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 laminarin (LAM) protected the liver from injury induced by lipopolysaccharides (LPS) and reduced TNF-α and NO₂ 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 NO₂.

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 NO₂ [10]. A particulate glucan from saccharomyces cerevisiae cell wall (Zymosan) stimulated production of TNF-α and NO₂ leading to shock in mice [11]. LAM has been shown to reduce the release of the proinflammatory factor arachidonate in response to zymosan [12].

Thus, blockage of dectin-1 by LAM could inhibit TNF-α and NO₂ 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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