A10 - NEURODEGENERATION AND NEUROPSYCHIATRIC DISORDERS

A 10-01 | Black seed oil reverses chronic antibiotic-mediated depression and social behavior deficits

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Chronic antibiotic use has been reported to impair mitochondrial indices, hypothalamus-mediated metabolic function, and amygdala-regulated emotional processes. Natural substances such as black seed (*Nigella sativa*) oil could be beneficial in mitigating these impairments. This study evaluated the role of black seed oil (BSO) on depression and sociability indices, redox imbalance, mitochondrial-dependent markers, and insulin expression in mice chronically exposed to ampicillin. Forty adult male BALB/c mice (30 ± 2 g) were either exposed to ampicillin (1000 mg/kg; for 3 weeks) or treated with BSO (2 mL/kg; for 3 weeks) following antibiotic exposure. Thereafter, animals were assessed for depressive-like behavior and social interaction. Animals were later euthanised, and whole blood and brain tissues were collected analysis. Animals displayed depressive-like behavior and impaired social interactive behaviors, and disruption of mitochondrial-dependent markers in plasma and hypothalamic tissue following chronic exposure to antibiotics. Furthermore, chronic antibiotic exposure downregulated insulin expression in the amygdala and hypothalamus. However, administration of the BSO improved depressive-like behavior, social interaction deficit in the antibiotic-exposed mice. Lipid peroxidation with increased plasma malondialdehyde (MDA) level was ameliorated while plasma superoxide dismutase (SOD) level was elevated following antibiotic exposure. Downregulation of plasma lactate, hypothalamic lactate, and hypothalamic creatinine kinase (CK) levels, as well as upregulation of insulin expression in the amygdala and hypothalamus were observed with BSO treatment compared to the untreated antibiotic group. Overall, findings from this study suggest the beneficial role of BSO as an adjuvant therapy in preventing and abrogating mood behavioral and metabolic impairments of chronic antibiotic exposure.

A 10-02 | From the molecular hallmarks to motor behavior: Characterization of a new transgenic mouse model for spinocerebellar ataxia type 2

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Spinocerebellar ataxia type 2 (SCA2) is a rare disease with no cure, and therefore patients depend on symptomatic and supportive treatments. It is a highly debilitating disease affecting predominantly the brain with symptoms that include motor and coordination impairment. SCA2 is caused by an abnormal expansion of the CAG triplet in the coding region of the ATXN2 gene. When it has above 33 CAG repeats, it originates a protein with an abnormally expanded glutamine tract. The mutant protein impairs several cellular functions, leading to neuronal degeneration and death. Several rodent models were developed to study the neuropathology and potential therapies for SCA2. However, most of them fail to mimic a complete SCA2 phenotype, taking too long to develop disease-related symptoms or failing to display neuronal-associated deficits.
Thus, we developed a novel transgenic SCA2 mouse model with the human ATXN2 gene encoding for 129 repetitions of CAG, under the control of a L7-6 promotor, which is a specific for Purkinje cells. We established 4 transgenic mouse lines, which are undergoing characterization. Every 4 weeks animals were submitted a battery of behavior tests, over a year. Animals were sacrificed in different time points for molecular and histological analysis, to access the presence of mutant protein aggregates and neurodegeneration. The characterization is still ongoing, but preliminary results indicate that animals display alterations in motor performance and signs of neuronal death, from an early age. This model will be of extreme importance for SCA2 to both evaluate novel therapeutic strategies and to investigate the molecular alterations underlying the pathology.

A 10-03 | Investigating the impact of apolipoprotein E4 on amyloid and tau pathology in an Alzheimer's disease mouse model

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