**Title**

The association between a genetic index for lameness resistance and the incidence of claw horn lesions in Holstein cows

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**Abstract**

**Objectives:** To determine the association between the Lameness Advantage genetic index and four outcomes: sole haemorrhage (SH), sole ulcers (SU), white line lesions (WL), and lameness during mobility scoring.

**Methods:** We enrolled 2,352 Holstein cows from four predominantly housed dairy herds in the UK. Cows were mobility scored and foot lesions recorded at four time points from before calving to late lactation. Cows were genotyped and genetic indexes were assigned to each cow following national genetic evaluations. Lameness records and genetic indexes were matched for 2,107 cows. Four separate multivariable logistic regression models, which included farm and parity as covariables, were used to quantify the association between the Lameness Advantage index and whether animals were affected by SH, SU, WL, or lameness.

**Results:** The odds ratios (95% confidence intervals) for one point increase in the Lameness Advantage index were 0.79 (0.72 - 0.86), 0.68 (0.59 – 0.78), 0.94 (0.84 – 1.04), and 0.82 (0.74 – 91) for SH, SU, WL, and lameness, respectively. The same trends were present when the sire’s Lameness Advantage index was evaluated in place of the animal’s own, although the strength of this association was generally weaker.

**Discussion:** The Lameness Advantage index is associated with SH, SU, and lameness, therefore selection on the Lameness Advantage index could be considered in herds aiming to reduce lameness. Where genomic testing of heifers is not conducted, sire Lameness Advantage index may still be effective to reduce SH and SU incidence.

**Introduction**

Farmers and veterinary surgeons regard lameness as one of the most important health and welfare concerns in dairy cattle (1,2), and lameness has been identified as the most pressing problem affecting the modern dairy industry in Europe (3). Foot lesions are the major cause of lameness in dairy cows (4–6) and directly impact the longevity, productivity, and fertility of affected animals (7–9).

Sole haemorrhage (SH), sole ulcers (SU), and white line lesions (WL) are often grouped under the collective term “claw horn lesions” (CHL) (5,10). Claw horn lesions have a high prevalence in dairy cattle (5,11,12) and, relative to other foot lesions, CHL have been associated with the most severe pain responses (13,14), economic impacts (15–17), and environmental consequences (18).

The phenotypic variation of CHL in a population represents the underlying risk of animals developing these lesions, this variation can be partitioned into genetic and environmental components (19). The proportion of phenotypic variation explained by genetic differences is referred to as the heritability. The heritability of CHL, based on underlying risk, has been estimated in a large number of studies which have recently been summarised by Heringstad et al. (2018) (20) as 0.07 – 0.09 for SH, 0.07 – 0.18 for SU, and 0.06 – 0.10 for WL; the heritability of lameness diagnosed from locomotion scoring has been estimated to be 0.15 (21). Therefore, although these heritability estimates are low, genetic selection could produce cumulative, long-term benefits to complement husbandry-based initiatives to reduce lameness.

Dairy farmers are generally motivated to reduce lameness (22–24) and lameness traits ranked highly when farmers were surveyed about their genetic selection preferences (25). Genetic traits relating to lameness can be considered as either direct traits, such as foot lesions, or indirect traits which include breed society classification traits such as leg conformation and gait assessment (20).

Historically, farmers wishing to reduce lameness in their herd through improved genetics could only select on indirect traits (26), but it is now recognised more broadly that selection on direct health traits can more successfully accelerate genetic gains (27). Consequently two approaches have evolved in recent years to develop effective genetic selection indexes which can reduce the incidence of lameness in dairy herds. Some countries have incorporated foot lesion records directly into selection indexes (27), but other countries, where the infrastructure to record foot-trimming lesions on a large-scale has not been established, have instead utilised farm records of lameness (28–30). In the UK, Pritchard et al. (2013) (28) demonstrated that farm records could be used as phenotypes for both clinical mastitis and lameness, however, there were concerns regarding the quality of farm lameness records. In that study (28), only a third of cow records used for the mastitis analysis were included in the lameness evaluations due to lack of lameness recording in individual herds. Furthermore, across the herds which were recording lameness, there was an apparent incidence of 15.8% over the first three lactations, which is lower than the average national prevalence of 34.9% (31), and dramatically lower than more directly comparable annual incidence rates (32,33). A further concern is that farm records may be skewed towards lesions which are consistently or severely associated with lameness (34), for example SU are associated with more severe lameness than SH (35), and therefore farm records may not reflect SH and SU with equal accuracy.

In 2018, a genetic selection index for lameness, termed “Lameness Advantage”, was published by the UK Agricultural and Horticultural Development Board (AHDB). The Lameness Advantage index is calculated using lameness events from farm records (collected via milk recording organisations) in combination with traits from type classification: Bone Quality, Locomotion, Feet and Legs (an overall assessment by the classifier incorporating Foot Angle, Rear Legs Side View, Locomotion, and Bone Quality), and Digital Dermatitis (36). Higher values of this index are associated with better genetic merit for lameness and an expected reduction in the incidence of lameness compared to lower values, however, this index has not yet been evaluated in independent data. A recent study in Ireland, reported that cows with a positive genetic index for lameness, in this case reflecting an increased genetic susceptibility, had a 37.5% increase in the odds of lameness compared to animals with a negative genetic index (37).

