Life Sciences
Volume 265, 15 January 2021, 118750

Lauric acid ameliorates lipopolysaccharide (LPS)-induced liver inflammation by mediating TLR4/MyD88 pathway in Sprague Dawley (SD) rats

Hidayat Ullah Khan [a], Khurram Aamir [a], Patricia Regina Jusuf [b], Gautam Sethi [c], Sreenivas Patro Sisinthy [d], Reena Ghildyal [e], Aditya Arya [b, f, g]

https://doi.org/10.1016/j.lfs.2020.118750

Abstract

Background

Lipopolysaccharide (LPS) is an endotoxin that leads to inflammation in many organs, including liver. It binds to pattern recognition receptors, that generally recognise pathogen expressed molecules to transduce signals that result in a multifaceted network of intracellular responses ending up in inflammation.

Aim

In this study, we used lauric acid (LA), a constituent abundantly found in coconut oil to determine its anti-inflammatory role in LPS-induced liver inflammation in Sprague Dawley (SD) rats.

Method

Male SD rats were divided into five groups ($n = 8$), injected with LPS and thereafter treated with LA (50 and 100 mg/kg) or vehicle orally for 14 days. After fourteen days of LA treatment, all the groups were humanely killed to investigate biochemical parameters followed by pro-inflammatory cytokine markers; tumour necrosis factor-α (TNF-α), interleukin-6 (IL-6), and IL-1β. Moreover, liver tissues were harvested for histopathological studies and evaluation of targeted protein expression with western blot and localisation through immunohistochemistry (IHC).

Results

The study results showed that treatment of LA 50 and 100 mg/kg for 14 days were able to reduce the elevated level of pro-inflammatory cytokines, liver inflammation, and downregulated the expression of TLR4/NF-κB mediating proteins in liver tissues.

Conclusion

These findings suggest that treatment of LA has a protective role against LPS-induced liver inflammation in rats, thus, warrants further in-depth investigation through mechanistic approaches in different study models.