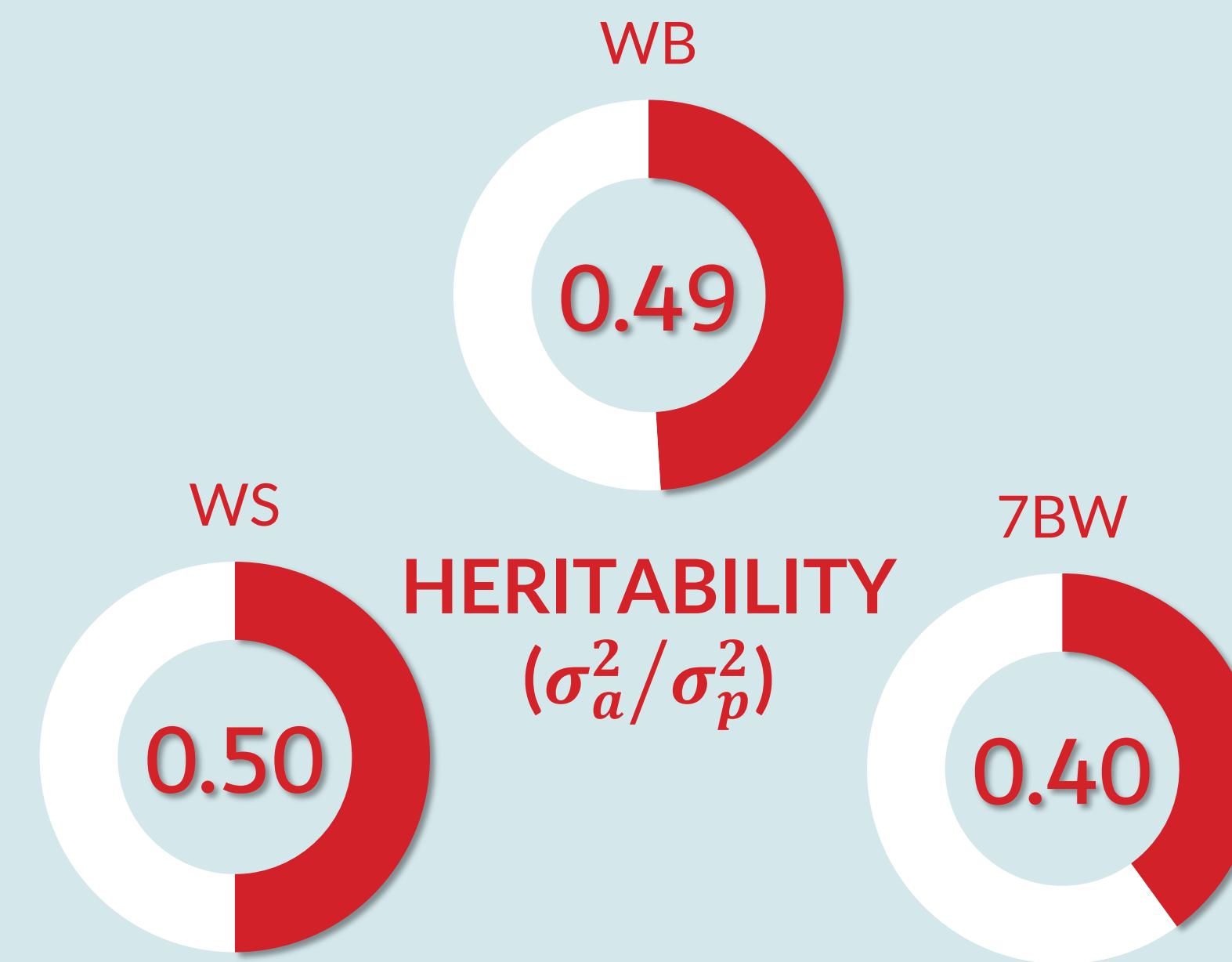


GENETIC BASIS OF WOODEN BREAST & WHITE STRIPING IN COMMERCIAL BROILERS

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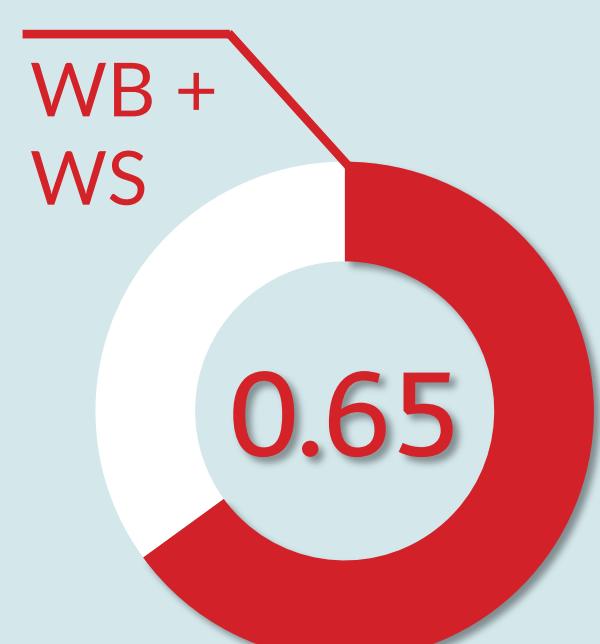
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ABBREVIATIONS

WB	wooden breast
WS	white striping
7BW	body weight at 7 weeks
SNP	single nucleotide polymorphism
GBS	genotyping by sequencing
QTL	quantitative trait loci
GRM	genomic relatedness matrix

