1521-0103/389/S3/supplement: ASPET 2024 Annual Meeting Abstract
The Journal of Pharmacology and Experimental Therapeutics

dx.doi.org/10.1124/jpet.339.130158
J Pharmacol Exp Ther 389: June 2024

Cultured adult mouse brain and spinal microglia exhibit differential proinflammatory responses: therapeutic implication for neurologic disorders

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Abstract ID 130158 **Poster Board 339**

The pathogenesis of a variety of neurologic disorders including Parkinson's and Alzheimer's disease may involve microglial activity. Microglia, as the resident immune cells in the central nervous system, are highly dynamic and exhibit a wide spectrum of morphological and functional characteristics under physiological and especially pathological conditions. In this study, we characterized the proinflammatory responses of brain and spinal microglia. Microglia isolated from adult C57BL/6J male mice using microglia/monocyte-specific antibody-conjugated immunobeads were cultured and then treated with Toll-like receptor-4 agonist bacterial endotoxin lipopolysaccharide to induce a proinflammatory phenotype. Global protein expression profiles were analyzed with mass spectrometry-based proteomic analysis. A total of more than five thousand proteins were identified and quantified in all groups, with nearly four hundred proteins demonstrating significant changes in profiled protein expression. While the changes in expression levels of the majority of the proteins in the canonical Toll-like receptor-4 mediated pathways were similar between activated brain and spinal microglia, a number of proteins including interleukin-18 showed stark differences. Bioinformatic Ingenuity pathway analysis further identified differences in predicted up- and down-stream pathways between brain and spinal microglia. Taken together, results from this study further unravel the molecular differences of microglia across different regions of the central nervous system, and facilitates the identification of critical proteins/pathways with potential therapeutic implications in treating neuroinflammation-related neurologic disorders.