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ABSTRACT BOOK

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affects motor neurons (MNs) in children and young adults following a decrease in the levels of functional SMN protein; this results in motor impairment, muscle atrophy and premature death. Although the genetic cause of SMA has been identified, many aspects of its pathogenesis remain elusive and novel biological targets are investigated to develop new therapeutics and to monitor the efficacy of existing treatments. Indeed, the current experimental therapies aim at restoring SMN protein levels; however, their long term effects are still under evaluation, especially in adults. We focus on mitochondria since already at early stages in SMA their function, number, area and transport are significantly altered in axons of spinal MNs. We characterized subcellular and mitochondrial alterations (size, amount, area, cristae length and density) by TEM in MNs from the SMA mouse model (SMNdelta7). Moreover, in order to understand the mechanisms underpinning mitochondrial dysfunctions, we isolated pure mitochondria from the spinal cord of mice. After 2D gel and MALDI-TOF mass spectrometry, we identified differentially expressed proteins among which Aconitase2 (Aco2) was significantly altered. Aco2 turned out to be also dysfunctional in SMA MNs by enzymatic assays. Aco2 is the responsible for the isomerization of citrate to isocitrate in the Krebs Cycle and in mitochondria it is a sensitive redox sensor of reactive oxygen and nitrogen species. Interestingly, its levels and activity are altered also in other neurodegenerations such as Parkinson's and Huntington's disease. In order to study Aco2 as a new target and read-out of therapies efficacy in peripheral non-invasive tissues, we cultured primary fibroblasts (MEFs). By MitoTracker staining and multidimensional analysis by MiNA toolset from ImageJ, we studied the number of mitochondria, their distribution and trafficking. We also measured Aco2 activity by enzymatic assay. Currently, we are performing these investigations in fibroblasts derived from patients and aged-matched controls. Altogether, we found that mitochondrial networks, anatomical structures, dynamics and activity are affected in spinal cord and MEFs of SMA mice. Since mitochondria take part in a plethora of processes to preserve cellular homeostasis and genomic integrity, Aco2 could represent a potential new target to implement *SMN*-dependent therapies for SMA but also a target for other neurodegenerative diseases.

REFERENCE

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IMPACT OF HONEY REGULAR INTAKE ON NEURODEGENERATION IN AN ANIMAL MODEL WITH DIET-INDUCED OBESITY

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The use of natural honey as a nutraceutical agent is associated with nutritional benefits and therapeutic promises. Honey flavonoids and phenolic acids can play a key role on health, due to the high antioxidant and anti-inflammatory properties. Up to date, it is unclear the effect of consumption of honey in obesity–related disorders, including neurodegeneration. The aim of the present study was to analyse the preventive effects of sicilian black bee chestnut honey daily intake on glucose dysmetabolism and neurodegeneration in mice fed high-fat diet (HFD). Three groups of mice were fed with standard diet (STD), HFD or HFD supplemented with honey (HFD-H) for 16 weeks. Glucose metabolism parameters, neuronal apoptosis (TUNEL assay and brain genes expression of Fas-L, Bim and P27) and central insulin resistance (cerebral cortex protein expression of pAKT, pERK and pGSK3 and microarray analysis) were analysed and compared between the different groups of animals. Fasting glucose, insulin levels, glucose tolerance and insulin sensitivity were significantly ameliorated in HFD-H mice compared to HFD, although the values were different from STD mice. Honey intake significantly reduced the HOMA index, which indeed was increased in HFD mice, suggesting a beneficial effect on insulin resistance. In addition, HFD mice showed a reduction in brain weight/body weight ratio, a significantly higher number of apoptotic nuclei in cerebral cortex, a higher gene expression of Fas-L, Bim and P27 (marker of neuronal apoptosis) in comparison with STD- and HFD-H mice, providing evidence for honey neuroprotective action. Moreover, honey intake significantly improved brain insulin resistance as demonstrated by PCR-array analysis showing upregulation of genes involved in insulin signalling (InsR, AdipoR and Irs1) and downregulation of genes involved in pro-inflammatory response (Rbp4, Cd36 and Stat3). In addition, in HFD-H mouse cortex, p-AKT and p-ERK protein expression was increased, while p-GSK3 was reduced in comparison with HFD cortex suggesting the ability of honey regular intake of protecting brain neurons from insulin resistance. In conclusion, the present results suggest a beneficial effect of the sicilian chestnut honey regular consumption on central nervous system in obesity conditions, by preventing onset of neurodegeneration and central insulin resistance.

HEREDITARY SPASTIC PARAPLEGIA AS NOVEL GENETIC NEURO-CHAPERONOPATHY

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Hereditary spastic paraplegia (HSP) is a term used to indicate a large group of clinically and genetically heterogeneous neurodegenerative disorders, involving the corticospinal tracts and characterized by slowly progressive spasticity and weakness in the lower limbs, associated with additional neurologic signs in complicated forms [1]. In recent years, an increasing number of missense mutations in the *HSPD1* gene, encoding for the mitochondrial chaperonin Hsp60, have been linked to the development of different forms of Spastic Paraplegia, making it as sortable in the vast and heterogeneous group of genetic neuro-chaperonopathies [2]. Among these, one of the most studied is the p.Val98Ile missense mutation, responsible for the onset of Hereditary Spastic Paraplegia 13 [3]. Other two disease-associated variants listed in public databases are the p.Glu129Lys and the p.Val287Ile. However, in current literature, there are no data about the molecular mechanisms underpinning the disease onset following these aminoacidic