PHENOTYPIC CORRELATION



GENOTYPIC CORRELATION

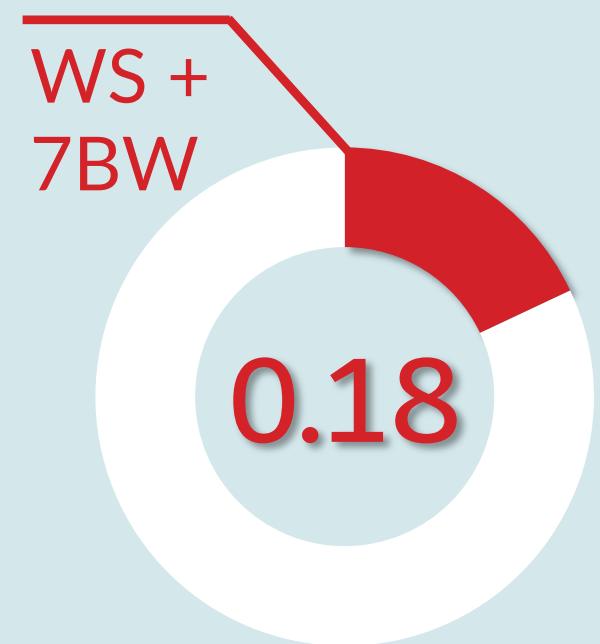
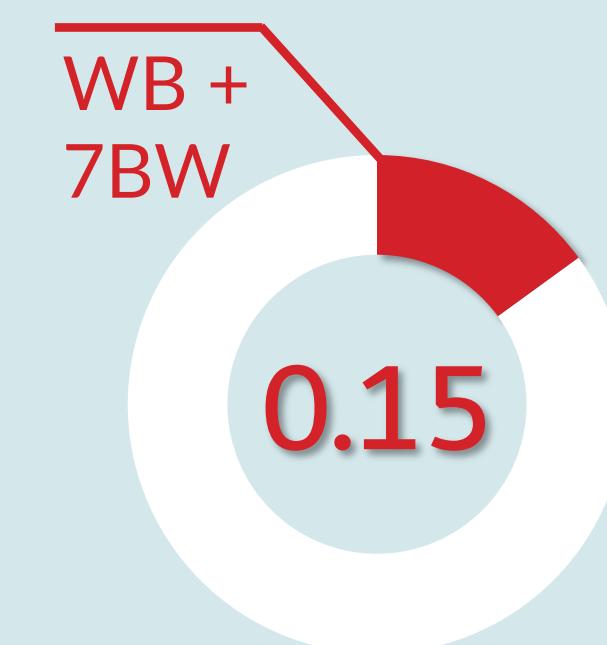
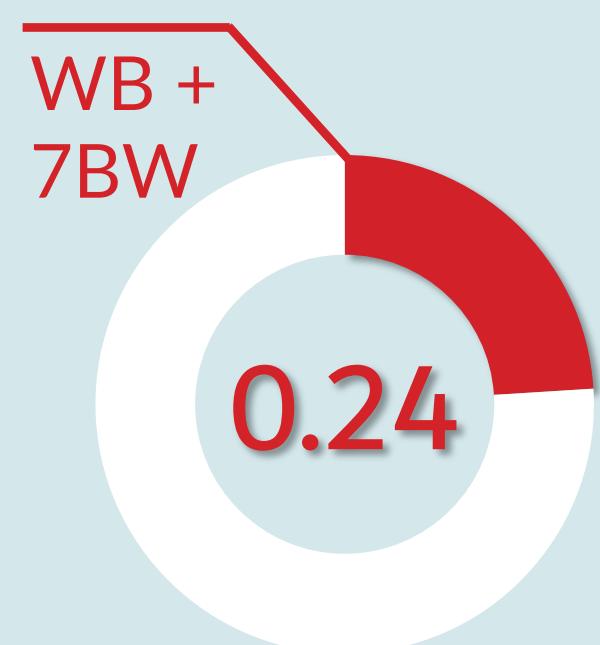
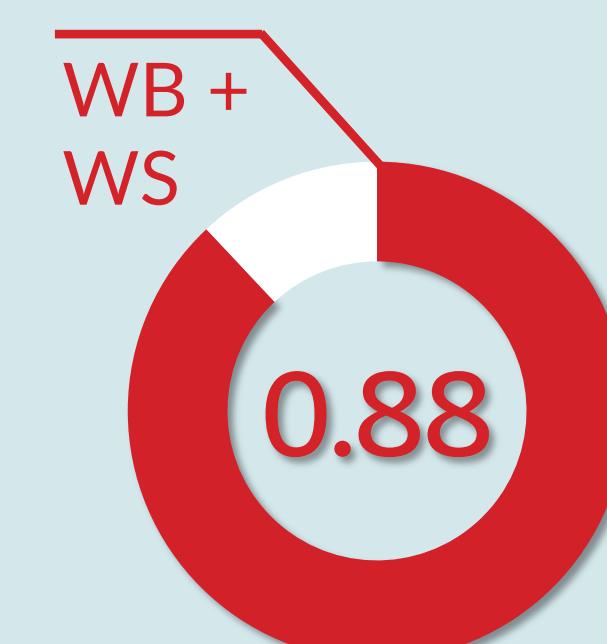


