Neuroscience 2015

Presentation Abstract

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Program#/Poster#:	408.05/J18
Presentation Title:	Comprehensive gene expression analysis of prefrontal cortex and hippocampus in social isolation rearing mouse model
Location:	Hall A
Presentation time:	Monday, Oct 19, 2015, 1:00 PM - 5:00 PM
Presenter at Poster:	Mon, Oct. 19, 2015, 1:00 PM - 2:00 PM
Topic:	++C.15.b. Genetics and genomics
Authors:	***K. UM**[1], A. KHAN[3], M. HARIHARAN[2], J. LUCERO[1], T. J. SEJNOWSKI[1,4], J. R. ECKER[2,4], S. B. POWELL[3,5], M. BEHRENS[1]; [1]Computat. Neurosci. Lab., [2]Genomic Analysis Lab., Salk Inst., La Jolla, CA; [3]Dept. of Psychiatry, UCSD, La Jolla, CA; [4]Howard Hughes Med. Inst., La Jolla, CA; [5]Res. Service, VA San Diego Healthcare Syst., San Diego, CA
Abstract:	Schizophrenia is a complex psychiatric disorder with heterogeneous symptoms including positive symptoms (hallucinations and delusions), negative symptoms (social withdrawal) and cognitive dysfunction (impaired

	learning and memory). Despite the rapidly increasing number of studies, pathophysiological mechanisms of schizophrenia remain elusive. To delineate the molecular events in schizophrenia, we performed comprehensive gene expression profiling and gene network analyses by rearing mice in social isolation from weaning, a preclinical neurodevelopmental model of schizophrenia. By single base level transcriptome analysis, we identified differentially expressed genes in the prefrontal cortex and hippocampus of socially isolated mice in the initial stages of post-weaning social isolation compared to group-housed control mice. Among these differentially expressed genes, 11 genes are common to both prefrontal cortex and hippocampus. Interestingly, a majority of the common genes are immediate early genes including Fos, Arc and Npas4, which suggests that brief social isolation elicits acute stress responses. Pathway analysis and disease network analysis of differentially expressed genes by Ingenuity Pathway Analysis tools indicated that they are involved in nNOS signaling and epileptic seizures, respectively, in both brain regions. To further study chronic responses to social isolation, we are currently examining brain transcriptome by RNA sequencing and a battery of schizophrenia-relevant behavior tests following long-term social isolation. Mice housed in social isolation through adolescence display behaviors relevant to schizophrenia, including deficits in prepulse inhibition of startle and impaired contextual fear conditioning. This study will help us facilitate understanding possible molecular mechanisms involved in schizophrenia and provide the basis for new biomarkers and improved treatment possibilities.
Disclosures:	**K. Um:** None. **A. Khan:** None. **M. Hariharan:** None. **J. Lucero:** None. **T.J. Sejnowski:** None. **J.R. Ecker:** None. **S.B. Powell:** None. **M. Behrens:** None.
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