ISAG 34th XI'AN 2014

ABSTRACT

2014.7.27 - 8.1

The 34th International Society for Animal Genetics Conference

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P1011 Genome-wide Association Studies with Somatic Cell Score in Chinese Holstein Cows. Xiao Wang, Peipei Ma, Qin Zhang, Jianfeng Liu, Dongxiao Sun, Xiangdong Ding, Yachun Wang, Yi Zhang, Shengli Zhang and Ying Yu (College of Animal Science and Technology, China Agricultural University)

Bovine mastitis is a costly disease in modern dairy farms worldwide. In general, Genome-wide association study (GWAS) for somatic cell scores (SCSs) or mastitis has been conducted with different association methods in different groups, while the identified single nucleotide polymorphisms (SNPs) are various. This study is attempted to identify SNPs of significant effects on mastitis resistance and susceptibility in Chinese Holstein cows. To avoid uncertain effects' influence, estimated breeding values (EBVs) of SCS based on a multiple trait random regression test-day model were provided as the phenotypes. A total of 2093 SCS EBVs of cows were performed with mixed model based on single locus regression model analysis (MMRA) while 1267 *vs* 667 were analyzed by ROADTRIPS software based on case-control association testing. After quality control, 43885 SNPs was totally available for MMRA in contrast to 43881 and 43887 SNPs for half of/one standard deviation (SD) of SCS EBVs for ROADTRIPS. In total, 54 significant SNPs on chromosome level were detected including 44 SNPs by MMRA, 5 SNPs by ROASTRIPS and the rest 5 SNPs by both methods, which are mainly located on the BTA 14 and X. *TRAPPC9* gene detected by both methods reveals the new candidate gene associated to mastitis resistance. In addition, GO analysis confirms one pathway participating in regulation of inflammatory response. To our knowledge, it is the first study aiming at unraveling the genetic mechanism of the mastitis resistance and susceptibility using a case-control association testing combined with MMRA based on a high density SNPs. Such findings herein provide novel methods for discovering candidate genes in dairy cattle. It was financially funded by the Earmarked Fund for Modern Agro-industry Technology Research System (CARS-37), the National Natural Science Foundation of China (31272420), the Fund for Basic Research from the Ministry of Education of the People's Republic of China (2011JS006) and the National Key Technologies R & D Program (2011BAD28B02).

P1012 Widespread differential maternal and paternal genome effects on fetal bone phenotype at mid-gestation. Ruidong Xiang (Robinson Institute, Research Centre for Reproductive Health, The University of Adelaide, South Australia, Australia. JS Davies Epigenetics and Genetics Group, School of Animal and Veterinary Sciences, Roseworthy Campus, The University of Adelaide, South Australia, Australia.), Alice Lee (Sansom Institute for Health Research, School of Pharmacy and Medical Sciences, University of South Australia, Adelaide, South Australia, Australia.), Tanja Eindorf (tanja.eindorf@student.adelaide.edu.au), Ali Javadmanesh and Mani Ghanipoor-Samami (Robinson Institute, Research Centre for Reproductive Health, The University of Adelaide, South Australia, Australia. JS Davies Epigenetics and Genetics Group, School of Animal and Veterinary Sciences, Roseworthy Campus, The University of Adelaide, South Australia, Australia.), Madeleine Gugger (JS Davies Epigenetics and Genetics Group, School of Animal and Veterinary Sciences, Roseworthy Campus, The University of Adelaide, South Australia, Australia.), Carolyn Fitzsimmons and Zbigniew Kruk (Robinson Institute, Research Centre for Reproductive Health, The University of Adelaide, South Australia, Australia. JS Davies Epigenetics and Genetics Group, School of Animal and Veterinary Sciences, Roseworthy Campus, The University of Adelaide, South Australia, Australia.), Wayne Pitchford (JS Davies Epigenetics and Genetics Group, School of Animal and Veterinary Sci-

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Parent-of-origin dependent (epi)genetic factors are important determinants of prenatal development that program adult phenotype. However, data on magnitude and specificity of maternal and paternal genome effects on fetal bone are lacking. We used an outbred bovine model to dissect and quantify effects of parental genomes, fetal sex and non-genetic maternal effects on the fetal skeleton and analyzed phenotypic and molecular relationships between fetal muscle and bone. Analysis of 51 bone morphometric and weight parameters from 72 fetuses recovered at Day153 gestation (54% term) identified six principal components (PC1-6) that explained 80% of the variation in skeletal parameters. Parental genomes accounted for most of the variation in bone wet weight (PC1, 72.1%), limb ossification (PC2, 99.8%), flat bone size (PC4, 99.7%) and axial skeletal growth (PC5, 96.9%). Limb length showed lesser effects of parental genomes (PC3, 40.8%) and a significant non-genetic maternal effect (gestational weight gain, 29%). Fetal sex affected bone wet weight (PC1, $P<0.0001$) and limb length (PC3, $P<0.05$). Partitioning of variation explained by parental genomes revealed strong maternal genome effects on bone wet weight (74.1%, $P<0.0001$) and axial skeletal growth (93.5%,$P<0.001$), while paternal genome controlled limb ossification (95.1%, $P<0.0001$). Histomorphometric data revealed strong maternal genome effects on growth plate height (98.6%, $P<0.0001$) and trabecular thickness (85.5%, $P<0.0001$) in distal femur. Parental genome effects on fetal bone were mirrored by maternal genome effects on fetal serum 25-hydroxyvitamin D (96.9%, $P<0.001$) and paternal genome effects on alkaline phosphatase (90.0%, $P<0.001$) and their correlations with maternally controlled bone wet weight and paternally controlled limb ossification, respectively. Bone wet weight and flat bone size correlated positively with muscle weight (r=0.84 and 0.77, $P<0.0001$) and negatively with muscle *H19* expression (r= −0.34 and −0.31, $P<0.01$). Since imprinted maternally expressed *H19* regulates growth factors by miRNA interference, this suggests muscle-bone interaction via epigenetic factors.

P1013 Generation of mammary gland bioreactor of lactoperoxidase in transgenic mice.

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