Assessment of genetic variability of resistance to gastrointestinal nematode parasites in Creole goats in the humid tropics

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ABSTRACT: Goat production is widespread in the tropics. Goats are very susceptible to gastrointestinal nematode infection, but there is less evidence of their genetic resistance. Genetic resistance of Creole goats to gastrointestinal nematodes has been studied at Guadeloupe in the French West Indies since 1995. The objective of this research was to investigate genetic variation for resistance to gastrointestinal nematode infection, in order to introduce this trait into breeding schemes. Genetic variability was assessed within a Creole experimental flock. Forty-nine sire groups were characterized at weaning and 55 during fattening after weaning. Kids were naturally infected, mainly by Haemonchus contortus and Trichostrongylus colubriformis. Fecal egg counts were determined once at weaning and every 6 and 7 wk after drenching during fattening. Blood samples were collected every 7 wk during fattening for determination of packed cell volume. Live weights were recorded at weaning and at the beginning and middle of every infection period during fattening. Genetic parameters were estimated using the REML for multivariate animal models. The heritability estimate for transformed fecal egg count was 0.37 ± 0.06 at weaning. During fattening, it increased from 0.14 ± 0.05 at 4 mo to 0.33 ± 0.06 at 10 mo. Heritabilities of packed cell volume ranged from 0.10 to 0.33. At weaning, maternal heritability of fecal egg count reached 0.26 and direct heritability 0.20. After 6 mo of age, maternal effects were found to be unimportant for fecal egg count and packed cell volume. Live weights presented significant genetic variability. Genetic relationships between fecal egg counts and live weight in infected pastures were never significant. Genetic correlations between packed cell volume and live weight decreased from 0.47 to 0.10 from weaning to 10 mo of age. These results demonstrated the feasibility of breeding for improved resistance to nematodes in Creole kids.

Key Words: Genetic Variation, Goats, Maternal Effects, Resistance, Strongylidae, Weight

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Introduction

Meat goat production is widespread in the tropics. It is also gaining in popularity in developed countries (Zajac et al., 1999), where dairy and fiber production are also economically important. Goats are markedly susceptible to infection with gastrointestinal nematodes. However, the frequency of anthelmintic resistance is higher than in sheep, with whom they share the same nematode parasites (Waller, 1997). Integrated control of strongylosis in goats is required due to anthelmintic resistance and new consumer preferences. Genetic resistance can be integrated into control systems, as shown in sheep (Barger, 1993; Waller, 1993).

To our knowledge, few experiments have been conducted on intrabreed genetic resistance to infection by gastrointestinal nematodes in goats. Some investigators suggested that genetic variation exists in this species (Rohrer et al., 1991; Patterson et al., 1996). However, other authors obtained very low genetic parameters under tropical conditions (Woolaston et al., 1992; Baker, 1998). Mechanisms of resistance in goats are sometimes suggested to differ from those in sheep, due to higher susceptibility and later establishment of immunity against strongyles (Pomroy et al., 1986).
The aim of this research was to investigate the genetic variability in resistance to gastrointestinal nematode parasites of Creole goats, an efficient meat breed of Guadeloupe (French West Indies) characterized by Alexandre et al. (1999). Fecal egg counts and packed cell volume were used to indicate resistance. We also desired a better understanding of the genetic control of resistance of Creole kids from weaning to the end of fattening on pasture. In this respect, evolution of genetic parameters with regard to age and maternal effects were tested. The specific objective was to estimate parameters required for genetic evaluation in order to introduce resistance into a breeding scheme for genetic improvement of goats.

Materials and Methods

Flock Management

The study was undertaken in the Creole goat flock of INRA-Gardel, from 1995 to 1998. Three annual climatic seasons were characterized: a wet and hot season, a dry and fresh season, and an intermediate season. The flock produced on average a 200-kid cohort every 4 mo. It was organized in 12 families. An animal belongs to the family of its father. Four bucks were culled after each mating season (three per year), so that each buck mated during three seasons and genetic links were provided between cohorts. Matings were planned to keep inbreeding as low as possible in the flock. The flock grazed all year on irrigated Digitaria decumbens pastures managed in a rotation system (7 d in, 28 d out per paddock). This system ensured good-quality forage and control of paddock contamination (Mandonnet et al., 1997). Kids were weaned at an average age of 82 d. After weaning, males and females were grazed on separate paddocks and were naturally infected. Regular drenching controlled infections by Coccidia at weaning and by Moniezia during fattening. Cowdriosis was completely controlled by twice-monthly acaricide application. Some cases of pneumonia and footrot were diagnosed during the wet season. Affected kids were removed from the experiment.

