Genetic analysis of calf health in Charolais beef cattle[1]

A. Vinet,[*,2] H. Leclerc,[†] F. Marquis,[‡] and F. Phocas[*]

*GABI, INRA, AgroParisTech, Université Paris-Saclay, 78 350 Jouy-en-Josas, France; [†]Institut de l'Elevage, GABI, 78 350 Jouy-en-Josas, France; and [‡]GIE Charolais France, Agropôle du Marault, 58 470 Magny-Cours, France

ABSTRACT: The objective of this study was to investigate the factors that influence calf health and survival in Charolais cattle. Data from 2,740 calves, originating from 16 French farms and observed from birth until 30 d of age, were analyzed using models that took account of direct genetic, maternal genetic, and common environmental effects. Both direct and maternal genetic parameters were estimated for birth weight (**BW**), calving ease (**CE**), neonatal vitality (**NV**), survival at 30 d (**Surv**), and umbilical infection and diarrhea at different ages (0 to 5 d: **Umb1** and **Diar1**; 6 to 20 d: **Umb2** and **Diar2**; and 21 to 30 d: **Umb3** and **Diar3**). The heritability values for direct and maternal genetic effects were, 0.026 (SE = 0.027) and 0.096 (SE = 0.042) for Surv, 0.280 (SE = 0.063) and 0.063 (SE = 0.038) for BW, 0.129 (SE = 0.041) and 0 for CE, 0.073 (SE = 0.035) and 0 for NV, 0.071 (SE = 0.038) and 0.017 (SE = 0.026) for Umb1, 0 and 0.082 (SE = 0.029) for Umb2, 0 and 0.044 (SE = 0.030) for Diar1, 0.016 (SE = 0.022) and 0.012 (SE = 0.026) for Diar2, and 0.016 (SE = 0.028) and 0 for Diar3, respectively. Significant genetic variability in beef cattle was thus revealed for five calf health traits: NV, Surv, Diar1, Umb1, and Umb2. In addition, for three traits (Surv, Diar1, and Umb2), maternal genetic effects clearly contributed more to health performance than direct genetic effects. Estimates of genetic correlation between traits varied markedly (from 0 to 1 in absolute values) depending on the traits in question, the age for a given trait, and the type (direct or maternal) of the genetic effects considered. These results suggest that not all health traits in Charolais cattle can be improved simultaneously, and breeders will therefore have to prioritize certain traits of interest in their breeding objectives. Overall, our results demonstrate the potential utility of collecting and integrating data on calf diseases, NV and survival in future beef cattle breeding programs. To ensure appropriate biological and genetic evaluations of calf health performance, it is important to accurately describe the phenotypes for diarrhea and umbilical infections (in terms of age ranges) and account for maternal genetic and common environmental effects that explain calf health performance traits. Further investigation and improved data collection are now necessary to maximize the efficiency of breeding schemes designed to simultaneously improve production and health traits.

Key words: diarrhea, disease resistance, mortality, navel illness, umbilical infection, vigor

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[2]Corresponding author: aurelie.vinet@inra.fr

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INTRODUCTION

The diseases that affect calves can have a substantial economic impact on beef and dairy farms, and most cases concern animals that are less than 1 mo old. The two most frequent diseases are diarrhea and respiratory disease (Sivula et al., 1996; USDA, 2010). Umbilical infection is the third most common disease during the first few months of life, affecting between 1.3% (Svensson et al., 2003) and 29% of newborn dairy calves (Virtala et al., 1996). As well as the costs of treating sick calves and the time this requires, the economic losses resulting from calf diseases may also include increased mortality (Sivula et al., 1996), reduced weight gain (Virtala et al., 1996) or longevity (Britney et al., 1984), and older age at first calving (Stanton et al., 2012). Previous studies had demonstrated the genetic component of diseases affecting calves (Heringstad et al., 2008; Henderson et al., 2011). Genetic and genomic selection therefore represent a potential strategy to increase resistance to these diseases, but necessitate the collection of additional phenotypic data beyond what is included in most of the national databases used for genetic evaluation. In addition, little research has been dedicated to the health of beef calves when compared to studies on dairy (and particularly Holstein) calves. Because French breeders were interested in improving the viability of Charolais beef calves, 16 herds were recruited for the purposes of this study, during which records were kept on calf health performance between birth and 1 mo of age. These records included birth weight (**BW**), calving ease (**CE**) score, vigor of the calf at birth [hereinafter referred to as neonatal vitality (**NV**)], survival, and all health events occurring between birth and 1 mo of age. The aim of the study was to estimate the genetic parameters for all these traits in order to answer three key questions relative to the value and feasibility of genetic selection to ensure young calf health: 1) Is it feasible to select calf health traits directly? 2) What pathologies should be included in such a health breeding scheme? 3) Can we select health traits indirectly through selection that focuses more generally on the performance of the young calf (BW, CE or NV)?

MATERIALS AND METHODS

Approval from the Animal Care and Use Committee was not required for this study because the data were obtained from existing national and breeders' databases.

Data

The herds included in this study were recruited from a network of 75 Charolais herds, mainly located in the regions of Burgundy and Vendée. These herds were participating in the on-farm DEGERAM project managed by the French Charolais breeding society in order to develop genomic selection schemes for new traits. The breeders recorded data on herd performance and transmitted it to the French Charolais breeding society during two successive birth campaigns, from August 2013 to July 2015. Data on the 30-d survival of all single newborn calves were extracted from the French national database which is used for on-farm genetic evaluations.

For the present study, we selected records from the herds that: 1) had reported at least three health events per herd-year, and 2) were genetically connected to each other by the use of at least one artificial insemination sire with progenies in a minimum of three herds. The final research dataset (Table 1) included data on 2,740 calves produced by 2,044 dams and 252 sires in 16 connected herds. The parent pedigrees of all calves had been registered for at least two generations of ancestors. Among the 252 sires, 112 bulls were also maternal grandsires of the calves, because they were sires of 839 dams. The two INRA experimental Charolais farms, located at Bourges (in the Berry region) and at Pin-aux-Haras (in Normandy) were among these 16 herds. All 16 herds were managed according to a standard suckler herd production system, in which calves were reared by their dams until 6 to 8 mo of age. With the exception of the INRA experimental unit in Bourges (where the animals were housed indoors throughout the year), the animals were indoors between late November and April and pastured outside for the other months of the year.

