Calf & Heifer Association. 2019. DCHA Gold Standards. Accessed. https://calfandheifer.org/wp-content/uploads/2020/09/DCHA_GoldStandards_2020_En_WEB-final.pdf.

Dairy Farmers of Canada. 2021. Pro Action 2021 Progress Report. Accessed. https://www.dairyfarmers.ca/Media/Files/proaction/proaction_progress_report_2021.pdf.

Dempster, E., and I. Lerner. 1950. Heritability of Threshold Characters. Genetics 35:212–236. https://doi.org/10.1093/genetics/35.2.212.

Donovan, G. A., I. R. Dohoo, D. M. Montgomery, and F. L. Bennett. 1998. Calf and disease factors affecting growth in female holstein calves in Florida, USA. Prev. Vet. Med. 33:1–10. https://doi.org/10.1016/S0167-5877(97)00059-7.

Fleming, A., E. A. Abdalla, C. Maltecca, and C. F. Baes. 2018. Invited review: Reproductive and genomic technologies to optimize breeding strategies for genetic progress in dairy cattle. Arch. Tierzucht 61:43–57. https://doi.org/10.5194/aab-61-43-2018.

Fleming, A., and B. Van Doormaal. 2020. New Genetic Evaluations Coming in December 2020. Accessed. https://www.cdn.ca/document.php?id=544.

Gibson, J.P., and S.C. Bishop. 2005. Use of molecular markers to enhance resistance of livestock to disease : a global approach Utilising and improving genetic resistance to disease 24:343–353.

Godden, S. M., J. E. Lombard, and A. R. Woolums. 2019. Colostrum Management for Dairy Calves. Vet. Clin. North Am. Food Anim. Pract. 35:535–556. https://doi.org/10.1016/j.cvfa.2019.07.005.

Gonzalez-Peña, D., N. Vukasinovic, J. J. J. Brooker, C. A. A. Przybyla, and S. K. K. DeNise. 2019. Genomic evaluation for calf wellness traits in Holstein cattle. J. Dairy Sci. 102:2319–2329. https://doi.org/10.3168/jds.2018-15540.

Gorden, P. J., and P. Plummer. 2010. Control, Management, and Prevention of Bovine Respiratory Disease in Dairy Calves and Cows. Vet. Clin. North Am. Food Anim. Pract. 26:243–259. https://doi.org/10.1016/j.cvfa.2010.03.004.

Haagen, I. W., L. C. Hardie, B. J. Heins, and C. D. Dechow. 2021. Genetic parameters of calf morbidity and stayability for US organic Holstein calves. J. Dairy Sci. 104:11770–11778. https://doi.org/10.3168/jds.2021-20432.

Hammon, H. M., W. Liermann, D. Frieten, and C. Koch. 2020. Review: Importance of colostrum supply and milk feeding intensity on gastrointestinal and systemic development in calves. Animal 14:s133–s143. https://doi.org/10.1017/S1751731119003148.

Heinrichs, A. J., B. S. Heinrichs, O. Harel, G. W. Rogers, and N. T. Place. 2005. A prospective study of calf factors affecting age, body size, and body condition score at first calving of holstein dairy heifers. J. Dairy Sci. 88:2828–2835. https://doi.org/10.3168/jds.S0022-0302(05)72963-5.

Henderson, L., F. Miglior, A. Sewalem, J. Wormuth, D. Kelton, A. Robinson, and K. E. Leslie. 2011. Short communication: Genetic parameters for measures of calf health in a population of Holstein calves in New York State. J. Dairy Sci. 94:6181–6187. https://doi.org/10.3168/jds.2011-4347.

Hulst, A. D., M. C. M. de Jong, and P. Bijma. 2021. Why genetic selection to reduce the prevalence of infectious diseases is way more promising than currently believed. Genetics 217:iyab024. https://doi.org/10.1093/genetics/iyab024.

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. https://doi.org/10.3168/jds.S0022-0302(05)72895-2.

Jamrozik, J., G. Kistemaker, B. Van Doormaal, C. Baes, and F. Miglior. 2021. Genomic Evaluation for Resistance to Fertility Disorders in Canadian Dairy Breeds. Interbull Bull. 102–110.

Jamrozik, J., G. J. Kistemaker, B. J. Van Doormaal, A. Fleming, A. Koeck, and F. Miglior. 2016. Genetic Evaluation for Resistance to Metabolic Diseases in Canadian Dairy Breeds. Interbull Bull. 9–16.

