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J ANIM SCI 2009, 87:2367-2375.
doi: 10.2527/jas.2008-1702 originally published online March 13, 2009

Intake and digestibility of naïve kids differing in genetic resistance and experimentally parasitized (indoors) with *Haemonchus contortus* in two successive challenges[1]

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ABSTRACT: We evaluated the effects of infection with *Haemonchus contortus* on feed intake, digestibility, fecal egg count, circulating eosinophils, and packed cell volume in Creole kids differing in genetic resistance (susceptible, S; resistant, R) to gastrointestinal parasitism and maintained on a similar level of nutrition. The experiment was carried out during 2 periods of 6 wk each differing in immunity development stage. In the first period (acquisition of immunity; period I), 22 naïve male kids (23.4 ± 0.65 kg of BW) were housed in individual boxes and fed a hay-based diet, and a primary infection was induced. In the second period (expression of immunity; period II), 15 of the initial 22 kids (28.4 ± 0.77 kg of BW) were submitted to a secondary infection. Housing and management were uniform throughout the experiment. For each period, measurements of intake and digestibility were made at 0, 2, and 4 wk postinfection (WPI) with a single dose of 10,000 infective larvae (L_3). The DMI and total-tract DM, OM, CP, NDF, and ADF digestibilities were determined using the total feces collection and ad libitum forage supply method. Fecal and blood samples were collected weekly to measure fecal egg count, circulating eosinophils, and packed cell volume. Infection with *Haemonchus contortus* decreased feed intake during period I. The absence of anorexia in period II was probably due to the acquired immunity of kids. The DMI was affected ($P = 0.05$) by genetic predisposition to resistance (626 vs. 583 ± 26 g/d, for R vs. S) and WPI, being greatest in the second WPI (693 vs. 614 and 657 g/d, for WPI-2 vs. WPI-0 and WPI-4, respectively). The latter was related to worm establishment phase and was linked to the lower total tract digestibilities at this point. Digestibilities were least at WPI-2. The fecal egg counts were greater ($P < 0.001$) in period I than II, and differences between S and R were evident after the fifth WPI in period II. Circulating eosinophils were greater ($P < 0.001$) in S vs. R. The results suggest that effects of these parasites on intake and digestibility are influenced by the individual genetic resistance and the immunological stage, and the strongest impact occurs between the second and the third WPI, a period during which the immune response is more pronounced, probably due to parasite maturation.

Key words: digestibility, goat, *Haemonchus*, immunity, intake, resistance

J. Anim. Sci. 2009. 87:2367–2375
doi:10.2527/jas.2008-1702

INTRODUCTION

Few published papers are available about effects of gastrointestinal parasitism on feed intake, digestibility, and their relationship with the immunological system in ruminants. Most of our understanding about the impact on ruminant nutrition has been derived from infections in sheep (Coop and Kyriazakis, 1999; Hoste et al., 2008). In goats, data on this topic remain scarce and there is little clarity about mechanisms underlying the relationship between nutrition and gastrointestinal parasitism. However, compared with sheep, goats seem to develop a reduced immune response against nema-

[1]We are grateful to the team of the Plateform Tropical de Experimentation Animal, INRA-Domaine Duclos, for their technical support. This study was financed by La Région Guadeloupe.

[2]These authors contributed equally to this study: J. C. Bambou in the design of the experiment, fecal egg count, and hematology determination, and E. González-García in the data processing and manuscript redaction. Both authors contributed equally to enrich the discussion.

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Received December 4, 2008.
Accepted March 9, 2009.