Performance records included BW, CE, NV (Table 2), and all health events occurring between

Table 1. Number of records, mean values and raw standard deviation with respect to calf health traits

Item	No.	Mean value	Raw standard deviation
Survival at 30 d, %	2,356	96.31	18.86
Birth Weight, kg	2,717	46.51	6.71
Calving Ease, pt	2,730	1.59	0.81
Neonatal Vitality, pt	2,423	1.45	0.65
Umb1[1], %	2,740	2.92	16.84
Umb2[1], %	2,740	3.47	18.30
Umb3[1], %	2,740	0.40	6.32
Diar1[2], %	2,740	5.26	22.32
Diar2[2], %	2,740	11.68	32.12
Diar3[2], %	2,740	1.57	12.43

[1]Umb1 = umbilical infections occurring between 0 and 5 d of age; Umb2 = umbilical infections occurring between 6 and 20 d of age; Umb3 = umbilical infections occurring between 21 and 30 d of age.

[2]Diar1 = diarrhea occurring between 0 and 5 d of age; Diar 2 = diarrhea occurring between 6 and 20 d of age; Diar3 = diarrhea occurring between 21 and 30 d of age.

Table 2. Definition and distribution of calving ease and neonatal vitality scores

	Calving ease			Neonatal vitality		
Score	Description	No.	Incidence, %	Description	No.	Incidence, %
1	No assistance	1,567	57.4	Very vigorous	1,509	62.3
2	Easy pull	827	30.3	Vigorous calf	780	32.2
3	Hard pull	218	8.0	Weak calf	98	4.0
4	Caesarian	118	4.3	Assisted calf	36	1.5

birth and 1 mo of age (Table 3). All observations were recorded directly by the breeders. For each health event, the breeder was asked to record the date of occurrence, the suspected disease, and the treatment administered to the calf. To be considered as a health event, treatment was not preventive but only given in response to calf illness.

Trait Definitions

CE conditions were evaluated using a score ranging from 1 (calving without assistance) to 4 (caesarean section) with intermediate values of 2 and 3 corresponding to calving with easy pull and calving with mechanical assistance, respectively. NV was estimated during the first 4 h after birth with a score ranging from 1 (very vigorous calf standing, walking and reaching for the udder within the first hour after birth) to 4 (assisted calf who requires help to stand and reach the udder) with intermediate scores of 2 (vigorous calf reaching the udder within 1 and 3 h of birth) and 3 (weak calf needing more than 4 h to reach the udder). Survival (**Surv**) was defined as a binary trait (0 = dead; 1 = alive), based on whether or not the calf was still alive at 30 d of age. Survival records were removed from the analysis if the calves were twins, born from an abortion or did not have a CE score.

Health events occurring at different time points were defined as binary disease traits (0 = no disease event; 1 = at least one disease event during the period in question) based on whether or not the calf had at least one health event recorded within the considered period. The time periods were defined in such a way as to account for existing knowledge on the different age-related causes of infections that cause diarrhea (Gruenberg, 2016). For example, infection by *Escherichia coli* is seen within the first 5 d of life, rarely later. Infection by rotavirus, coronavirus, and other viruses is frequently observed in calves that are 5–15 d old. Cryptosporidiosis is seen in calves older than 5 d but most commonly during the second and third weeks of life, while coccidiosis only appears after 3 wk of age (Gillhuber et al., 2014). Thus, the health events considered for the genetic analysis were umbilical infections occurring between 0 and 5 d of age (**Umb1**) or between 6 and 20 d of age (**Umb2**), and diarrhea occurring between 0 and 5 d of age (**Diar1**), between 6 and 20 d of age (**Diar2**), or between 21 and 30 d of age (**Diar3**). The frequency of navel illness between 21 and 30 d of age (**Umb3**) was too low (Table 1) to estimate genetic parameters; this was also the case for other diseases (including respiratory diseases) recorded in the 2 yr of data that we examined (Table 3).

The incidence risk of diseases was calculated as the number of calves treated for the first time for a disease before 1 mo of age divided by the number of calves enrolled in the 24-mo period of the study. The incidence risk of disease within a given age category (birth to 5 d, 6 to 20 d, and 21 to 30 d) reflected the number of calves treated for the first time during that age interval divided by the number of calves eligible for treatment.

Statistical Analyses

Theoretically, the discrete nature of health traits should be taken into account in genetic

Table 3. Number of cases and incidence of health events between birth and 30 d of age according to the ranking of health events

	First event		Second event		Third event		Fourth event		Fifth event	
Item	No.	Inc[1], %	No.	Inc[1], %	No.	Inc[1], %	No.	Inc[1], %	No.	Inc[1], %
Number of calves at ith event	793	28.9	204	25.7	42	20.6	11	26.2	2	18.2
With diarrhea	418	15.3	98	12.4	22	10.8	3	7.1	-	-
With umbilical infection	143	5.2	42	5.3	6	2.9	1	2.4	-	-
With respiratory disease	57	2.1	15	1.9	3	1.5	-	-	1	9.1
With other diseases	175	6.4	49	6.2	11	5.4	7	16.7	1	9.1
Total number of calves	2,740		793		204		42		11	

[1]Inc = Incidence (percentage of calves affected out of the total number of calves enrolled in the study).

evaluation by applying a threshold model (Gianola and Foulley, 1983). However, when dealing with small herds, or if some scores are absent within certain herd-years, the herd-year effects in threshold models cannot be estimated as fixed effects; instead, they are considered to be random. This type of statistical treatment of herd-year effects is incorrect from a theoretical point of view as it creates errors in the ranking of animals based on their estimated breeding values (Phocas and Laloë, 2003). Because of this, the discrete nature of most of the traits under analysis was ignored, as a linear model can perform as well as, or better than, a threshold model when the amount of information per level of effect is low. This is especially true when confounding effects are suspected between the herd genetic level and herd management performance due to moderate genetic links between beef cattle herds (Phocas and Laloë, 2003). In addition, estimates of genetic correlations are not affected by the statistical treatment (linear or threshold model) of the categorical trait (Kadarmideen et al., 2003). The following mixed linear animal model was therefore considered for each trait:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{Qm} + \mathbf{Wc} + \mathbf{e}$$

where **y** is the vector of observations, **b** is a vector of fixed effects, and **u**, **m**, **c**, and **e** are random vectors representing direct genetic breeding value, maternal genetic breeding value, common maternal environmental effects, and residual effects, respectively. **X**, **Z**, **Q**, and **W** are the corresponding incidence matrices.

