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GENETIC PARAMETERS OF PUBERTY ESTIMATED USING TWO GENETICALLY DIVERGENT GROUPS OF HOLSTEIN-FRIESIAN DAIRY HEIFERS

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SUMMARY

Genetic parameters for age at puberty were estimated for Holstein-Friesian dairy heifers in an experimental herd comprised of genetically divergent lines for fertility. Despite the non-random population structure, the estimated heritability of age at puberty was approximately 13%, based on behavioural observations, and between 41% and 76% based on blood progesterone levels. Although the phenotypic correlation between these two different measures was moderate (~0.5), the genetic correlation was much stronger (~0.9). In addition, the genetic correlations between age at puberty traits and fertility BV was ~-0.3, which suggests that age at puberty estimates may aid in the genetic evaluation of lowly heritable fertility traits.

INTRODUCTION

Previous studies of the onset of puberty, defined as age at first behavioural oestrus, estimated its heritability as approximately 0.27 in NZ beef cattle (Morris *et al*. 2000; Amyes and Morris 2009), and Martin *et al*. (1992) reported an average of 0.4 across nine beef cattle studies (ranging from 0.07 to 0.67). Fewer studies have reported the heritability of age at puberty in dairy cattle, but one available estimate was 0.09 (Morris and Hickey 2004). While the onset of puberty itself is an important trait for herd management purposes, its influence on reproductive traits is also of interest since genetic correlations may aid in the genetic improvement of lowly heritable fertility. In the NZ dairy industry, the fertility breeding value (BV) is comprised of several distinct traits such as PM21 (presented for mating within 21 days of planned start of mating; h^2=0.05) and CR42 (calving rate in 1st 42 days after planned start of calving; h^2=0.03). In NZ beef cattle, favourable genetic correlations have been reported between heifer age at puberty and several reproductive traits, such as scrotal circumference in NZ beef bulls (-0.25), pregnancy rate (-0.23) and calving date (0.57) (Morris and Amyes 2010).

Oestrus onset may instead be directly measured and defined as the age when blood progesterone (P4) concentration has reached a certain threshold. McNaughton *et al.* (2005) used a criterion of P4 >1ng/mL for 2 of 3 consecutive weekly samples in NZ dairy heifers. Age at puberty determined in this way may have an advantage over behavioural measurements due to its quantitative accuracy, and may thus provide a better estimate of heritability. No heritability estimate for P4-based age at puberty in dairy heifers is currently known.

The objective of this study was to estimate genetic parameters of several age at puberty (AP) traits in dairy cattle, including their heritabilities and genetic correlations with fertility BVs. These AP traits would be determined via either observational oestrus or several P4-based criteria.

The data used was from a physiological study of NZ dairy heifers in which the experimental herd was genetically divergent on fertility (Meier *et al.* 2017). This divergence could introduce bias into any genetic analyses, so an auxiliary objective of this study was to evaluate a basic method to account for this.

MATERIALS AND METHODS

Study animals. The study population consisted of 527 Holstein-Friesian heifers born across 379 herds between June and September 2015 and produced by mating low or high fertility BV dams and sires to generate divergent genotypes (Low BV heifers: n=252, μ=-5.12, σ=1.37; High BV heifers:

n=275, μ=5.00, σ=0.74). These heifers were reared in four mobs, each consisting of a mixture of high and low BV heifers, with the ratio per mob being no more than 40:60 either way.

Data. From April to November 2016, P4 was determined in weekly collected plasma samples. Tail-paint or heat mount detectors (Kamar Inc., Zionsville, IN USA) were used, and checked approximately weekly to quantify mounting activity associated with expression of oestrus. From these data, we defined six potential indicators of age at puberty: (AP1) when P4 first reached 1.0 ng/mL; (AP2) when P4 first reached 1.0 ng/mL for 2 consecutive weekly samplings; (AP3) when P4 first reached 1.0 ng/mL for 2 of 3 consecutive weekly samplings; (AP4) when P4 first reached 0.7 ng/mL; (AP5) when P4 first reached 0.7 ng/mL for 2 consecutive weekly samplings; and, (APK) when either the heat mount detector was activated, tail-paint was mostly worn off (scored 1 on a 1-5 rubbing scale; 5 being as new), or the animal was visibly in oestrus.

