"*Metabolomic characterization and bioactivity assays of Momordica charantia (bitter melon) juice*"

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Momordica charantia, also known as bitter gourd, is a climber plant that is widely cultivated in Asia, India, East Africa, and South America. Although *M. charantia* is known for its anti-diabetic and anti-inflammatory activities, its mechanism of action is yet to be fully understood. The current study aimed to examine the bioactivity of *M. charantia* in modulating lipopolysaccharide (LPS)-induced inflammation. RAW 264.7 murine monocytic macrophages were treated with *M. charantia*, and real-time PCR was applied to analyze the expression of genes involved in inflammatory pathway and glucose metabolism in the lipopolysaccharide (LPS) activated RAW264.7 cells treated with or without *M. charantia* water extract. The results showed that the mRNA level of pro-inflammatory cytokines such as *IL6*, *TNF-α*, *IL1β*, glycolytic gene such as *HK2* and the main glucose transporter *GLUT1* were upregulated significantly by LPS. Notably, these changes in gene expression can be inhibited by *M. charantia* treatment. Furthermore, *M. charantia* was found to inhibit LPS-induced on NF-κB (p65) nuclear translocation, which was attributed to defects in IκB-α phosphorylation. As upregulated glucose metabolism has been associated with inflammation event in RAW264.7 cells, the metabolic effects of *M. charantia* was further investigated with end-point glucose and lactate analysis with extracellular medium. Consistently, our results showed that induction with LPS increased the consumption of glucose and lactate production in activated RAW264.7 cells, and the upregulated glycolysis can be inhibited by *M. charantia* treatment. Taken together, the current results provide new insight into the anti-inflammatory activity of *M. charantia* treatment, which can be associated with downregulation of glucose metabolism. The current findings contribute to the accumulating evidences showing the potential of *M. charantia* in providing therapeutic effect to inflammation and inflammation-related disorders.

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