For all traits, the **b** vector considered the fixed effects of the contemporary group and age of the dam. Depending on the trait, sex and twinning effects were also fitted in the models. A sex effect was significant with respect to calf BW, CE, NV, umbilical infection and survival at 30 d. A twinning effect was only significant for NV, BW and CE. The effect of vaccination of the dam against diarrhea could not be tested because it was completely confounded with the effect of contemporary groups given that all or none of the groupmates were vaccinated altogether. The 62 contemporary groups were defined as all calves born in a given herd during a given season of a given year. For each birth year, three birth seasons were considered: from August to October, from November to January and from February to May. No calves were born in June or July. Contemporary group sizes ranged from 5 to 125 calves. A variable proportion of contemporary groups were not informative (all values equal to 1 or 0 within group) for the analysis of binary traits (ranging from 40% for Surv to 70% for Diar3). There were 10 classes of dam age: classes 1 and 2 corresponded to primiparous dams aged less than 3 yr old and more than 3 yr old at calving, respectively. Classes 3, 4, and 5 corresponded to second parity cows whose ages were 3, 4, and 5 yr, respectively. Classes 6, 7, and 8 corresponded to third parity cows aged 4, 5, and 6 yr, while classes 9, 10, and 11 corresponded to fourth and more parity cows aged 5, 6, and more than 6 yr old.

(Co)variance component estimations were run using ASREML software while applying an Average Information REML algorithm (Gilmour et al., 1995, 2009). Analyses were performed using a BLUP multitrait animal model. A preliminary univariate analysis was performed to select the best genetic model for each of the nine traits and obtain univariate estimates of genetic parameters. Because of the small size of the dataset and the strong genetic correlations between some of the nine traits, it was not possible to run a single nine-trait analysis; it was very difficult to achieve convergence of the REML log-likelihood in analyses containing more than five traits. As a result, 10 different five-trait analyses were performed in order to obtain at least 2 different estimates of any of the two-by-two correlations between traits. Some correlations were fixed at the boundary value of 0.999 or −0.999 by the software in order to maximize the likelihood of the data. In these cases, bi-variate analyses were performed to check that the results were unchanged and that corresponding correlations were at the boundaries of the parameter space.

The results for a given parameter (and its standard error) were averaged over all the analyses performed on the corresponding trait. Estimates of correlations between direct and maternal effects (within or across traits) are always highly sensitive to any difference in data structure design, and it is difficult to distinguish a biased estimate from a true biological correlation between direct and maternal effects (Robinson, 1996a,b). For this reason, we did not attempt to estimate these correlations using our small dataset.

RESULTS AND DISCUSSION

Phenotypic Means

The phenotypic mean and raw standard deviation are shown in Table 1 for the 10 analyzed traits. With a rate of slightly over 96%, our results regarding calf survival at 30 d were clearly higher than the national average (93%) for Charolais calves

recorded between 2005 and 2011 (Leclerc et al., 2016). In the 16 experimental herds, the mortality rate between birth and 30 d was within the range of the national value for perinatal mortality among Charolais calves. In France as a whole (Leclerc et al., 2016), 3.8% of Charolais calves die within the first 48 h, while in the experimental herds this rate was only 1.5%. The survival of male calves was 2 percentage points below that of females (95% vs. 97%; Fig. 1). This sex difference in survival is partly due to birth difficulties linked to weight, as males weigh an average of 3 kg more at birth. Average BW values were similar to those recorded at a national scale in Charolais herds (Guerrier and Leudet, 2015). The distributions of CE and NV scores in our dataset are shown in Table 2. Nearly 88% of calves were born without any assistance or only an easy pull (CE < 3) and 84.5% were vigorous at birth (NV < 3). In the experimental herds, calving events not requiring assistance were 11 points below the rate of 68% observed at a national scale (Guerrier and Leudet, 2015), while the proportion of caesarians was close to the national average (4%).

No health events were recorded for 1,947 of the 2,740 calves in the analyzed dataset, while 1,052 health events were recorded among the remaining 793 calves. In the current study, the total incidence risk of umbilical infections during the first month of life was about 6%, which is consistent with recent observations of U.S. Holstein calves (Henderson et al., 2011). This risk was higher than that reported (1.3%) for Swedish dairy heifers at 3 mo of age (Svensson et al., 2003), but significantly lower than previous results reported by Virtala et al. (1996) in U.S. Holstein heifer calves (27% during the first month of life, including a 13% risk of umbilical hernia). The average age of occurrence was 7 d, with approximately 1.14 infections declared for each of the 169 calves in which umbilical disease was reported (Fig. 2). Most umbilical infections were first declared within the first 3 wk of life; only 0.4% of umbilical infections were reported between 21 and 30 d. Sivula et al. (1996) had shown that the risk of enteritis was highest during the first 3 wk of life in Holstein heifers, while that of pneumonia was highest at 10 wk of age. In our study, the incidence risk for respiratory disease was only 2.7% during the first month of life in Charolais calves (Table 3), whereas Mahmoud et al. (2017) reported a 28% risk between birth and 2 mo of age in Holstein calves. The frequency of umbilical infections between 21 and 30 d of age (Umb3) was too low (Table 1) to estimate genetic parameters; this was also the case for other diseases (including respiratory diseases) recorded in the 2 yr of data that we examined (Table 3).

In the current study, about 17% of calves experienced at least one diarrheic event, with most occurring between 6 and 20 d of age (Table 1). The average age of onset was around 10 d (Fig. 2), with an average of 1.19 events among the 456 calves in which diarrhea was reported. As for umbilical infections, a wide range of values has been reported in the literature regarding the incidence risk of diarrhea during the first 3 mo of life in Holstein calves: from 10% (Svensson et al., 2003) to 29% (Virtala et al., 1996).

