Commercial Layer-type Chickens and Newcastle Disease Virus Infection: Toward Genetic Selection of More Resilient Chickens

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Summary and Implications

Exotic Newcastle Disease Virus (NDV) causes major losses due to extremely quick mortality in chickens after exposure to the virus. In places where this virus is not effectively controlled through vaccination and biosecurity, people rely heavily on poultry to provide protein and income. Losses from NDV contribute to worldwide hunger and poverty. It may be possible to use genetic selection to produce chickens that not only have a stronger immune response in the face of NDV challenge but also respond better to vaccination. In order for genetic selection to be successful, two major elements are required: differences in immune response between chickens and genetic control of these differences. This study clearly demonstrates the existence of both these factors. These findings provide strong possibility for the ability of genetic selection to produce chickens that are more resistant to NDV and thereby lessen the burdens of hunger and poverty.

Introduction

Exotic strains of NDV can cause severe losses due to mortality, in excess of 80%. Newcastle Disease Virus, in its velogenic form, is endemic in many parts of the world, including Africa. These regions do not have agricultural outputs on the same scale as in the US and people rely heavily on village poultry to provide protein and income for their families.

Not every chicken responds equally to viral infections and some of this variation likely is due to genetic differences between birds. Thus, some chickens may have 'better' genetics than others for responding to viral infections. Animals that respond to infection favorably can be selected as parents to produce the next generation and subsequently improve the response of their offspring to NDV.

One method to determine how well a chicken responds to a viral challenge is to measure antibody level following infection. Antibodies are produced by the immune system in response to infection and help stop the virus from infecting more cells. Another method is to measure viral load, or viral content that is present in the bird. The amount of virus measured in a chicken across the course of infection can be an indication of how quickly the chicken's immune system is able to clear the virus after infection.

Chickens that are capable of producing more antibodies and interfering with viral replication more quickly tend to recover from the infection quicker and experience less internal damage. These chickens are less likely to die from infection and would be able to provide a source of protein and income.

This experiment measured differences in antibody production to NDV and viral content levels between chickens and estimated the genetic control of these traits. Body weights were also recorded during the study period to quantify the impact of viral infection on growth.

Materials and Methods

In this experiment, 540 birds from a commercial layer line were infected with a mild (LaSota) strain of NDV at 21 days of age. The virus was administered via the ocular-nasal route. Antibody titers were measured using a commercial ELISA kit before and after a 10-day course of infection. Counting copies of the virus' genetic material in lacrimal fluid (via RT-qPCR) 2 and 6 days post infection was used to quantify the rate of viral clearance. Growth rate post infection was calculated using a regression analysis of body weight at 0, 6, and 10 days-post-infection on age.

DNA samples were collected from all birds to determine genotypes for 326,000 genetic markers across the genome using the Affymetrix 600k SNP chip. Statistical methods were used to determine how much of the antibody production, viral load, and growth were affected by genetics versus environmental effects. A calculation known as heritability measures the proportion of variation in phenotype (in this case antibody titer) that is due to the variation in genetics (measured from genotypes on the 600k SNP chip).

Results and Discussion

Results showed that all infected chickens produced antibodies to NDV, measured 10 days after viral challenge. A range of titers was observed (Figure 1). The heritability for antibody titer post infection was 0.10 meaning that 10% of the difference in response to NDV between the chickens in this experiment was due to genetic diversity within the group. This heritability measurement is substantial enough for selection of improved antibody production to be successful.

Viral load measured at 2 and 6 days post infection demonstrated a reduction from day 2 to day 6. Similar to antibody titer, a range of load was observed at both time

points (Figure 2). Heritability of viral load at 2 and 6 days post infection was estimated to be 0.19 and 0.05 respectively.

Growth post-infection was also a variable trait within the experimental group (Figure 3). Heritability of growth post-infection was estimated at 0.19, indicating genetic selection could result in birds that continue to grow during an NDV infection.

Acknowledgments

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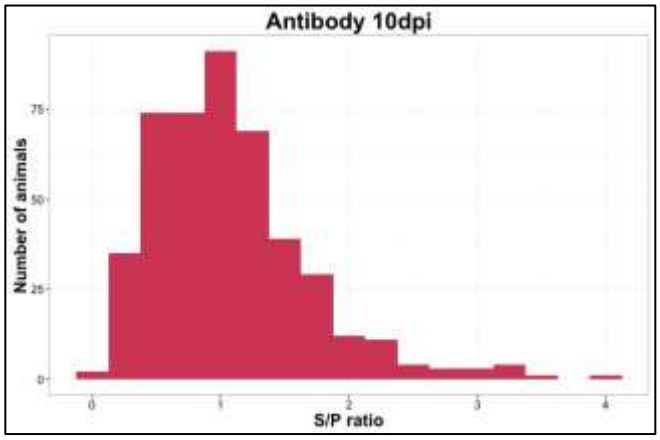

Figure 1. Distribution of antibody titers 10 days post infection

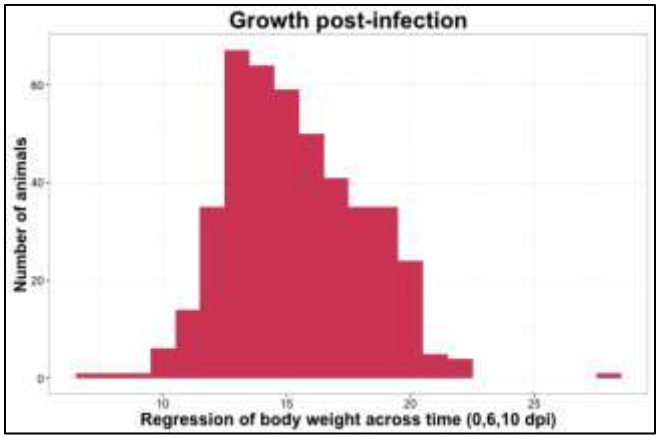

Figure 3. Distribution of growth post-infection

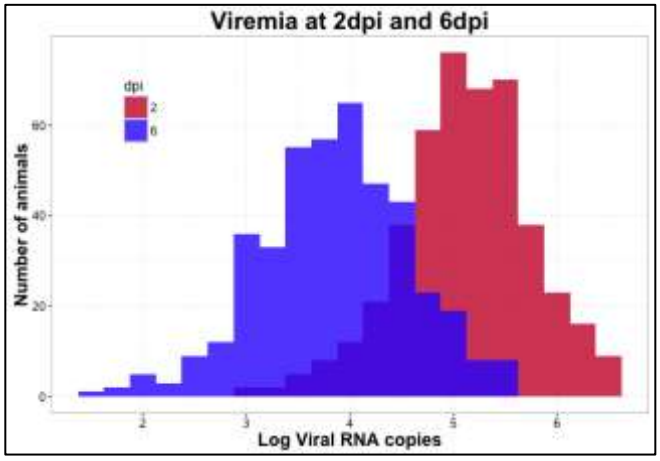

Figure 2. Distribution of viral load 2 and 6 days post infection