

## GENETIC DIFFERENCES OF *ASCARIDIA GALLI* EGG OUTPUT IN LAYING HENS

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### INTRODUCTION

Lately, animal welfare issues and changes in consumer's demands have resulted in an increased number of birds being kept in alternative production systems such free-range and floor husbandry where the animals are not separated from their faeces. This change has resulted in a renewed importance for helminthoses. The economic impact of endoparasites and the consequences of antiparasitic treatments from chemical residues on food products and the environment as well as the occurrence of drug resistance, has led to an increasing interest in genetic selection for parasite resistance in different host species as an alternative method of control (Gauly and Erhardt, 2001). Results of several studies indicated that some chicken breeds may be more resistant to parasitic infection than others (Ackert *et al.*, 1935 ; Buchwalder *et al.*, 1977). Heritabilities for parasite resistance have not been estimated in chickens so far. Therefore the aim of this study was to estimate the heritability of *A. galli* resistance in two commercial lines. This trait can be of importance for animals kept in alternative and organic farming systems.

### MATERIAL AND METHODS

**Animals and management.** Eighteen week old white (Lohmann LSL, n = 60) and brown (Lohmann Brown, n = 60) layers, born from 8 (6 to 12 hens/sire) and 3 sires (20/sire), respectively, marked with numbered wing tags and reared under helminth-free conditions by a commercial breeder (Lohmann Tierzucht GmbH, Cuxhaven, Germany) were housed in pairs in battery-cages (650 cm<sup>2</sup>/hen) at the Research Station of the Department of Animal Breeding and Genetics, Giessen. A commercial layer diet and water were provided ad libitum. No anthelmintic treatments were given before and during the trial. All the layers were helminth-free at the beginning of the study as confirmed by faecal examinations before artificial infections.

### Experimental infection, faecal egg counts (FEC), clinical examinations and performance.

At the age of 20 weeks, 46 white and 40 brown hens were infected orally with 250 embryonated *A. galli* eggs. Individual faecal samples were collected in monthly intervals and examined by a modified McMaster technique with saturated sodium chloride solution using the MSD counting chamber, adapted to detect minimum egg counts of 50 eggs per gram of faeces. Layers were weighed monthly. Eggs were collected and weighed daily. The mean laying performance and egg weights were calculated for two hens per cage.

**Statistical analyses.** Individual FEC were transformed to log<sub>10</sub> to correct for heterogeneity of variance and produce normally distributed data and the geometric means were calculated. Heritabilities of log-transformed data were estimated with the programm VCE4 version 4.2.5

by Neumaier and Groenefeld (1998) under consideration of the whole relationship matrix. The following model was used :  $y_{ijk} = \mu + s_i + m_j + e_{ijkm}$  ( $y_{ijk} = \log \text{FEC}$  ;  $\mu$  = overall mean ;  $s_i$  = random sire effect,  $m_j$  = fixed laying month effect ;  $e_{ijkm}$  = residual error). FEC at the first date of sampling (first laying month), taken 30 days *p.i.* were excluded from all calculations.

## RESULTS AND DISCUSSION

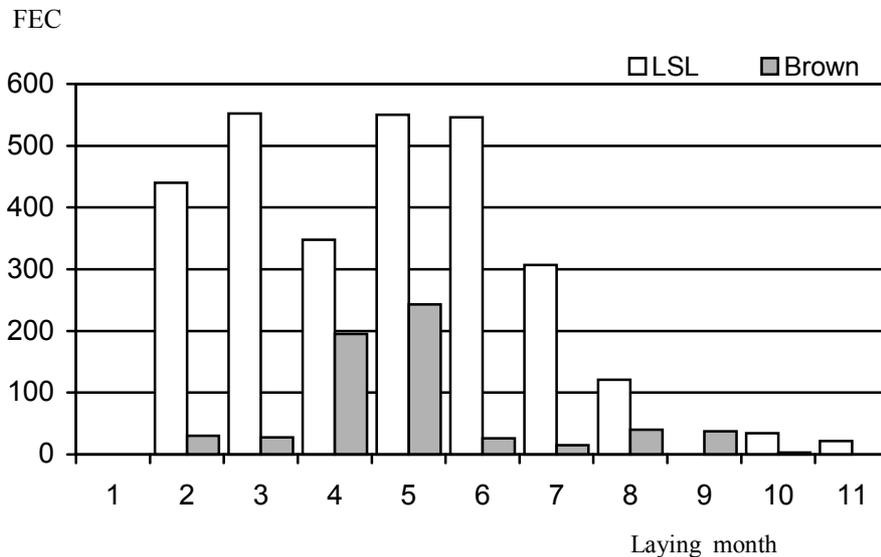
**Ascaridia egg shedding.** All control hens remained uninfected until the end of the trial. The first *A. galli* eggs were detected 2 months *p. i.* in faecal samples. All infected animals developed patent roundworm infection by 5 (brown line) or 6 (white line) months *p. i.* and all shed roundworm eggs at least at two sampling dates. In total, 69.1 % of droppings from white hens (440 samples) and 33.7 % of droppings from brown hens (436 samples) were found to be egg-positive ( $p < 0.001$ ). The differences can not be explained by a different rate of infection because all birds did excrete *A. galli* eggs on at least two occasions. The highest percentage of animals with positive egg counts occurred later in Lohmann Brown hens, which could be caused by differences in the duration of the histotropic phase before the final maturation in the lumen. This phase can vary between 3 and 54 days (Ikeme, 1971).

