receptor abnormalities in schizophrenia may help explain prior epidemiologic data relating the risk for this illness to altered rates of autoimmune disorders, prenatal infection and familial leukemia. Colony stimulating factor 1 receptor (CSF1R) gene encodes a tyrosine kinase growth factor receptor for CSF1, the macrophage and monocyte specific growth factor. CSF1R gene is located at chromosome 5q32, a region that was suggested to be linked to childhood onset schizophrenia. CSF1R gene mutation has been associated with microglial development. In this study, we investigated the genetic association between schizophrenia and single nucleotide polymorphisms (SNPs) of the CSF1R gene.

Methods: 219 Korean schizophrenia patients and 379 control subjects were enrolled for this study. We genotyped four SNPs (rs216138, rs10079250, rs2228422 and rs1986027) of the CSF1R gene by direct sequencing. All patients were evaluated by the Operational Criteria Checklist for Psychotic Illness. Multiple logistic regression models (that is, co-dominant, dominant, and recessive) were performed to generate odds ratios, 95% confidence intervals, and p values.

Results: The genotype frequencies of rs1986027 showed significant association between schizophrenia and control groups [p<0.011 in the co-dominant model (T/T vs. C/C); p=0.003 in the recessive model (T/T vs. C/C + C/T)]. For the SNP rs10079250, significant association was found in the recessive model [(C/C vs. T/T + C/T); p=0.035]. There was no significant association between other two SNP polymorphisms and schizophrenia.

Conclusions: Our study is the first to report an association of the CSF1R gene polymorphisms with schizophrenia. We found significant association between CSF1R polymorphism and schizophrenia in Korean population.

Key Words: CSF1R, cytokine, schizophrenia, association.

PM454

Association analysis between (AAT)n repeats in the cannabinoid receptor 1 (CNR1) gene and smooth pursuit eye movement (SPEM) abnormality in Korean patients with schizophrenia.

Min Jae Kim, MD1, Jioun Kim, MD2, Chae-Ri Kim, MD1, Jin-Wan Park, MD2, Sang-Woo Han, MD1, Jaeuk Huang, MD1, Yeon Jung Lee, MD1, Sung-II Woo, MD1

1Department of Psychiatry, College of Medicine, Soonchunhyang University, Soonchunhyang University Hospital, Seoul, Korea 2 Sachun Hamnæum Hospital

Abstract

Objective: According to previous studies, the cannabinoid receptor 1 (CNR1) gene could be an important candidate gene for schizophrenia which is located on chromosome 6q14-q15. The association between CNR1 polymorphisms and schizophrenia is actively being investigated, and some studies have linked the AAT-trinucleotide repeats in CNR1 gene with risk of schizophrenia. Meanwhile, smooth pursuit eye movement (SPEM) has been regarded as one of the most consistent endophenotype of schizophrenia.

In this study, we investigated the association between the AAT-trinucleotide repeats in CNR1 gene and smooth pursuit eye movement abnormality in Korean patients with schizophrenia. We also investigated allele frequencies of AAT-repeat polymorphisms on CNR1 gene in each group. A logistic regression analysis was performed to find the association between SPEM abnormality and AAT-trinucleotide repeats in each group.

Results: The natural logarithm value of signal/noise ratio (Ln S/N ratio) of good SPEM function group was 4.34±0.29 and that of poor SPEM function group was 3.21±0.70.

In total, 7 types of trinucleotide repeats were identified, each containing 7, 10, 11, 12, 13, 14, and 15 repeats, respectively. (AAT)13 allele was most frequently observed, with a frequency of 30.5%. The frequencies of the other repeat alleles (in the decreasing order) were as follows: (AAT)12, 30.5%, (AAT)7, 24.3%, (AAT)7, 19.8%, and (AAT)7, 11.1%.

However, no significant associations were found between the number of AAT-repeat polymorphisms of the CNR1 gene and SPEM function.

Conclusions: No significant associations were found between AAT-trinucleotide polymorphisms and SPEM abnormality in Korean patients with schizophrenia.

PM455

Genetic variants in Chromogranin B is associated with the Risk of Schizophrenia in Korean male population

Jioun Kim, MD1, Min Jae Kim, MD1, Chae-Ri Kim, MD1, Jin-Wan Park, MD1, Sang-Woo Han, MD1, Jaeuk Huang, MD1, Yeon Jung Lee, MD1, Sung-II Woo, MD1

1Department of Psychiatry, College of Medicine, Soonchunhyang University, Soonchunhyang University Hospital, Seoul, Korea 2 Sachun Hamnæum Hospital

Abstract

Schizophrenia is a devastating mental disorder with high heritability estimate up to 80%. Secretory pathway of peptide hormones and neuropeptides in brain is regulated by chromogranin proteins. Chromogranin B (CHGB), a member of chromogranin family gene, is proposed as one of the candidate genes for the risk of schizophrenia. In a genome wide association study performed in a Japanese population, genetic variant (microsatellite: D20S95) near CHGB could be a potential genetic marker for the schizophrenia development.

In the current study, 15 SNPs of CHGB were genotyped in 310 schizophrenia patients and 604 healthy controls to investigate the association with the schizophrenia susceptibility. Statistical analysis has revealed that four genetic variants (rs446659, rs6133278 (D145N), rs910122 (R178Q), rs2821) were associated with the reduced risk of schizophrenia (OR=0.72–0.78, p=0.002-0.02). In the subgroup analysis, five genetic variants (rs236141, rs446659, rs6085323, rs910122 (D178Q), rs2821) and a haplotype (ht3) showed more protective effect on the schizophrenia in male subjects (OR=0.52-0.74, p=0.002-0.05), but not in female subjects.

Our results demonstrated that genetic variants in CHGB showed gender-specific effect to the reduced risk of schizophrenia, which could be a useful preliminary result for further study.

Keywords: Single nucleotide polymorphisms (SNPs), Chromogranin B (CHGB), schizophrenia, gender-specific marker, male

PM456

Aberrant cortico-cerebellar connectivity of the default mode network in individuals at ultra-high risk for psychosis: a resting-state fMRI study

Minji Bang1,2, Chongwon Pae3,4, Hae- Jeong Park3,4, Suk Kyoung An1,2 (*correspondence: ansk@yuhs.ac)