It was hypothesised that the association between Lameness Advantage genetic index and actual frequency of claw horn lesions would be weak due to the quality of farm lameness records; therefore the primary objective of our study was to quantify this relationship in a cohort of dairy cattle with accurate foot lesion records. A further objective was to screen for associations between other selection indexes and claw horn lesions or lameness, in order to evaluate whether selection on type traits could still be utilised to reduce lameness.

**Materials and Method**

**Ethical approval**

The study was conducted following ethical approval by the University of Liverpool Research Ethics Committee (VREC269a, VREC466ab).

**Study design and population**

A prospective, cohort study on four dairy herds in the UK was designed to record foot lesions at four time points during a lactation cycle. Herds were selected based on convenience and practicalities of frequent visits and data collection. Herds A to C housed lactating cows all-year-round, milked cows three times daily and recorded 305-day milk yields of approximately 11,000 - 11,500 L. Herd D housed early lactation and high-yielding cows all-year-round and lower yielding cows were grazed during the summer; cows were milked twice daily and the 305-day milk yield was approximately 9,000 L. Parous cows on all herds were routinely foot-trimmed twice a year prior to drying off and 60 - 120 days after calving. On all herds, lactating cows were regularly footbathed after milking. Herd A footbathed cows three times a week with either copper sulphate or formalin; herd B footbathed cows twice daily with formalin, herd C footbathed cows daily with either copper sulphate or formalin and herd D footbathed three times a week with formalin.

**Data collection**

A total of 2,352 Holstein cows which were expected to calve between April and September 2019 were prospectively enrolled prior to calving with no additional inclusion or exclusion criteria applied. Data were collected by qualified veterinary surgeons during weekly or twice weekly visits to each herd from February 2019 to July 2020. Animals were assessed at four time points relative to their calving date: prior to calving (mean: -55 days, standard deviation (SD): 18), immediately after calving (mean: +5 days, SD: 3), in early lactation (mean: +84 days, SD: 14), and finally in late lactation (mean: +200 days, SD: 31). Enrolments continued until the final assessments in late lactation began, at which point additional enrolments stopped as data collection at four time points simultaneously was not feasible.

All cows were mobility scored according to the AHDB system from 0 (sound) to 3 (severely lame) (38,39). Cows were restrained in a foot-trimming crush and, depending on the assessment time point and the foot-trimming schedule in each herd, either functionally foot-trimmed or lightly trimmed to allow visualisation of foot lesions. In either case, CHL on each claw were recorded based on the ICAR claw health atlas (40). Over 90% of foot lesion identification and recording was performed by a single researcher.

All cows were genotyped and genetic indexes for cows and their sires were provided by the AHDB in the form of genomic predicted transmitted abilities, following calculation in the August 2021 national evaluation. In addition to the Lameness Advantage index, the other genetic indexes available for analysis were: Profitable Lifetime Index (PLI), Lifespan, Type Merit, Digital Dermatitis, Feet and Legs, Locomotion, Condition Score, Milk (kg), Fat (kg), Protein (kg), Fat (%), Protein (%), Somatic Cell Count (SCC), Mammary, Mastitis, Fertility Index, TB Advantage, Calf Survival, Maintenance, Stature, Chest Width, Body Depth, Angularity, Rump Angle, Rump Width, Rear Leg Side View, Foot Angle, Fore Udder Attachment, Rear Udder Height, Udder Support, Udder Depth, Front Teat Placement, Rear Teat Placement, Teat Position Side, Temperament, and Milking Speed.

**Statistical analysis**

Four independent outcomes were defined to reflect the susceptibility or resistance of an animal to SH, SU, WL, or lameness. Animals which were affected by a lesion or lameness at any time point were regarded a susceptible, and animals which were unaffected at every assessment were regarded as resistant. Therefore, the repeated records from each animal were essentially used in order to reduce misclassification bias by increasing the robustness of a “resistant” classification. The aim of statistical analysis was to quantify the association between the four outcomes and genetic indexes by fitting logistic regression models in a descriptive capacity.

Lameness data collected during the study was matched by cow ear tag or herd book number to their published genetic indexes. Matched records were available for 2,107 cows out of the 2,352 with lameness data. Descriptive and statistical analyses were conducted in R (41).

Lesion records from all assessments were used to categorise cows as either affected (i.e., susceptible), if the lesion had been present on any foot at any time point during the study, or unaffected (i.e., resistant) if the lesion had been absent throughout. At each assessment, cows were considered affected with SU if there was any ulceration in the sole area of the foot; cows were considered affected with SH if haemorrhage was ≥2cm diameter or dark pink/purple in colour; cows were considered affected with WL if there had been discoloration or separation of the white line which was still present after limited trimming. Similarly, mobility scores from each time point were summarised by the maximum recorded mobility score across the whole study period. This maximum mobility score was dichotomised to indicate either the animal had always been recorded as “non-lame” (maximum mobility score 0 or 1) or the animal had been recorded “lame” at least once (maximum mobility score 2 or 3). Finally, when adjusting for the confounding effects of parity, the parity of each cow was grouped into an ordinal variable (1 to 5) where the top level included 5th parity or greater.

