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Demyelinating Disorders 2

41
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Demyelinating Disorders 2
RGC-32 as a potential marker of relapse and response to treatment with glatiramer acetate in multiple sclerosis

H. Rus[a], A. Kruszewski[a], C. Cudrici[a], V. Nguyen[a], W. Royal[a], C. Bever[a], V. Rus[b]. [a]*Neurology, University of Maryland School of Medicine, Baltimore, USA;* [b]*Medicine, University of Maryland School of Medicine, Baltimore, USA*

Background: We have previously shown that response gene to complement (RGC)-32 and FasL mRNA expression are significantly lower in the peripheral blood mononuclear cells (PBMCs) of multiple sclerosis (MS) patients during relapses than in stable patients.
Objectives: We have now investigated the combined roles of RGC-32, FasL, and CDC2 as possible biomarkers of relapse and predictors of response to glatiramer acetate (GA) treatment in relapsing-remitting MS patients.
Material and Methods: Over the course of 2 years, a cohort of 15 GA-treated MS patients was clinically monitored using the Expanded Disability Status Scale, and blood samples were collected at 0, 3, 6, and 12 months. Target gene mRNA expression was measured in patients' isolated PBMCs by real-time quantitative PCR.
Results: During relapses MS patients had a decreased expression of RGC-32 ($P < 0.0001$) and FasL ($P < 0.0005$) mRNA but no change in CDC2 when compared to stable MS patients. As compared to non-responders, responders to GA treatment had significantly higher mRNA levels of RGC-32 ($P < 0.0001$) and FasL ($P < 0.003$) but no change in CDC2. Receiver operating characteristic analysis was used to assess the predictive power of each putative biomarker. The predictive values of relapse were 90% for RGC-32, and 84% for FasL; the predictive values of responsiveness to GA treatment were 85% for RGC-32 and 85% for FasL.
Conclusion: Our data suggest that RGC-32 and FasL could serve as potential markers for the prediction of MS relapses and the evaluation of patients' response to GA therapy.

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42
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Demyelinating Disorders 2
Lithium induced amelioration of a rat model of multiple sclerosis through the suppression of glycogen synthase kinase (GSK)-3 signaling

T. Shin[a], M. Ahn[b], C. Park[a], K. Jung[c], J. Kim[d], C. Moon[e]. [a]*Anatomy, College of Veterinary Medicine, Jeju, Korea;* [b]*College of Medicine, College of Medicine, Jeju, Korea;* [c]*Eco-friendly Material Research Center, Korea Research Institute of Bioscience and Biotechnology, Jeongeup, Korea;* [d]*Anatomy, Faculty of Medicine University of the Ryukyus, Okinawa, Japan;* [e]*Anatomy, College of Veterinary Medicine Chonnam National University, Gwangju, Korea*

Background: Glycogen synthase kinase (GSK)-3 is one of important molecules in the course of cell activation in the central nervous system diseases.
Objective: The aim of this study was to evaluate whether lithium, an inhibitor of GSK-3 beta, ameliorates rat paralysis with acute monophasic experimental autoimmune encephalomyelitis (EAE).
Material and Methods: EAE was induced in Lewis rats through immunization of guinea pig myelin basic protein (MBP) and complete Freund's adjuvant. Lithium was administered in immunized rats.Tissues and sera of rats were collected for the cytokine assay and signals by western blot analysis and immunohistochemistry.
Results: Lithium treatment significantly delayed the onset of EAE paralysis and ameliorated its severity compared with those of vehicle treated group.
Conclusion: Taken all into considerations, lithium, an inhibitor of GSK-3 beta, is a potential anti-inflammatory drug to suppress acute autoimmune diseases including rat EAE, possibly through the inactivation of GSK-3 signal cascades. (*supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education (Grant number: 2014R1A1A2055965)).

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43
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Microglial activation correlates with disease progression in multiple sclerosis

L. Airas[a], E. Rissanen[a], J. Tuisku[b], M. Gardberg[c], J.O. Rinne[b]. [a]*Division of Clinical Neurosciences, Turku University Hospital, Turku, Finland;* [b]*Turku PET Centre, Turku University Hospital, Turku, Finland;* [c]*Pathology, Turku University Hospital, Turku, Finland*

Background: Activation of microglia is considered a crucial step in CNS response to injury. Measuring microglial activation *in vivo* is possible using PET imaging and radioligands binding to 18 kDa translocator protein (TSPO).
Objective: The aim of this cross-sectional study was to investigate how activation of microglia relates to certain milestones related to MS progression.
Patients and methods: MS patients with secondary progressive (SPMS,n = 10) or relapsing remitting (RRMS,n = 10) disease, and

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