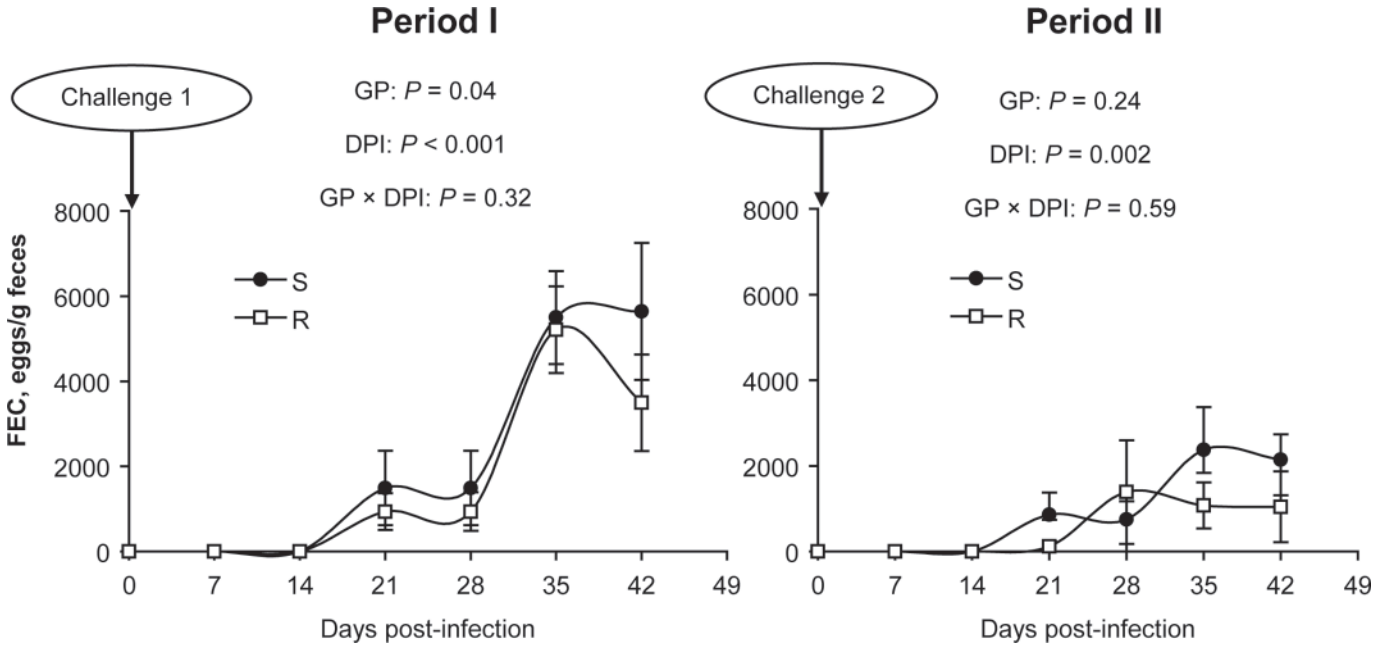

Figure 1. Weekly evolution of fecal egg counts (FEC) in resistant (R) and susceptible (S) Creole kids after 2 single experimental infections with 10,000 infective *Haemonchus contortus* larvae. Period I = first challenge, corresponding to the acquisition of immunity stage; period II = second challenge, corresponding to the expression of immunity stage. Periods differed at $P < 0.001$. GP = genetic predisposition to gastrointestinal parasitism; DPI = days postinfection. Error bars represent the SE at each point for R or S kids.

todes, which has implications for epidemiology, pathology, and control of infections (Hoste et al., 2008).

Mandonnet et al. (2001) demonstrated intrabreed variability in resistance to gastrointestinal parasitism in Creole kids, ensuring that this factor must be considered when evaluating impacts on this breed. Their work (Mandonnet et al., 1997, 2003, 2005) constitutes 1 of 2 attempts at the international level to select goats based on response to gastrointestinal parasitism; the other was in Scotland with Cashmere goats (Vagenas et al., 2002).

Response to gastrointestinal parasitism is dependent on immunity stage, physiological condition, nutritional status, and nutrient requirements of the host at the time infection occurs; therefore, different reactions would be expected among breeds of the same species (sheep; Zaralis et al., 2008) or between naïve and previously infected animals, even when subject to the same nutrition and parasite burden.

Our hypothesis was that genetic predisposition (**GP**) to gastrointestinal parasites and the immunity stage of the host at the beginning of the infection affects the magnitude of the effects of parasites on feed intake and diet digestibility of Creole kids. This hypothesis was tested in resistant (**R**) and susceptible (**S**) kids, and during a primary (**period I**) and secondary (**period II**) challenge with infective larvae ($\mathbf{L_3}$) of *Haemonchus contortus* (**HC**).

MATERIALS AND METHODS

The experiment was conducted at Institut National de la Recherche Agronomique (**INRA**) Animal Research Unit, Guadeloupe (French West Indies). All animal care, handling techniques, and procedures as well as the license for experimental infection and blood sampling were approved by INRA, according to the certificate of authorization (number A-971-18-01) to experiment on living animals issued by the French Ministry of Agriculture, before the initiation of the research.

Animals, Management, and Experimental Design

The study was carried out with growing male Creole kids during 2 consecutive periods of 6 wk each, the first (period I) from October 8 to November 19, 2007 and the second (period II) from December 17, 2007 to January 28, 2008; there was a lapse of 4 wk between finishing period I and beginning period II. Although the acquisition of immunity is a continuous process, the 2 periods were considered as separate according to our objectives and taking into account previously established criteria and definitions for the development of the nutrient partitioning framework (Coop and Kyriazakis, 1999; Athanasiadou et al., 2008). Thus, period I and period II were respectively associated with the acquisition and expression of immunity phases. The expression of immunity after the second challenge was confirmed by the decreased fecal egg count (**FEC**) compared with FEC values after the first challenge (see Figure 1; compare period I vs. II).

All kids were born indoors at INRA-Domaine Gardel (north of Guadeloupe) during the summer season of 2006 and were brought into a naturally illuminated and ventilated shed in INRA-Domaine Duclos (South) 1

Table 1. Genetic resistance indexes of the kids selected for the experiment and classified as resistant (n = 11) or susceptible (n = 11) to gastrointestinal parasitism according to their breeding value

Animal classification	Breeding value for [log (FEC, eggs/g + 15)][1]
Susceptible	0.156
Resistant	−0.286
SEM	0.08
P-value[2]	<0.0001
Distance between susceptible and resistant, genetic SD	2.99

[1]FEC = fecal egg count. Breeding value based on individual, ascendants, and pedigree historical performance. The breeding value for FEC of the Creole goat flock in Guadeloupe has been estimated since 1979 at Institut National de la Recherche Agronomique, Domaine Gardel, under natural mixed infection under grazing conditions.