The Pearson’s correlation coefficient between Lameness Advantage index and other genetic indexes was calculated. The unadjusted relationship between Lameness Advantage index and period prevalence (42) of CHL and lameness was calculated by binning the index, based on the distribution within our dataset, into: ≤ -2.0, > -2.0 ≤ -1.0, > -1.0 ≤ -0.5, > -0.5 ≤ +0.5, > +0.5 ≤ +1.0, > +1.0 ≤ +2.0, and > +2.0. This relationship was further evaluated after adjusting for the effects of herd and parity by fitting a multivariable logistic regression model with herd, parity and Lameness Advantage index as covariables; herd and parity were included as categorical variables and Lameness Advantage index as a continuous variable. The same model was fit to the four different outcomes which categorised cows as unaffected/affected by SH, SU, WL, and lameness as described above. The assumptions of logistic regression were assessed using the *performance* package (43). Specifically, log-linearity was assessed by inspecting scatter plots of genetic index against logit values; multicollinearity was assessed by calculating the variance inflation factor for each explanatory variable, and residual distribution was assessed by examining binned residual plots. Goodness of fit was assessed using the Hosmer-Lemeshow test and the explanatory power of the model was assessed by calculating the coefficient of discrimination (Tjur’s R2) (44). The model-adjusted probabilities of each outcome for different values of the Lameness Advantage index were calculated using the *ggeffects* package (45), this was displayed for different herds whilst averaging the effect of parity, and for different parities whilst averaging the effect of herd.

The same approach of fitting multivariable logistic regression models to each of the four outcomes was repeated using the sire Lameness Advantage index in place of the animal’s own index. The only change made to this model was to reduce the parity variable to three levels to maintain adequate numbers of observations per level, consequently cows were considered as either first parity, second parity, or third parity and greater. Finally, all other genetic indexes in the dataset were screened for an association with one of the four outcomes by fitting each one in turn in multivariable logistic regression models which also included farm and parity (five levels) as before. Given the lower prior probability of finding an association during this final part of the analysis, and the large number of hypotheses tested for each outcome (36 genetic indexes), associations were only considered statistically significant if the regression coefficient for the genetic index had a p-value lower than 0.05 following Bonferroni correction (i.e. 0.05/36). Therefore, the adjusted significance level for the association between a genetic index, other than Lameness Advantage, and one of the four outcomes was set at 0.0014.

**Results**

Lameness data and genetic indexes were available for 2,107 cows, representing 90% of the cows with lameness data. The reasons for missing genetic index data were either genotyping failures or mismatches between pedigree and genotyping information such as parental identification. A total of 1,818 cows could be matched to sires which had a published Lameness Advantage index, resulting in 280 different sires in this dataset.

The parity distribution and period prevalence of lesions and lameness were similar in the 2,352 cows with lameness data we had collected and the 2,107 cows with both lameness data and genetic indexes (Table 1). The mean Lameness Advantage index was +0.5 (SD: 1.1) and ranged from -3.1 to +4.4; younger animals tended to have higher values than older animals (Table 2). The reliability of the Lameness Advantage index, calculated in the validation step of the genetic evaluations as the squared correlation between genomic merit and average relative performance, ranged from 0.46 to 0.60 (mean: 0.55, SD: 0.03). The mean Lameness Advantage index in sires represented in this dataset was +1.0 (SD: 1.9) and ranged from -5.8 to +6.3. In cows which had a Lameness Advantage index value close to the genetic average (-0.5 to +0.5), the period prevalence of SH, SU, WL, and lameness was 33.0%, 11.1%, 21.3%, and 23.5% respectively. In all cases there was a clear trend that cows with a lower value of Lameness Advantage had a higher period prevalence of each outcome and vice versa (Table 3).

The multivariable logistic regression models were intended to quantify the relationship between the Lameness Advantage index and each outcome after adjusting for the effects of parity and herd. There were no violations in the assumptions regarding log-linearity, multicollinearity, and residual distributions. In all models, the Hosmer-Lemeshow test statistic was not statistically significant (*p* > .05) indicating acceptable fit to the data. The explanatory power of each model was generally low, the coefficients of discrimination (Tjur’s R2) were 0.07, 0.11, 0.04, and 0.15 for SH, SU, WL, and lameness, respectively. The odds ratios (95% confidence intervals (CI)) for the Lameness Advantage index (for one point increase) were 0.79 (95% CI = 0.72 - 0.86), 0.68 (95% CI = 0.59 – 0.78), 0.94 (95% CI = 0.84 – 1.04), and 0.82 (95% CI = 0.74 – 91) for SH, SU, WL, and lameness, respectively (Table 4). Model-adjusted probabilities were calculated and indicated an average relative risk increase between a Lameness Advantage index of -1 compared to +1 of 29% (absolute risk increase (ARI) = 12%), 100% (ARI = 10%), 12% (ARI = 3%), and 33% (ARI = 7%) for SH, SU, WL, and lameness, respectively. Subsequently, model-adjusted probabilities of each outcome were displayed for each herd after averaging the effect of parity (Figure 1), and for each parity after averaging the effect of herd (Figure 2).