According to Sivula et al. (1996) and the USDA (2010), the majority of deaths among preweaned Holstein heifers can be attributed to diarrhea and other digestive problems, followed by respiratory problems. Calf diarrhea can be caused by both infectious agents and non-infectious factors (such as the housing environment, an inadequate intake of colostrum and

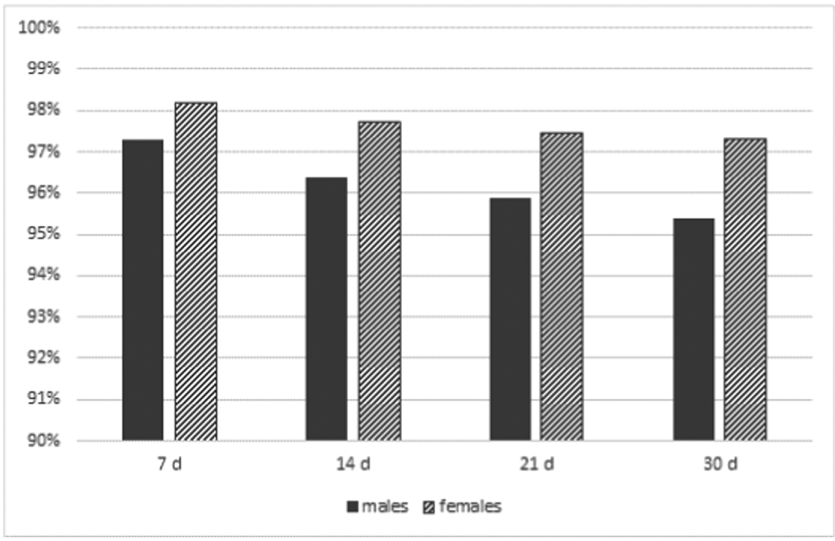

Figure 1. Survival at 7, 14, 21, and 30 d of age as a function of calf sex.

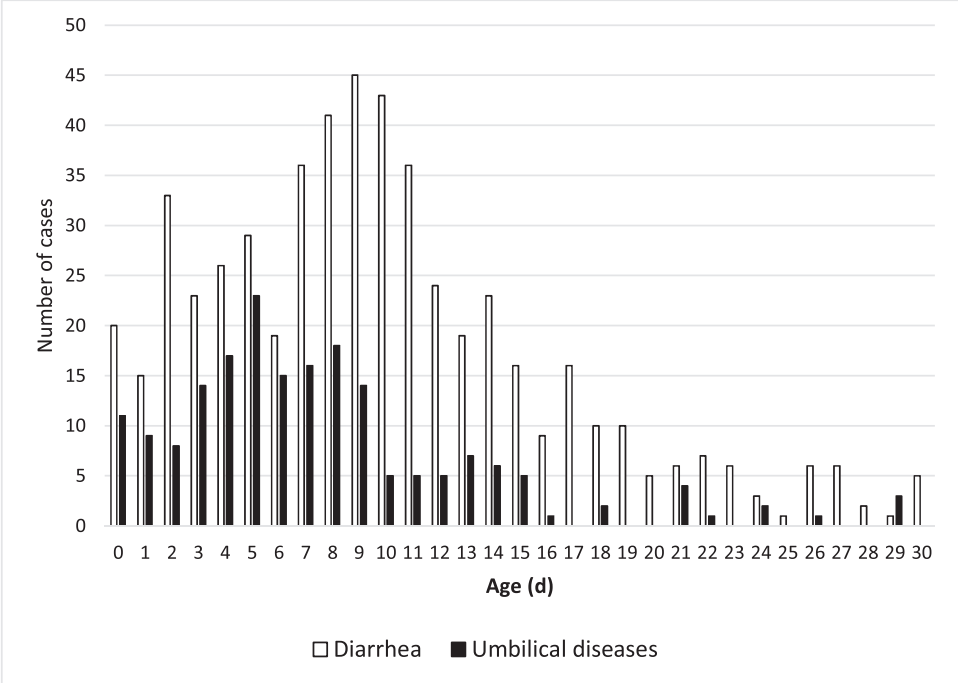

Figure 2. Distribution of calf age (d) at the onset of diarrhea or umbilical disease event.

waste milk). Multiple enteric pathogens are involved in the development of this disease and coinfection is frequently observed in diarrheic calves. Since the commercial availability of vaccines against *Escherichia coli K99*, rotavirus and coronavirus (Snodgrass et al., 1982; Crouch et al., 2000), *Cryptosporidium* has emerged as the principal diarrheic agent in young calves (e.g., de Graaf et al., 1999). This protozoan parasite is the etiological agent of cryptosporidiosis, which is one of the major causes worldwide of moderate-to-severe diarrhea in both humans and livestock. Cattle have been considered to be the primary reservoir for oocysts of zoonotic *Cryptosporidium parvum*, which is one of two species of *Cryptosporidium* that have been reported as being responsible for most human infections (Ryan et al., 2014). No drug therapy is yet available and the high resistance of oocysts to a range of environmental conditions and chemical treatments make cryptosporidiosis difficult to control (e.g., de Graaf et al., 1999). In France, data suggest that *C. parvum* is the main infectious agent behind neonatal diarrhea in both beef and dairy calves (Naciri et al., 1999). Improving the resistance of cattle to *C. parvum* would therefore be beneficial not only to cattle and their breeders, but also to all humans.

Univariate Genetic Analysis of Traits

A univariate analysis was performed to select the best genetic model for each of the nine traits and to obtain univariate estimates of genetic parameters (Table 4). A preliminary study (results not presented) demonstrated that the estimate of genetic variance of any of the two disease traits (diarrhea or umbilical infection) was markedly decreased when events between 0 and 30 d were considered as a single trait. The three time periods (0 to 5 d; 6 to 20 d; 21 to 30 d) were therefore defined in such a way as to account for existing knowledge on the different age-related causes of diarrhea, but also were consistent with time points allowing to detect the most important genetic variations in the expression of the disease traits. Moving only of a few days any time points led also to less consistent results in the partitioning of direct and maternal genetic variances across the different time periods. These preliminary results suggested that calves answered to different causes of infections through various biological pathways.

