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IMMUNOMODULATORY AND PROTECTIVE EFFECTS OF EXTRACTS FROM GREEN LEAVES AND RHIZOMES OF *P. OCEANICA* (L.) DELILE ON RAW 264.7 MACROPHAGES AND A HUMAN BLOOD-BRAIN BARRIER MODEL

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Bioactive compounds from marine biodiversity exert several beneficial effects on human health (*e.g.,* anti-inflammatory and antioxidant). In particular, extracts derived from green leaves (GLE) and rhizomes (RE) of *P. oceanica* have been shown to exert antitumoral activity *in vitro* against HepG2 cells[1]. Their prominent polyphenolic content prompted us to assess the potential anti-inflammatory effect on LPS-treated mouse RAW 264.7 macrophages and TNFα-treated endothelial cells of an *in vitro* model of human blood brain barrier (BBB)[2] . No cytotoxic effect and a reduction of nitrite production by LPS-treated macrophages were found after 24 h-treatments with increasing concentrations of both extracts. A differential immuno-modulatory activity of both extracts was revealed by qPCR and Western blot assays. Subsequently, shifting the focus from the peripheral level, to investigate their potential anti-inflammatory effect at the central nervous system level, an *in vitro* model of inflamed BBB consisting of a human endothelial cells/pericytes co-culture exposed to TNFα[3], was used. Even though both extracts appeared ineffective in reducing inflammation, interestingly they did not alter BBB integrity but played a protective role reducing the TNFα-induced permeability alteration and counteracting the release of nitrites. Noteworthy, only RE up-regulated both mRNA and protein expression of molecular markers of tight and adherens junctions, leading to a recovery of protein delocalization after exposure to TNFα. These results prompt further investigation to detail the potential immunomodulatory role of GLE and RE and to unveil the molecular cascade responsible for the observed beneficial effect on BBB integrity.

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VASOSTATIN 1 MODULATES AUTISM-LIKE BEHAVIORS AND HIPPOCAMPAL NEUROINFLAMMATION IN BTBR MICE

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Autism spectrum disorder (ASD) is a neurodevelopmental disorder that causes behavioral impairments along with cardiometabolic disease, immune system dysregulation and neuroinflammation[1,2]. Vasostatin 1 (VS1), the most conserved fragment of chromogranin A, is implicated in the inhibition of angiogenesis and inflammation[3]. In the present study, we evaluated the effects of an intraperitoneal treatment of VS1 for 4 weeks on social and cognitive deficits, and on repetitive behaviors of an idiopathic autism model (BTBR). Furthermore, expression profiles of some pro-inflammatory cytokines (IL-1β, IL6) and NF-kB were investigated in the hippocampus, a brain region involved in social and cognitive deficits[4]. Interestingly, VS1 reversed sociability deficits in BTBR by increasing time spent in the chamber with the stranger ($p<0.001$) and sniffing it ($p<0.001$) rather than the novel object in the three-chamber test. VS1 also reduced self-grooming behaviors ($p<0.001$) as well as rescuing memory impairments as indicated in the novel object recognition test by a higher discrimination index ($p<0.001$) with respect to BTBR treated with a saline solution. Such behavioral effects were related to reduced expression levels of both IL-6 and IL-1β ($p<0.001$) as well as NF-kB ($p<0.01$) in the hippocampus of BTBR mice treated with VS1. This is a first study highlighting a potential therapeutic role of VS1 as indicated by its involvement in decreasing the pro-inflammatory response with consequent improvement of the behavioral deficits typical of ASD.

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