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Brain-derived neurotrophic and immunologic factors: Beneficial effects of Riboflavin on motor disability in murine model of multiple sclerosis

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In the present study, C57BL/6 female mice (n=56) were used to explore the neuroprotective effects of riboflavin in motor disability of experimental autoimmune encephalomyelitis (EAE) as a model of multiple sclerosis. The animals were assigned into 7 groups: Sham operated 1 (SO1), healthy mice received PBS (Phosphate Buffer Saline); Sham operated 2 (SO2), healthy mice received PBS and riboflavin; Sham treatment 1 (ST1), EAE mice received water; Sham treatment 2 (ST2), EAE mice received sodium acetate buffer; Treatment 1 (T1), EAE mice received interferon beta-1a (INFβ-1a); Treatment 2 (T2), EAE mice received riboflavin; Treatment 3 (T3), EAE mice received INFβ-1a and riboflavin. After EAE induction, scoring was performed based on clinical signs. By detecting score 0.5, riboflavin at 10 mg/kg of body weight and/or INFβ-1a at 150 IU/g of body weight administration were started for two weeks. The brain and spinal cord levels of brain-derived neurotrophic factor (BDNF), interleukin-6 (IL-6), and interleukin-17A (IL-17A) were studied using real-time PCR and ELISA methods. BDNF expression and protein levels were increased in the brain and spinal cord of the T3 group compared with the other groups ($P<0.01$). IL-6 and IL-17A expression were increased in the brains of the T3 and T1, respectively, compared to the other groups ($P<0.01$). Daily clinical score was reduced significantly by riboflavin in both effector and chronic phases of the disease compared with that of controls ($p<0.05$). Our findings showed that riboflavin is capable for suppressing the neurological disability mediated by BDNF and IL-6.

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Recent treatments for cardiac arrhythmias: Confusion of ganglionated plexi (GP), fibrosis and myocardial structural diseases areas, leading to similarity of detected signals (complex atrial fractionated electrograms)

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Atrial Fibrillation (AF) is a major life threatening disease which is still refractory to all pharmaceutical compounds. Catheter ablation procedure is currently used as an 'easy and safe' clinical intervention targeting the early activation sites that are triggering the initiation of arrhythmias. However, the catheter ablation procedure is still considered as ineffective treatment for long-term curing of arrhythmias and for preventing their recurrences. Despite the high technologies for mapping, detecting and delivering the right energy sources to any location in the human cardiac heart chambers insulating the right triggers areas for AF still the main challenges to all currently existing devices companies (ex. Biosense Webster (subsidiary of J&J; Carto Solution), St Jude Medical Inc, (Ensite system), Boston Scientific, Medtronic (with Cryocath Technologies, Atricure Inc, etc). Detecting the sites of early activations and the locations of the triggers ganglia are still confused with other structural diseases such as fibrosis, which are limiting those devices to achieve the detection of the right hit (ex. triggering GP). Thus, it adds complications and risks to the long-term inefficacy of the catheter ablation procedure, in addition to major side effects and complications to this minimally invasive clinical procedure. 'NerveC' is developing a high end assembly able to detect selectively the GPlocations. Thus, it will limit the confusion during mapping of the heart for electrophysiological (EP) study and for catheter ablation procedure for offering long-term cure from cardiac arrhythmias, such as atrial fibrillation, a major disease that affects our aging population. Therefore, by introducing 'NerveC' to the EP clinical practice will help to detect selective EP signals generated by the autonomic nerve (=GP) using the FFT (Fast Fourier Transformation), offering a unique method to specifically targeting the rotors triggering the cardiac AF.

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