Results and Discussion

The FEC, EOSI and PCV significantly varied across the experimental infection ($P < 0.001$). Infection type did not affect FEC and PCV. FEC increased until the end of the infection (D35) and was two fold higher in R than in S kids ($P < 0.0001$). The PCV decreased significantly ($P < 0.0001$) but no difference was observed between R and S kids. These results suggest that experimental infection with a single dose of 10,000 *H. contortus* L_3 could lead to a standardized genetic evaluation design for resistance to strongyles in Creole goats. Infection type affected EOSI ($P < 0.05$), basically at D42 (792. 10^3 cells/mL vs 513.10^3 cells/mL, in TI and SI respectively). These data are so far consistent with a role of eosinophils in the killing of infective larval stages, but not adults, of most helminths (Meeusen *et al.*, 2005). The post-weaning grazing management affected FEC ($P = 0.0003$) but not EOSI and PCV. During experimental infection, FEC was higher in animals from grazing group 25% than 75% and 100% ($P < 0.0001$). No interaction of grazing management $\times$ genetic status was observed. Here we showed that without any effect on genetic status, the level of infection of kids during the post-weaning period could help in preventing severe infection rate at the adult stage. The results suggest that stimulation of the immune system in young animals increase the efficiency of the protective immune response at a further stage. This result needs further studies to understand the mechanisms underlying this observation.

Conclusion

Standardized genetic evaluation design for resistance to gastrointestinal strongyles in Creole goats could be realized with a single dose of 10,000 *H. contortus* L3. The level of post-weaning natural parasitism at pasture would not influence the genetic status evaluation. More generally our results suggest that it would be better to expose young kids to a high level of gastrointestinal parasitism in order to increase to basal level of resistance thereafter.

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Assessment of genetic variability of resistance to heartwater in Creole goats

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Introduction

In the Caribbean as in many other parts of the tropical area, gastrointestinal strongylosis (GIS) and heartwater are the two main threats on small ruminant production and especially on goat production. Even if several studies have been done on heartwater immune response with a protective cellular immune response, the protective mechanisms are still unclear. A collaborative research program has been carried out in Guadeloupe between CIRAD and INRA to analyse resistance to GIS and heartwater, including their relationship. The first step of this program is to validate the hypothesis of a genetic variability in resistance to heartwater.

Material and methods

Eleven Creole bucks were evaluated on the resistance/susceptibility of their offsprings to an *Ehrlichia ruminantium* standardized subletal infection inducing 70% of mortality (Vachiery *et al.*, 2006). Two susceptible and 2 resistant bucks were chosen among them and were randomly mated to 22 does. Forty-three kids, allocated in 2 cohorts of 21 and 22 kids, were separated from mother and reared indoors in order to avoid transfer of mother immunity and tick infestation until the challenge at yearling. The intensity of the disease was quantified using clinical reaction indices (incubation period, intensity of fever, nervous signs, death) as already described by Vachiery *et al.* (2006). Clinical indices were indicators of intensity of the disease. Clinical scores were defined as the sum of each clinical reaction indices per animal and per day, added up over the period. A square root transformation was applied in order to normalize residual variances. Repeated measures

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between day 10 and day 25 post infection were taken into account using the MIXED procedure of SAS software version 8.1 (SAS institute Inc., 1999). A quadratic polynomial was used to model the evolution with time. Sex, cohort, rank of infection within cohort, buck effects were investigated in the analyses. An auto-regressive covariance structure provided the best fit of the score.

Results and Discussion

No effect of sex and rank of infection were shown on clinical scores. The 2 cohorts differed in the quadratic coefficient, which is in the mortality rate after infection. This coefficient was significantly higher ($P < 0.05$) in the second cohort although the challenge doses were the same.

The buck effect was significant for all the coefficients of the polynomial (Figure 1). Ten days after infection, susceptible bucks had already higher scores than the resistant one ($P < 0.01$), that is, displayed more important clinical reactions. During the period, scores of resistant bucks increased quicker than susceptible ones ($P < 0.001$) but also stabilized quicker ($P < 0.0001$).

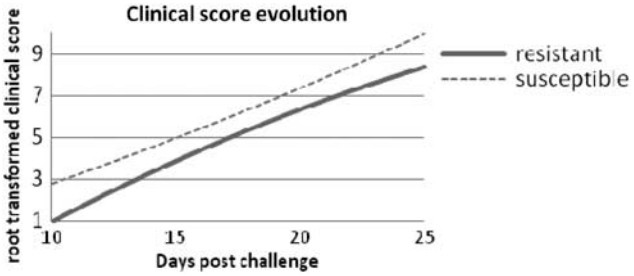

Figure 1 Evolution of the square root transformed clinical scores in resistant and susceptible bucks after standardized challenge with *E. ruminantium*.

Conclusions

Genetic variability on resistance to heartwater was suspected in previous works of Bensaid *et al.* (1993). To our knowledge, this study is the first to assess this variability. The aim of the project was also to link this variability physically with areas of the genome and to propose hypotheses for the underlying resistance mechanisms. A candidate gene approach is in progress in collaboration with the Institute of Genetics of Bern University. The following step of the program is to verify how this trait is correlated to GIS resistance in Creole goats.

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