The results of the multivariable logistic regression models which included the sire’s Lameness Advantage index in place of the animal’s own index are presented in Table 5. With the exception of WL, there was a generally weaker effect of the sire’s Lameness Advantage index compared to the animal’s own index, although 95% confidence intervals overlapped.

The correlation between all published genetic indexes were calculated. Only three indexes had a Pearson’s correlation coefficient with Lameness Advantage greater than 0.4: Digital Dermatitis, PLI and Lifespan. Correlations between all genetic indexes are provided as supplementary materials (Supplementary Table 1), but of note was the low and positive correlation (r = 0.09, 95% CI = 0.04 – 0.13) between Lameness Advantage and the genetic index for milk production. All remaining genetic indexes were screened for an association with one of the four outcomes after adjusting for the effect of farm and parity (Supplementary Table 2). Genetic indexes which had a statistically significant association after adjusting for multiple testing (significance level: 0.0014) are presented in Table 6.

**Discussion**

**Key results**

Our primary objective was to validate the Lameness Advantage genetic index with respect to claw horn lesion development in a cohort of dairy cows. The Lameness Advantage index is calculated from national genetic evaluations and primarily determined by an animal’s genotype, with additional information from an animal’s pedigree, farmer-recorded lameness events, and breed society classifying results. As the foot lesions and mobility scores recorded during our study were independent of the Lameness Advantage index calculation, we used these records to independently evaluate this genetic index.

Our results showed the Lameness Advantage index was associated with CHL development and lameness; for every one-point increase in Lameness Advantage there were reduced odds of an animal having SH, SU, or lameness during our study. We observed a similar, but generally weaker, trend using the sire’s index in place of the animal’s own. It should be noted that as 95% confidence intervals overlapped between the odds ratios of an animal’s own and animal’s sire Lameness Advantage index, by definition our results are also compatible with the Lameness Advantage index of both animal and sire having equivalent effects, although this is less likely. The strength of association between Lameness Advantage and CHL, using either the animal’s own index or the sire’s, followed the general trend in heritability estimates of CHL, where SU is typically reported to have the highest heritability and WL the lowest (20). These results highlight the potential of genetic selection on the Lameness Advantage index to complement strategies to reduce the incidence of SH and SU in UK dairy herds.

**Interpretation**

In this study, the odds of SU decreased by 32% for every one-point increase in Lameness Advantage index, after adjusting for the effects of parity and herd (Table 4); the odds of SU decreased by 18% when the sire Lameness Advantage index was assessed in the same way (Table 5). For context, a recent study reported a 20% reduction in odds of a SU in cows which had been preventatively foot trimmed prior to drying-off (46). Preventive foot-trimming is widely considered a key part of SU prevention, so on the strength of the association between SU and the Lameness Advantage index, we believe it is advisable to also include genetic selection as part of SU prevention programmes. Furthermore, one of the major barriers to lameness control is often cited to be the cost of interventions (22–24,47). However, the direct costs of selecting on a genetic index, particularly from a bull proof as opposed to an animal’s own genotype, are negligible in comparison to other interventions which often include re-designing housing or increasing foot-trimming frequency.

The magnitude of the potential reduction in SU incidence that could be achieved through genetic selection would result in a substantial improvement in both animal welfare and farm efficiency. From an animal welfare perspective, SU are recognised as one of the major causes of lameness in dairy cattle, a condition which is a painful and highly representative of their welfare (48–50). Additionally, this reduction in SU would be of economic benefit with every case of SU reported to cost farmers between $232 and $622 depending on the severity of the lesion (51).

All animals in our study had been genotyped and we observed the strongest associations between an animal’s own Lameness Advantage index and the odds of SU development. Genomic testing and selection of females has risen over recent years (52) and improved profitability can offset the costs of genotyping (53,54), particularly when combined with breeding programmes which use sexed or beef semen (55–59). The results of this study indicate an additional financial return from the genomic selection of heifers may include the reduction in SU incidence, and this could be realised, at least in part, during the first lactation. As genetic gains are slow, farmers can be reluctant to engage with genetic selection for lameness reduction (22,60); therefore the reduced chance of SU development within the first lactation could present a compelling incentive to consider this approach.

The relationship between Lameness Advantage and SU frequency appeared to be strongest in older cows (Figure 2). Our interpretation of this trend is that genetic resistance to SU may become increasingly important in older cows because risk of CHL development increases cumulatively with age (61,62). Therefore, as there is a drive to increase the longevity of dairy cows (63,64), breeding cows with good genetic merit for lameness is a clear priority.