[2]Kruskal-Wallis test.

mo before the beginning of the experiment; kids were housed in individual pens (2.0 × 1.0 m) until the end of the study.

In the flock at INRA Domaine Gardel, the pedigree of each animal was available since the foundation generation was established in 1979. Fecal samples were collected regularly (during wk 6 and 7 after drenching) at 7 and 11 mo of age for genetic evaluation on the average of 2 FEC measures. Thus, the breeding value for FEC of each kid of the flock at 11 mo of age, under natural mixed infection on pasture conditions, was regularly estimated. Such estimation was made taking into account their own individual performances, the performances of their ascendants, and their pedigree. The records used were then back-transformed value logarithm of parasite eggs/g of feces. The 11 R and 11 S kids initially used in the current study were selected on basis of their extreme breeding value (see Table 1) with regard to their cohorts. The average breeding value of the 2 groups was distant ($P < 0.0001$) at 2.99 genetic SD of FEC at 11 mo of age.

Period I started with 22 male worm-free (naïve) Creole kids (23.8 ± 0.65 kg of BW; 15 mo old), selected on the basis of their genetic index (Mandonnet et al., 2001) as R (n = 11; 24.3 ± 0.93 kg of BW) or S (n = 11; 23.4 ± 1.25 kg of BW). Period II continued with 15 kids (28.4 ± 0.77 kg of BW; 17 mo old; R, n = 8, 28.2 ± 1.02 kg of BW; S, n = 7, 28.5 ± 1.24 kg of BW) from the initial 22; 7 kids were slaughtered for in vitro studies about parasite balance and morphology at abomasum (results not corresponding to the objectives of this paper). The selection criterion for slaughter of kids was their FEC values; kids were categorized as low, average, and high FEC, and we selected 2 or 3 animals per group.

On the first day of each period and before the morning meal (0730 h), we individually challenged all kids with a single dose of 10,000 L_3 of HC to monitor the effects of induced infection on feed intake and diet digestibility during the subsequent weeks. The L_3 were obtained 42 d before the challenge. Cultures of feces taken from anthelmintic-susceptible strain were harvested from feces of donor Creole goats monospecifically infected (Bambou et al., 2008) with isolates previously obtained from Creole goats reared on pasture in different farms in Guadeloupe (Aumont and Cabaret, 1999). A standard Baerman procedure (Urquhart et al., 1988) was used. After harvesting, L_3 were stored at 4°C in tap water (1,000 L_3/mL) and used within 3 wk of collection. Each infective dose was suspended in 10 mL of water and was administered orally using a syringe.

During the entire experiment, animals received a diet composed of ad libitum access to 75-d-old *Dichantium* spp. hay and restricted concentrate (100 g/d). Measurements of intake and digestibility were made at 0, 2, and 4 wk post-oral infection (**WPI**); feed intake, BW changes, FEC, and blood variables were measured weekly throughout the experiment.

Feed Intake and In Vivo Total Tract Digestibility

The total feces collection (with fecal trays placed behind the kids) and ad libitum forage supply method was used. Pooled samples of hay, supplementary concentrate, and feces (10% per day of the wet weight) were also collected daily for chemical analyses (Table 2). Each digestibility measurement was based on a 5-d period for sample collection. Offered and refused feed were individually recorded weekly to determine voluntary DMI and to enable 115% of that respective value to be offered the following week. The in vivo apparent total tract DM, OM, CP, NDF, and ADF digestibilities were determined.

FEC and Blood Sampling

Fecal and blood samples were collected before challenge in WPI-0. To determine FEC, fecal samples of approximately 10 g were collected weekly after experimental infection directly from the rectum of each kid. The feces were kept in plastic tubes to avoid contamination and immediately transported to the laboratory in refrigerated vials. All samples were individually analyzed

Table 2. Average determined chemical composition (DM basis) of diet components

	Hay[1]						
	Period I[3]			Period II[4]			
Item, %	WPI-0[5]	WPI-2[6]	WPI-4[7]	WPI-0	WPI-2	WPI-4	Concentrate[2]
DM	92.7	92.3	91.2	90.2	90.5	90.4	93.4
OM	93.7	93.7	93.0	95.8	94.5	95.3	97.0
CP	4.4	5.3	5.5	8.1	8.3	8.1	16.5
NDF	76.5	76.3	79.2	72.1	73.6	73.8	17.6
ADF	45.6	41.8	44.3	37.7	37.5	38.8	4.8
ADL	7.0	5.3	7.0	5.1	4.5	4.7	0.7
Hemicellulose	30.9	34.5	34.8	34.4	36.1	35.0	—
Cellulose	38.6	36.5	37.3	32.6	33.0	34.1	—

