conferenceseries.com JOINT EVENT
10th International Conference on
Genomics and Molecular Biology
&
6th International Conference on
Integrative Biology
May 21-23, 2018 Barcelona, Spain

J Mol Genet Med 2018, Volume 12
DOI: 10.4172/1747-0862-C2-028

Prediction analysis of transient receptor potential ion channel and acetylcholine receptor genes in b lymphocytes from chronic fatigue syndrome/myalgic encephalomyelitis patients

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Calcium (Ca2+) and acetylcholine (ACh) signaling are important in B cell activation and potential antibody development. The aim of the study was to examine the effects of key genes responsible for these mechanisms from transient receptor potential (TRP) ion channels and acetylcholine receptors (AChRs) in isolated B cells from chronic fatigue syndrome/myalgic encephalomyelitis patients (CFS/ME). Flow cytometric protocols were used to determine B cell purity, followed by single-nucllеotide polymorphisms (SNP) and genotype analysis from 21 TRP ion channel genes and 9 AChR genes examined by iPLEX Gold assay. Exome analysis was conducted using Illumina HiSeq platform and SNP association and genotype was determined using ANOVA (Analysis of Variance) and PLINK analysis. The SNP predictions on splicing events were realized using Automated Splice Site and Exon Definition Analysis server. Eleven CFS/ME patients (mean age 31.8±SD 5.5 years) defined according to the Fukuda criteria and 11 non-fatigued controls (mean age 33.9±5.1 years) were included. Seventy-eight SNPs were associated with CFS/ME: 35 were mAchR M3, the remaining were nAChR delta, nAChR alpha 9, TRPV2, TRPM3, TRPM4, mAChRM2 and mAChRM5. Mutations in the above genes can create or abolish splicing cryptic sites, which could induce important consequences on protein expression. The mutations can also strengthen or weaken binding sites by affecting the affinity with spliceosome elements, consequently inducing an increase or decrease in protein expression. These findings warrant further examination of the above genes in a larger cohort to investigate their potential role in CFS/ME.

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