In addition to the log-likelihood values of the selected models, log-likelihood values of the full models accounting for common maternal environmental effects, direct and maternal genetic effects, and log-likelihood values of a model taking no account of maternal genetic effects are presented in Table 4 in order to validate the relevance of the random effects selected for each trait. As demonstrated by Clément et al. (2001), a reduced model (with one or more existing effects omitted, such as the maternal genetic effect) caused variable bias of the true value, arising from confusion between different variance components. By contrast, fitting unnecessary random effects yielded neither biased estimates (genetic parameters relative to these effects being

Table 4. Log-likelihood values of full and reduced models for the choice of random effects and univariate estimates of direct (h^2d) and maternal (h^2m) heritability, proportion (c^2) of phenotypic variance (σ_P^2) due to common maternal environmental effects under the selected model (standard errors are in brackets)

	LLamc[3]	LLac[3]	LLsel[3]	h^2d	h^2m	c^2	σ_P^2
Survival	2,565.75	2,564.35	2,565.75	0.023 (0.027)	0.070 (0.042)	0.088 (0.060)	0.035 (0.001)
Birth weight	398.48	396.64	398.48	0.279 (0.063)	0.063 (0.038)	0.168 (0.046)	26.396 (0.817)
Calving ease	−671.19	−671.24	−671.19	0.129 (0.041)	-	0.113 (0.041)	0.560 (0.016)
Neonatal vitality	152.68	152.68	152.68	0.073 (0.035)	-	0.168 (0.040)	0.292 (0.009)
Umb1[1]	3,341.75	3,341.57	3,341.75	0.071 (0.038)	0.017 (0.026)	-	0.027 (0.001)
Umb2[1]	3,106.87	3,104.36	3,106.87	-	0.082 (0.029)	-	0.032 (0.001)
Diar1[2]	2,750.90	2,749.63	2,750.90	-	0.044 (0.030)	0.046 (0.048)	0.042 (0.001)
Diar2[2]	1,955.20	1,955.09	1,955.20	0.016 (0.022)	0.012 (0.026)	0.100 (0.045)	0.077 (0.002)
Diar3[2]	4,111.04	4,111.04	4,111.04	0.016 (0.028)	-	0.041 (0.040)	0.015 (0.0004)

[1]Umb1 = umbilical infection occurring between 0 and 5 d of age; Umb2 = umbilical infection occurring between 6 and 20 d of age.

[2]Diar1 = diarrhea occurring between 0 and 5 d of age; Diar 2 = diarrhea occurring between 6 and 20 d of age; Diar3 = diarrhea occurring between 21 and 30 d of age.

[3]LLamc = log-likelihood of the full model including direct (a) and maternal (m) genetic effects and common environmental effects (c); LLac = log-likelihood of a model including only direct (a) genetic effects and common environmental effects (c); LLsel = log likelihood of the selected model depending on the trait.

either equal to zero or nonestimable) nor substantial losses affecting the accuracy of the estimates. We therefore considered any random effect that might explain at least 1% of phenotypic variance (even with a large standard error) in the most parsimonious model with a log-likelihood very close to that of the full model. Our results showed that maternal genetic variance was null for all disease traits recorded during the period between 21 and 30 d. Nor was maternal genetic variance significant (less than 1% of phenotypic variance) for CE and NV in our dataset. As a result, the maternal genetic component was not considered for CE, NV and Diar3 in the final multitrait analysis. Our preliminary univariate analysis also showed that no common maternal environmental variance existed for any umbilical disease trait. This effect was not therefore considered in the model of resistance to umbilical infection. Furthermore, no direct genetic effect was found for Diar1 and Umb2.

We verified that direct and maternal heritability values, as well as the ratio between common maternal environmental variance and phenotypic variance, were consistent between univariate and multitrait analyses for each of the nine traits.

Proportion of Phenotypic Variances Explained by Direct Genetic Effects and Maternal Genetic Effects Versus Common Environmental Effects

The direct heritability values for BW and CE (Table 5) were low to moderate, with estimates within the range of most values that have previously been reported in different beef cattle breeds (Varona et al., 1999; Phocas and Laloë, 2004). In particular, a comparison with previous results from a large French Charolais dataset (Phocas and Laloë, 2004) revealed that the values for direct heritability were quite similar in both cases. However, our estimates of maternal heritability different markedly from published results: while the 2004 analysis of Charolais cattle obtained estimates of 12% and 3% for CE and BW, respectively, in the present study we detected no maternal heritability for CE and a value close to 6% for BW. These differences could be explained by the limited number of years covered by the records in the current dataset and by our assumption of a genetic independence of direct and maternal genetic effects for all traits. The previous study had indeed estimated the correlation between direct and maternal genetic effects to be around −0.4 (Phocas and Laloë, 2004).

We did not find any maternal genetic variance regarding NV (Table 5), while the maternal common environmental variance component (Table 6) was quite a bit higher (16.6% of phenotypic variance) than direct heritability (7.8%). As far as we know, the only existing estimates of genetic parameters for calf vigor have been based on a binary trait (vigorous/weak calf during the first 24 h) in Brahman (Riley et al., 2004) and Nelore (Schmidek et al., 2013) cattle. Although we found a value for direct heritability (8% vs. 9%) which was very similar to that determined by Riley et al. (2004), their study also showed a significant maternal heritability of 10%, which differs substantially from our current estimate. Schmidek et al. (2013) also detected significant maternal heritability (8%), but very low direct heritability (1%).

