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Review

Neuroinflammation in the Central Nervous System: Exploring the Evolving Influence of Endocannabinoid System

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Abstract: Neuroinflammation is a complex biological process that typically originates as a protective response in the brain. This inflammatory process is triggered by the release of pro-inflammatory substances like cytokines, prostaglandins, and reactive oxygen and nitrogen species from stimulated endothelial and glial cells, including those with pro-inflammatory functions, in the outer regions. While neuronal inflammation is common in various central nervous system disorders, the specific inflammatory pathways linked with different immune-mediated cell types and the various factors influencing the blood-brain barrier significantly contribute to disease-specific characteristics. The endocannabinoid system consists of cannabinoid receptors, endogenous cannabinoids, and enzymes responsible for synthesizing and metabolizing endocannabinoids. The primary cannabinoid receptor is CB1, predominantly found in specific brain regions such as the brainstem, cerebellum, hippocampus, and cortex. The presence of CB2 receptors in certain brain components, like cultured cerebellar granular cells, Purkinje fibers, and microglia, as well as in the areas like the cerebral cortex, hippocampus, and cerebellum is also evidenced by immunoblotting assays, radioligand binding, and autoradiography studies. Both CB1 and CB2 cannabinoid receptors exhibit noteworthy physiological responses and possess diverse neuromodulatory capabilities. This review primarily aims to outline the distribution of CB1 and CB2 receptors across different brain regions and explore their potential roles in regulating neuroinflammatory processes.

Keywords: neuroinflammation; proinflammatory cytokines; endocannabinoid system; microglia; cannabinoid receptor 1; cannabinoid receptor 2

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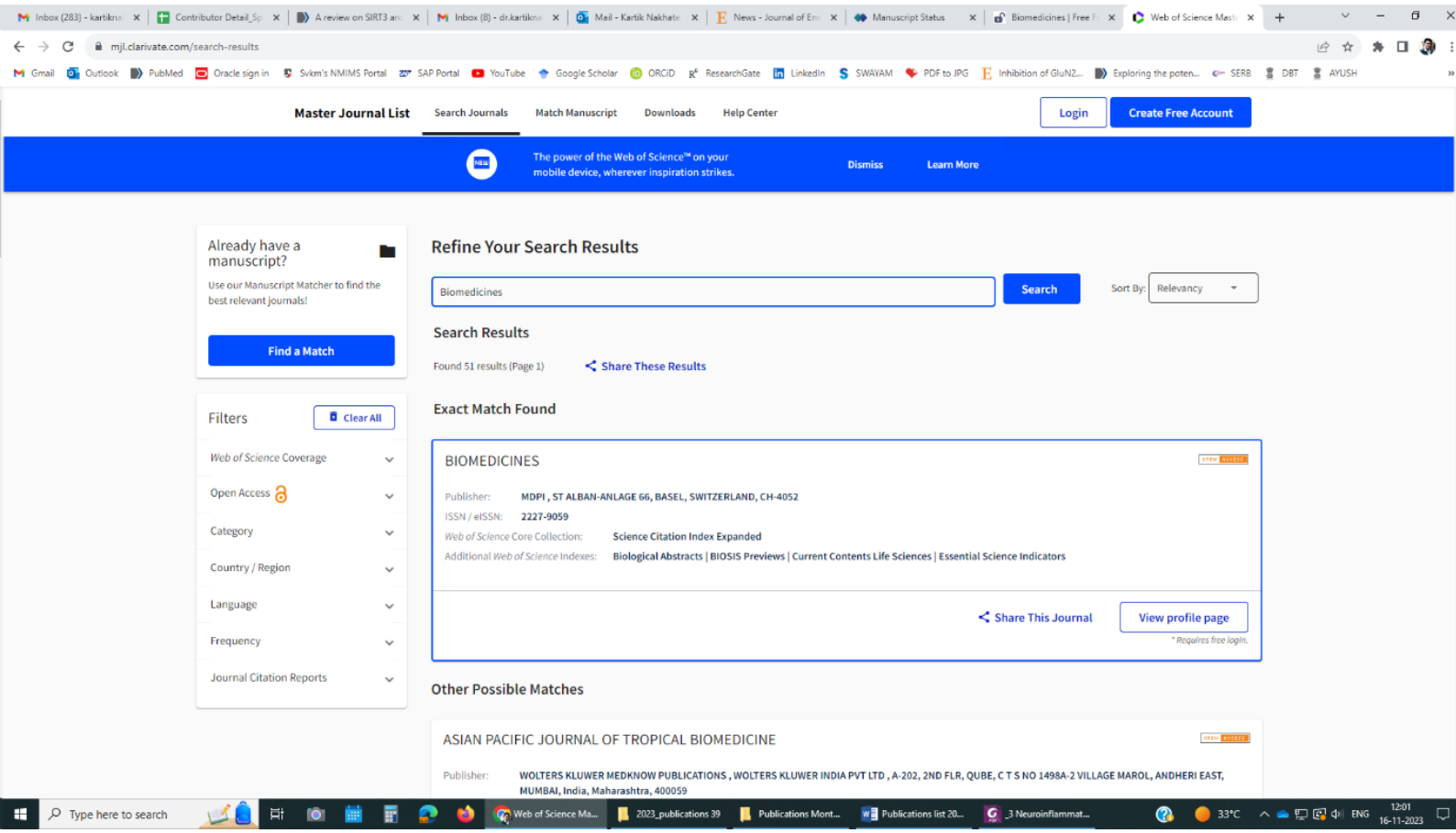
1. Introduction

Neuroinflammation is a multifaceted biological framework that normally originates as a brain's protective response, with the goal of providing safeguarding. Nevertheless, it is possible to develop into a protracted immune system activation, culminating in an unfavorable clinical state [1]. The inflammation of the central nervous system (CNS), which includes the brain and spinal cord, is frequently referred to as neuroinflammation [2]. The expulsion of pro-inflammatory chemicals such as cytokines, prostaglandins, and reactive oxygen and nitrogen species from stimulated endothelium and glial cells triggers neuroinflammatory process. Following this, pro-inflammatory cells from the outer regions enter the CNS [3]. As a consequence, the inflammation of neurons can cause swelling, tissue damage, and neurological dysfunction, as well as acceleration, which leads to memory loss and the onset of neurodegenerative disorders. Hazardous physiological waste products, troublesome self-proteins in autoimmune reactions, the aging process, pathogenic load caused by viruses and bacteria, as well as traumatic neurological and spinal cord lesions,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