Genetic selection requires accurate selection indexes to be available and in turn, genetic selection indexes are dependent on phenotype accuracy. The Lameness Advantage index utilises farmer-recorded lameness to allow more direct selection for lameness reduction than through conformation traits alone (28), however, the recording of lameness in farm records has repeatedly been highlighted to be poorer than other health conditions (28–30). Despite the promising results we observed, we still believe it is important to encourage better recording of lameness on farms to provide useful phenotypes for national genetic evaluations. In the future, direct lesion traits, such as foot-trimming records, may be available for genetic evaluations, as they are in other countries (65–67), and ultimately this is likely to be the approach which maximises genetic improvements to reduce lameness (27).

We observed associations between genetic indexes other than Lameness Advantage and the development of CHL or lameness. There were associations between lameness and Fertility Index, Lifespan, and PLI, and likewise between SCC value and SU development. However, despite the low p-values the magnitude of these associations was negligible and we do not consider these results to be of particular importance. It is worth noting, however, the lack of association between PLI and CHL development. Therefore, although Lameness Advantage is included in the PLI, selection on PLI alone is unlikely to result in reductions in CHL incidence. Of the top ten Holstein bulls for PLI listed on the AHDB website in November 2021 (68), four have a Lameness Advantage index greater than +2.0 so it is possible to select for both high PLI and good Lameness Advantage; this is the approach we would advise to farms looking to use breeding decisions as part of lameness reduction programs. Having said that, the correlations between Lameness Advantage and production indexes were low and positive, indicating that selecting on Lameness Advantage alone does not risk sacrificing productivity.

Lameness, as determined by mobility scoring, was associated with three genetic indexes other than Lameness Advantage: Angularity (OR: 1.32, 95% CI: 1.14 – 1.53), Legs and Feet (OR: 0.75, 95% CI: 0.64 – 0.89), and Locomotion (OR: 0.75, 95% CI: 0.64 – 0.89). Angularity, also called “dairy form”, refers to the openness between ribs and is recognised to correlate with body condition (higher angularity is associated with lower body condition) and locomotion (69), therefore this association seems plausible. The Feet and Legs index includes Locomotion, in addition to other linear conformation traits, and unsurprisingly these two genetic indexes are highly correlated with each other (Supplementary Table 1). The association between these three genetic indexes and lameness, but not CHL development, could be explained by an association with foot lesions other than CHL, which we did not evaluate in this study, or these genetic indexes could relate more closely to gait than CHL development. The absence of an association with CHL development suggests that although selecting on type traits such as Angularity, Legs and Feet, and Locomotion may reduce the prevalence of visibly lame cows, it is unlikely to reduce the incidence of CHL.

A study which compared farmers’ stated preferences for genetic selection with actual selection practices reported that although farmers reported health traits to be the most important, selection on these traits was less frequent, and the opposite effect was observed for type traits (70). Our data indicate that type traits alone will not be as effective at reducing CHL frequency and this result, alongside the validation of the Lameness Advantage index, should be communicated to farmers wishing to breed for reduced lameness.

We observed a strong and unexpected association between the Digital Dermatitis genetic index and SH and SU development. This result warrants further investigation. The most straightforward explanation for this result would be if SH/SU are highly genetically correlated with digital dermatitis (DD); however, previously reported genetic correlations have ranged from -0.15 to 0.12 for DD and SH and from -0.19 to 0.56 for DD and SU (20). Although the standard errors of previous genetic correlation estimates are large, the magnitude of these correlations suggests this explanation of our results is unlikely. Furthermore, the highest positive correlation reported of 0.56 (71) is an outlier among previous studies with the next highest correlation reported to be 0.15 (72), and the genetic correlation between DD and SH/SU is frequently reported to be negative (67,73,74) making it harder to accept this as an explanation for the association we observed in our study.

It is possible that DD is a risk factor for SH/SU in the absence of a shared genetic background. For this mechanism to exist, the DD index must first have a strong association with DD development, and preliminary analysis of our data indicates that this could be the case (data not shown). However, it would then be necessary for DD development to substantially increase the risk of SH/SU development, which previous studies have not identified, although analysis of sufficiently longitudinal and detailed foot lesion data is lacking. It is therefore theoretically possible that DD in younger animals, such that it is identified during breed society classification, increases the risk of the animal subsequently developing SH or SU; but there are reasons to be sceptical about this hypothesis including the low rates of concurrent DD and SH/SU reported (73,75,76) and the implausibly high attributable risk required for this mechanism to hold. We consider it more likely, therefore, that SH/SU may be conditionally associated with DD via an unknown factor which has an association with both DD and SH/SU but is only genetically correlated with DD.

