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HERITABILITY OF RESISTANCE TO BACTERIAL INFECTION IN COMMERCIAL MEAT RABBIT POPULATIONS

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ABSTRACT

This study was undertaken to determine if routine observational comments on commercial rabbits, including disease incidence, could be used to identify genetic variation for bacterial infection. In two populations heritability for the infection trait (incidence of bacterial infection) was low but significantly different from zero (0.044±0.010 and 0.034±0.006), when the measurement was treated as a continuous variable. Using threshold models the heritability was higher, in the range of 0.14 to 0.37. The incidence of infection in the populations was 7% (rabbits scored at 10 weeks of age) and 4% (rabbits scored at 9 weeks of age), respectively. There appears to be no significant maternal genetic component for the disease trait. Liveweight was heritable in the two populations (0.180±0.034 and 0.159±0.020, respectively) and showed a significant maternal genetic effect (0.1). The genetic correlation between disease incidence and liveweight was low (-0.13) and could only be estimated in one group. Further studies investigating different statistical approaches are warranted and further work is required to refine the scoring system used and define the optimum age of measurement.

Key words: disease resistance, heritability, Pasteurella.

INTRODUCTION

Farmed meat rabbits have the potential to play an important role in income diversification for businesses in rural Australia. The industry is currently worth A$2.4 million dollars and is predicted to double in the next 5 years (CIE 2003). As a developing industry it faces a number of challenges, one of which is disease control. Diseases are largely caused by bacterial infection with *Pasteurella multocida* and *Staphylococcus aureus*. Capital investment in the industry has been low compared to industrial rabbit farming in Europe. Sheds have simple ventilation systems and little temperature control. Scale of production is small, such that batch production of rabbits, using artificial insemination and regular destocking of sheds for cleaning, has yet to be widely adopted.

In Europe, where rabbit farming is well established, approaches to disease control rely on environmental modification such as strict quarantine, batch production of rabbits allowing regular destocking and cleaning of sheds, and a high level of shed hygiene. In addition, there is routine inclusion of antibiotics in rabbit feed and treatment of breeding stock with antibiotics. Some of these methods are under challenge with increasing public health concerns regarding the feeding of antibiotics to livestock. Even with these controls *Pasteurella multocida* infection still occurs, albeit at low incidence and severity.

In Australia and Europe improving genetic resistance of rabbits to bacterial infection has advantages. Genetic variation for bacterial infection has been reported for a range of livestock species (RAADSMA 1995; HERINGSTAD *et al.* 2000) including footrot and fleece rot in sheep, and mastitis in dairy species. There are reports of genetic variation for more general disease expression in pigs (HENRYON *et al.* 2001). In rabbits there are reports of genetic resistance to *Pasteurella*, BASELGA *et al.* (1988) estimating heritability of lung damage, caused by infection, to be 0.12 to 0.28. Our study was undertaken to determine if routine observational comments made on live commercial rabbits, including disease incidence, could be used to identify genetic variation for bacterial infection.

MATERIALS AND METHODS

Animals and data

The study was undertaken in two populations of meat rabbits, housed separately and selected independently for live weight at slaughter age. Population 1 had 281 sires with an average of 62 progeny. Population 2 had 1186 sires with an average of 40 progeny. At 10 and 9 weeks of age, respectively, for the two populations, each rabbit was weighed and inspected for general health and well-being. If a problem was noted for a rabbit, the major cause was recorded. The recording was not specifically to score rabbits for the absence or presence of disease. In Pop. 1, 6.9% of animals had a comment related to bacterial infection and in Pop. 2 the incidence was 3.9%.

Comments that were classified as related to bacterial infection were – respiratory problems, snuffles, eye infection, wry neck, abscesses and any other form of infection (nail, injury, genital). Rabbits with no comment or a comment unrelated to infection were given a score of 0. Rabbits with a comment indicating the presence of an infection were given a score of 1. Rabbits that died prior to the weighing date were omitted from the analysis regardless of cause of death. Numbers that died from infection were small (16 and 13 rabbits in Pop 1 and 2, respectively), and their omission would not unduly bias the results. Sex was determined at time of weighing.

Fixed effects were sex, week+year of birth, parity of dam and number weaned per litter. Sub-classes of each effect were formed to give similar numbers per class. In Pop. 1 the range in values for parity was from 1 to 12 and for number weaned was from 1 to 10. In Pop. 2 the range in values for parity was 1 to 12 and for number weaned was 1 to 14. In both populations parity sub-classes of 1, 2, 3, 4, 5, 6, 7, 8 and 9-12 were formed. In Pop.