[1]Hay of 75-d-old *Dichantium* spp. grass, with forage grown with irrigation and inorganic fertilization.
[2]Offered daily in a fixed individual amount (100 g/d) as supplement.
[3]Primary infection; considered as acquisition of immunity period (period I) for the naïve kids; 6 wk in length.
[4]Secondary infection; considered as expression of immunity period (period II); 6 wk in length.
[5]Week postinfection number 0: beginning week immediately after experimental challenge with 10,000 infective *Haemonchus contortus* larvae.
[6]Second week after experimental challenge with 10,000 infective *H. contortus* larvae.
[7]Fourth week after experimental challenge with 10,000 infective *H. contortus* larvae.

using a modified McMaster method for rapid determination (Aumont et al., 1997), and FEC was expressed as the number of eggs/gram of feces. Blood samples (4 mL) were individually collected once weekly by jugular venipuncture from each Creole kid using disposable syringes and 20-gauge needles. A 2.5-mL aliquot of each blood sample was placed in commercial anticoagulant tubes (EDTA tubes, Becton Dickinson, Plymouth, UK); the remainder was placed in a tube without additive, left to clot at room temperature for 1 h, and then centrifuged for 5 min at 5,000 × *g* (4°C). Serum was then frozen at –20°C until analysis. Blood samples previously placed in EDTA-coated tubes were used to measure the number of circulating eosinophils (**EOS**) according to the method of Dawkins et al. (1989) and counted with a Malassez cell counter. The packed cell volume (**PCV**) was measured using the capillary microhematocrit method.

Chemical Analyses of Feeds, Orts, and Feces

The DM contents of feeds, orts, and feces were determined by oven drying (type SE-79, Le Matériel Physic Chimique Flam et Cie, MPC, Neuily S/Marine, France) to a constant weight at 60°C for 48 h (AOAC, 1997), and ash content was determined by heating samples at 550°C for 4 h according to AOAC (1997). The OM content was calculated by difference. Dry samples were obtained for further chemical analyses and were ground (model SK100 confort Gußeisen, F. Kurt Retsch GmbH & Co., Haan, Germany) to pass through a 1-mm stainless steel screen. The CP content was calculated after N determination by combustion using the micro-Dumas method (NA2100 Protein, CE Instruments, ThermoQuest S.p.A., Milano, Italy).

The methods of Van Soest et al. (1991) were followed to determine NDF, ADF, and ADL (sequentially) on an ash-free basis using the Ankom[200] Fiber Analyzer incubator (Ankom Technology, Fairport, NY). The hemicellulose and cellulose contents of ingredients were calculated as the differences between NDF and ADF, and between ADF and ADL, respectively.

Statistical Analysis

Data were analyzed by repeated measures ANOVA (PROC MIXED, SAS Inst. Inc., Cary, NC), considering the immunity period, the GP to gastrointestinal parasitism, the WPI, and their interactions as fixed effects. This was done for the whole data set (including data from both periods). Second, because of the significant effect of period in the statistical analysis conducted on the entire data set, subsequent analyses were performed on each period separately. For all traits, kid was the experimental unit because they were individually fed and managed, and was included in the model as a random effect. Significance was declared at $P \leq 0.05$; comparisons between means were tested by the least squares means procedure with adjustment for multiple comparisons (Tukey-Kramer test; compound symmetry covariance parameter).

RESULTS

The chemical composition of the hay and concentrate for both experimental periods is shown in Table 2. During period II hay quality changed with a greater content of CP and decreased values of cell wall components (NDF, ADF, ADL), which may have influenced feed intake and digestibility compared with period I.

Table 3. Least squares means of DMI, apparent total tract digestibility, and BW of Creole kids during the acquisition of immunity period (period I) after a primary oral infection with a single dose of 10,000 third-larvae stage (L_3) *Haemonchus contortus*

	WPI-0[1]		WPI-2[2]		WPI-4[3]			*P*-value		
Item	R[4]	S[4]	R	S	R	S	SEM	WPI	GP[4]	GP × WPI
DMI, g/d	579	552	671	617	628	581	26.0	0.01	0.05	0.87
Digestibility, g/kg of DM										
DM	630	620	600	610	621	630	9.8	0.03	0.62	0.71
OM	649	645	620	627	638	646	9.5	0.04	0.61	0.79
CP	582	577	542	573	578	580	12.5	0.15	0.34	0.31
NDF	547	519	504	500	559	565	9.9	<0.001	0.33	0.22
ADF	535	520	456	440	520	529	12.0	<0.001	0.46	0.52
BW, kg	24.3	23.4	25.9	24.1	24.3	23.4	0.76	0.46	0.19	0.87

[1]Week postinfection (WPI) number 0: beginning week immediately after experimental challenge with 10,000 infective *H. contortus* larvae.
[2]WPI 2: second week after experimental challenge with 10,000 infective *H. contortus* larvae.
[3]WPI 4: fourth week after experimental challenge with 10,000 infective *H. contortus* larvae.
[4]Genetic predisposition (GP) to gastrointestinal nematode resistance: kids indexed by pedigree as resistant (R) or susceptible (S).

However, differences in total DMI between periods were likely to be linked to the progressive natural increase in feed intake capacity with growth (kids were 5.2 kg heavier in period II).

There were significant effects of GP ($P = 0.02$), WPI ($P < 0.001$), and period ($P < 0.001$) for total DMI, whereas no significant interactions between these variables were observed. Greater average DMI were recorded for R kids compared with S kids and during the second WPI in both periods (Tables 3 and 4).