**FEC values.** The following mean  $\log_{10}$  FEC ( $\pm$  SE) were calculated for the infected groups : white hens, 2.38 ( $\pm$  0.04) ; brown hens, 1.89 ( $\pm$  0.03). FEC and mean log FEC differed significantly between the two lines ( $p < 0.01$ ) and between hens from some sires ( $p < 0.05$ ). In white hens FEC increased from the 2<sup>nd</sup> to the 3<sup>rd</sup> date of sampling, decreased at the 4<sup>th</sup> and increased again. Egg counts decreased significantly ( $p < 0.05$ ) after the 6<sup>th</sup> month. In brown hens FEC were significantly ( $p < 0.01$ ) lower at all sampling dates (figure 1). Because the infection dose was identical for both but the mean egg counts differed, either the rate of worm establishment or the fecundity of established worms was different. Both could be suppressed by host immunity which may have developed sooner and more efficiently in Lohmann Brown causing the rapid decrease of samples with egg counts. The lower mean FEC of the Lohmann Brown hens can be explained by their different genetic background. This is in agreement with earlier studies where heavy breeds proved to be more resistant when compared with White Leghorns (Ackert *et al.*, 1935). Chickens kept under conditions with a higher risk of reinfection, such as free-range, could establish a higher worm burden and FEC. The development of protective immunity in the hens might explain the differences between hens, lines and sires. The decrease of FEC over the study period could also be caused by an increased immunity or because reinfection was not possible given the age of the worms. The life-span of *A. galli* can vary from 9 to 14 months (Hiepe and Schuster, 1992)

**Clinical observations, body and egg weight and laying performance.** No signs of clinical disease were observed in any animal during the study, which may be explained by the low infection dose and resulting low worm burdens. Higher worm counts can cause health problems in chickens (Permin *et al.*, 1997). Birds were kept in cages separated from their faeces which minimized the possibility of reinfection.

The relative body weight development of brown birds was not significantly different between the control and the infected groups, while infected white hens showed on average a 2.3 % lower body weight development rate ( $p < 0.05$ ) than non infected control birds. *A. galli* infections can have a negative effect on weight gain in chickens (Ackert, 1931 ; Gauly *et al.*,

2001). The relationship between parasite and weight in white hens shows the potential impact of the parasite on economically important traits. No significant differences of the laying performance and mean egg weight were found between infected and control groups of both lines. This may be different in animals with higher infection rates. Laying performance was 91 % for the white and 86 % for the brown hens during the 11 months laying period. Mean egg weight was 59.2 g ( $\pm 0.38$ ) (white hens) and 62.7 g ( $\pm 0.43$ ) (brown hens).



**Figure 1. Mean Ascaridia egg counts in white (Lohmann LSL) and brown (Lohmann Brown) laying hens artificially infected with 250 *A. galli* eggs at an age of 20 weeks during the following laying period**

**Heritability of FEC.** The estimated heritabilities for total FEC was 0.0 for brown hens and 0.13 (SE : 0.029) for white hens. Heritabilities estimated for FEC for months 2 to 7 were 0.10 (SE : 0.041) and 0.19 (SE : 0.039), respectively. The value agrees with heritabilities estimated for nematode resistance in sheep, where breeders have started to integrate this parameter into breeding programs (Gray, 1997 ; Kominakis and Theodoropoulos, 1999 ; Gauly and Erhardt, 2001). The lower FEC of the Lohmann Brown did lead to a lower value of heritability. The estimated heritabilities for the period of higher excretion (2 to 7 months) was therefore higher. The lower value compared with the white may also reflect the limited number of sires.

**CONCLUSION**

In conclusion, it should be possible, to select for *A. galli* resistance in both chickens lines. This approach may be of importance in poultry husbandry relying on alternative and organic housing systems.

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