Experimental Design

Kids born in the flock between 1995 and 1998 were used in this research, which began at weaning and continued to the end of fattening. Therefore, all the bucks and does used during this period were characterized for resistance to gastrointestinal nematodes through performance of their progeny. The animals were drenched 6 wk after the beginning of the kidding period and at weaning. Fecal samples were collected at weaning. During the postweaning or fattening period, kids were drenched every 8 wk with levamisole (12 mg/kg). Levamisole was effective (Barré et al., 1997) until the three latest cohorts, when worm populations became resistant (Aumont, unpublished data). Fecal samples were collected after 6 and 7 wk of each infection period. Blood samples were collected on each animal every 7th wk. Live weights were recorded at weaning, during fattening, at drenching, and in the middle of the infection period.

Analysis of Fecal and Blood Samples

Fecal egg counts were estimated using a modified McMaster method for rapid determination (Aumont et al., 1997). In addition fecal cultures were prepared to assess the composition of nematode burdens. Packed cell volume was measured by the capillary microhematocrit method.

Statistical Analysis

At weaning, 1,202 kids born of 49 sires and 271 does were available for analysis (Table 1). Thus, there were an average of 24.5 kids per sire and 4.4 kids per dam. During fattening, 979 kids sired from 55 bucks and 297 does, or an average of 17.8 kids per sire and 3.3 kids per dam, were involved in the experiment (Table 1). Not all kids that have data recorded during fattening also have data recorded at weaning, because data collection began later at weaning than at fattening. Due to mortality, fewer records were available at 10 mo of age (end of fattening) than at 4 mo of age (beginning of fattening).

As usually reported (Woolaston and Piper, 1996), the fecal egg count variable was transformed. The very skewed distribution of fecal egg count implied a fourth root transformation in order to normalize the variance. At 4, 6, 8, and 10 mo of age, analyses were performed using the mean of two fecal egg count measurements made 1 wk apart, because single measurements were...
found to have low repeatability in preliminary analyses. For fecal egg count at weaning and packed cell volume during fattening, the analyses were performed on single measurements. The fixed effects were tested using SAS software (SAS Inst. Inc., Cary, NC). Parity of the dam, combined effect of cohort-sex, and the combined effect of birth-rearing rank were added in the models when significant, along with any significant first-order interactions. Age or weight at weaning was included as a covariate when it was significant.

A sire model was tested on fecal egg count at 4, 6, 8, and 10 mo of age using SAS (SAS Inst. Inc.) software. At each age, the relationship between the P-value of the sire effect and infection level of the cohort-sex group was then presented in Figure 2. Regression coefficients and coefficients of determination were similarly estimated.

Variance and covariance components for genetic and residual effects were estimated by animal models using REML VCE package (Groeneveld, 1993) according to the following models:

(Model A) \[ y = Xb + Za + e \]

where

\[
E\left[ \begin{array}{c} a \\ e \end{array} \right] = \begin{bmatrix} 0 \\ 0 \end{bmatrix} \quad \text{and} \quad Var\left[ \begin{array}{c} a \\ e \end{array} \right] = \left[ \begin{array}{cc} G_0 \otimes A & 0 \\ 0 & R_0 \otimes I \end{array} \right]
\]

(Model B) \[ y = Xb + Z_1 a + W_1 i + Z_2 m + W_2 p + e \]

where

\[
\begin{bmatrix} a \\ m \\ i \\ p \\ e \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad Var\begin{bmatrix} a \\ m \\ i \\ p \\ e \end{bmatrix} = \begin{bmatrix} A_0^2 & A_0 & 0 & 0 & 0 \\ A_0 & A_0 & 0 & 0 & 0 \\ 0 & 0 & I_{\sigma_i^2} & 0 & 0 \\ 0 & 0 & 0 & I_{\sigma_p^2} & 0 \\ 0 & 0 & 0 & 0 & I_{\sigma_e^2} \end{bmatrix}
\]

