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Andrographolide attenuates activation of NF-κB, JNK signaling pathways and chemokines gene expression in astrocytes: An implication for anti neuroinflammation therapy

Siew Ying Wong, Peter T H Wong and **Mitchell K P Lai**
National University of Singapore, Singapore

Substantial evidence gained over the past two decades has supported the conclusion that neuroinflammation is associated with Alzheimer's disease (AD) pathology. Therefore, keeping neuroinflammatory responses regulated with anti-inflammatory compounds could be beneficial in ameliorating the disease conditions. In our study, we investigated andrographolide, a bioactive molecule isolated from *Andrographis paniculata* with anticancer and anti-inflammatory activities. Recent work suggested that andrographolide can inhibit the inflammatory actions of microglia, but its effects on chemokine production and other molecular markers of reactive astrogliosis are unclear. Using rat primary astrocytes, we showed that andrographolide treatment attenuated release of chemokine CCL5 andupregulation of glial fibrillary basic protein (GFAP) after IL-1β stimulation. Moreover, treatment of andrographolide before and after LPS stimulation inhibited the elevation of CCL5, CXCL5, CXCL1, CXCL10, CX3CL1, and CCL2 chemokines mRNAs, suggesting potential effects of andrographolide in attenuating leukocytes recruitment into the brain during neuroinflammation events. Andrographolide also decreased activation of c-Jun N-terminal kinase (JNK) and nuclear factor-κB p65(NF-κB), the key regulator of inflammation. These results suggested andrographolide being a potential anti-neuroinflammation compound and it should be further assessed for its efficacy in conditions characterized by significant neuroinflammation, including AD.

Biography

Siew Ying Wong is from National University of Singapore (NUS). She is currently a 4th year Postgraduate student with a strong interest in neuroinflammation research. Knowing that neuroinflammation is implicated in the pathology of many neurodegenerative diseases, she started looking for potential therapeutics for neuroinflammation diseases and molecules mediating neuroinflammation. As limited therapeutics is available for neurodegenerative diseases, she hope with her research work and contributions, she could soon find therapeutics that could ameliorate disease conditions and improve living quality of patients.

littleharmony2004@gmail.com