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Veterinary Parasitology 128 (2005) 141–148
veterinary parasitology
www.elsevier.com/locate/vetpar

Age-related differences of *Ascaridia galli* egg output and worm burden in chickens following a single dose infection

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Received 11 March 2004; received in revised form 13 October 2004; accepted 12 November 2004

Abstract

Ninety white chickens (Lohmann LSL) were reared under helminth-free conditions and divided into five groups. Four groups were artificially infected with 250 embryonated *Ascaridia galli* eggs at the age of 6, 12, 18 or 24 weeks. Ten birds were kept as uninfected controls. Six and 10 weeks after infection (p.i.), individual faecal egg counts (FEC) were performed. The birds were slaughtered after the second sampling and their gastrointestinal tracts were examined for the presence of adult *A. galli*.

The FEC increased from the first to the second sampling significantly in all the infected groups. The highest increase was shown in the group infected at 12 weeks of age, whereas the increase in the other groups was relatively moderate. However, the total worm burden and mean FEC at the second sampling were highest ($p < 0.01$) in those birds infected at an age of 12 or 18 weeks. The serum protein and triiodothyronine (T3) levels did not differ significantly ($p > 0.05$) between any of the groups. Thyroxine (T4) was significantly different between the groups infected at 6 and 18 weeks of age ($p < 0.05$), and those at 6 and 24 weeks of age ($p < 0.01$). The thyroid hormone levels correlated significantly with the FEC.

Age does not seem to play a major role in resistance to *A. galli* infections in layers, whereas a bird's hormonal and immune status, related to laying activity, seems to have a significant negative impact on resistance.

Keywords: *Ascaridia galli*; Age resistance; FEC; Chicken parasitological disease

1. Introduction

Ascaridiosis is a cause of economic losses in poultry free-range and floor production systems (Permin et al., 1997; Ponnundurai and Chellappa, 2001). The nematode's direct life cycle and the environmental resistance of its eggs favour infections under these conditions (Permin and Ranvig, 2001). *Ascaridia galli* can cause a reduction in growth rate, weight loss (Hiepe and Schuster, 1992; Ramadan and Znada, 1991) and damage to the intestinal mucosa, leading to blood loss and secondary infection (Ackert and Herrick, 1928). Toxins of *A. galli* adversely influence enzyme systems in the intestinal mucosa and interfere with the normal absorption of nutrients in the intestine (Vassilev et al., 1973).

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doi:10.1016/j.vetpar.2004.11.023

Various studies have indicated that some chicken breeds are more resistant to *A. galli* infections than others (Gauly et al., 2001a,b; Schou et al., 2003). In addition to the genetic factors involved in the establishment and survival of *A. galli* in the intestine, other factors seem to influence the resistance against the parasite in the chicken. Johnson et al. (1974) have shown that immunosuppressive drugs significantly increase the numbers of *A. galli* and the incidence of infection. While Egerton and Hansen (1955) have found that chickens produce a humoral factor in response to an *A. galli* infection. These authors also concluded from their studies that both an age-related immunity and tolerance in chickens toward *A. galli* exist. These findings confirmed earlier studies by Herrick (1926) and Ackert and Herrick (1928), where birds at four months of age were more resistant than one-month-old chicks. Ackert et al. (1935a,b) determined the differences in age resistance on the length of the nematodes removed from the chickens. Tongson and McCraw (1967) described a decrease in the total *A. galli* burden in male white Leghorn chicks infected at the age of 2, 4, 8, 12 or 16 weeks.

Resistance to *A. galli* infections in relation to age and reproductive activities in layers have not been described. Therefore, the aim of this study was to estimate the effects of age and reproductive status in relation to resistance to a single *A. galli* infection in white chickens (Lohmann LSL).

2. Materials and methods

2.1. Animals and management

Ninety white chickens (Lohmann LSL) marked with numbered wing tags were reared under helminth-free conditions. At the age of 6 weeks they were randomly allocated into four different groups with 20 birds per group and housed in battery cages (650 cm^2/hen) in a windowless barn at the Research Station of the Department of Animal Breeding and Genetics, Giessen. The remaining 10 birds were kept as controls under same conditions. A commercial diet and water were provided ad libitum. The energy levels of the diets were 12 (1–3 weeks of age) and 11.4 MJME (>3 weeks of age). The protein levels were 21.0% (1–3 weeks of age), 18.5% (4–8 weeks of age), 14.5% (9–16 weeks of age) and 17.5% (>16 weeks of age).

The birds were vaccinated against Newcastle disease. The light programme followed recommendations for commercial chickens. The light decreased from 24 h (days 1–2) to 16 h (days 3–6), and then stepwise to 8 h in week 8 of life. The hours were then increased after the age of 19 weeks stepwise to 16 h at the age of 25 weeks. The light intensity was between 2 and 3 W/m^2. Anthelmintic treatment was not given before or during the trial.

2.2. Experimental infection

Twenty birds were infected orally with 250 embryonated *A. galli* eggs at the age of 6 (group 1), 12 (group 2), 18 (group 3) or 24 (group 4) weeks. The eggs had been harvested from the uteri of adult roundworms and incubated at 20 °C in a 4% potassium bichromate solution for 14 days before infection. This procedure was repeated for every group. Additional ten birds were kept as uninfected controls.

2.3. Parasitological measurements

Individual faecal samples were collected 6 and 10 weeks after the infection (p.i.). The total number of samples taken per group was 40. Faecal egg counts (FEC) were performed using a modified McMaster technique (MAFF, 1986) with saturated sodium chloride solution using the MSD counting chamber, adapted to detect minimum egg counts of 50 eggs per gram of faeces.

All the birds were slaughtered after the second sampling. The gastrointestinal tract was removed, opened longitudinally, and washed in tap water. The contents were poured into a sieve (pore size 100 μm), washed and examined for the presence of adult *A. galli*. All adult worms were counted and sex was determined by morphological parameters (James and Ackert, 1931). Length was measured with a ruler. Worms were weighed on an electronic scale with a precision of ±0.05 g.

On the day of slaughtering, blood was taken from all the birds. The blood was centrifuged (3600 U/min, 10 min) and the serum removed and frozen at −18 °C until analysis. Serum thyroxine (T4) and triiodothyronine (T3) concentrations were determined using a