**Limitations**

As only four herds were included in this study, the applicability of the results to other herds requires careful interpretation (further discussed in “Generalisability” section). Genetic indexes were only available for 90% of cows with CHL and mobility score records and, although the genetic merit of the missing animals is unknown, the distribution of lesion prevalence across parities appeared to be similar with and without these animals (Table 1). This study analysed the relationship between the Lameness Advantage index and the risk of CHL or lameness development during a single lactation. It therefore does not relate to the performance of an animal over its entire lifetime which would be a more appropriate phenotype in order to fully assess the influence of genetic merit, but one that is logistically much more challenging to obtain. Equally, although the accuracy of our phenotypes was improved by using repeated records for each animal, it is still possible that lesions could have been missed if they occurred transiently between assessment time points. Multivariable models were designed to be descriptive and therefore not evaluated as predictive models. We would expect the predictive performance of these model to be poor, as suggested by the low coefficients of discrimination; this is because it would be unlikely that a genetic index could predict the phenotype when the heritability estimates of these lesions suggest that the majority of phenotypic variance is not due to genetics (20).

**Generalisability**

Generalisability from a study which includes four herds is limited, however, we discuss relevant details of the study herds to allow interpretation of the potential applicability of these results to other herds. This study included four dairy herds which were all commercially run with operating practices common to many British dairy farms, but could not be considered representative of the full spectrum of dairy farms. Within these four herds, three were operating relatively intensive systems of zero grazing and three times a day milking. In a recent survey of 53 randomly selected dairy herds in the UK, 36% housed milking cows all year round (77). A survey of 863 dairy herds in 2012, also in the UK, reported that 23% of herds housed early lactation and high-yielding groups all year round (78). We did not observe any differences in trends between the three farms which housed all milking cows and the remaining herd which was managed with a combination of housed and grazed groups (Figure 1).

The overall period prevalence of lame cows (i.e. based on repeated mobility scores) ranged from 18.5% to 33.3% over the four herds; this is similar to previous reports in British dairy herds and suggests the four herds in our study have an average or below average prevalence of lameness (79,80). The prevalence of CHL has historically only been reported for lame animals or from foot-trimming records. Therefore previous reports may not have a reliable numerator, due to under-reporting of mild lesions, or a reliable denominator, due to over-representation of lame cows. In studies using foot-trimming records, the prevalence of CHL has been reported to range from 5 – 59% for SH, 5 – 19% for SU, and 4 – 18% for WL (71–73,75,76,81–83). It is therefore possible that our study had a population of cows with an unusually high prevalence of CHL despite an average or below average prevalence of lame cows. However, we think this is unlikely and would consider our results to accurately represent the true frequency of CHL in these herds as foot lesions were recorded at repeated time points in cows assessed specifically for this purpose.

**Conclusions**

We have performed a study which highlights the potential of the Lameness Advantage genetic index to facilitate breeding cows with better resistance to lameness. We found differences in the frequency of claw horn lesions and lameness in cattle associated with this index, particularly for sole haemorrhage and sole ulcers. In comparable populations, genetic selection on the Lameness Advantage index is likely to translate to a reduced risk of cows developing sole haemorrhage and sole ulcers, although we would expect the greatest reductions to occur through a combination of genetic selection and husbandry-based improvements.

**Declarations**

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*Ethics approval*

The study was conducted following ethical approval from the University of Liverpool Veterinary Research Ethics Committee.

*Consent for publication*

Consent was obtained from participating farms prior to the start of the study.

*Availability of data and material*

The datasets generated and analysed during the current study are available at reasonable request.

*Competing interests*

All authors declare that they have no competing interests.

*Contributorship statement*

MB collected field data, performed statistical analyses and wrote the first draft of the manuscript, BG, AA, CB collected field data, MW advised on study design and provided genetic evaluations, BL advised on study design and statistical analysis, MC advised on study design and lead the calculation of breeding values for AHDB, AP advised on study design, GB advised on study design and statistical analysis, GO (corresponding author) designed and supervised the study. All authors critically evaluated the manuscript and approved the submitted version.

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Table 1. Period prevalence of claw horn lesions and lameness in the whole population of animals with lameness data and the final study population used for analysis.

|  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | | Whole study population with lesion records (N = 2,352) | | | | | | | | |
| N | SH | | SU | | WL | | Lameness | |
| Parity | 1st | 610 | 38.2% | | 4.6% | | 15.9% | | 6.1% | |
| 2nd | 730 | 22.2% | | 4.0% | | 20.5% | | 14.7% | |
| 3rd | 394 | 35.8% | | 12.4% | | 20.6% | | 27.0% | |
| 4th | 315 | 43.2% | | 22.9% | | 26.0% | | 40.5% | |
| 5th | 303 | 45.5% | | 30.4% | | 35.3% | | 51.2% | |
|  | | Population with lesion records and genetic indexes (N = 2,107) | | | | | | | | |
|  | | N | | SH | | SU | | WL | | Lameness |
| Parity | 1st | 583 | 37.2% | | 4.5% | | 16.1% | | 6.0% | |
| 2nd | 589 | 23.3% | | 4.8% | | 20.7% | | 14.6% | |
| 3rd | 362 | 34.8% | | 13.0% | | 20.2% | | 25.2% | |
| 4th | 290 | 39.7% | | 23.1% | | 24.1% | | 38.1% | |
| 5th | 283 | 44.5% | | 29.3% | | 35.3% | | 51.2% | |

