Nutritional sensitivity of correlations between estimated breeding values for faecal egg counts and resistance to parasites in periparturient ewes

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Introduction
Only at times of metabolisable protein (MP) scarcity, resistance to gastrointestinal parasites was lower in highly productive Mules than in the less productive Blackface ewes (Kidane et al 2009). Thus, genetic differences in parasite resistance may be more pronounced when MP is scarce. Estimated breeding values (EBV) for faecal egg counts (FEC) may describe within-breed variation in parasite resistance. Here we tested whether FEC are positively correlated with EBV for FEC at times of MP scarcity only.

Materials and methods
Single (-1) and twin (-2) bearing pure-bred Suffolk ewes (n=16) with an EBV for FEC ranging from -0.37 to +0.91, were trickle infected with 10,000 Teladorsagia circumcincta larvae from day -44 onwards (day 0 is parturition). Their body weight (kg) and condition score on day-22 were 61.1±1.9 and 1.9±0.1, and 66.0±1.4 kg and 1.9±0.1, respectively. From day-22, ewes were fed at 0.9 times metabolizable energy requirement and at either 0.8 (LP-) or 1.3 (HP-) times MP requirements (AFRC, 1993). Ewes and lambs were weighed weekly and within 12h post lambing. Ewe FEC (in eggs per gram (epg) faeces) were assessed twice weekly, and log(FEC+1) was used for statistical analyses. Ewe body weight gain (g/day) and relative litter daily gain (g/day/kg) was estimated by linear regression. FEC were analysed using a repeated measures 2 x 2 factorial ANOVA (REML). Ewe weight gain, post parturition ewe body weight, litter birth weight and relative litter weight gain were analysed using a 2 x 2 factorial ANOVA (REML). Pearson’s correlations were calculated between EBV and mean FEC during late pregnancy and during lactation.

Results
Feeding treatment and litter size did not significantly interact for any performance measures taken. Treatments did not affect late pregnancy body weight gain and ewe body weight at parturition, which averaged at 206±23 g/day and 58.1±1.0 kg, respectively. However, during lactation, ewe body weight gain averaged at -56 and +59 g/day for single- and twin-rearing ewes (s.e.d. 42.4 g/day; P<0.01) and -81.2 and +83.1 g/d for LP and HP ewes, respectively (s.e.d. 42.4 g/day; P<0.001). Lactational weight gain of twin-rearing HP ewes (HP2) was different from zero (P<0.05). Feeding treatment did not affect litter birth weight, which averaged 5.1 and 6.9 kg for single and twin litters, respectively (s.e.d. 0.34 kg; P<0.001). Litter size did not affect relative litter weight gain, which averaged 36.7 and 47.0 g/d/kg for LP and HP ewes, respectively (s.e.d. 4.95 g/day/kg; P<0.05).

Figure 1 show the backtransformed FEC of the ewes and their correlation with EBV for FEC on each time point. Time did not interact with feeding treatment and litter size for FEC. During late pregnancy, FEC tended to increase (P=0.06), whilst during lactation, FEC first reduced and then gradually increased until the end of the experiment (P<0.001). During late pregnancy and lactation, single-rearing ewes had lower FEC than twin-rearing ewes (P<0.01). However, feeding treatment and litter size interacted (P<0.05); MP feeding did not affect FEC in single-rearing ewes but HP2 ewes had higher FEC than LP2 ewes. Ewe EBV for FEC correlated significantly with observed FEC during lactation only, and when calculated across lactation only for LP ewes (LP1: r=0.70; LP2: r=-0.81; P<0.05) but not for HP ewes (HP1: r=-0.39; HP2: r=0.16; P>0.35).

Conclusion
Response in litter gain to MP supply suggests that MP was limiting for LP ewes. However, in contrast to the expectation (Coop and Kyriazakis, 1999), MP supplementation did not reduce FEC but increased ewe body weight gain in twin-rearing HP ewes, which also had a lower than expected litter birth weight. This could suggest that at times of low body condition score, low weight gain may be prioritised over immunity to parasites. The presence of significant correlations between FEC and EBV for FEC in LP ewes only supports the view that genetic superiority in terms of resistance to parasites may only be observed at times of protein scarcity.

Acknowledgements
A. Kidane received a post graduate scholarship from Quality Meat Scotland, Meat Promotion Wales and English Beef and Lamb Executive, and P. Sakkas from Hellenic State Scholarship Foundation. SAC receives support from Scottish Government.

References