There were interactive effects ($P < 0.01$) between period and WPI for total tract apparent digestibilities. Such differences for OM, CP, NDF, and ADF digestibilities may be checked by comparing values of Tables 3 and 4.

The FEC and PCV were also affected ($P < 0.001$ and $P = 0.009$, respectively) by the interaction of period × WPI with the FEC being greater from 28 d postinfection (**DPI**) in period I, whereas the PCV was greater at 7 and 14 DPI in period II (Figures 1 and 2). The EOS were greater ($P = 0.03$) in S kids when compared with R during period II (125 vs. 100 ± 9.0; Figure 3).

Acquisition of Immunity Period (Period I)

Table 3 presents the results from the analysis of period I, after a primary infection of the naïve kids. The DMI were affected by WPI, ($P = 0.01$) being greater at WPI-2, whereas GP influenced ($P = 0.05$) DMI with R kids demonstrating less effect on appetite depression compared with S kids. There were no GP effects nor an interaction of GP × WPI for nutrient digestibility, indicating that effects on this variable during period I were more dependent on the evolution of parasite establishment in the gastrointestinal tract (WPI), irrespective of the genetic resistance in individual kids. Except for CP ($P = 0.15$), all digestibility values were less at WPI-2 than at WPI-0 and WPI-4.

The FEC remained at zero during the first 2 wk of measurement and were greater ($P = 0.04$) in period I for S vs. R. The PCV decreased with time ($P < 0.001$) without being affected by GP. The EOS were not affected in period I by GP with the exception of 14 DPI ($P < 0.001$) on which S kids had greater values (Figure 3).

Table 4. Least squares means of DMI, apparent total tract digestibility, and BW of Creole kids during the expression of immunity period (period II) after a secondary oral infection with a single dose of 10,000 third-larvae stage (L_3) *Haemonchus contortus*

	WPI-0[1]		WPI-2[2]		WPI-4[3]			*P*-value		
Item	R[4]	S[4]	R	S	R	S	SEM	WPI	GP[4]	GP × WPI
DMI, g/d	666	659	767	718	742	676	33.7	0.07	0.15	0.67
Digestibility, g/kg of DM										
DM	701	701	662	682	657	660	7.5	<0.001	0.23	0.37
OM	707	707	673	693	664	669	7.9	<0.001	0.21	0.41
CP	578	573	514	557	508	513	11.1	<0.001	0.13	0.10
NDF	687	689	661	676	663	660	11.4	0.07	0.65	0.72
ADF	698	700	663	685	669	674	10.5	0.02	0.26	0.61
BW, kg	29.3	28.7	31.4	31.0	32.7	31.7	0.78	0.02	0.47	0.98

[1]Week postinfection (WPI) number 0: beginning week immediately after experimental challenge with 10,000 infective *H. contortus* larvae.
[2]WPI 2: second week after experimental challenge with 10,000 infective *H. contortus* larvae.
[3]WPI 4: fourth week after experimental challenge with 10,000 infective *H. contortus* larvae.
[4]Genetic predisposition (GP) to gastrointestinal nematode resistance: kids indexed by pedigree as resistant (R) or susceptible (S).