(Model C) \[ y = Xb + Za + W_1 i + W_3 q + e \]

where

\[
\begin{bmatrix} a \\ i \\ q \\ e \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad Var\begin{bmatrix} a \\ i \\ q \\ e \end{bmatrix} = \begin{bmatrix} A_0^2 & 0 & 0 & 0 \\ 0 & I_{\sigma_i^2} & 0 & 0 \\ 0 & 0 & I_{\sigma_q^2} & 0 \\ 0 & 0 & 0 & I_{\sigma_e^2} \end{bmatrix}
\]

where \( y \) is the vector of observations, \( b \) is the vector of significant fixed effects, \( a \) is the vector of random direct additive genetic effects, \( m \) is the vector of random maternal additive genetic effects, \( p \) is the vector of random maternal environmental permanent effects, \( i \) is the vector of random individual environmental permanent effects, \( q \) is the vector of random residual effects, \( X, W_1, W_2, W_3, Z, Z_1, Z_2 \), and \( Z_3 \) are the incidence matrices connecting \( y \) to the effects in the model, \( G_0 \) is the additive genetic variance-covariance matrix between traits, \( R_0 \) is the residual variance-covariance matrix between traits, \( A \) is the relationship matrix among animals, and \( I \) is the identity matrix. The variances of the random effects \( a, m, i, p, q, \) and \( e \) are \( \sigma_a^2, \sigma_m^2, \sigma_i^2, \sigma_p^2, \sigma_q^2, \) and \( \sigma_e^2 \), respectively, and \( \sigma_{am} \) is the covariance between the random effects \( a \) and \( m \).

Preliminary univariate analyses were performed at each age and showed that maternal effects (genetic or environmental) did not significantly influence fecal egg count, packed cell volume, or live weight during fattening. A more basic model (Model A) was then fitted in multivariate analyses. Maternal effects at weaning were also dropped because no genetic correlation could be estimated with the other ages. Two multivariate analyses were performed on fecal egg count with Model A: one with records at weaning and at 4 and 6 mo of age and the other with the four traits of fattening. This data partitioning was chosen because few kids were recorded at weaning and at 8 or 10 mo of age. Therefore, no genetic correlation could be estimated among those variables.

Because genetic correlations were high among ages, we considered fecal egg count at 6, 8, and 10 mo and the four packed cell volume variables as repeated measures of the same traits, and we tested maternal component of variance with univariate analyses (Model B). We also tested maternal component of variance for fecal egg count at weaning with Model B, but without the individual environmental permanent effect (i). Note that the VCE package does not provide any standard errors of estimates when maternal effects are included in the model. Fecal egg count and packed cell volume repeatability parameters were estimated \textit{a posteriori} among measurement times with Model C, but without the \( q \) effect for packed cell volume.

\textbf{Results}

Fecal cultures indicated that kids excreted \textit{Haemonchus contortus}, \textit{Trichostrongylus colubriformis}, and \textit{Oesophagostomum colombianum} eggs. The proportions of these three species of nematodes varied during the course of the experiment (Figure 1). \textit{H. contortus} was proportionally the main species during the first 2 yr, and \textit{T. colubriformis} became predominant the last year.

The infection level in the flocks (each level of the cohort-sex effect) ranged between 106 eggs/g (back-transformed value) and 5,245 eggs/g. Figure 2 shows the relationship between the level of infection and the significance of the sire effect (probability of obtaining this effect under the null hypothesis) tested in each
cohort-sex group. Sire effects, and presumably additive genetic effects, became more significant as infection level increased. We standardized fecal egg count variables and verified that heterogeneity of variance had no impact on the estimates of genetic parameters. To be consistent with the other published results, non-standardized fecal egg count variables are shown here.

Estimates of heritability for each fecal egg count trait and phenotypic and genetic correlations among those traits obtained with Model A are shown in Table 2. At weaning, the heritability estimate was 0.37 ± 0.06. It increased during fattening from 0.14 ± 0.07 at 4 mo to 0.33 ± 0.06 at 10 mo of age. The genetic correlations were positive and moderate to high. The highest correlation coefficients among fecal egg counts were obtained between measures made at weaning, 4 mo, and 6 mo and also at 8 and 10 mo. Using Model C, the overall heritability of fecal egg count was 0.14 ± 0.03, the variance of the individual environmental permanent effect (i) was 0.13 ± 0.02, and the variance of the worm population effect (q) was 0.01 ± 0.01. Therefore, the repeatability of fecal egg count was 0.28 at a 1-wk interval and 0.27 at a 2-mo interval.

