GENETIC PARAMETERS OF PRODUCTION TRAITS AND RESISTANCE TO DIGESTIVE DISORDERS IN A COMMERCIAL RABBIT POPULATION

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ABSTRACT

A Bayesian analysis was performed in order to estimate the genetic parameters of a binary trait based on the observed signs of enteropathy, 63-day body weight, carcass yield and perirenal fat percentage in a commercial paternal line. There were 53,222 rabbits inspected for signs of disease and 2,646 slaughtered rabbits for carcass traits from 1999 to July 2007. The incidence of digestive disorder was 8% of the population and 66% of these rabbits died prior to 63-day weighing. Estimated heritabilities were equal to 0.08, 0.36, 0.24 and 0.64 for the disease trait, 63-day body weight, carcass yield and perirenal fat percentage, respectively. The genetic correlations between the disease trait, on one hand, and the 63-d body weight and the carcass yield, on the other hand, were negative (-0.19 and -0.34, respectively) and so favorable. The genetic correlation between the disease score and the perirenal fat percentage was close to zero (-0.07). Therefore, it would be possible to reduce the incidence of enteropathy by including this binary trait in a breeding program, if its relative economic value is high enough to warrant its inclusion.

Key words: Genetic parameters, Carcass traits, Resistance to disease, Digestive disorders.

INTRODUCTION

Intestinal pathology is one of the most important problems encountered in rabbit production and occurs mainly in young rabbits after weaning (Marlier *et al.*, 2003). Digestive disorders are responsible for significant mortality and morbidity (depression of growth and poor feed conversion). Virus, bacteria and intestinal parasites can induce enteropathy. Since 1996 epizootic rabbit enterocolitis has emerged as the major cause of rabbit losses and is associated with a specific clinical pattern that can be defined but the pathogens responsible for this disease have not yet been identified. In commercial populations clinical symptoms of digestive disorders (diarrhoea, bloated abdomen) can be routinely observed and recorded without identifying any clear pathogenic origin.

The major challenge in breeding for any disease is to find measures of resistance that are practical to undertake and have a genetic component. Due to these difficulties much of the research into disease resistance is focused on identifying gene markers. Such studies are a major investment for any livestock industry, and for small industries such as meat rabbits, are less feasible. An alternative approach is to observe signs of disease and investigate the utility of using relatively easy and cheap quantitative measures to improve resistance (Eady *et al.*, 2007).

For a meat rabbit breeding program to improve resistance to disease, we need to identify a measurable and heritable trait related to disease incidence and to estimate the genetic correlations of this trait with the other traits of economic importance in the breeding objective, in order to set up an adequate selection strategy. The goal of this study was to estimate genetic parameters for a binary trait based on the observed signs of enteropathy and production traits in a commercial paternal line.

MATERIALS AND METHODS

Animals and data

The study was undertaken in the AGP39 paternal line (Grimaud Frères Sélection, La Corbière, Roussay, France), which is widely used in terminal crosses in French rabbit production. Matings involved approximately 120 bucks and 270 does each year. The population is reared in overlapping generations and divided in 14 family groups. After weaning at 31 days of age, rabbits were placed in collective cages with 5 individuals each and fed a commercial pelleted food until 63 days of age. The food was restricted to approximately 80 % of average *ad libitum* intake from day 31 to 58 of age and for the final 4 days the rabbits were fed *ad libitum.*

Each rabbit was weighed and inspected for general health and well-being at 63 days of age. Rabbits dying before 63 days were also inspected for signs of disease that may have contributed to their death. If multiple signs of disease were observed for a rabbit (e.g. snuffles and diarrhea), a judgement was made as to the major cause of death and this sign only was recorded.

In addition to the information “live” or “dead before 63 day weighing”, 2 classes of symptoms describing digestive disorders were used: diarrhea and “various digestive disorders”. Rabbits dead or alive with no comment or a comment unrelated to digestive disorders were given a score of 1. Rabbits dead or alive with a comment indicating the presence of a digestive disorder were given a score of 2. There were 53,222 rabbits measured for the study, from 1999 to July 2007. The incidence of digestive disorder was 8% of the population (alive or dead animals until 63 days with a score equal to 2) and 66% of these rabbits died prior to 63 day weighing.

For each dam, 2 to 4 healthy offspring of the first litter were slaughtered in a commercial slaughterhouse, after the 63 day weighing at the breeding farm. The rabbits were weighed again just before slaughter, and after 24 hours of chilling the cold carcasses were weighed. The perirenal fat was removed from the carcass and weighed. Carcass yield (chilled carcass weight/live weight at slaughter), and perirenal fat percentage (weight/chilled carcass weight) were calculated.

Model and statistical analysis

Preliminary univariate analyses using a general linear model (GLM) procedure were performed first to determine the significance of the fixed effects in the form of the ANOVA method, using ASREML (Gilmour *et al.,* 2006). An animal model was used for 63 day weight, carcass yield and perirenal fat percentage. A threshold sire model using a probit transformation was used for the binary trait disease score. For 63-d body weight and disease score the effects retained in the final model were sex, batch (88 classes), parity of dam (5 classes) as fixed effects, and common litter environement as a random effect. The fixed effects of age at weighing (3 classes) and litter size at weaning (8 classes) were also included in the model for weight at 63 days. For carcass yield and perirenal fat percentage, the model included the fixed effect of slaughter batch.