Table 5. Genetic parameters for the nine calf health traits: direct and maternal (in italics) heritability on the first and second lines of the diagonal, direct genetic correlations in the upper triangle and maternal genetic correlations in the lower triangle (standard errors are in brackets)

	Surv[1]	BW[2]	CE[3]	NV[4]	Umb1[5]	Umb2[5]	Diar1[6]	Diar2[6]	Diar3[6]
Surv[1]	**0.026 (0.03)** *0.096 (0.04)*	−0.34 (0.42)	−0.90 (0.48)	−0.53 (0.56)	−0.32 (0.52)	-	-	−0.71 (1.01)	0.999 (ne)
BW[2]	*−0.48 (0.37)*	**0.270 (0.06)** *0.057 (0.03)*	0.86 (0.09)	−0.05 (0.26)	0.13 (0.24)	-	-	−0.35 (0.50)	0.32 (0.45)
CE[3]	-	-	**0.147 (0.04)** *0 (ne)*	0.05 (0.26)	0.10 (0.26)	-	-	0.17 (0.54)	−0.04 (0.51)
NV[4]	-	-	-	**0.078 (0.04)** *0 (ne)*	0.27 (0.33)	-	-	0.999 (ne)	−0.73 (0.58)
Umb1[5]	*0.52 (0.50)*	*0.29 (0.54)*	-	-	**0.081 (0.04)** *0.019 (0.02)*	-	-	−0.07 (0.61)	0.94 (0.68)
Umb2[5]	*0.34 (0.27)*	*0.18 (0.32)*	-	-	*−0.68 (0.87)*	**0 (ne)** *0.079 (0.02)*	-	-	-
Diar1[6]	*0.48 (0.39)*	*−0.999 (ne)*	-	-	*0.49 (0.78)*	*−0.68 (0.32)*	**0 (ne)** *0.048 (0.03)*	-	-
Diar2[6]	*0.59 (0.53)*	*−0.31 (0.61)*	-	-	*−0.999 (ne)*	*−0.85 (0.49)*	*0.33 (0.52)*	**0.016 (0.02)** *0.024 (0.02)*	0.33 (0.94)
Diar3[6]	-	-	-	-	-	-	-	-	**0.020 (0.03)** *0 (ne)*

[1]Surv = survival at 30 d.

[2]BW = birth weight

[3]CE = calving ease score

[4]NV = neonatal vitality

[5]Umb1 = umbilical infections occurring between 0 and 5 d of age; Umb2 = umbilical infections occurring between 6 and 20 d of age.

[6]Diar1 = diarrhea occurring between 0 and 5 d of age; Diar 2 = diarrhea occurring between 6 and 20 d of age; Diar3 = diarrhea occurring between 21 and 30 d of age.

As for survival, our estimate of direct heritability was low (2.6%) but within the range of most values found in the literature (Fuerst-Waltl and Sorensen, 2010; Leclerc et al., 2016). Maternal genetic (Table 5) and common environmental (Table 6) effects were also found to affect survival at 30 d. To our knowledge, the only other estimate of maternal genetic variance in the literature was reported by Schmidek et al. (2013), who estimated similar values for both direct (5%) and maternal (3%) heritability regarding survival at 30 d. Instead, maternal genetic heritability (9.6%) was almost four times higher in our study than the direct heritability of early survival (Table 5). Regarding calf mortality until weaning, Bunter and Johnston (2014) estimated a negligible direct heritability in comparison to maternal heritability for calf mortality in tropically adapted beef breeds managed under extensive production systems.

Table 6. Proportion of phenotypic variance due to common maternal environmental effects (on the diagonal) and correlations between common environmental effects (above the diagonal)

	Surv[1]	BW[2]	CE[3]	NV[4]	Diar1[5]	Diar2[5]	Diar3[5]
Surv[1]	0.065 (0.06)	0.35 (0.34)	0.23 (0.39)	0.05 (0.33)	−0.85 (0.86)	0.03 (0.50)	−0.22 (0.64)
BW[2]		0.255 (0.04)	0.30 (0.16)	0.20 (0.15)	0.34 (0.28)	0.18 (0.20)	−0.17 (0.26)
CE[3]			0.117 (0.04)	−0.01 (0.22)	−0.38 (0.45)	−0.31 (0.25)	0.05 (0.42)
NV[4]				0.166 (0.04)	0.69 (0.53)	0.05 (0.22)	0.42 (0.39)
Diar1[5]					0.044 (0.04)	0.999 (ne)	−0.79 (0.86)
Diar2[5]						0.087 (0.04)	−0.16 (0.48)
Diar3[5]							0.043 (0.04)

[1]Surv = survival at 30 d.

[2]BW = birth weight

[3]CE = calving ease conditions

[4]NV = neonatal vitality

[5]Diar1 = diarrhea occurring between 0 and 5 d of age; Diar 2 = diarrhea occurring between 6 and 20 d of age; Diar3 = diarrhea occurring between 21 and 30 d of age.

One of the principal diseases that affect calves in both dairy and beef herds is bovine respiratory disease (BRD). Unfortunately, the recording period for our study was not sufficiently long after calf birth to obtain a sufficient number of cases that would enable a genetic analysis of BRD in Charolais cattle. To date, only five studies have attempted to quantify the genetics of susceptibility to BRD at a relatively large scale in beef (Snowder et al., 2005; Schneider et al., 2010) or dairy populations (Heringstad et al., 2008; Berry et al., 2014; Mahmoud et al., 2017). Estimates of direct heritability for BRD in preweaned beef calves ranged from 0.11 (Schneider et al., 2010) to 0.22 (Snowder et al., 2005). Because of the magnitude of this variation, we encourage breeders to continue recording calf health events until weaning.

Genetic effects influencing resistance to diarrhea seemed to be markedly dependent on calf age; only maternal genetic effects were detected for diarrhea within the first 5 d of life (Diar1), while only direct genetic effects were detected in the case of later-onset diarrhea (Diar3). A mixture of the two types of genetic effects—direct and maternal—was observed for diarrhea declared in the intermediate age range (Diar2). For direct genetic effects, heritability ranged from 0 for early diarrhea to 2% for late diarrhea, with standard errors as large as the estimates (Table 5). Conversely, the heritability of maternal genetic effects ranged from 4.8% for early to 0% for late diarrhea (Table 5). Significantly, higher proportions of phenotypic variance for diarrhea were explained by maternal common environmental effects (Table 6) rather than any genetic effects (Table 5). This was especially true for the Diar2 trait, for which the maternal common environment, maternal genetic and direct genetic effects explained 8.7%, 2.4%, and 1.6% of phenotypic variance, respectively. A newborn beef calf only receives maternal antibodies and cell-mediated immunity via passive transfer by consuming colostrum. The degree to which maternal genetic and common environmental effects affect calf resistance to early and intermediate age diarrhea is therefore closely related to the consumption by calves of high-quality colostrum in sufficient quantities (Cho and Yoon, 2014).