Heritability estimates, from Model A, for packed cell volume ranged from 0.10 to 0.33 during fattening (Table 3). Genetic correlations were greater than 0.68 using Model A. The repeatability of packed cell volume was 0.20 at a 2-mo interval and the estimate of its overall heritability was 0.12 using Model C. Due to nonoptimal convergence, standard errors of estimates were not meaningful.

At weaning, maternal genetic effects on fecal egg count were found to be significant (h²m = 0.26) with Model B, and similar in magnitude to the direct genetic effects (h²a = 0.20). The genetic correlation between direct and maternal effects was negative (σ²am = −0.64). After weaning, maternal genetic effects on fecal egg counts between 6 and 10 mo of age were found to be unimportant. Parameters estimates from Model B were h²a = 0.18, I² = 0.08 (permanent individual environment variance), h²m = 0.06, and σ²am = −0.48. The conclusion was similar for repeated packed cell volume measures between 4 and 10 mo of age: h²a = 0.04, I² = 0.09, h²m = 0.04, and σ²am = 0.08 in Model B.

Genetic parameter estimates for weights recorded at weaning and 4, 6, 8, and 10 mo of age are shown in Table 4. Heritability of these traits was moderate and genetic correlations were close to unity. No meaningful standard errors were estimated.

Estimates of the genetic correlations between fecal egg count, packed cell volume, and live weight are given in Table 5 at each age of measurement. Correlations between fecal egg count and packed cell volume fluctuated from −0.06 to −0.67. Fecal egg count and live weight at each age of measurement were never significantly correlated. The estimated genetic correlations between packed cell volume and live weight decreased from 0.47 to 0.10 during fattening.

**Discussion**

Resistance to strongyles seems to be a heritable trait in Creole goats under natural challenge with *H. contortus* and *T. colubriformis*. At 10 mo of age, our heritability values (0.33 ± 0.06) are similar to the estimate of Jackson et al. (1999) (0.37 ± 0.18) for fecal egg count of Scottish Cashmere goats exposed to natural and artificial infection with *Teladorsagia circumcincta* and *T. vitrinus*. In a dual-purpose goat flock infected with *H. contortus*, Rohrer et al. (1991) estimated heritability values at 10 to 12 mo of age of 0.40 fecal egg count and 0.22 for packed cell volume. Our estimates are in the same range (or slightly lower) than those reviewed by Baker et al. (1992) and Morris (1998) in sheep studies. Under tropical conditions, environmental variability is often large, so very few significant heritability esti-
mates have been obtained for fecal egg count and packed cell volume measurements following mixed infection in goats (Woolaston et al., 1992; Baker, 1998). In Guadeloupe, the experimental conditions and flock management were relatively standardized, and this may explain why we found significant heritability estimates.

Resistance to strongyles in goats seems to be under a degree of genetic control similar to that previously observed in sheep. Direct genetic variability in fecal egg count increased when the kids aged and reached the highest value at 10 mo of age in our experiment. The tendency is similar for packed cell volume, except for an unexplained low estimate at 8 mo of age. Regulation and mechanisms of resistance may take place gradually with repeated contacts with the parasites. Baker et al. (1994) showed the same evolution in sheep and concluded that genetic resistance develops with age. The high genetic correlations between two adjacent measurements of fecal egg count or packed cell volume are a further evidence of this rule in Creole goats.

Maternal genetic effects have little impact on resistance after weaning. The genetic correlations between adjacent fecal egg count or packed cell volume measurements were equal to or higher than 0.70. Thus, it was appropriate to consider them as repeated measures of the same trait in analyzing maternal genetic effects. However, our ability to separate direct and maternal effects was limited because few dams that were recorded as kids also had progeny with records (Gerstmayr, 1992): 21% at weaning and 13% during fattening. Our preliminary results suggest that maternal genetic control of fecal egg count might be as important as individual genetic control before weaning. These two genetic effects are antagonistic, as usually reported for growth traits (Robinson, 1996), but the nature of this maternal effect remains unknown. After 6 mo of age, maternal effects were found to be unimportant for fecal egg count and packed cell volume measures, whether treated as permanent environmental or genetic effects. Therefore, postweaning genetic resistance can be evaluated on individual performances only. These results are consistent with those reported in Merino sheep at 6 mo of age by Woolaston and Piper (1996) and in Dorper and Red Maasai sheep at 8 mo of age by Baker et al. (1998). However, Baker (1998) did not observe any maternal or direct genetic variability for fecal egg count at weaning, in contrast to our results.