Genetic parameters were estimated in a Bayesian framework using Gibbs sampling, by the TM software developed by the authors (available on request). A threshold (probit) model was used for the disease score, using data augmentation (e.g. Sorensen and Gianola, 2002). Flat priors were used for variance components and fixed effects. Statistical inferences were derived from the samples of the marginal posterior distribution, using two chains of 100,000 iterations. The first 10,000 iterations of each chain were discarded, and samples of the parameters of interest were saved for each of 20 iterations. Statistics of marginal posterior distributions were calculated directly from the samples. Convergence was assessed by visual inspection of the chains.

RESULTS AND DISCUSSION

Means, standard deviations and coefficients of variation of 63-d body weight, carcass yield, perirenal fat percentage and disease score are given in Table 1.

Table 1: Number of records (*N*), means, standard deviations of the means (SD) and coefficients of variation (CV) of 63-d body weight, carcass yield, perirenal fat percentage and disease score

	N	Mean	SD	CV
63-d body weight (g)	45023	2622	287	10.9
Carcass yield %	2646	56.6	1.6	2.8
Perirenal fat %	2646	1.8	0.4	22.2
Disease score	50579	1.08	0.27	25.0

Table 2 shows posterior averages and standard deviations for heritabilities, genetic and phenotypic correlations. Heritabilities of 63-d body weight and carcass yield were moderate (0.36 and 0.24, respectively). Heritability of perirenal fat percentage was larger (0.64). These results are consistent with the values reported in the literature (Lukefahr *et al.,* 1996; Su *et al.,* 1999; Larzul *et al.,* 2005). The positive genetic correlation between 63-d body weight and perirenal fat percentage (0.24) was equal to the value estimated by Larzul *et al.* (2005) but the negative genetic correlation between 63-d body weight and dressing yield (-0.24) is inconsistent with the positive value reported by the same authors. Nevertheless Su *et al.* (1999) reported also a negative genetic correlation between post-weaning daily gain and dressing yield (-0.22).

Table 2: Posterior averages and standard deviations (±) for heritabilities (on the diagonal), genetic correlations (above the diagonal), phenotypic correlations (below the diagonal) for production traits and disease score

	63-d body weight	Carcass yield %	Perirenal fat %	Disease score
63-d body weight	0.36 ± 0.02	-0.24 ± 0.08	0.24 ± 0.06	-0.19 ± 0.10
Carcass yield %	0.10 ± 0.03	0.24 ± 0.06	0.01 ± 0.11	-0.34 ± 0.17
Perirenal fat %	0.25 ± 0.02	0.10 ± 0.02	0.64 ± 0.04	-0.07 ± 0.12
Disease score	-0.42 ± 0.01	-0.10 ± 0.09	0.06 ± 0.07	0.08 ± 0.02

The heritability of disease score (0.08±0.02) was low compared to the estimate of 0.21±0.16 for a similar trait (Garreau *et al.,* 2006) in experimental rabbits. However, the experimental rabbits were

inoculated with ERE and mortality and morbidity were much higher, in excess of 50% and 80% respectively. These results suggest that the heritability of ERE may vary with disease incidence and care should be taken in the choice of which estimate to use in a commercial breeding program as predicted gain and genetic trends may be over estimated if the incidence of the disease is low.

Using the same scoring system in the same population Eady *et al.* (2004) estimated the heritability of incidence of bacterial infection: the reported values (0.04±0.01 with linear model and 0.13±0.04 with a probit threshold model) were close to the present estimated heritability for resistance to ERE. This last study and the present one confirm that routine observational signs of disease can be used as heritable indicator traits for resistance to disease. The relative economic value (REV) given to these traits has now to be defined, so that their merit for use in a breeding program can be assessed. An initial REV for resistance to ERE is reported by Eady and Garreau (2008). A similar approach was used by Eady and Garreau (2007) to investigate the relative economic values of functional traits in rabbit production in Australian conditions.

The genetic correlations between disease score, on the one hand, and the 63-d body weight and carcass yield, on the other hand, were negative and so favorable. The genetic correlation between disease score and perirenal fat percentage was close to zero.These correlations make possible the introduction of this criterion into the breeding objective without unduely compromising genetic progress for the other characters. Estimates of genetic correlations between production traits and disease resistance in rabbits are scarce in the literature, especially between carcass traits and disease resistance for which no results have yet been reported. Eady *et al.* (2004) found also a negative genetic correlation (-0.13) between 70-d body weight and bacterial disease incidence in an other commercial population of the same breeding company. These correlations could be overestimated if phenotypes interact among themseleves. If disease affects growth but growth does not affect disease, the relationship is called recursive. Legarra and Granié (2006) demonstrated how to fit simple recursive models with animal breeding tools and demonstrated that ignoring the possibility of recursive models can lead to overestimated genetic correlations. The value of using such models should be investigated in further studies, in order to better understand the biological relationships between disease resistance and production traits.

CONCLUSIONS

This study demonstrates that routine observational data on non-specific digestive disorders in a commercial line can be a useful indicator of resistance to disease, the trait appearing to have a significant genetic component. Genetic correlations between disease score, on the one hand, and 63-day body weight and carcass yield on the other hands were favourable, suggesting a possible introduction of the disease trait into the breeding objective without compromising the genetic gain for other traits of economic importance. An accurate estimation of the economic value of the trait is required and more work is needed to take into account the possible recursive relations between the disease trait and the production traits. This study provided also some new estimated genetic parameters among production traits using Bayesian and Gibbs sampling methodology.

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