P1121 / #4166

Topic: AS08 Diseases of The Nervous System (including, infective and psychiatric)

EFFECTS OF HIGH COPPER LEVELS ON OXIDATIVE STRESS PARAMETERS IN U-87 MG GLIOBLASTOMA CELL LINE

Lara Stoeberl [1], Letícia Cordeiro Koppe De França [1], Aline Pertile Remor [2], Viviane Glaser [1]

[1] *Federal University of Santa Catarina, Biological And Agronomic Sciences Department, Curitibanos, Brazil*
[2] *University of West Santa Catarina, Campus Joaçaba, Joaçaba, Brazil*

Copper (Cu) is an essential element to cell functions, but high Cu levels catalyze the production of hydroxyl radicals mainly by Fenton reaction, resulting in oxidative stress. Considering Cu toxicity and that this metal could triggers cell death by cuproptosis, some strategies with Cu-content compounds have been suggested in the literature to prevent tumor growth. In this way, the aim of this study was to investigate the effects of high Cu levels on oxidative stress parameters in a human glioblastoma cell line (U-87 MG). Cells were exposed to copper sulfate (CuSO4) (100, 300 and 600 μM) for 24 or 48h. Glutathione peroxidase (GPx), glutathione reductase (GR), catalase (CAT) e superoxide dismutase (SOD) activities were measured. Non-protein thiols groups (NPSH), proteins thiols groups (PSH), and superoxide levels ($O2^{-}$) were also evaluated. Results indicated that high Cu levels decreases GPx, GR, and CAT activities after 24h or 48h of exposure to CuSO4. Regarding SOD activity, it was observed an increase in the activity of this enzyme within 24h of Cu exposition, but after 48h the enzyme activity was decreased. NPSH and PSH levels were reduced after 24h of Cu exposure, and $O2^{-}$ generation was increased. Taken together, these results suggest that excess Cu could induce modifications in the cell antioxidant system that trigger oxidative stress in human glioblastoma cell line, which could be used as a target against glioblastoma growth.

Declaration of Interest Statement: None

https://doi.org/10.1016/j.ibneur.2023.08.1127

P1122 / #1743

Topic: AS08 Diseases of The Nervous System (including, infective and psychiatric)

TRANSAURICULAR VAGAL NERVE STIMULATION EFFECTS ON PRO- AND ANTI-INFLAMMATORY CYTOKINES IN AN EXPERIMENTAL MODEL OF AUTISTIC MICE

Hale Gök Dağıdır [1,2], Neslihan Bukan [1], Ayşen Çalıkuşu [2], Ece Alim [3], Özen Akarca [3], Meltem Bahcelioglu [2,3]

[1] *Gazi University, Biochemistry, Ankara, Turkey*
[2] *Gazi University, Neurology, Ankara, Turkey*
[3] *Gazi University, Anatomy, Ankara, Turkey*

Autism is a very diverse neurodevelopmental condition, and its frequency has risen considerably in recent years. This study uses bilateral transcutaneous auricular vagus nerve stimulation in an autism animal model to study neuroinflammation. The autism model was created by giving valproic acid to C57BL/6 pregnant mice on day 12.5 of the embryonic period. The study included three groups: Autistic Transcutaneous Auricular Stimulation Group (ATASG), Autistic Control Group (ACG), and Healthy Control Group (HCG). Each group included 16 mice (N = 48), 8 females and 8 males. Transauricular vagus nerve stimulation was performed under general anesthesia. Pro- and anti-inflammatory cytokines were measured in blood and brain tissues in three groups using ELISA. Blood and brain tissue NLRP3 and IL 1 Beta, IL 6, IL 10, IL 22, TNF-alpha levels were measured. TNF-alpha, IL10, IL22, and NLRP 3 parameters in blood and brain tissue were significantly different across groups ($p < 0.05$). While the underlying causes of autism have not been adequately understood, the disorder is currently diagnosed based on behavioral criteria. Early diagnosis is essential, hence the development of biomarkers is of utmost importance. Autism's etiology and possible biomarkers involve inflammation. The vagus nerve, may be a promising autism treatment target. Further studies are needed to better understand this neurodevelopmental condition, whose frequency is rising, to uncover biomarkers, to diagnose it early, and to create treatments.

Declaration of Interest Statement: This study was supported by Gazi University Scientific Research Projects Coordination Unit project number TDK-2021-7245.

https://doi.org/10.1016/j.ibneur.2023.08.1128

P1123 / #3922

Topic: AS08 Diseases of The Nervous System (including, infective and psychiatric)

FRIZZLED 1 AND WNT1 AS NEW POTENTIAL THERAPEUTIC TARGETS IN THE TRAUMATICALLY INJURED SPINAL CORD.

Pau González, Carlos González-Fernández, Virginia Pérez, Alfredo Maqueda, Tomás Sánchez-Gutierrez, Francisco Javier Rodríguez

Hospital Nacional de Parapléjicos, Laboratory Of Molecular Neurology, Toledo, Spain

Despite the experimental evidence pointing to a significant role of the Wnt family of proteins in physiological and pathological rodent spinal cord functioning, its potential relevance in the healthy and traumatically injured human spinal cord as well as its therapeutic potential in spinal cord injury (SCI) are still poorly understood. To get further insight into these interesting issues, we first demonstrated by quantitative Real-Time PCR and simple immunohistochemistry that detectable mRNA expression of most Wnt components, as well as protein expression of all known Wnt receptors, can be found in the healthy human spinal cord, supporting its potential involvement in human spinal cord physiology. Moreover, evaluation of Frizzled (Fz) 1 expression by double immunohistochemistry showed that its spatio-temporal and cellular expression pattern in the traumatically injured human spinal cord is equivalent to that observed in a clinically relevant model of rat SCI and suggests its potential involvement in SCI progression/outcome. Accordingly, we found that long-term lentiviral-mediated overexpression of the Fz1 ligand Wnt1 after rat SCI improves motor functional recovery, increases myelin preservation and neuronal survival, and reduces early astroglial reactivity and NG2+ cell accumulation, highlighting the therapeutic potential of Wnt1 in this neuropathological situation. Supported by the MICINN/AEI/10.13039/501100011033 and by “ERDF A way of making Europe” (Grant number RTI2018-097775-BI00), the “Instituto de Salud Carlos III” (Grant number DTS19/00129; ERDF co-funded), and the “Consejería de Educación, Cultura