In this trial, one detrimental effect of gastrointestinal nematode infection was a loss of 11% of the live weight at 325 d of age (Mandonnet et al., 2000). Therefore, a potential benefit of breeding Creole goats for resistance should be the reduction of this negative impact. Our results strongly argue for the feasibility of including resistance into a breeding scheme after elements of the testing protocol are defined. The repeatability estimates for fecal egg count and packed cell volume are low and in agreement with a number of published results for sheep (Baker et al., 1998; Bouix et al., 1998). Because repeatability of fecal egg count at a 1-wk interval is low, the resistance or susceptibility status of a kid is difficult to determine using only one measure. It was necessary and more informative to use the mean of two measures. Repeatabilities at a 2-mo interval (Model C) were greater than the overall heritabilities of fecal egg count (0.27 vs 0.14) and packed cell volume (0.20 vs 0.12). This variability of permanent individual environ-

### Table 2. Genetic parameters of fourth root transformed fecal egg counts from kids at weaning (82 d of age) and at 4, 6, 8, and 10 mo of age (heritability estimates on the diagonal, genetic correlations above and phenotypic correlations below)

<table>
<thead>
<tr>
<th>Age</th>
<th>Weaning</th>
<th>4 mo</th>
<th>6 mo</th>
<th>8 mo</th>
<th>10 mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weaning</td>
<td>0.37 ± 0.06</td>
<td>0.75 ± 0.21</td>
<td>0.81 ± 0.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo</td>
<td>0.24</td>
<td>0.14 ± 0.07</td>
<td>0.82 ± 0.27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 mo</td>
<td>0.25</td>
<td>0.14</td>
<td>0.17 ± 0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 mo</td>
<td>0.23</td>
<td>0.38</td>
<td>0.17 ± 0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 mo</td>
<td>0.18</td>
<td>0.34</td>
<td>0.46</td>
<td>0.33 ± 0.06</td>
<td></td>
</tr>
</tbody>
</table>

### Table 3. Genetic parameters for packed cell volume of measured on 4, 6, 8, and 10 mo of age (heritabilities on the diagonal, genetic correlations above, and phenotypic correlations below; no standard errors available)

<table>
<thead>
<tr>
<th>Age</th>
<th>4 mo</th>
<th>6 mo</th>
<th>8 mo</th>
<th>10 mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 mo</td>
<td>0.20</td>
<td>0.92</td>
<td>0.79</td>
<td>0.68</td>
</tr>
<tr>
<td>6 mo</td>
<td>0.37</td>
<td>0.22</td>
<td>0.95</td>
<td>0.88</td>
</tr>
<tr>
<td>8 mo</td>
<td>0.22</td>
<td>0.39</td>
<td>0.10</td>
<td>0.99</td>
</tr>
<tr>
<td>10 mo</td>
<td>0.29</td>
<td>0.48</td>
<td>0.49</td>
<td>0.33</td>
</tr>
</tbody>
</table>

### Table 4. Genetic parameters for live weight recorded for kids at weaning and at 4, 6, 8, and 10 mo of age (heritabilities on the diagonal, genetic correlations above, and phenotypic correlations below; no standard errors available)