*SH: sole haemorrhage, SU: sole ulcer, WL: white line lesion; Lameness: mobility score 2 or 3.*

Table 2. The average and distribution of Lameness Advantage index in female animals by year of birth.

|  |  |  |  |
| --- | --- | --- | --- |
| Year of birth | N | Lameness Advantage | |
| Mean (SD) | Range |
| 2007 - 2012 | 159 | -0.44 (1.17) | -2.88 – +2.21 |
| 2013 - 2014 | 415 | -0.07 (1.17) | -2.62 – +3.59 |
| 2015 - 2016 | 940 | +0.61 (1.03) | -3.10 – +3.72 |
| 2017 - 2018 | 593 | +0.90 (0.84) | -1.43 – +4.40 |

*SD: standard deviation*

Table 3. Unadjusted period prevalence of claw horn lesions and lameness for ranges of Lameness Advantage index

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Lameness Advantage index | N | SH | SU | WL | Lameness |
| ≤ -2.0 | 39 | 48.7% | 56.4% | 59.0% | 61.5% |
| > -2.0 ≤ -1.0 | 174 | 45.4% | 23.0% | 29.9% | 42.5% |
| > -1.0 ≤ -0.5 | 187 | 44.4% | 23.5% | 20.9% | 32.3% |
| > -0.5 ≤ +0.5 | 630 | 33.0% | 11.1% | 21.3% | 23.5% |
| > +0.5 ≤ +1.0 | 364 | 33.5% | 9.3% | 17.3% | 16.5% |
| > +1.0 ≤ +2.0 | 554 | 30.9% | 6.0% | 20.0% | 13.7% |
| > +2.0 | 159 | 24.5% | 5.0% | 23.3% | 15.1% |

*SH: sole haemorrhage, SU: sole ulcer, WL: white line lesion; Lameness: mobility score 2 or 3*

Table 4. Multivariable logistic regression for claw horn lesion presence or lameness based on mobility score using the animal’s own Lameness Advantage index (N = 2,107)

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | | SH | | | SU | | | WL | | | Lameness | | |
| OR | 95% CI | *p* | OR | 95% CI | *p* | OR | 95% CI | *p* | OR | 95% CI | *p* |
| Lameness Advantage (animal) | | 0.79 | 0.72 – 0.86 | <.001 | 0.68 | 0.59 – 0.78 | <.001 | 0.94 | 0.84 – 1.04 | .210 | 0.82 | 0.74 – 0.91 | <.001 |
| Parity | 1 | *Reference* | | | | | | | | | | | |
| 2 | 0.41 | 0.31 – 0.53 | <.001 | 0.96 | 0.55 – 1.68 | .889 | 1.24 | 0.91 – 1.68 | .170 | 2.72 | 1.81 – 4.16 | <.001 |
| 3 | 0.73 | 0.55 – 0.97 | .032 | 2.88 | 1.73 – 4.87 | <.001 | 1.30 | 0.91 – 1.84 | .143 | 5.05 | 3.34 – 7.80 | <.001 |
| 4 | 0.84 | 0.62 – 1.15 | .284 | 4.91 | 2.99 – 8.24 | <.001 | 1.59 | 1.10 – 2.29 | .012 | 8.49 | 5.57 – 13.21 | <.001 |
| ≥5 | 0.96 | 0.70 – 1.32 | .802 | 6.10 | 3.70 – 10.29 | <.001 | 2.72 | 1.90 – 3.89 | <.001 | 13.85 | 9.05 – 21.66 | <.001 |
| Herd | A | *Reference* | | | | | | | | | | | |
| B | 0.30 | 0.19 – 0.48 | <.001 | 0.36 | 0.19 – 0.71 | .002 | 0.32 | 0.20 – 0.51 | <.001 | 0.85 | 0.47 – 1.64 | .618 |
| C | 0.68 | 0.41 – 1.11 | .123 | 0.35 | 0.17 – 0.73 | .004 | 0.53 | 0.32 – 0.88 | .014 | 0.57 | 0.30 – 1.14 | .098 |
| D | 0.77 | 0.72 – 0.86 | <.001 | 0.26 | 0.59 – 0.78 | .001 | 0.27 | 0.15 – 0.48 | <.001 | 0.77 | 0.39 – 1.559 | .470 |

*The intercept (standard error) for each model was: SH: 0.65 (0.24); SU: -1.80 (0.36); WL: -0.58 (0.25), and Lameness: -2.39 (0.34).*

*OR: Odds ratio; CI: confidence interval; SH: sole haemorrhage, SU: sole ulcer, WL: white line lesion; Lameness: mobility score 2 or 3*

Table 5. Multivariable logistic regression for claw horn lesion presence or lameness based on mobility score using the Lameness Advantage index of the sire of each animal (N = 1,818)