FIGURE 1. WOODEN BREAST

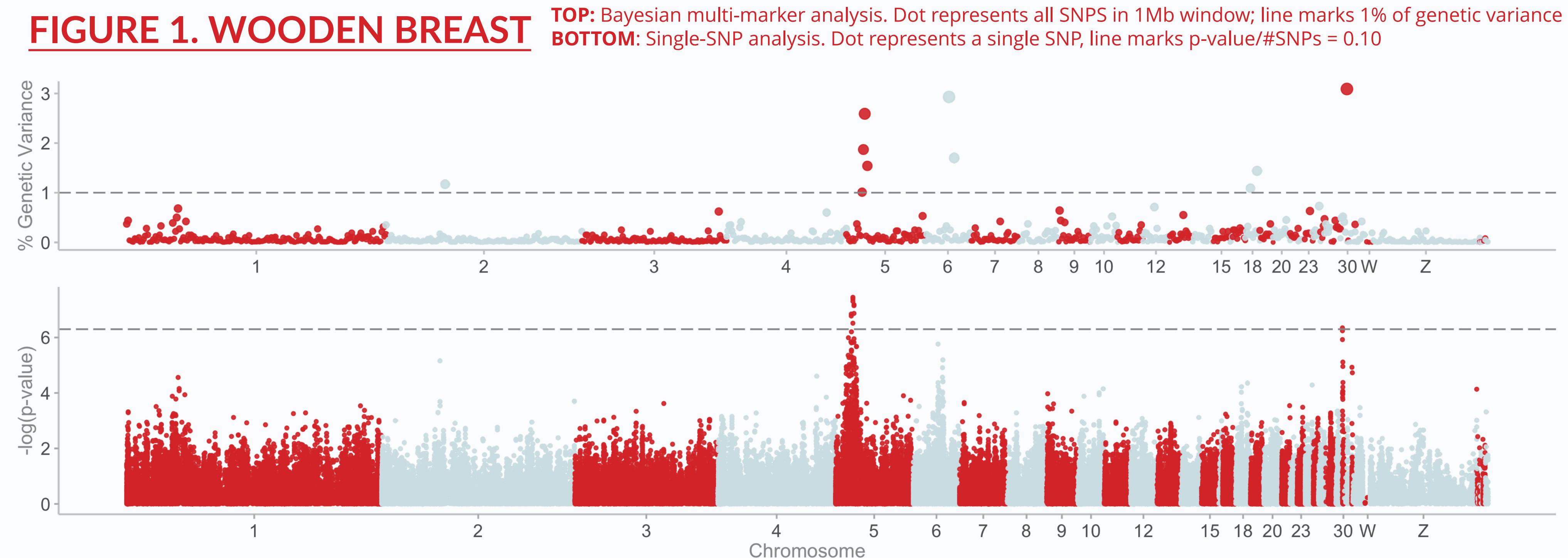
