Abstract/Session Information for Program Number 2167

Session Information

Session Title: Statistical Genetics and Genetic Epidemiology **Session Type:** Poster

Session Location: Exhibit Hall E **Session Time:** Wed 4:30PM-6:30PM, Thu 4:30PM-6:30PM, Fri 10:30AM-12:30PM

Abstract Information

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Abstract Content

Genome-wide association scan for height in 6,671 individuals from Finland and Sardinia. *S. Sanna[1,2], A.U. Jackson[1], G. Usala[2], C.J. Willer[1], M. Dei[2], L.L. Bonnycastle[3], S. Lai[2], Y. Li[1], M. Uda[2], M.R. Erdos[3], H. Shen[4], A. Shuldiner[4], A. Cao[2], R.M. Bergam[5], D. Schlessinger[2,6], F.S. Collins[3], M. Boehnke[1], G.R. Abecasis[1], R. Nagaraja[5], K.L. Mohlke[7]* 1) Dept Biostatistics, Univ Michigan, Ann Arbor, MI; 2) National Human Genome Research Institute, Bethesda, MD; 3) Istituto di Neurogenetica e Neurofarmacologia (INN), CNR, Cagliari, Italy; 4) University of Maryland, School of Medicine, Baltimore, MD; 5) Keck School of Medicine of USC, Los Angeles, CA; 6) Gerentology Reasearch Center, NIA, Baltimore, MD; 7) Dept Genetics, University North Carolina, Chapel Hill, NC.

Height represents a classic example of a highly heritable quantitative trait. In our sample, heritability analysis shows that genes can explain >80% of the variation in height. Nevertheless, with the exception of a few rare Mendelian syndromes, gene-identification has proved difficult despite many parallel mapping efforts. Genetic influences on height are probably due to the contribution of several loci of small effect. We have carried out a meta-analysis of genome-wide association results from two different groups, ProgeNIA and FUSION. The first sample consist of 4,305 individuals from 570 families from Sardinia, the second includes 2,366 mostly unrelated Finnish individuals. Since the two groups worked with two different platforms (Illumina 300K and Affymetrix 500K respectively), SNPs appearing only in one platform were imputed to allow direct comparison of results across studies. To control inflation of type I error due to outliers and departure from normality, quantile normalization was applied to each trait prior the analysis. In both GWA scans, we evaluated the additive effect of each SNP, adjusting the model for familiality and covariates. In our combined results, the top associated SNP (p=4.0*10-7) maps to a region of LD containing several genes, including one previously implicated in growth. Replication is ongoing, but preliminary results on 2017 Finnish and 858 Amish samples support our initial finding (p=1.7*10-3), with the same direction of effect. Further detailed SNP analysis of the region is necessary to refine the responsible gene.

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