J Alzheimers Dis Parkinsonism 2016, 6:3(Suppl)
http://dx.doi.org/10.4172/2161-0460.C1.019

Role of glia in inflammation and Alzheimer's disease

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During decades, glia cells have been considered such as protective and nutrient cells taking care neurons in the brain. In this decade, many scientists have published different roles for astrocytes, oligodendroglia, microglia and endothelial cells. The main aim of this study was to show the role of glia in Alzheimer's disease using transgenic APP/Presenilin 1 and comparing with Wild type mice. We detect increase in inflammatory genes in wild type mice compared with transgenic one, demonstrating a chronic inflammation in those mice. Also we noted increase in *CCL3* and *CCL4* genes involved in brain demyelination compared with wild type mice, which can explain the cleaning job of astrocytes in transgenic mice trying to eliminate Aβ1-42 plates. By microarray *CCR8*, *CX3CL1* and *CXCR3* genes were significantly high expressed in wild type compared with transgenic mice, showing us the proper positioning of activated T cells with adhesive, trafficking and migratory functions in wild type. In fact we detect also a significant increase of IL-3 in transgenic mice compared to wild type indicating activation of T cells and induction of proliferation and differentiation of T cells in transgenic mice compared with wild type. In our study, integrin activation, cytoskeletal changes and chemotactic migration was also altered in transgenic mice compared to wild type. For instants, astrocytes play important roles such as protector of neurons in front of inflammation imbalance and regenerating damage intake. Further we detect presence of tumour resistant gene in transgenic mice, *ABCF1*, without any expression of this gene in wild type and on the contrary expression of *CCL12*, cancer gene, in wild type without any expression in transgenic mice. These last data indicate a resistance of transgenic mice to cancer compared to wild type mice. In the future the study of the communication between all brain cells will be necessary to understand much neurodegenerative illness and the protection of stem natural cells of our young brain will be the next frontier.

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Association of genetic polymorphisms of claudin-1 with small vessel vascular dementia

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Introduction & Aim: The most recent hypothesis that explains the development of small vessel disease vascular dementia (VaD) emphasises the role of blood-brain barrier (BBB) dysfunction. However, while environmental risk factors like hypertension and Type 2 diabetes mellitus are known to contribute to BBB dysfunction, the molecular mechanism of this process is unclear. It is hypothesised that certain genetic polymorphisms of the BBB tight junction claudin-1 protein, in combination with adverse environmental risk factors, increase the risk of BBB dysfunction and small vessel disease.

Methods: In this case-control study, 47 control participants, with a Mini Mental State Exam (MMSE) score of above 27, and 36 VaD participants were recruited and completed a questionnaire on their medical history and lifestyle factors. Blood was also collected and three single nucleotide polymorphisms (SNPs), rs17501010, rs9290927 and rs893051 of claudin-1 genotyping were analysed by real-time polymerase chain reaction (PCR) assay.

Results: A significant higher prevalence was found in the VaD group compared to controls in the GT genotype of SNP rs17501010 (p=0.011) and the AT genotype of SNP rs9290927 (p=0.012). Stratified analysis also showed that individuals with both the variant genotype of any of the 3 SNPs (rs17501010, rs9290927 and rs893051) of claudin-1 and Type 2 diabetes mellitus, have a significantly higher risk of developing small vessel VaD.

Conclusion: The claudin-1 polymorphisms rs17501010 and rs9290927 are significantly associated with VaD. Moreover, gene-environment interaction between claudin-1 polymorphisms and Type 2 diabetes mellitus plays a synergistic role on the development of small vessel VaD.

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