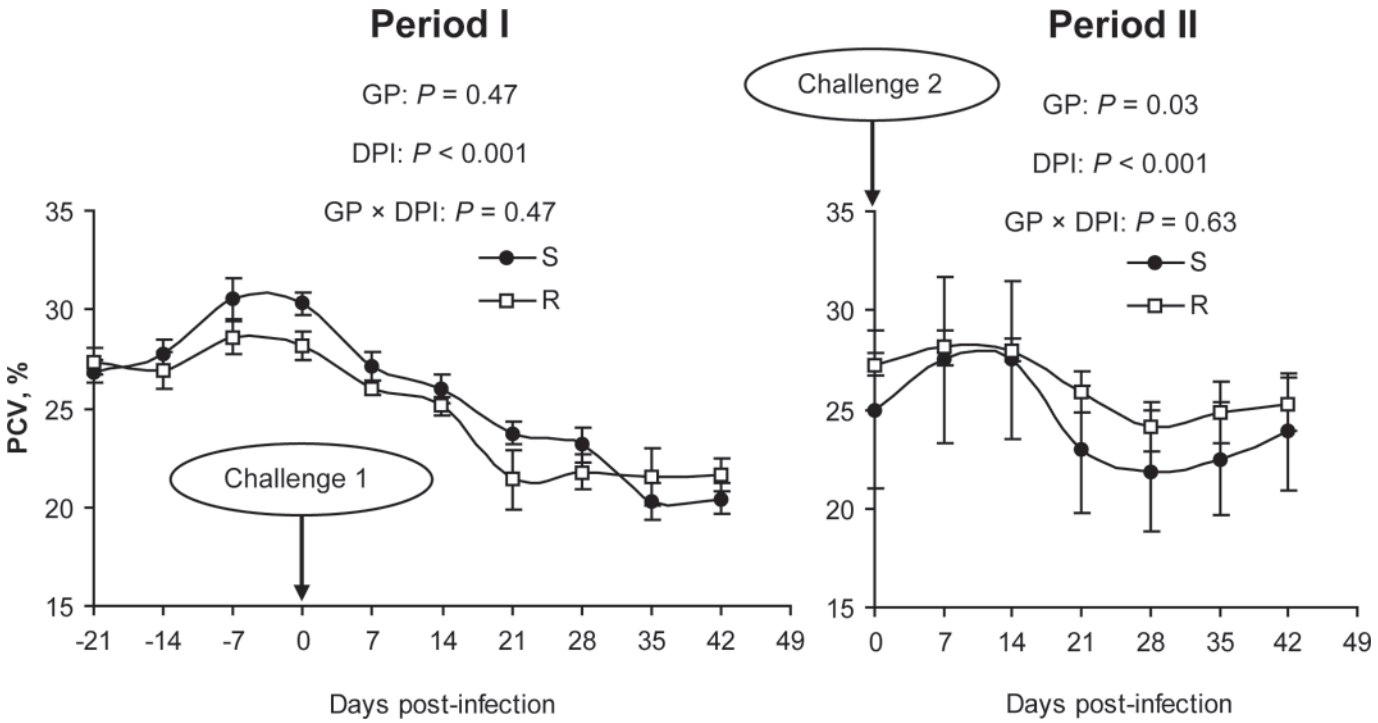

Figure 2. Weekly evolution of pack cell volumes (PCV) in resistant (R) and susceptible (S) Creole kids after 2 single experimental infections with 10,000 infective *Haemonchus contortus* larvae. Period I = first challenge, corresponding to the acquisition of immunity stage; period II = second challenge, corresponding to the expression of immunity stage. Periods differed at $P < 0.001$. GP = genetic predisposition to gastrointestinal parasitism; DPI = days postinfection. Error bars represent the SE at each point for R or S kids.

Expression of Immunity Period (Period II)

The results from period II are presented in Table 4. There were no differences in the performance of R and S kids for any variable, except for circulating EOS, which were significantly greater ($P < 0.001$) in S than in R kids.

In contrast to period I, there was no effect of GP, WPI, or their interactions on DMI. The digestibilities of DM, OM, CP ($P < 0.001$), and ADF differed accord-

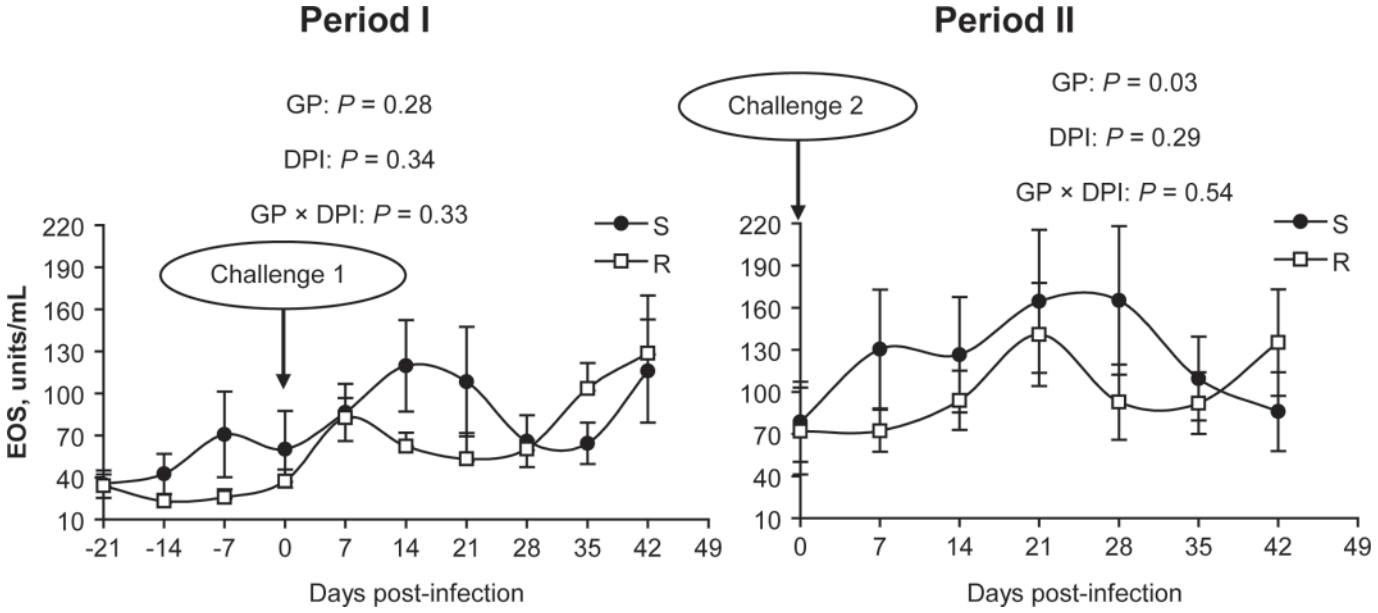

Figure 3. Weekly evolution of eosinophils (EOS) in resistant (R) and susceptible (S) Creole kids after 2 single experimental infections with 10,000 infective *Haemonchus contortus* larvae. Period I = first challenge, corresponding to the acquisition of immunity stage; period II = second challenge, corresponding to the expression of immunity stage. Periods did not differ, $P = 0.42$. GP = genetic predisposition to gastrointestinal parasitism; DPI = days postinfection. Error bars represent the SE at each point for R or S kids.

ing to WPI, consistently decreasing as WPI advanced (WPI-0 > WPI-2 > WPI-4).

