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Spirulina platensis Suppressed iNOS and Proinflammatory Cytokines in Lipopolysaccharide-Induced BV2 Microglia

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Abstract: The disease burden of neurodegenerative diseases is on the rise due to the aging population, and neuroinflammation is one of the underlying causes. *Spirulina platensis* is a well-known superfood with numerous reported bioactivities. However, the effect of *S. platensis* Universiti Malaya Algae Culture Collection 159 (UMACC 159) (a strain isolated from Israel) on proinflammatory mediators and cytokines remains unknown. In this study, we aimed to determine the anti-neuroinflammatory activity of *S. platensis* extracts and identify the potential bioactive compounds. *S. platensis* extracts (hexane, ethyl acetate, ethanol, and aqueous) were screened for phytochemical content and antioxidant activity. Ethanol extract was studied for its effect on proinflammatory mediators and cytokines in lipopolysaccharide (LPS)-induced BV2 microglia. The potential bioactive compounds were identified using liquid chromatography-mass spectrometric (LC-MS) analysis. Ethanol extract had the highest flavonoid content and antioxidant and nitric oxide (NO) inhibitory activity. Ethanol extract completely inhibited the production of NO via the downregulation of inducible NO synthase (iNOS) and significantly reduced the production of tumor necrosis factor (TNF)-α and interleukin (IL)-6. Emmotin A, palmitic amide, and 1-monopalmitin, which might play an important role in cell signaling, have been identified. In conclusion, *S. platensis* ethanol extract inhibited neuroinflammation through the downregulation of NO, TNF-α and IL-6. This preliminary study provided insight into compound(s) isolation, which could contribute to the development of precision nutrition for disease management.

Keywords: *Spirulina platensis*; neuroprotective; anti-neuroinflammation; antioxidants; nitric oxide; BV2 microglia

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Citation: Ngu, E.-L.; Tan, C.-Y.; Lai, N.J.-Y.; Wong, K.-H.; Lim, S.-H.; Ming, L.C.; Tan, K.-O.; Phang, S.-M.; Yow, Y.-Y. *Spirulina platensis* Suppressed iNOS and Proinflammatory Cytokines in Lipopolysaccharide-Induced BV2 Microglia. *Metabolites* **2022**, *12*, 1147. https://doi.org/10.3390/metabo12111147

Academic Editors: Fan Zhang and Fei Gan

Received: 24 October 2022
Accepted: 18 November 2022
Published: 20 November 2022

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1. Introduction

The global life expectancy was reported to increase to 73 years in 2017, accompanied by an increase in age-related disease burdens, including neurodegenerative diseases. As the major neurodegenerative disease, Alzheimer's disease (AD) has contributed to a 38.3% increment in disability-adjusted life-years (DALYs) within 10 years [1]. In addition, the number of death caused by AD has increased from 1.004 million in 2010 to 1.639 million in 2019, making AD the sixth leading cause of global deaths [2]. Neurodegenerative diseases are characterized by gradual neuronal loss due to brain injuries and pathological aging, which exaggerates age-related cognitive decline [3]. Neuroinflammation is one of our body's defense mechanisms, which maintains body homeostasis and protects the central nervous system (CNS) against pathogenic insults [4]. As the primary mediator