1 number weaned sub-classes of 1-5, 6, 7, 8 and 9-10 were formed, and in Pop. 2 the number weaned sub-classes were 1-3, 4, 5, 6, 7, 8, 9 and 10-14.

Statistical analysis

Disease incidence and live weight were analysed using a linear model for the observed data. The statistical package used for all analyses was ASReml (GILMOUR *et al.* 2002). A separate analysis was undertaken for each population. The significance of fixed effects and their first order interactions were estimated, where possible, for each trait. Significant fixed effects were retained in the final models. First order interactions of fixed effects were not significant and were omitted from the final models. The model to determine the significance of fixed effects also included random effects of animal and litter. Once significant fixed effects were determined, the model was expanded to include the additional random effect of a maternal genetic component. The following animal model was used to estimate direct and maternal genetic components of variance for the disease and liveweight traits:

$$Y_{hijklmno} = \mu + A_i + M_j + L_k + r_l + s_m + t_n + u_o + e_{ijklmno}$$

where Y is the trait; μ is the common mean; A_i is the random effect of the *i*th animal; M_j is the random effect of the *j*th dam; L_k is the random effect of the *k*th litter; r_l is the effect of the *l*th week+year; s_m is the effect of the *m*th sex (female of male); t_n is the effect of *n*th parity class u_o is the effect of the *o*th number weaned class and $e_{ijklmno}$ is the normally distributed random error.

Variance components were estimated by the restricted maximum likelihood procedure. From the ratio of appropriate variance components, heritability of each trait was estimated. Correlations were estimated from bivariate analyses, fitting significant fixed effects for each trait independently. Approximate standard errors for heritabilities came from the ASReml analysis. In addition to the linear model, which assumes the trait to be a continuous variable, two threshold models (logit and probit) were used to analyse disease incidence. The three approaches were compared using a sire model with random litter effect and the same fixed effects as in the animal model.

RESULTS AND DISCUSSION

Heritability for the infection trait, when treated as a continuous variable, was low but significantly different from zero (0.044±0.010 for Pop. 1 and 0.034±0.006 for Pop. 2). There appeared to be no significant maternal genetic component for the disease trait. Liveweight was heritable, 0.180±0.034 and 0.159±0.020 for Pop. 1 and 2, respectively. In contrast to the disease trait, there was a significant maternal genetic effect of approximately 0.1. If an animal model is to be used, the correct model for analysing liveweight must include this additional random genetic effect, as omitting it will result in inflated heritability estimates, as observed with these data (Table 1).

For the disease trait the fixed effects of sex and parity of dam were significant in both populations (P<0.01) but number weaned was not. Males consistently showed a higher incidence of infection than females. The pattern of infection for parity classes was not consistent across the two populations, showing a steady decrease with increasing parity in Pop. 1 but a bi-modal pattern in Pop. 2 (Figure 1). For liveweight males were heavier than females by 16g and 11g for Pop. 1 and 2, respectively.

Table 1. Fixed effects and direct genetic effect (heritability, h^2), common litter effect (c^2) and maternal genetic effect (m^2) for infection and liveweight.

	Week +Year	Sex	Dam parity	Litter size	H^2	c^2	m^2
Infection (0 or 1 score treated as continuous variable)							
Pop. 1	**	**	**	ns	0.044±0.010	0.047±0.006	
Pop. 2	**	**	**	ns	0.034±0.006	0.063±0.004	
Infection with maternal genetic effect (0 or 1 score treated as continuous variable)							
Pop. 1	**	**	**	ns	0.041±0.013	0.046±0.006	0.006±0.006
Pop. 2	**	**	**	ns	0.023±0.007	0.062±0.004	0.011±0.005
Live weight (grams)							
Pop. 1	**	**	**	**	0.375±0.028	0.097±0.007	
Pop. 2	**	**	**	**	0.364±0.020	0.161±0.006	
Live weight with maternal genetic effect (grams)							
Pop. 1	**	**	**	**	0.183±0.034	0.085±0.007	0.101±0.022
Pop. 2	**	**	**	**	0.159±0.020	0.130±0.006	0.091±0.014

