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# Comment on: Dietary supplementation with laminarin, a fermentable marine β (1–3) glucan, protects against hepatotoxicity induced by LPS in rat by modulating immune response in the hepatic tissue

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I wish to suggest another interpretation for the data published by Neyrinck et al. [1] The paper demonstrated that the beta-glucan larminarin (LAM) protected the liver from injury induced by lipopolysaccharides (LPS) and reduced TNF-α and $NO_2$ secretion. The authors suggested that this was due to the direct effect of beta-glucan on immune cell function alongside an indirect effect of dietary fibre properties.

However, the LPS used by Neyrinck et al. was derived from pathogenic *Escherichia coli* and as such may have itself been contaminated with beta-glucans. Pathogenic bacteria, e.g., *Pneumocystis carinii* are known to contain beta-glucans [2]. Beta-glucan has been found in the cell wall of pathogenic *E. coli* but was absent in non-pathogenic *E. coli* [3,4]. Beta-glucan has also been detected in periplasmic material of pathogenic *E. coli* [5].

Therefore, activation of dectin-1 by contaminating particulate beta-glucans may have participated in the inflammation that Neyrinck et al. attributed solely to LPS. The activated dectin-1 pathway could have acted synergistically with the TLR pathway to increase production of TNF-α and $NO_2$.

LAM as well as soluble beta-glucan has different role with particle beta-glucan [6]. It blocks beta-glucan receptor rather than stimulates it [6–8]. It has previously been used to block the action of dectin-1 activated by mycobacteria to reduce interleukin-12 production [9]. The results from Neyrinck et al. are consistent with the role of LAM as a beta-glucan receptor blocker. In the absence of LAM, stimulation of beta-glucan receptors would induce TNF-α, interleukins and $NO_2$ [10]. A particulate glucan from *saccharomyces cerevisiae* cell wall (Zymosan) stimulated production of TNF-α and $NO_2$ leading to shock in mice [11]. LAM has been shown to reduce the release of the proinfammatory factor arachidonate in response to zymosan [12].

Thus, blockage of dectin-1 by LAM could inhibit TNF-α and $NO_2$ production. If true, this opens the possibility of using beta-glucan receptor blocking agents like LAM to treat other inflammatory diseases such as arthritis. This would be in addition to the already established use of beta-glucan like lentinan for the treatment of cancer and infectious diseases. Structurally modification of LAM may enhance its beta-glucan receptor blocking effect, and this warrants further research.

## References

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