Resistance to gastrointestinal nematodes during the periparturient period is sensitive to specific amino acid deficiency

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Introduction Periparturient resistance to parasites in previously immune mammals often breaks down, resulting in elevated levels of parasitism. The underlying periparturient relaxation in immunity (PPRI) may have a nutritional basis (Coop and Kyriazakis, 1999); immune rats, re-infected with *Nippostrongylus brasiliensis* given access to low crude protein (CP) diets showed an augmented degree of PPRI compared to their high CP counterparts (Jones *et al.*, 2009; Sakkas *et al.*, 2009). As host responses to dietary CP can be seen as responses to essential amino acids, we assessed sensitivity of PPRI to reduced availability of two essential amino acids, i.e. leucine and methionine. Leucine is relatively abundant in proteins of up-regulated immune responses in response to CP supply (Houdijk and Athanasiadou, 2003), and supplemental methonine has been shown to improve resistance to *N. brasiliensis* (Cummings *et al.*, 1986).

Materials and methods Forty rats were infected with 1600 *N. brasiliensis* larvae prior to mating, and upon parturition, allocated to one of four feeding treatment groups, balanced for parturition body weight (n=10). Diets LP and HP were formulated to supply 150 and 250 g CP per kg, respectively. LP protein was methionine-enriched casein, and HP was made by including purified amino acids at levels found in methionine-enriched casein at the expense of starch/sucrose. Diets HP-L and HP-M were identical to HP but without extra leucine and methionine, respectively. The iso-energetic diets were fed at ~90% of metabolizable energy intake on *ad libitum* fed diets (Jones *et al.*, 2009). Parturition litter size was adjusted to 12 pups and dams were re-infected with 1600 *N. brasiliensis* larvae on day 2 of lactation. Dams and litters were weighed daily to estimate litter and dam growth (using linear regression) until either on 6 or 9 days post infection when worm burdens (number and sex) were assessed as a proxy for the degree of PPRI. Worm burdens were log-transformed prior to statistical analysis, and reported as backtransformed means with 95% CI. Results were analysed using ANOVA through REML. Main effects of feeding treatments are reported as interactions with sampling time were not significant.

Results Figures 1, 2 and 3 shows that feeding treatment affected litter growth, dam growth and worm burdens, respectively (P<0.005). HP litters grew faster than LP and HP-L litters, which in turn grew faster than HP-M litters. In a similar fashion, HP dams had higher weight gains than LP and HP-L dams, which in turn grew faster than HP-M dams. HP dams had lower worm burdens than LP, HP-L and HP-M dams, whilst worm burdens for the latter three groups did not differ. Worm burden composition was affected by time only; across feeding treatments, female worm percentage was 55.6% on day 6 and 64.0% on day 9 (s.e.d. 3.3%; P<0.05).

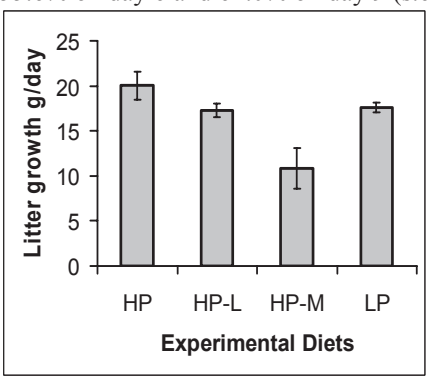

Figure 1 Litter growth

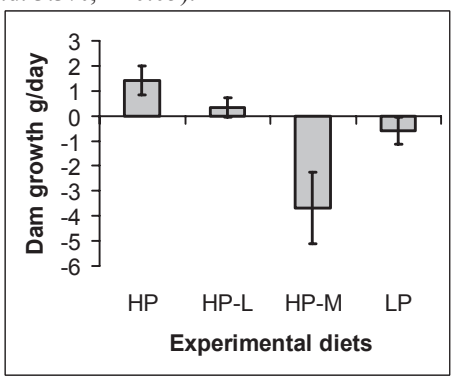

Figure 2 Dam growth

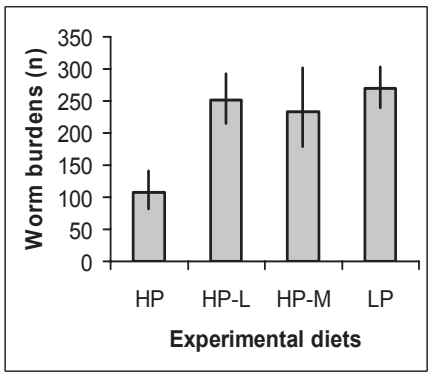

Figure 3 Worm burdens

Conclusion The response in litter gain to increased protein supply suggests that protein was limiting for LP dams. As expected, (Coop and Kyriazakis, 1999), LP dams had consequently higher worm burdens than HP dams. The responses to the deficiency in leucine and methionine may suggest that the underlying PPRI is sensitive to the reduced availability of these specific amino acids. Further studies using this model may be required to identify if the responses observed are the consequence of feeding imbalanced protein *per se* and could lead to identification of an optimal amino acid composition required to reduce the degree of PPRI.

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