

Liver and whole blood transcriptome response to chronic heat exposure in laying hens

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CDH2, MEF2A, FoxO3, MAT1A, and TNNT2) were identified to regulate muscle growth involving in pathways like muscle organ development and muscle organ maintain. Eight genes (FABP5, ENPP3, NGEF, INSIG1, PCSK1, PLPPR1, ADORA1, and ACSL1) involving pathways like PPAR signalling were identified to regulate lipid deposition.

Key Words: ducks, integrative genomics, candidate gene

53 Liver and whole blood transcriptome response to chronic heat exposure in laying hens. F. Jehl¹, A. Rau¹, C. Désert^{2,3}, M. Boutin^{2,3}, K. Muret^{2,3}, S. Leroux⁴, D. Esquerré⁵, C. Klopp⁶, D. Gourichon⁷, F. Pitel⁴, A. Collin⁸, S. Lagarrigue^{2,3}, and T. Zerjal*¹, ¹GABI, INRA, AgroParisTech, Université Paris-Saclay, Jouy-en-Josas, France; ²INRA, UMR1348 Physiologie, Environnement et Génétique pour l'Animal et les Systèmes d'élevage, Saint-Gilles, France; ³Agrocampus-Ouest, UMR1348, Rennes, France; ⁴UMR INRA/INPT ENSAT/INPT ENVT-GenPhySE, Castanet Tolosan, France; ⁵INRA, Plateforme GENOTOUL, Castanet-Tolosan, France; ⁶INRA, SIGENAE, Castanet-Tolosan, France; ⁷IN-RA-PEAT, Nouzilly, France; ⁸URA, INRA, Nouzilly, France.

Adaptation to heat exposure is required to maintain animal welfare and productivity under high ambient temperature (AT) conditions. In this study we investigate the effects of chronic heat exposure (5 weeks at a constant temperature of 32°C) on the liver and whole blood transcriptome of brown egg layers from 2 divergent lines selected for low (R-) and high (R+) residual feed intake. The R+ and R- hens were equally distributed among 2 temperature-controlled chambers and reared under thermo-neutrality (22°C). At 28 wk of age the AT of one chamber was increased to 32°C until 33 wk of age, when 32 animals (8 per line and treatment) were slaughtered. Total RNA was obtained from the liver and blood and was sequenced using the Illumina HiSEqn 3000, yielding an average per sample of 90 million paired-end reads. The reads were mapped to the Gallus gallus-5 reference genome by STAR software and counted by RSEM software using the Ensembl V87 GTF annotation. Comparisons between the two AT groups were made using the edgeR-robust R/Bioconductor package. Patterns of AT-specific differential expression were largely shared by the two lines, and no evidence of temperature × line interactions were observed. In liver, a total of 229 differentially expressed genes (DEG) were identified (adjusted *P*-values < 0.05) with respectively 104 and 125 over and under expressed in the heat-exposed compared to the control group. In blood, 960 DEG were identified between the two AT groups with 479 and 481 over and under expressed. Most DEG were tissue specific, and only 18 genes were DE in both liver and blood. Ingenuity Pathway Analysis revealed that many of the DEG in liver were associated with amino acid and lipid metabolisms and energy production. Key genes involved in *fatty acid* β -oxidation, ketogenesis, cholesterol biosynthesis were under-expressed in the heat-exposed animals. In blood, many of the DEG were associated with cell related functions. Based on the DEG expression profile, down-regulation was observed for the PI3K/AKT, the VEGF and the PDGF signalling pathways involved in cell survival and growth, vasculogenesis and angiogenesis. Taken together, these results indicate a tissue-specific response to heat exposure.

Key Words: heat exposure, liver, blood, RNA-seq, laying hens

54 Mapping QTLs affecting Marek's disease by selective DNA pooling in eight lines across 15 generations. E. Lipkin*¹, J. Smith², D. Burt², M. Soller¹, and J. Fulton³, ¹Dept. of Genetics, Silberman Life Sciences Institute, The Hebrew University of Jerusalem, Jerusalem, Israel; ²The Roslin Institute and Royal (Dick) School of Veterinary Studies, The University of Edinburgh, Easter

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Marek's disease (MD) is a major disease affecting the poultry industry. The aim of the present study was to map QTLs, genes and mutations affecting chicken MD mortality. Selective DNA pools were made of 9,391 sires of 8 lines and 15 generations per line, that had progeny test results for MD mortality. The pools were genotyped using an Affymetrix 600 K chicken SNP array. Allele frequencies were obtained by raw intensities of alleles A and B, B% = B/(A+B). The significance of allele frequency differences between sires with low or high progeny mortality was obtained using empirical SE. Moving average of -LogP and Log drop were used to define QTL regions (QTLRs). A total of 42 QTLRs were found among the eight lines, averaging 32 QTLs per line and ranging from 0 to 11. Overlap and proximity were used to condense the QTLRs to 28 QTLRs shared across families. The shared QTLRs averaged 1.2 Mb in length and covered a total of 33.3 Mb (3.1% of the entire genome). RNASeq and bioinformatics analyses of the QTLRs identified differentially expressed and functional candidate genes. Markers from the candidate genes were used to individually genotype 9,391 individuals from the same lines. Association analysis of the individual genotyping confirmed most of the QTLs found by the pools. Putative quantitative trait genes (QTG) and candidate causative nucleotides (QTN) were identified. This study confirmed most of the QTLRs found by the F6 study reported elsewhere at this meeting.

Key Words: poultry, genome-wide association, quantitative genetics, animal health, complex trait

55 Genome-wide association study of complex traits in response to Newcastle disease virus in chickens. K. Rowland*¹, H. Zhou², R. Gallardo³, T. Kelly^{2,3}, A. Wolc^{1,4}, and S. J. Lamont¹, ¹Iowa State University, Department of Animal Science, Ames, IA, USA; ²University of California-Davis, Department of Animal Science, Davis, CA, USA; ³University of California-Davis, School of Veterinary Medicine, Davis, CA, USA; ⁴Hy-Line International, Dallas Center, IA, USA.

Newcastle disease (ND) causes up to 80% mortality in chickens in developing countries where velogenic NDV strains are endemic. Genetic improvement of disease resistance, complementary to vaccination, has the potential to be an important tool to reduce the impact of NDV. We hypothesise that many genes regulate NDV response in chickens. Our specific objective was to identify genetic markers associated with NDV resistance (reduced viral load, high antibody titer) so these markers can be applied in a genetic selection program benefiting areas of endemic NDV challenge. The experiment was replicated across three hatches from 150 dams of a commercial egg-laying line, Hy-Line Brown. We inoculated with NDV, La Sota strain, on day 21 of age by an ocular-nasal route. Virus load was estimated from viral mRNA level in lachrymal fluid by qRT–PCR. Systemic antibody response to NDV in serum was measured by ELISA. Genomic DNA was genotyped via Affymetrix 600K chicken SNP array. Analyses of viral RNA and antibody levels confirmed response of challenge groups to the virus and lack of response in control groups. ASReml estimated heritabilities of 0.34, 0.34, 0.105, 0.117, 0.19, and 0.05 for hatch weight, day 31 body weight, 0 and 10 days post-infection (dpi) antibody levels, and 2dpi and 6dpi viral load, respectively. Genome-wide associations with 250 kb windows were tested using the Gensel program. BayesC was used to estimate variance components, subsequently, BayesB (pi=0.999) tested associations. Several QTL regions were found to confirm previously reported QTL for these traits. Identification of novel SNPs and regions will provide insights into genetic control of response to NDV infection in chickens as well as the genetic archi-

ISAG 2017 Abstract Book 15