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Autoantibodies against brain neurons in Systemic Lupus Erythematosus

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Systemic lupus erythematosus (SLE), is an autoimmune disease characterized by multiple autoantibodies (AutoAbs). Neuropsychiatric syndromes appear when SLE affects brain neurons. However, the physiophatological mechanisms involved in Neuropsychiatric Lupus (NP-SLE) are unknown as well as the specific biomarkers. The project aim was to identify AutoAbs to neuronal epitopes possibly involved in specific NP-SLE clinical features. The search of AutoAbs was done through immunohistochemistry on perfused rat brains (4% paraformaldehyde), using sera from patients affected by: (1) NP-SLE (n=25), (2) SLE without neuropsychiatric features (n=39), (3) Multiple Sclerosis as no-autoimmune disease (MS, n=22) and age-matched controls (n=82). When sera were tested (1:60–600), a high percentage of patients contained AutoAbs labeling neurons of the cortex, hippocampus and cerebellum (72% and 30.7% in NP-SLE and SLE, respectively) while control subjects and MS patients were negative. Patients' sera stained a high number of perikarya within the cortex as well as within hippocampus and cerebellum (ImageJ optical density: p= 0.0003 and 0.00000001 respectively, positive patients vs. controls). To investigate the neuonal types involved in the autoimmune reaction, we carried out double stainings with the relevant neurotransmitters/proteins. No colocalization profiles were found with antibodies to the vesicular acetylcholine transporter, GAD-65 or glutammate but we observed that the staining of patients' sera was largely revealed within the same cells labeled also by the anti NeuN antibody in all the areas studied (cortex, hippocampus and cerebellum). Many patients showed a virtually 90% of colocalization profile, while other ranged 50–75%. The NeuN protein is mainly localized in the nuclei of specific brain neurons while NeuN antibodies have applied in the differential morphological diagnosis of cancer. The role of AutoAbs to NeuN in our cohort of patients remains to be identified. In conclusion, we found high titer neuronal AutoAbs in the majority of the analyzed NP-SLE/SLE patients, probably reacting against the NeuN protein.