<table>
<thead>
<tr>
<th>Age</th>
<th>Weaning</th>
<th>4 mo</th>
<th>6 mo</th>
<th>8 mo</th>
<th>10 mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weaning</td>
<td>0.33</td>
<td>0.97</td>
<td>0.96</td>
<td>0.94</td>
<td>0.90</td>
</tr>
<tr>
<td>4 mo</td>
<td>0.90</td>
<td>0.32</td>
<td>0.97</td>
<td>0.92</td>
<td>0.88</td>
</tr>
<tr>
<td>6 mo</td>
<td>0.85</td>
<td>0.95</td>
<td>0.34</td>
<td>0.98</td>
<td>0.96</td>
</tr>
<tr>
<td>8 mo</td>
<td>0.76</td>
<td>0.87</td>
<td>0.93</td>
<td>0.29</td>
<td>0.98</td>
</tr>
<tr>
<td>10 mo</td>
<td>0.70</td>
<td>0.80</td>
<td>0.87</td>
<td>0.95</td>
<td>0.23</td>
</tr>
</tbody>
</table>
ment is slight. This confirms the results observed in two previous sheep studies (Albers et al., 1987; Gruner et al., 1995). Levamisole may affect the immunity of kids, as observed in the immune response of uninfected lambs (Cabaj et al., 1995). The drenching of kids seems to erase a part of the acquired immunity against infective larvae. At each new infection, the animal must reactivate its immunity against strongyles, mainly on its genetic abilities. The worm burden increased at the end of our experiment, probably due to resistance to levamisole, and disrupted our observations. Therefore, the genetic resistance status of kids should be evaluated after drenching and reinfection. Then, their previous contacts with the parasites would not interfere.

The genetic correlations between resistance and live weight in Creole goats were not significant or slightly favorable as already reported for most of the studied sheep populations (Albers et al., 1987; Baker et al., 1992; Eady et al., 1994). Breeding for resistance in Creole goats will not decrease genetic ability for growth in infected pastures. Moreover, genetic variability is available for selection on live weights in this breed when it is reared in a semi-intensive system (irrigated and fertilized Digitaria decumbens pastures). The same genetic basis seems to control live weight during the post-weaning fattening period in our conditions.

The main gastrointestinal nematodes were *H. contortus* and *T. colubriformis*. The suspected resistance to levamisole (G. Aumont, unpublished data) in those two species during the 3rd yr of experimentation induced an accumulation of parasite burden in the last cohorts of kids. The infection level increased significantly. Finally, a wide range of infection levels was observed during this experiment. Therefore, linear regression was estimated between infection and significance of sire effect (indicator of genetic variability) on fecal egg count measures. Our results show increased genetic variability in host resistance as the infection level of animals increases. Thus, if the infection level is sufficiently high, then genetic evaluation and segregation into susceptible and resistant animals will be easier, as suggested by Woolaston et al. (1992) and Woolaston and Piper (1996). Our conditions were favorable to show genetic variability for resistance. However, we must now verify the mechanism whereby *T. colubri-

### Table 5. Estimates of genetic correlations between fecal egg count, packed cell volume, and live weight of kids at weaning and at 4, 6, 8, and 10 mo of age

<table>
<thead>
<tr>
<th>Age</th>
<th>Fecal egg count and packed cell volume</th>
<th>Fecal egg count and live weight</th>
<th>Packed cell volume and live weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weaning</td>
<td></td>
<td>−0.00</td>
<td></td>
</tr>
<tr>
<td>4 mo</td>
<td>−0.14 ± 0.21</td>
<td>0.19 ± 0.20</td>
<td>0.47 ± 0.10</td>
</tr>
<tr>
<td>6 mo</td>
<td>−0.47 ± 0.17</td>
<td>−0.03 ± 0.15</td>
<td>0.28 ± 0.12</td>
</tr>
<tr>
<td>8 mo</td>
<td>−0.67 ± 0.17</td>
<td>−0.14 ± 0.15</td>
<td>0.07 ± 0.18</td>
</tr>
<tr>
<td>10 mo</td>
<td>−0.06 ± 0.14</td>
<td>−0.09 ± 0.14</td>
<td>0.10 ± 0.14</td>
</tr>
</tbody>
</table>

**Implications**

Overall, our results demonstrate that breeding for improved resistance to nematodes in Creole kids is feasible. The genetic control of this trait is very similar to what is known in sheep: moderate heritability, increasing genetic variability with age, and no maternal genetic influence after weaning. Some elements of the genetic evaluation design are proposed to breeders: importance of drenching and infection level, independent evaluations of resistance, and live weight. Economic benefits are expected from including kid resistance into breeding schemes (e.g., higher weight gain and lower mortality rate during fattening) but still need to be quantified. Relationships with resilience and with susceptibility of does during the periparturient period also remain to be investigated.

**Literature Cited**


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