The full pedigree of these animals was extracted from the New Zealand Dairy Industry Good Animal Database (DIGAD), which consisted of 10,992 records, up to 18 generations deep. Also extracted from DIGAD were the fertility BVs for the 527 heifers estimated in the most recent national animal evaluation (January 2017). Although these BVs are already genetic estimates based on pedigree linkages rather than own data, they were used as a response variable in the models, and are referred to as the "fertility BV trait" in this study.

Variance component estimation. The data came from a population that is genetically divergent for fertility and, therefore, not normally distributed for this trait. Because fertility is likely to be genetically correlated with puberty, the herd is likely to be genetically divergent (and, therefore, not normally distributed) for puberty also, and so the genetic variance components and heritability of puberty will be overestimated by a standard univariate mixed model. To account for this, a two-model approach was used: the first (standard) model included mob as the only fixed effect; the second model included fixed effects for both mob and fertility group (low or high). Both models included a pedigree-based random animal effect. The second model, by absorbing some of the puberty variation into the fertility group effect, will underestimate the variance components for puberty. Consequently, the first and second models provide upper and lower bounds for the genetic variance of age at puberty, and thus a range for heritability. However, due to the potential for some confounding of mob effect with fertility group (as mobs were not exactly balanced), the upper heritability bound may still contain some downwards bias. If there is no genetic correlation between fertility and puberty, these upper and lower bounds ought to be approximately the same.

Bivariate mixed models (having fixed mob effect and random animal effect) were fitted with pairs of puberty definitions as response variables to estimate both the phenotypic and genetic covariances and correlations between them. Assuming linearity of genetic covariance between the fertility and age at puberty traits, the divergent population structure will not affect genetic covariance estimates, and so standard bivariate mixed models (without fertility group effect) were used.

In order to estimate genetic correlations between fertility and puberty traits, a Pearson correlation was used, in which puberty genetic variance was estimated from a (standard) univariate mixed model and fertility genetic (co)variances were estimated from a bivariate fixed effect model (mob effect; no random effect), with fertility BV and a puberty trait as response variables. As the fertility BVs are already genetic estimates, the residual variance in fertility and residual covariance from the bivariate fixed effect model are estimates of the genetic (co)variances of fertility. Standard errors are not readily available for this genetic correlation, but they ought to be of a similar magnitude to those of the other genetic correlations.

ASReml (Gilmour *et al*. 2015) was used to perform all model analyses.

RESULTS AND DISCUSSION

The lower and upper bounds for heritability estimates of the six defined puberty traits and their standard errors are presented in Table 1. Generally, the difference between lower and upper estimates was about 10% for P4-based puberty, although the high standard errors may indicate that this range is larger than calculated. Nonetheless, the two-model approach does seem to provide a useful range of potential heritabilities. Observation-based puberty (APK) unexpectedly had an upper bound which was slightly lower than its lower bound, although given the large standard errors, this is not a significant anomaly. The fact that these two heritability bounds are almost identical indicates, initially at least, a weak genetic correlation between APK and fertility.

Table 1. Upper and lower bounds of heritability estimates of puberty traits (± s.e.)

	AP1	AP2	AP3	AP4	AP5	APK
h^2_{Upper}	0.63 ± 0.17	0.76 ± 0.19	0.71 ± 0.18	0.53 ± 0.16	0.76 ± 0.19	0.13 ± 0.10
h^2_{Lower}	0.49 ± 0.16	0.66 ± 0.19	0.62 ± 0.18	0.41 ± 0.15	0.67 ± 0.19	0.14 ± 0.10