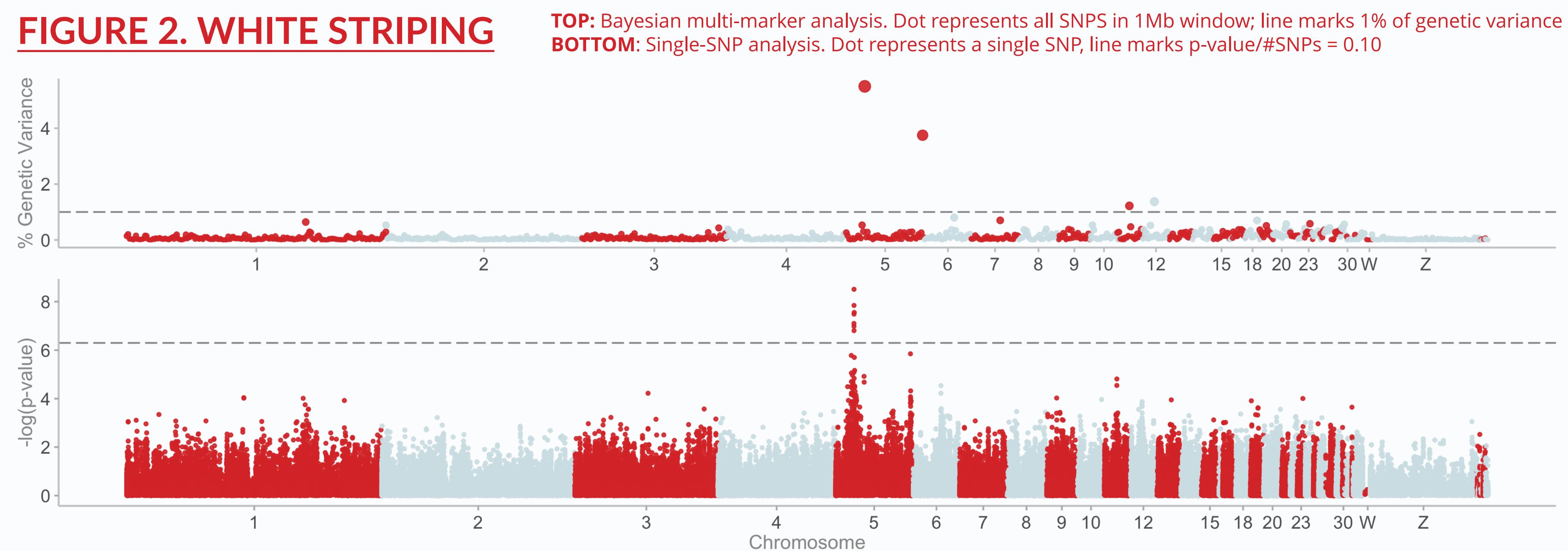


