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Heritability of bovine respiratory disease in weaned beef calves

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Introduction

Bovine respiratory disease (BRD) is one of the most common cattle diseases and the leading cause of morbidity and mortality in feedyards (White and Larson, 2020). The U.S. feedlot industry loses approximately $1 billion annually due to post-weaning BRD (Griffin *et al.*, 2010). Most economic losses are the result of mortality and depressed feedlot performance, but BRD also increases feed, labor, and medication costs (Wilson *et al.*, 2017). An aggregation of stressors, viral infections, and bacterial infections contribute to the disease complex (Krehbiel, 2020). Good management practices are key to minimizing the impacts of stress, but most of these factors are unavoidable in the beef industry. Genetic selection for improved BRD resilience offers valuable opportunities to promote better health, productivity, and efficiency in feedlots.

Several challenges exist in creating a national genetic evaluation for BRD. The segmentation of the beef industry leads to the loss of data when ownership of cattle is transferred, making it difficult to retain pedigree and early-life information on feedlot animals. Currently, there are no BRD phenotyping guidelines or standardized trait recording. Common measurements include BRD incidence, susceptibility, and resilience, all of which differ in definition to some degree. The issue is further exacerbated by the fact that cattle are adept at hiding illness, hindering visual detection of BRD and potentially introducing errors in phenotyping. Previous work has shown variable but low heritability estimates for BRD incidence (Buchanan *et al.*, 2016; Schneider *et al.*, 2010; Snowder *et al.*, 2007; Speidel *et al.*, 2014), but the data collection and modelling procedures differ between these studies. An evaluation of different analyses is necessary to determine the trait definition and statistical model that provide the most potential for genetic improvement. The objective of this study was to compare heritability estimates between linear and threshold models for BRD incidence.

Materials & Methods

Data. Historical health records on weaned beef calves born from 2016 to 2024 but excluding 2020 were provided by a seedstock operation in Southwest Iowa. Data format varied by year but generally included treatment dates and, when applicable, death date and reason for death. Initial data processing was performed using R software version 4.1.1 (R Core Team, 2021). Calves with unknown birth dates or weaning contemporary groups were removed from the dataset. Animals with no treatment record were assumed to have never received treatment for BRD. After cleaning, data on 5,676 bulls, steers, and heifers were available for analysis. Three pure breeds and three composites were represented in the dataset. Pure breeds were Angus, Simmental, and South Devon, and crosses included Angus x Simmental, Angus x South Devon, and Angus x Simmental x South Devon. Contemporary groups were constructed using birth year, sex, and weaning group. The final dataset included 172 contemporary groups with an average size of 33 ± 28.7. A 3-generation pedigree was used containing 14,613 animals, 300 unique sires, and 2,371 unique dams. Parent generations ranged from 1 to 8 for sires and 1 to 9 for dams.

Statistical analysis. Disease incidence was evaluated as a binary phenotype defined as never receiving treatment (0) or receiving one or more treatments for BRD (1). Genetic analyses were performed using ASReml version 4.2 software package (Gilmour *et al.*, 2021). Variance components for BRD incidence were obtained using univariate animal models, where animal was included as a random effect, and fixed effects included contemporary group, breed, and date of birth as a covariate. Two models were used to estimate the heritability of BRD incidence. The first model analyzed BRD incidence linearly and treated the observed response as continuous. Due to the binary nature of BRD, the second model converted the underlying normal distribution using a probit link function in order to evaluate the binary response on the underlying scale (Gianola and Foulley, 1983). The univariate model for both analyses was

$$\boldsymbol{y} = \boldsymbol{X\beta} + \boldsymbol{Zu} + \boldsymbol{e} \quad (1)$$

where $\boldsymbol{y}$ was the response vector of observations, $\boldsymbol{\beta}$ was the solution vector for fixed effects, $\boldsymbol{u}$ was the vector of additive genetic effects, $\boldsymbol{X}$ and $\boldsymbol{Z}$ represented the incidence matrices for fixed and random effects, respectively, and $\boldsymbol{e}$ was the residual error. Means and variances followed the assumptions

$$E(\boldsymbol{y}) = \boldsymbol{X\beta}, \quad (2)$$

$$E(\boldsymbol{e}) = E(\boldsymbol{u}) = \boldsymbol{0} \quad (3)$$

$$var\begin{bmatrix}\boldsymbol{u}\\ \boldsymbol{e}\end{bmatrix} = \begin{bmatrix}\boldsymbol{A}\sigma_a^2 & \boldsymbol{0}\\ \boldsymbol{0} & \boldsymbol{I}\sigma_e^2\end{bmatrix} \quad (4)$$

where $\boldsymbol{A}$ was Wright's numerator relationship matrix, $\boldsymbol{I}$ represented an identity matrix with dimensions equal to the number of animals with records, σ_a^2 was the additive genetic variance, and σ_e^2 was the residual variance. The residual variance was fixed to one in the probit model (Gianola and Foulley, 1983). Pearson and Spearman rank correlations between estimated breeding values (EBV) were performed to quantify the relationship between predictions from each model.

Results

Summary statistics are listed in Table 1. Variance component and heritability estimates are presented in Table 2. The linear model produced a heritability estimate of 0.049 ± 0.020. A slightly higher heritability estimate of 0.074 ± 0.026 was achieved with the threshold model. As illustrated in Figure 1, EBVs from the probit model showed greater variation than those from the linear model. Variances (± SE) of EBVs were 0.0007 ± 0.0002 and 0.0054 ± 0.0006 for the linear and probit models, respectively. The Pearson correlation coefficient between EBVs from the linear and threshold models was 0.99, indicating a strong linear relationship between model predictions. The Spearman rank correlation coefficient was also 0.99, reflecting close agreement in animal rankings between the models.

