Genetic Basis of Resistance to Avian Influenza

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Summary and Implications

Two high pathogenic avian influenza (HPAI) outbreaks have severely affected the poultry industry on the American continent within the last four years; a 2012 H7N3 outbreak in Mexico and a 2015 H5N2 outbreak in the US. Blood samples were collected from survivors of each outbreak plus age and genetics matched non-affected controls. As surviving birds could contain natural genetic mutation(s) that make them resistant to HPAI, the goal of the present study was to identify genomic regions associated with resistance to HPAI and to determine whether resistance regions are the same for different virus strains. Four genomic regions were identified for the H5N2 outbreak and five different regions were identified for the H7N3 outbreak. The apparent different genomic regions of resistance for different virus strains is a challenge for the poultry industry, as it requires a more diversified strategy for improving resistance to AI.

Introduction

Highly pathogenic avian influenza (HPAI) outbreaks have a devastating impact on the poultry industry, causing reduced egg production, nearly 100% mortality, and mandated euthanization/depopulation of infected flocks. HPAI outbreaks differ significantly from low pathogenic avian influenza virus (LPAI) infections which often show only subclinical symptoms. These pathogenicity differences between HPAI and LPAI, as well as between different HPAI strains, may be related to specific characteristics of virus strains and host genetics. Identification of genetic basis of the HPAI resistance of chickens is an active field of research and would be very beneficial for the industry.

Materials and Methods

Samples for this study were obtained following two different HPAI outbreaks in multiple commercial layer facilities in Mexico and the US. A total of 12 flocks were sampled in Mexico and 3 flocks in Iowa. In total, 845 individuals were used in this study, 564 survivors and 412 age- and genetics-matched controls (random individuals from uninfected flocks). Genotypes at 600,000 genetic markers across the genome were determined using the Affymetrix SNP chip. Statistical analysis was performed to identify genomic differences between survivors and controls. As no single genetic marker showed large differences, the effects were summarized in 1 Mb windows across the genome.

Results and Discussion

Figures 1 and 2 show regions associated with HPAI survival for the H5N2 and H7N3 outbreaks, respectively.

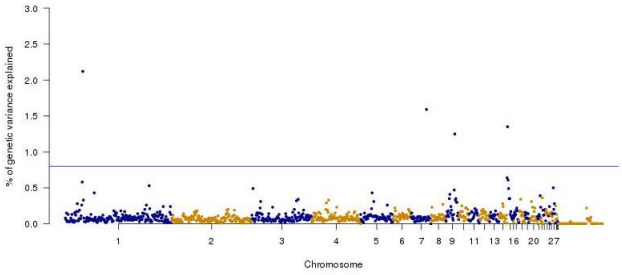

Figure 1. Windows explaining highest percentage of genetic variance for H5N2 survival.

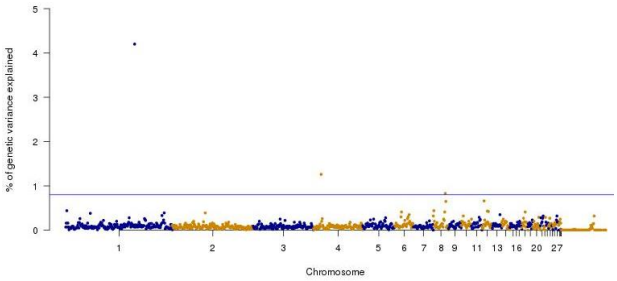

Figure 2. Windows explaining highest percentage of genetic variance for H7N3 survival.

Several genomic regions were identified as potentially associated with resistance to HPAI, but none had a very large effect. The strongest associations were located on chromosomes 1 (32 Mb), 7 (28 Mb), 9 (14 Mb) and 15 (1 Mb) for H5N2 strain and on 1 (126 Mb), 4 (14 Mb) and 8 (23 Mb) for H7N3 strain. A number of genes connected to immune response are located in these regions. Different genetic regions were identified as associated with resistance to H7N3 vs H5N2 virus. Identification of genomic regions associated with HPAI and further analysis of genes located in these regions can provide targets for genetic selection for resistance to HPAI, identify candidate genes involved in viral resistance mechanisms and provide targets for vaccine development.

Acknowledgments

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