In period II, FEC was not affected ($P = 0.24$) by GP and remained at zero until 14 DPI, afterwards increasing with time in both lines with a lesser slope than period I. There were differences ($P = 0.03$) between S and R kids in PCV (Figure 2) with S showing greater values. The PCV also decreased ($P < 0.001$) with time after infection in period II. The EOS were greater ($P < 0.03$) in S kids and showed a peak between 21 and 28 DPI (Figure 3).

DISCUSSION

Information about the variability in response to gastrointestinal parasitism that could be linked to genetic components is still scarce in goats (Hoste et al., 2008). Including resistance to these pathologies as an objective of genetic improvement schemes for Creole goats has been one of the main research goals for the INRA Animal Research Unit (URZ) team over the last several decades (Aumont, 1996; Aumont et al., 1997; Mahieu et al., 2008). Thus, the Creole goat is now considered a good model for highlighting mechanisms and genes of resistance to gastrointestinal strongyles in small ruminants.

Our results support findings by Mandonnet et al. (2001), who demonstrated the heritability of resistance to strongyles and the feasibility of increasing resistance through a selection program in Creole goats. Such genetic differences related to the immune response characteristics (GP) had significant effects on feed intake and immune effectors after the artificial infection implemented in our study. Thus, the hypothesis that GP influences the magnitude of negative effects of gastrointestinal parasitism, when the kids were categorized as R or S according to their pedigree, was confirmed.

However, differences in FEC were only evident after 35 DPI in both periods, being greater at this point in S kids. We think that such differences in FEC only after this point are probably related to the delay for the immune response effects on the worm population. Also, it remains a possibility that an effect on female prolificacy occurs. Thus, a possible modulation of genetic resistance against adult worms rather than against the incoming parasites could be hypothesized. This may also reflect the necessity for a longer monitoring period (>6 wk) in future infection protocols with similar aims, if we want to check effects from the incoming L_3 until the last phases of adult worms.

Possible inconsistency in our findings with the difference between R and S, normally found at 6 to 7 WPI at grazing (selection criteria) in Domaine Gardel, must be carefully interpreted because the different prevailing conditions, which could make the comparison irrelevant. During the selection process, the genetic evaluation is performed on grazing and with 7-mo-old nonnaïve animals that were naturally exposed to HC infection since they began to eat grass. In contrast, in the current study the first challenge was conducted in naïve kids.

In other traits, differences were more evident in period I during which R kids experienced greater DMI and less FEC after 35 DPI. An explanation could be that similar FEC before 35 DPI do not necessarily reflect that animals have similar worm burdens. It is possible that, during the first challenge, R kids limit worm establishment in the abomasum but not the female prolificacy. Thus, a smaller number of worms established in the abomasum may have a smaller effect on intake.

The results of this experiment are in general agreement with our previous findings obtained under natural grazing conditions. Bambou et al. (2006) observed a more intensive immune response against HC in R kids that had greater immunoglobulin response than S at 49 DPI, when FEC were relatively small. Surprisingly, they found that, whereas R presented less FEC than S at 49 and 56 DPI, the FEC were not correlated to the parasite (female) prolificacy at 56 DPI. Furthermore, R harbored 20 times more L_4 than S, and their young/adult ratio was 15 times greater. Such results may indicate that resistance occurred by controlling worm maturation, but a question still remains on the precise function of immunoglobulin response in this process.

Despite the lack of clarity of such mechanisms for depressing appetite and DMI during infection, Dakkar (1995) tried to describe other possible factors that could be implicated in the process. Local inflammation and pain in sites of the gastrointestinal tract affected by the infection, changes in absorption sites, and the decrease of availability for absorption of AA, as well as problems of motility in the GIT and of digesta transit may all be correlated to the depression of voluntary feed intake (anorexia) experienced in S kids. A more integrated approach to the occurrence of anorexia in parasitic diseases has been recommended by Kyriazakis et al. (1998). Its duration perhaps depends on the rate of development of the immunity, which can be from a few days in protozoan infections or up to several weeks in helminthes parasitism. Its extent is independent of the nutrition of the host (i.e., feed quality and quantity), which does not seem to affect the rate of establishment or development of parasites in nonimmune hosts. This is consistent with the approach of our experimental design looking for genetic effects while maintaining a similar level of nutrition.

Kyriazakis et al. (1998) also refers to a host-specific, perhaps genetically determined, response to parasitic infections in terms of a reduction in their feed intake that could also be related to a decrease in energetic efficiency. They highlighted the untested but attractive possible role of anorexia to enhance the immune response of the host.