FIGURE 2. WHITE STRIPING



INTRODUCTION

Wooden breast (WB) and white striping (WS), the two major myopathies of fast-growing broilers, continue to threaten global poultry production due to their severe impacts on meat quality. Tightly associated with economic traits such as growth rate, feed efficiency, and breast muscle yield, these muscle disorders present an exceptional challenge to producers, as dietary or management strategies against WB and WS generally impair performance. An understanding of the genetic basis of these myopathies is therefore critical for developing a long-term solution.

The objective of this study is to estimate genetic parameters for WB and WS and identify genetic variants associated with the two myopathies in broiler chickens. The results described herein improve our understanding of the genetic architecture of these myopathies and suggest that WB and WS would respond to selection.



ACKNOWLEDGEMENTS

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METHODS

A total of 1,194 mixed male (n=564) and female (n=630) Cobb 500 broilers from the same breeding population of 15 sires and 200 dams were raised as two separate hatches to seven weeks of age and evaluated for WB, WS, and body weight.

Whole blood from progeny and sires was used for total DNA extraction and genotyping with restriction enzyme-based low-depth GBS. SNPs were filtered to keep those with a minimum read depth of 5 for at least 50% of samples and a minor allele frequency of 5%. Samples were filtered to only include those with a minimum read depth of 5 for at least 50% of SNPs.

A GRM was constructed from variant data using the R package KGD. Genotype posterior probabilities were estimated using the Bayesian genotype caller polyRAD, with population structure and linkage disequilibrium as a prior, and were subsequently converted to allele dosages for use in association analyses.

Variance components and heritabilities were estimated from the GRM using ASReml 4 with fixed effects of sex, poultry house, and age at necropsy. QTL were detected using two separate approaches – Bayesian multi-marker regression and single-SNP analysis. Bayesian models involved first estimating the proportion of markers with null effect (π) using BayesC π followed by estimation of genetic variance explained by each 1-Mb window with BayesB.

RESULTS

After quality control, 198,778 SNPs with mean depth of 10.86 remained and were used for all analyses.

Heritability was estimated to be moderate for all traits: 0.49 ± 0.07 for WB, 0.50 ± 0.06 for WS, and 0.40 ± 0.06 for body weight. Genetic correlation between WB and WS was high (0.88 ± 0.04), but genetic correlation between either WB or WS and body weight was low (0.15 ± 0.11 and 0.08 ± 0.11 , respectively).

Multi-marker analysis of WB found ten 1-Mb regions that each explained $>1\%$ of genetic variance (Figure 1 top). Together, these eight regions explained 18.4% of the genetic variance for WB. Multi-marker analysis of WS (Figure 2 top) identified four 1-Mb regions that each explained $>1\%$ of genetic variance.

Single-SNP analyses (Figure 1 bottom, Figure 2 bottom) were consistent with results from BayesB and identified SNPs with genome-wide significance even using the very conservative Bonferroni multiple testing correction.

FUTURE WORK

Significant regions will be scrutinized to identify candidate genes and sequencing data will be compared with gene transcripts to detect potential functional variants in protein coding genes. Linkage disequilibrium will be estimated from variant data to determine if coverage in the present study was sufficient for detecting QTL.