Human Molecular Genetics, 2020, Vol. 29, No. 16 2812

doi: 10.1093/hmg/ddy229
Advance Access Publication Date: 28 June 2018
Corrigendum

CORRIGENDUM

Open chromatin profiling of human postmortem brain infers functional roles for non-coding schizophrenia loci

John F. Fullard[1], Claudia Giambartolomei[1], Mads E. Hauberg[1,2,3,4], Ke Xu[5], Georgios Voloudakis[1], Zhiping Shao[1,6,7], Christopher Bare[8], Joel T. Dudley[5], Manuel Mattheisen[2,3,4], Nikolaos K. Robakis[1,6,7], Vahram Haroutunian[1,6,9] and Panos Roussos[1,5,9,*]

[1]Department of Psychiatry, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA, [2]Department of Biomedicine, [3]Centre for Integrative Sequencing (iSEQ), Aarhus University, Aarhus, Denmark, [4]The Lundbeck Foundation Initiative of Integrative Psychiatric Research (iPSYCH), Denmark, [5]Department of Genetics and Genomic Science and Institute for Multiscale Biology, [6]Department of Neuroscience, [7]Center for Molecular Biology and Genetics of Neurodegeneration, [8]Flow Cytometry Center of Research Excellence, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA and [9]Mental Illness Research, Education, and Clinical Center (VISN 2 South), James J. Peters VA Medical Center, Bronx, NY, USA

*To whom correspondence should be addressed at: Icahn School of Medicine at Mount Sinai, Department of Psychiatry and Department of Genetics and Genomic Science and Institute for Multiscale Biology, One Gustave L. Levy Place, New York, NY 10029, USA. Tel: +1 212 8248982; Fax: +1 646 5379583; Email: panagiotis.roussos@mssm.edu

Human Molecular Genetics, 2017, 26(10), 1942–1951.
doi: 10.1093/hmg/ddx103.

Following publication we became aware of a significant error in our original manuscript, relating to the identification of a putative risk allele affecting the expression of SNX19 in schizophrenia. In the text, for SNP rs10750450, we mistakenly refer to the reference allele (G) and risk allele (T). However, based on the GWAS schizophrenia results, it is, in fact, the T allele that is the reference allele and the G that is the risk i.e. the G allele leads to increased SNX19 expression based on expression quantitative trait loci (eQTL) analysis. Upon discovering the error, we re-sequenced the plasmid DNAs used in our in vitro assays. We also tested two additional SNPs, not included in our initial experiments, and although we confirm an allelic effect for each of the identified SNPs, the impact in vitro is opposite to that predicted by eQTLs, i.e. the protective alleles display increased luciferase expression relative to the risk alleles in vitro whereas, in the brain, it is the risk alleles that are predicted to increase transcription. One possible explanation for our observations is that testing plasmid encoded enhancers in HEK cells might not accurately reflect the mechanism of action of these enhancers in the context of the human brain. In addition, the risk variant resides within an enhancer element containing binding sites for two proteins, ZNF354C and ZSCAN10 (ZNF206, Zfp206 in mouse, respectively), both of which are thought to act as transcriptional repressors (1). However, there is evidence that ZSCAN10 can also function as an activator (2).

All other data in the manuscript, including the ATAC-seq experiments, is unaffected by this error.

1. Wang, Z. X., Kueh, J. L., Teh, C. H., Rossbach, M., Lim, L., Li, P., Wong, K. Y., Lufkin, T., Robson, P. and Stanton, L. W. (2007) Zfp206 is a transcription factor that controls pluripotency of embryonic stem cells. *Stem Cells*, **25**, 2173–2182.
2. Yu, H. B., Kunarso, G., Hong, F. H. and Stanton, L. W. (2009) Zfp206, Oct4, and Sox2 are integrated components of a transcriptional regulatory network in embryonic stem cells. *J Biol Chem*, **284**, 31327–31335.