

MEETING ABSTRACT

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EHMTI-0262. Dysregulation of inflammatory pathways in a familial hemiplegic migraine 1 mouse model after the induction of cortical spreading depression

B de Vries^{1*}, EE Eising¹, R Shyti¹, LS Vijfhuizen¹, LAM Broos¹, PAC 't Hoen¹, MD Ferrari², EA Tolner¹, AMJM van den Maagdenberg¹

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Background

Familial Hemiplegic Migraine type 1 (FHM1) is a rare monogenic subtype of migraine with aura caused by mutations in the CACNA1A gene. In FHM1 knock-in mouse models these mutations increase the susceptibility for cortical spreading depression (CSD): the underlying mechanism of the migraine aura.

Aim

To study the consequences of CSD in a migraine-relevant context, we measured cortical gene expression profiles in FHM1 and wild-type mice 24 hours after CSD induction.

Method

Expression profiles were generated using deep-Serial Analysis of Gene Expression (SAGE) sequencing, a tag-based next-generation sequencing method for gene expression profiling. Relevant expression changes were validated by qPCR experiments.

Results

Our data show that CSD induces differential expression of genes involved in inflammatory pathways in both the FHM1 and wild-type mice. However, we identified a gene set that is up-regulated upon CSD specifically in the FHM1 migraine mouse model. Genes from this gene set are involved in inflammatory and interferon-related

signaling, and were often found up-regulated in immune-stimulated conditions.

Conclusion

Differential expression of genes involved in inflammatory pathways in the brain of FHM1 migraine mice compared to wild-type mice upon CSD, indicates that CSD affects the brain differently in a genetically predisposed animal which may help increase our understanding of migraine pathophysiology.

No conflict of interest

Authors' details

¹Human Genetics, Leiden University Medical Center (LUMC), Leiden, Netherlands. ²Neurology, Leiden University Medical Center (LUMC), Leiden, Netherlands.

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¹Human Genetics, Leiden University Medical Center (LUMC), Leiden, Netherlands

Full list of author information is available at the end of the article