The heritability of P4-based puberty (AP1-AP5) was moderate/high (41%-76%), whereas the heritability of APK was low (~13%). The low APK heritability is consistent with previous findings (Morris and Hickey 2004), and the large difference in heritability estimates between these two types of puberty measures is likely due mostly to higher measurement error in APK. The heritability of P4-based puberty relying on when P4 first reached 1.0 or 0.7 ng/mL (i.e. AP1 or AP4) was lower than those utilising consecutive P4 data (i.e. AP2, AP3 or AP5), possibly due to a higher prevalence of initial false positives. Defining puberty onset using a 2 of 3 criterion (AP3) reduced heritability by 4-5% compared with the first of 2 consecutive weeks with elevated P4 (AP2), which may mean that this AP3 estimate has a similar problem to AP1 and AP4. Using the more sensitive P4 threshold of 0.7 ng/mL did not change heritability when considering 2 consecutive detections (AP5), although when considering the first detection only (AP4), this more sensitive measure had an even lower heritability than the AP1 estimate.

Table 2. Genetic correlations (below diagonal) and phenotypic correlations (above diagonal) between age-at-puberty (AP) and fertility traits, derived from bivariate models (± s.e.)

	AP1	AP2	AP3	AP4	AP5	APK
AP1		0.972 ± 0.003	0.972 ± 0.003	0.969 ± 0.003	0.972 ± 0.003	0.485 ± 0.037
AP2	1.000 ± 0.003		*	0.943 ± 0.006	*	0.505 ± 0.037
AP3	1.000 ± 0.003	*		0.946 ± 0.005	*	0.514 ± 0.035
AP4	1.000 ± 0.004	1.000 ± 0.007	1.000 ± 0.006		0.947 ± 0.005	0.477 ± 0.037
AP5	0.999 ± 0.003	*	*	0.999 ± 0.007		0.500 ± 0.037
APK	0.830 ± 0.217	0.925 ± 0.217	0.888 ± 0.205	0.770 ± 0.244	0.896 ± 0.200	
Fertility†	-0.333	-0.285	-0.295	-0.335	-0.279	-0.252

* Log-likelihood failed to converge
† Standard errors not available for fertility genetic correlations

Genetic and phenotypic correlations between puberty traits, and their genetic correlations with fertility BV are presented in Table 2. Correlations between P4-based puberty traits (AP1-AP5) are

close to 1, as expected, with phenotypic correlations slightly lower than genetic correlations. Phenotypic correlations between APK and AP1-AP5 are moderate at ~0.5, but the genetic correlations are high at 0.77-0.92. Although P4-based puberty and observation-based puberty ought to be genetically the same or strongly associated traits, P4 fluctuations may occur prior to *corpus luteum* (CL) formation, thus resulting in false positives. As previously discussed, the AP2 trait ought to be the best measure for avoiding false positives, and the fact that it has the highest genetic correlation with APK indicates that it is succeeding in doing so, and is thus a good measure of the genetics of puberty onset (i.e. oestrus).

Genetic correlations between fertility and puberty traits are consistent at approximately -0.3, including that of APK, despite this latter measure having similar lower and upper heritability bounds. This low/moderate correlation agrees with previous findings (Morris and Amyes 2010). Of note is that this correlation is stronger for the P4-based puberty traits which are more sensitive to early P4 levels (i.e. AP1 or AP4), indicating that the fertility BV trait may have a stronger genetic association with P4 increase in general than with the stable P4 increase at oestrus.

CONCLUSIONS

Although the data is limited in size and divergent in nature, the analysis undertaken here has yielded useful preliminary results which are consistent with current literature. Age at puberty as determined via behavioural oestrus observations has a low heritability and a moderate genetic correlation with fertility. Age at puberty as determined via blood P4 measurement has a high heritability; a moderate genetic correlation with fertility; and, given appropriate threshold criteria, appears to be able to capture the genetic signal of CL formation associated with puberty onset, as behavioural oestrus does. P4-based puberty may thus be of good use for the genetic improvement of puberty and/or fertility. Although P4 measurement may have limited practicality on a national scale under traditional selection, it may be quite feasible within the reference population of a genomic selection scheme. Consequently, these findings indicate that further investigation to establish more robust genetic parameter estimates and assessment of feasibility are warranted.

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