** P<0.01.

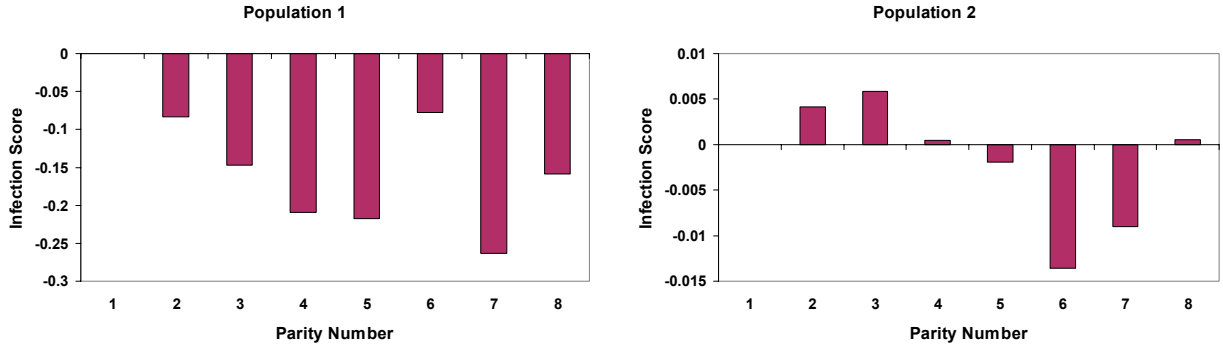

Figure 1. Pattern of infection score with increasing dam parity.

The genetic correlation between disease incidence and liveweight was low in Pop. 1 (-0.13) and not significantly different from zero. In Pop. 2 it could not be estimated, most likely due to non-positive definite variance structure.

Heritability estimates for the infection trait varied from low to moderate depending on the statistical model used (Table 2). The use of a threshold model gave higher heritability as expected, as the transformation to the underlying liability scale reduces the variance of

measurement error (FALCONER 1981). However, the estimates of >0.35 seem improbably high, as the expected magnitude of increase in heritability when going from a continuous to a threshold model would be in the order of 3-fold, for the incidence levels reported here. The use of simulation to investigate the optimum model is warranted, followed by confirmation of realised heritability via selection experiments.

Table 2. Direct genetic effect (heritability, h^2) and common litter effect (c^2) for incidence of bacterial infection using a continuous variable model and logit and probit threshold models, with random effects of sire and litter.

	h^2	c^2
Continuous variable model		
Pop. 1	0.042±0.012	0.056±0.006
Pop. 2	0.035±0.008	0.070±0.004
Logit threshold model		
Pop. 1	0.379±0.106	0.276±0.035
Pop. 2	0.351±0.090	0.356±0.029
Probit threshold model		
Pop. 1	0.133±0.037	0.088±0.015
Pop. 2	Non-estimable	

The value of future data may be improved; in this study the purpose of its collection was not specifically to score for the presence or absence of disease and the predominant symptom was recorded, rather than all symptoms. In research populations (EADY and GARREAU, unpublished data), where incidence of bacterial infection (approximately 10% of animals) has been scored systematically in growing rabbits, heritability estimates are in the range of 0.08 to 0.1, when analysed as a continuous variable.

The incidence of infection will also influence heritability, with low incidence tending to reduce heritability estimates. In practical terms, the usefulness of a routine scoring system may be dependant on the incidence at time of observation. However, with a systematic assessment of rabbits, incidence is likely to be in excess of 5-6% and may also increase with age (EADY and GARREAU, unpublished data), suggesting assessment at 10 weeks rather than 9 weeks may be better. Further information is required for the commercial enterprise in this study to introduce selection for disease resistance, and there are plans to collect more data across a range of ages (8-10 weeks of age).

The single estimate of the genetic relationship between infection incidence and liveweight is slightly favourable but not significantly different from zero. The lack of a significant effect of number of kits weaned on the disease trait suggests it is not strongly correlated with litter size. If it is the case that disease resistance is not strongly genetically linked with other production traits, then it will need to be included as an additional selection trait in its own right to make improvement. Further investigation of the relationship between disease resistance in growers and disease resistance in

breeding does is warranted. There may be a stronger case for a relationship between doe longevity and disease resistance.

CONCLUSIONS

Routine observational data on disease incidence in growing rabbits may be a useful indicator of genetic resistance to bacterial infection, the trait appearing to have a significant genetic component. The magnitude of heritability estimates (0.03 to 0.37) varied with statistical model, higher values resulting from the use of threshold models. Overall, heritability may be improved if rabbits are scored systematically for disease incidence. From initial estimates the correlation of disease with growth rate is slightly favourable, but not statistically different from zero, and there appears to be no strong relationship with litter size. Further studies investigating different statistical approaches are warranted and further work is required to refine the scoring system used and define the optimum age of measurement.

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