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | | SH | | | SU | | | WL | | | Lameness | | |
| OR | 95% CI | *p* | OR | 95% CI | *p* | OR | 95% CI | *p* | OR | 95% CI | *p* |
| Lameness Advantage (sire) | | 0.90 | 0.84 – 0.95 | <.001 | 0.82 | 0.75 – 0.88 | <.001 | 0.91 | 0.85 – 0.97 | .003 | 0.89 | 0.83 – 0.94 | <.001 |
| Parity | 1 | *Reference* | | | | | | | | | | | |
| 2 | 0.43 | 0.35 – 0.57 | <.001 | 1.04 | 0.58 – 1.84 | .906 | 1.35 | 0.97 – 1.88 | .076 | 2.81 | 1.83 – 4.41 | <.001 |
| ≥3 | 0.93 | 0.73 – 1.19 | .570 | 5.06 | 3.28 – 8.14 | <.001 | 1.86 | 1.39 – 2.52 | <.001 | 9.21 | 6.31 – 13.86 | <.001 |
| Herd | A | *Reference* | | | | | | | | | | | |
| B | 0.31 | 0.19 – 0.50 | <.001 | 0.35 | 0.19 – 0.70 | .002 | 0.32 | 0.20 – 0.52 | <.001 | 0.84 | 0.46 – 1.60 | .570 |
| C | 0.73 | 0.44 – 1.22 | .232 | 0.34 | 0.17 – 0.72 | .004 | 0.48 | 0.28 – 0.81 | .006 | 0.53 | 0.27 – 1.06 | .063 |
| D | 0.93 | 0.52 – 1.66 | .804 | 0.21 | 0.08 – 0.53 | .001 | 0.32 | 0.17 – 0.60 | <.001 | 0.58 | 0.27 – 1.29 | .175 |

*The intercept (standard error) for each model was: SH: 0.60 (0.25); SU: -1.73 (0.35); WL: -0.52 (0.25), and Lameness: -2.33 (0.35).*

*OR: Odds ratio; CI: confidence interval; SH: sole haemorrhage, SU: sole ulcer, WL: white line lesion; Lameness: mobility score 2 or 3*

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | OR (95% CI) from multivariable model including parity and herd. | | | |
|  | SH | SU | WL | Lameness |
| Angularity | 1.066  (0.947 – 1.201) | 1.188  (0.990 – 1.429) | 1.031  (0.900 – 1.181) | **1.316**  **(1.138 – 1.525)** |
| Digital dermatitis | **0.584**  **(0.453 – 0.751)** | **0.490**  **(0.345 – 0.693)** | 0.978  (0.739 – 1.295) | 0.606  (0.455 – 0.807) |
| Fertility index | 0.977  (0.958 – 0.997) | 0.958  (0.931 – 0.986) | 0.987  (0.965 – 1.009) | **0.957**  **(0.935 – 0.980)** |
| Legs and Feet | 1.031  (0.896 – 1.187) | 1.081  (0.879 – 1.331) | 1.043  (0.890 – 1.224) | **0.753**  **(0.637 – 0.889)** |
| Lifespan | 0.999  (0.996 – 1.001) | 0.996  (0.993 – 0.999) | 1.001  (0.998 – 1.004) | **0.994**  **(0.991 – 0.997)** |
| Locomotion | 1.084  (0.943 – 1.246) | 1.137  (0.927 – 1.395) | 1.075  (0.919 – 1.259) | **0.751**  **(0.636 – 0.885)** |
| PLI | 1.000  (0.999 – 1.000) | 0.998  (0.997 – 1.000) | 1.000  (0.999 – 1.001) | **0.998**  **(0.997 – 0.999)** |
| SCC | 1.017  (1.003 – 1.031) | **1.040**  **(1.018 – 1.062)** | 1.003  (0.987 – 1.018) | 1.026  (1.009 – 1.044) |

Table 6. Multivariable logistic regression screening for associations between all genetic indexes and claw horn lesion presence or lameness (N = 2,107). Results are presented as the odds ratio and 95% confidence interval for the genetic index, adjusted for parity and herd. Only genetic indexes with at least one association which was statistically significant at the adjusted significant level of 0.0014 are presented (bold).

*OR: Odds ratio; CI: confidence interval; SH: sole haemorrhage, SU: sole ulcer, WL: white line lesion; Lameness: mobility score 2 or 3; PLI: Profitable Lifetime Index; SCC: Somatic Cell Count*

**Figure 1.** The model-adjusted probabilities of sole haemorrhage (SH), sole ulcer (SU), white line lesion (WL) and lameness based on mobility score (Lameness). The probability of each outcome is displayed against the animal’s Lameness Advantage genetic, results are presented separately for each herd using the average effect of parity.

**Figure 2.** The model-adjusted probabilities of sole haemorrhage (SH), sole ulcer (SU), white line lesion (WL) and lameness based on mobility score (Lameness). The probability of each outcome is displayed against the animal’s Lameness Advantage genetic index, results are presented separately for each parity using the average effect of herd.

**Supplementary Table 1.** The Pearson’s correlation between genetic indexes.

**Supplementary Table 2.** Multivariable logistic regression screening for associations between all genetic indexes and claw horn lesion presence or lameness (N = 2,107). Results are presented as the odds ratio and 95% confidence interval for the genetic index, adjusted for parity and herd.