The relatively high level of nutrition applied in our experiment could help to emphasize the relevance of the individual genetic factors in this tropical breed. However, as our kids were advancing in age (from 15

to 17 mo old), an increase in genetic variability of the resistance to HC could be expected, as has been reported after 10 mo of age (Mandonnet et al., 2001), thus making it more difficult to perceive differences between the experimental groups. Also, a better regulation and development of the mechanisms of resistance is thought to develop with age and repeated contacts with parasites (Baker et al., 1994, 1998). This was demonstrated by Mandonnet et al. (2001) with the high genetic correlations obtained between 2 adjacent measurements of FEC or PCV in Creole goats. Inter- and intrabreed differences in the immune responses to gastrointestinal parasitism, and especially with HC infections, have been also demonstrated in several studies with sheep (Terefe et al., 2007).

The lack of effects on DMI during period II remains unclear. An interpretation could be that S kids had developed immunity, thus diminishing the possible negative effects of parasitism on appetite and intake during period II. However, the fact that a single-challenge protocol was used and that anorexia was reported to last for weeks makes this hypothesis doubtful. Nevertheless, apparent nutrient digestibilities, which were greater than in period I (with the exception of CP digestibility), continued to be influenced by WPI, with the least values being obtained at WPI-2, the phase of establishment of parasites in the digestive tract.

The increase in DMI observed in the second WPI was consistent with the decrease in nutrient digestibility in the same week. We speculate that this may be because it is at this moment that animals start to suffer the strongest negative effects of the establishment of the HC population in the abomasum leading to competition for nutrients between host and parasites that momentarily could enhance feed intake to compensate for the depression in nutrient digestibility. This may have occurred in our experiment, in contrast to the criterion of depression in intake, because of the high level of nutrition used. The peak of such a process is likely to occur around 2 or 3 wk after infection (Dakkar, 1995; Coop and Kyriazakis, 1999), and the result would depend on the immunity status of the host, leading to a resilient (maintaining productivity while still infected) or an acutely infected animal (Kyriazakis and Houdijk, 2006).

Because circulating EOS were greater in S than in R kids, our results are in some ways contradictory with the available literature. In a previous study (Bambou et al., 2008) we did not observe correlation between EOS and resistance. In the present study, we confirmed this observation. In our model (Creole kids), EOS seems to perform more as a marker of the level of infection rather than a trait associated with resistance. In agreement with this suggestion Wildblood et al. (2005) demonstrated in vitro that HC produced EOS-chemoattractant activity (with identity that remains unknown), challenging the hypothesis that EOS are host-protective effector cells. They raised the possibility that nematodes (i.e., HC, *Teladorsagia circumcincta*, *Ostertagia ostertagi*, or *Trichostrongylus*) actively encourage recruitment of EOS. The magnitude of the chemotactic response was also directly related to larval numbers. Such results provide evidence that pathogenic gastrointestinal nematodes produce factor(s) that directly influence EOS migration. This may be important for in vivo pathogenesis and could provide novel targets for future control strategies. The chemoattractant activity could derive either from constitutively expressed worm products or indirectly through interactions with other cell types present in the mixed bone marrow cell populations. Thus, the precise nature of the interaction between parasite and EOS remains an enigma that will require new research efforts.

It was also noticeable that CP digestibility did not differ between GP or among WPI during period I. According to Dakkar (1995), parasitism of the abomasum (i.e., with HC) reduces rumen fermentation by almost 30% and generates an important depression in OM digestibility and CP digestibility, more so than intestinal parasitism, the maximum intensity being around 2 to 3 wk after infection, as in our study. In contrast, the infestation site did not seem to influence DM, energy, and sugar digestibilities. Nutrient absorption problems are difficult to identify due to the relevant capacity of intestines in ruminants to compensate for losses at the site of infection by increasing nutrient absorption in distal sites. The effects are influenced by the level of CP in the diet; at greater CP concentrations, less negative effects of parasitism on DMI are expected. A suitable level of CP in the diet could help to avoid negative effects on DMI, so it would be interesting to replicate the study but offering different levels of CP. The different trend observed for the digestibility of CP is likely to be influenced by the already known modifications of nitrogen metabolism in infected animals. The CP is the most critical nutrient because many components of the immune system, such as immunoglobulins and mucoproteins, and cellular products, such as leukotrienes, are proteinaceous in nature; they would be expected to use protein resources, especially in quantitative terms (Coop and Kyriazakis, 1999). In HC infections these losses can represent as much as 10% of the circulating blood volume per day.

Dietary CP and the increase in the catabolism of proteins from muscles, bone medulla, wool, and skin, to satisfy the increase in the metabolic demands, are expected to be used to replace cells in the epithelium and to produce a large quantity of mucus at the infection site, leading to increases in endogenous N losses into the gut (apparent absorption of CP can fall to 25% in sheep; Dakkar, 1995). The amount of nonreabsorbed endogenous N may be considerable and a proportion will be excreted in feces, but the majority will be fermented in the hind-gut and absorbed as ammonia or just excreted as urea, and will not be available for metabolic processes. Such reduction in absorption in addition to the endogenous losses results an increase in CP proportion in the digestive contents and in the

feces, which could explain the linear decrease in CP digestibility in infected kids, basically during the expression of immunity period.

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