When considering resistance to umbilical infections (Table 5), the genetic effects involved in early umbilical infection (Umb1) were mainly direct (8.1%), whereas those involved in later umbilical infections (Umb2) were exclusively maternal effects (7.9%). Such a result is unusual because maternal effects usually have more impact on performance at a younger age. No maternal common environmental variance was detected for either Umb1 or Umb2.

As far as we know, our study is the first to have estimated genetic parameters for calf diarrhea and umbilical infections in beef calves, as well as the first to have generated estimates of maternal genetic and common environmental variances for diseases affecting both beef and dairy calves. This first report on maternal parameters should be considered as preliminary results because our dataset lacked multigenerational data that would have enabled an accurate separation of direct and maternal effects.

To date, only a few studies have estimated direct genetic parameters for diseases in the calves of dairy or dual-purpose breeds, and most of these estimates focused on BRD. For example, Henderson et al. (2011) estimated the heritability of umbilical (14%) and respiratory (9%) diseases in Holstein heifers, and more recently, Mahmoud et al. (2017) estimated the heritability of diarrhea (6%) and respiratory diseases (7%) in a large-scale study of German Holstein calves monitored from birth to 2 mo of age. Our results revealed the importance of accurately describing both diarrhea and umbilical infections (in terms of age ranges) and accounting for both maternal genetic and environmental effects when modeling disease traits in order to define relevant biological and genetic evaluations of calf health performance.

Correlations Between Calf Traits

Despite the high standard errors for many of the correlation estimates (Table 5), they were very consistent between different multitrait models (less than 0.2 point of variation). The only exceptions were the maternal genetic correlation between BW and Umb1 (which ranged from 0 to 0.6 depending on the analysis) and the common maternal environmental correlations (Table 6) between BW and CE (ranging from 0.1 to 0.5) or BW and Surv (between 0.2 and 0.6). Most residual environmental correlations were close to zero, with SE ranging from 0.02 to 0.06 for estimates below 0.08 in absolute values (results not shown). The only exceptions were residual environmental correlations between Umb1 and Umb2 (0.13; SE = 0.027), CE and NV (0.20; SE = 0.045), and CE and BW (0.22; SE = 0.055), corresponding to low phenotypic correlations of 0.11, 0.14, and 0.21, respectively. All other phenotypic correlations were lower in absolute values (results not shown).

Correlations Between Surv and BW, CE, and NV Birth Traits

As evidenced by the moderate degree of favorable environmental correlation between CE and NV, we were able to confirm that Charolais calves assisted at birth were less vigorous than those which were unassisted; this was consistent with the phenotypic trends previously observed in Brahman (Riley et al., 2004) and Holstein (Barrier et al., 2012) calves. However, our results indicated that this trend could only be due to environmental factors, since no significant common maternal environmental (Table 6) or direct genetic (Table 5) correlations were detected between these traits. We also estimated correlation values between direct genetic effects and between environmental (both common maternal and residual) effects for CE and BW, as has generally been reported in the literature. However, our estimate of the direct genetic correlation between BW and CE (Table 5) was higher (0.86) than the previous estimate (0.66) published for French Charolais cattle (Phocas and Laloë, 2004). The common maternal environmental correlation between NV and Surv was null (Table 6). By contrast, the favorable direct genetic correlation between NV and Surv indicated that calves which were more vigorous at birth had a higher probability of surviving to 1 mo of age (Table 5). This result supported previous phenotypic results obtained in Brahman cattle, where 39% of the calves that did not survive until weaning (excluding stillbirths) displayed weak NV (Riley et al., 2004). Therefore, early calf death could potentially be reduced by means of genetic improvement targeting NV rather than survival, as the former trait has a significantly higher heritability. The strong direct correlation between Surv and CE found during our study (Table 5) was consistent with the phenotypic results obtained by Lombard et al. (2007), who showed that dystocia led not only to a rise in stillborn dairy calves but also an increase in the risk of subsequent mortality up to 30 d.

Correlations Between the Umb1, Umb2, Diar1, Diar2, and Diar3 Disease Traits

When we considered resistance to umbilical infections, we found that the correlation between maternal genetic effects involved in early (Umb1) and later (Umb2) umbilical infections was clearly negative (−0.68), indicating that maternal genetics may have opposing effects on disease resistance in the two age ranges. However, this antagonism needs to be further confirmed on a larger scale. Future studies should also examine the correlation between the direct genetic effects for Umb1 and maternal genetic effects for Umb2 in order to obtain a clearer understanding of the genetic determinism of umbilical infections. The bacterial agents most commonly encountered are not specific to umbilical infection; rather they are micro-organisms that cause suppurative infections, whose presence is generally a sign of the microbial environment or poor local hygienic conditions. As well as causing local infection and inflammation, these bacteria can spread by via the blood into joints, the liver or other organs, causing severe complications and impairing the calf growth (Virtala et al., 1996) or longevity in the longer term (Britney et al., 1984). Unfortunately, we lacked sufficient information to interpret our results regarding umbilical infections in greater detail.

Regarding susceptibility to diarrhea, the estimated correlation between the maternal genetic effects of Diar1 and Diar2 was moderately positive (0.33), as was the correlation between the direct genetic effects of Diar2 and Diar3 (0.33; Table 5). Common maternal environmental effects for Diar1 and Diar2 were almost completely positively linked (Table 6), whereas common maternal environmental effects for Diar1 and Diar3 were strongly negatively correlated (−0.79) and those of Diar2 and Diar3 were almost uncorrelated.

Calf diarrhea is a complex and multifactorial disease. Environmental factors associated with the occurrence of calf diarrhea are linked to peripartum calving management and feeding, calf immunity, herd size, and environmental stress or contamination (Cho and Yoon, 2014). *Escherichia coli K99*, rotavirus, coronavirus, and *Cryptosporidium* are the most important enteropathogens associated with diarrhea in calves younger than 1 mo (e.g., Gillhuber et al., 2014). Differentiating these bacterial, viral and parasitic agents is only possible using a diagnostic test, not by clinical examination. Coinfection is frequently observed in diarrheic calves, although a single primary pathogen may be the cause in some cases. Large-scale testing in the field is too expensive, so it is important to base preventive measures and the control of calf diarrhea on a thorough understanding of the complexities of the disease (Cho and Yoon, 2014). Because genetic resistance to diarrhea at early, intermediate and later ages seems to be moderately, but favorably, correlated, selective breeding may constitute an effective means of preventing some diarrhea outbreaks. In particular, breeding efforts that take advantage of the strong maternal heritability of

early diarrhea resistance may be sufficient to yield a significant response, and indirect selection criteria need to be proposed in order to improve the direct genetic resistance of calves against diarrhea at later stages.