Koeck, A., F. Miglior, D.F. Kelton, and F.S. Schenkel. 2012. Health recording in Canadian Holsteins : Data and genetic parameters 4099–4108. doi:https://doi.org/10.3168/jds.2011-5127.

Leblanc, S. J., K. D. Lissemore, D. F. Kelton, T. F. Duffield, and K. E. Leslie. 2006. Major Advances in Disease Prevention in Dairy Cattle. J. Dairy Sci. 89:1267–1279. https://doi.org/10.3168/jds.S0022-0302(06)72195-6.

Lipschutz-powell, D., and J.A. Woolliams. 2012. Bias, accuracy, and impact of indirect genetic effects in infectious diseases 3:1–10. doi: https://doi.org/10.3389/fgene.2012.00215.

Macdonald, K.A., J.W. Penno, A.M. Bryant, and J.R. Roche. 2005. Effect of Feeding Level Pre- and Post-Puberty and Body Weight at First Calving on Growth, Milk Production, and Fertility in Grazing Dairy Cows 3363–3375. doi:https://doi.org/10.3168/jds.S0022-0302(05)73020-4.

Mahmoud, M., T. Yin, K. Brügemann, and S. König. 2017. Phenotypic, genetic, and single nucleotide polymorphism marker associations between calf diseases and subsequent performance and disease occurrences of first-lactation German Holstein cows. J. Dairy Sci. 100:2017–2031. https://doi.org/10.3168/jds.2016-11767.

Malchiodi, F., A. Koeck, S. Mason, A. M. Christen, D. F. Kelton, F. S. Schenkel, and F. Miglior. 2017. Genetic parameters for hoof health traits estimated with linear and threshold models using alternative cohorts. J. Dairy Sci. 100:2828–2836. https://doi.org/10.3168/jds.2016-11558.

McCorquodale, C. E., A. Sewalem, F. Miglior, D. Kelton, A. Robinson, A. Koeck, and K. E. E. Leslie. 2013. Short communication : Analysis of health and survival in a population of Ontario Holstein heifer calves. J. Dairy Sci. 96:1880–1885. https://doi.org/10.3168/jds.2012-5735.

Miglior, F., R. Finocchiaro, F. Malchiodi, A. Fleming, L. Brito, C. F. Baes, J. Jamrozik, P. Martin, J. Chesnais, and F. S. Schenkel. 2016. Enhancing the data pipeline for novel traits in the genomic era: from farms to DHI to evaluation centres. ICAR Tech. Ser. ●●●:23–30.

Miglior, F., A. Fleming, F. Malchiodi, L. F. Brito, P. Martin, and C. F. Baes. 2017. A 100-Year Review: Identification and genetic selection of economically important traits in dairy cattle. J. Dairy Sci. 100:10251–10271. https://doi.org/10.3168/jds.2017-12968.

Misztal, I., S. Tsuruta, D. Lourenco, Y. Masuda, I. Aguilar, A. Legarra, and Z. Vitezica. 2014. Manual for BLUPF90 Family Programs. Accessed. http://nce.ads.uga.edu/wiki/doku.php?id=documentation.

Murray, B. 2011. Optimizing Calf Survival at Birth. Accessed November 22, 2022. http://www.omafra.gov.on.ca/english/livestock/dairy/facts/optbirth.htm.

Negussie, E., I. Strandén, and E. A. Mäntysaari. 2008. Genetic analysis of liability to clinical mastitis, with somatic cell score and production traits using bivariate threshold–linear and linear–linear models. Livest. Sci. 117:52–59. https://doi.org/10.1016/j.livsci.2007.11.009.

Nor, N. M., W. Steeneveld, T. Van Werven, M. C. M. M. Mourits, H. Hogeveen, N. Mohd Nor, W. Steeneveld, T. van Werven, M. C. M. M. Mourits, and H. Hogeveen. 2013. First-calving age and first-lactation milk production on Dutch dairy farms. J. Dairy Sci. 96:981–992. https://doi.org/10.3168/jds.2012-5741.

Phanse, Y., C. Wu, A.J. Venturino, C. Hansen, K. Nelson, S.R. Broderick, H. Steinberg, and A.M. Talaat. 2020. A Protective Vaccine against Johne ' s Disease in Cattle 1–18.

R Core Team. 2021. R: The R Project for Statistical Computing. Accessed. https://www.r-project.org/.

RSPCA. 2021. RSPCA welfare standardsfor Dairy cattle.

Schinwald, M., K. Creutzinger, C. B. Winder, D. Haley, and D. L. Renaud. 2022. Predictors of diarrhea, mortality, and weight gain in male dairy calves. J. Dairy Sci. 105:5296–5309. https://doi.org/10.3168/jds.2021-21667.