Table 1. Number of animals by BRD incidence status, where 0 represents no treatment and 1 indicates at least one treatment for BRD.

BRD Incidence	N
0	4030
1	1646

Table 2. Univariate linear and probit model variance component and heritability estimates (± SE) for BRD incidence.

Model	Additive variance	Residual variance	Heritability
Linear	0.008 ± 0.003	0.165 ± 0.004	0.049 ± 0.020
Probit	0.079 ± 0.030	1.00	0.074 ± 0.026

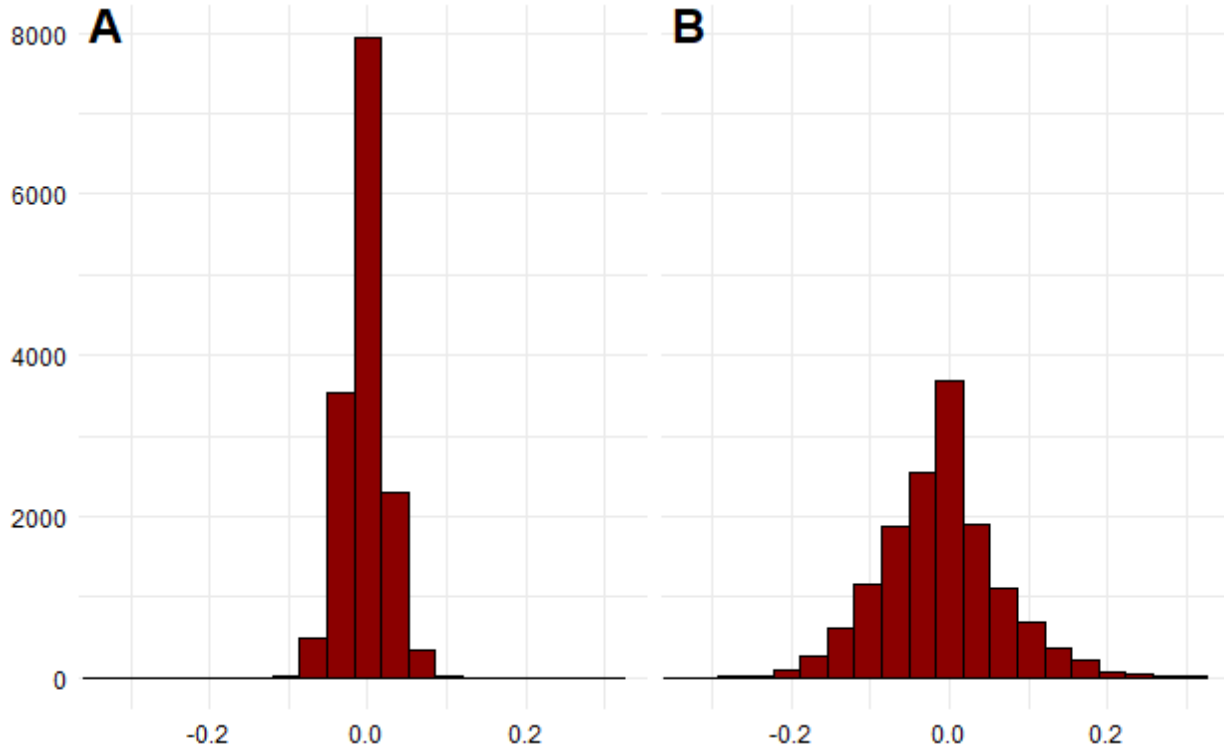

Figure 1. Distribution of estimated breeding values for BRD incidence from (A) linear and (B) probit models.

Discussion

A slightly higher heritability estimate was obtained using the probit model. The heritability estimate of 0.074 ± 0.026 agrees with similar studies reported in the literature (0.07 – 0.08; Schneider *et al.*, 2010; Snowder *et al.*, 2007). Given the binary nature of BRD incidence, a threshold model would be better suited to the data than a linear model due to the variance structure and latent liability scale associated with the binary response (Ødegård *et al.*, 2010). Additionally, the greater variance of EBVs resulting from the probit model reinforces the conclusion that the probit model performs better than the linear model in analyzing BRD incidence. Both models predict the same trait, but the scale for EBVs for the probit model demonstrates greater variance. The very strong Pearson correlation between EBVs from the models indicates an almost perfect linear relationship between the predictions. Consistent with the Pearson correlation, the Spearman rank correlation was also high, indicating that animal re-ranking was minimal. Genetic progress should be analogous between the models. This is in agreement with literature showing that while a threshold model is statistically more appropriate for binary data, a linear model can be a practical

alternative that reduces computational requirements and generates a very similar genetic trend, providing disease prevalence is not extreme and sufficient data is available (Hidalgo *et al.*, 2024).

Conclusions

This study shows that genetic selection for improved weaned calf health is plausible. Heritability of BRD incidence was estimated to be low with both linear and probit models. Heritability was slightly higher when estimated with the probit model, but the models otherwise performed similarly and gave highly correlated EBVs. Due to the binary status of BRD incidence, the threshold model would better fit the data. However, the probit model is significantly more computationally demanding with large datasets or when genomics are included, making the linear model a sufficient alternative. While this study focused on BRD incidence, other BRD traits have yet to be fully explored. Disease severity, characterized by number of treatments, may capture additional between-animal differences that are not observable with the simpler incidence trait. Further research is warranted to investigate alternative trait definitions and statistical procedures that may provide greater benefit to breeding programs and genetic improvement.

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