The direct genetic effects underlying the resistance of calves to early umbilical infection (Umb1) were strongly correlated (0.94), with the direct effects linked with resistance to late diarrhea (Diar3), while the association with Diar2 was close to zero. Likewise, the maternal genetic effects associated with the resistance of calves to early umbilical infections were also favorably, but moderately, correlated (0.49) with maternal genetic resistance to early diarrhea. By contrast, a strong negative correlation (tending towards −1) was estimated between maternal genetic resistance to Umb1 and Diar2. In addition, strongly unfavorable maternal genetic correlations were estimated between Umb2 and both Diar1 (−0.68) and Diar2 (−0.85). These results suggest that selection to improve maternal genetic resistance to late umbilical infection may adversely affect resistance to diarrhea, as well as maternal genetic resistance to early umbilical infection. Until these results can be corroborated or disputed using a larger dataset, we would not recommend the use of Umb2 as a selection criterion for the improvement of general calf health. Instead, our data suggest that selection on both maternal and direct genetic resistance to early umbilical infection (Umb1) could potentially improve resistance to early umbilical infection and early and late diarrhea.

Correlations Between Birth Traits or Survival and Disease Traits

BW and CE were not clearly associated with any of the five disease traits ($|r| < 0.4$), regardless of the genetic (direct or maternal) or environmental (residual or maternal common) effects considered. The only exception to this concerned the maternal genetic correlation between BW and Diar1, which tended towards −1 (Table 5), meaning that the maternal genetic effects which increased BW had a favorable effect on reducing the occurrence of early diarrhea. Similarly, McCorquodale et al. (2013) showed that Holstein heifer calves with lower BW were more susceptible to illness during the first 3 wk of life. However, because BW and Surv were unfavorably linked during our study in terms of both direct and maternal genetic effects (Table 5), BW does not seem to represent a useful selection criterion to improve survival or any calf disease trait.

As a general rule, survival during the first month of life is strongly dependent on both dystocia and the onset of diarrhea. Diarrhea may be fatal by causing dehydration and acidosis that could result in anorexia and ataxia of the calves (e.g., Cho and Yoon, 2014). In 3-mo-old Holstein heifer calves, Sivula et al. (1996) estimated case-fatality risks at approximately 18%. Dystocia increases susceptibility to environmental pathogens that may cause calf diarrhea (Larson and Tyler, 2005). Here, we found conflicting results depending on the type of effects (direct genetic, maternal genetic, or common environmental) and the age range affected by the diarrhea outbreak. Maternal common environmental effects for Surv or NV and Diar1 (and to a lesser extent Diar3) were clearly favorably associated, while maternal common environmental effects for Surv and NV appeared to be uncorrelated (Table 6). Instead, maternal genetic effects for Surv were unfavorably linked to those improving resistance in any of the three diarrhea traits (Table 5). In terms of direct genetic effects, correlation estimates demonstrated opposing trends depending on the age range for the diarrhea outbreak: the direct genetic potential for survival revealed a largely favorable genetic association with direct genetic resistance to Diar2, whereas it exhibited a strongly unfavorable association with direct genetic resistance to Diar3 (Table 5). Because estimates of direct heritability for Diar2 and Diar3 were very low (1.6% and 2%, respectively), with large standard errors (2% and 3%, respectively), we strongly question the observation that a higher genetic susceptibility to late diarrhea seemed to be associated with a better direct potential for calf survival. However, correlation estimates between direct genetic effects for NV (a trait with significant direct heritability) and Diar2 versus Diar3 displayed the same trends as those for Surv and Diar2 versus Diar3. Such counterintuitive results need to be further investigated in a study at a larger scale. In their 2013 study, McCorquodale et al. estimated that the direct heritability for treatment due to illness during the first 8 d of life was 7%, with a negative genetic correlation (−0.4; SE = 0.5) with survival at 4 mo of age, indicating that calves with a higher genetic susceptibility to early disease had a lower genetic survival potential. In our study, lower direct genetic survival was not only associated with lower genetic susceptibility to Diar2 but also, to a lesser extent, with lower genetic susceptibility to Umb1 (Table 5). Conversely, maternal genetic effects that improved calf survival were unfavorably linked to the maternal genetic effects involved in any disease resistance

traits, with correlation estimates ranging from 0.34 (SE = 0.27) for Umb2 to 0.59 (SE = 0.53) for Diar2 (Table 5). These results were counterintuitive and require further investigation and validation in the context of large-scale studies.

CONCLUSION

Disease prevention in livestock can yield a range of benefits—for consumers, by improving animal welfare, and by increasing productivity for farmers. Our study revealed significant genetic variability in beef cattle for the following calf health characteristics: NV, survival at 1 mo of age, early diarrhea, and umbilical infections at early and later ages. In addition, maternal genetic effects were clearly more important than direct genetic effects in explaining survival, early diarrhea and umbilical infections between 6 and 20 d of age. These initial results will need to be confirmed by future large-scale studies of beef calf health genetics. In the current era of genomics, it is now possible to perform direct genomic selection for disease resistance in calves because of the degree of genetic variation present in calf health traits if an appropriately managed reference population to evaluate several tens of thousands of phenotyped and genotyped calves is available. However, not all health traits can easily be improved simultaneously, so Charolais breeders are now faced with the major task of developing prioritized breeding objectives. For instance, selection to improve maternal genetic resistance to one type of calf infection may adversely affect resistance to other types. In addition, breeding goals should consider health traits not only directly through the use of health phenotypes, but also indirectly by using correlated traits such as calf survival or NV. Last but not least, breeders must be aware of the potential for antagonistic genetic correlations between animal health and production traits. There is a current lack of large-scale studies in cattle—particularly beef cattle—in which precise estimates of genetic correlations between all performance traits can be determined. Such efforts will be necessary in the future in order to implement efficient breeding schemes which can improve both production and health traits.

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