Sischo, W. M., D. W. Hird, I. A. Gardner, W. W. Utterback, K. H. Christiansen, T. E. Carpenter, C. Danaye-Elmi, and B. R. Heron. 1990. Economics of disease occurrence and prevention on California dairy farms: A report and evaluation of data collected for the national animal health monitoring system, 1986–87. Prev. Vet. Med. 8:141–156. https://doi.org/10.1016/0167-5877(90)90007-5.

Smith, B. 2016. Bayesian Output Analysis Program (BOA) for MCMC. Accessed. http://www.jstatsoft.org/v21/i11.

Stanton, A. 2011. An evaluation of the impact of management practice on the health and welfare of dairy Heifer calves. University of Guelph, Guelph.

Svensson, C., and J. Hultgren. 2008. Associations between housing, management, and morbidity during rearing and subsequent first-lactation milk production of dairy cows in southwest Sweden. J. Dairy Sci. 91:1510–1518. https://doi.org/10.3168/jds.2007-0235.

Urie, N. J., J. E. Lombard, C. B. Shivley, C. A. Kopral, A. E. Adams, T. J. Earleywine, J. D. Olson, and F. B. Garry. 2018. Preweaned heifer management on US dairy operations: Part V. Factors associated with morbidity and mortality in preweaned dairy heifer calves. J. Dairy Sci. 101:9229–9244. https://doi.org/10.3168/jds.2017-14019.

USDA. 2018. Dairy 2014: Health and Management Practices on U.S. Dairy Operations, 2014. Accessed November 22, 2022. https://www.aphis.usda.gov/animal_health/nahms/dairy/downloads/dairy14/Dairy14_dr_PartIII.pdf.

Van Doormaal, B. 2007. Calf Survival : A New Trait of Interest. Accessed. https://www.cdn.ca/document.php?id=128.

van Staaveren, N., E. Hyland, K. Houlahan, C. Lynch, F. Miglior, D.F. Kelton, F.S. Schenkel, and C.F. Baes. 2023. Recording of calf diseases for potential use in breeding programs: a case study on calf respiratory illness and diarrhea. https://doi.org/10.1139/cjas-2022-0112. doi:https://doi.org/10.1139/CJAS-2022-0112.

Virtala, A. K., G. D. Mechor, Y. T. Grohn, and H. N. Erb. 1996. The Effect of Calfhood Diseases on Growth of Female Dairy Calves During the First 3 Months of Life in New York State. J. Dairy Sci. 79:1040–1049. https://doi.org/10.3168/jds.S0022-0302(96)76457-3.

Waltner-Toews, D., S. Martin, and A. Meek. 1986. The effect of early calfhood health status on survivorship and age at first calving. Can. J. Vet. Res. 50:314–317.

Wathes, D. C., J. S. Brickell, N. E. Bourne, A. Swali, and Z. Cheng. 2008. Factors influencing heifer survival and fertility on commercial dairy farms *. Animal 2:1135–1143. https://doi.org/10.1017/S1751731108002322.

Wilson-welder, J. H., P. Boggiatto, J. E. Nally, E. I. Wafa, D. P. Alt, R. L. Hornsby, A. Frank, D. E. Jones, S. C. Olsen, N. B. Bowden, and A. K. Salem. 2020. Bovine immune response to leptospira antigen in different novel adjuvants and vaccine delivery platforms. Vaccine 38:3464–3473. https://doi.org/10.1016/j.vaccine.2020.02.086.

Windeyer, M. C., K. E. Leslie, S. M. Godden, D. C. Hodgins, K. D. Lissemore, and S. J. LeBlanc. 2014. Factors associated with morbidity, mortality, and growth of dairy heifer calves up to 3 months of age. Prev. Vet. Med. 113:231–240. https://doi.org/10.1016/j.prevetmed.2013.10.019.

Zhang, H., K. Wang, T. An, L. Zhu, Y. Chang, W. Lou, A. Liu, G. Su, L. F. Brito, and Y. Wang. 2022. Genetic parameters for dairy calf and replacement heifer wellness traits and their association with cow longevity and health indicators in Holstein cattle. J. Dairy Sci. 105:6749–6759. https://doi.org/10.3168/jds.2021-21450.

Zhang, W., L. Chang, and D. Brito. 2019. Mortality-Culling Rates of Dairy Calves and Replacement Heifers and Its Risk Factors in Holstein Cattle. Animals (Basel) 9:730. https://doi.org/10.3390/ani9100730.

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