ABSTRACT
Mechanism of Sepsis Improvement after Zinc Supplementation in Lipospolysaccharide Exposure Rodent

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Background:
Severe sepsis increases pro-inflammatory cytokines and damage to the intestinal mucosa, causing systemic translocation to the commensal bacteria. Low zinc levels were also found in patients with sepsis.

Objective
To explain the mechanisms of sepsis improvement after zinc administration through cytokine regulation and improvement of intestinal mucosal

Material and Method:
Samples of 40 rats were randomized into 4 group of Control, LPS, LPS-Zinc, and Zinc . Blood sampling in 2-hour after LPS or placebo administration to measure zinc level. Zinc was administered in LPS-Zinc and Zinc group, placebo was given in other groups. Blood sampling in 8, 24 and 72 hours to measure NFkB monocyte expression, TNF-α, IL-6, IL-10, TGF-β, MDA, MMP-8 by sandwich-ELISA method and in 72 hours also measured the zinc content by AAS method and jejunal tissue by SEM analysis. Statistical analyzes used were one-way Anova, Kruskall Wallis, Mann-Whitney, Paired-t tests, Wilcoxon Signed Rank Test and path analysis.

Results
There were decreased in NFkB monocyte expression, levels of TNF-α, IL-6, MDA, and MMP-8 serum in the LPS-Zinc group compared to the LPS group and elevated levels of IL-10, TGF-β in the LPS-Zinc group compared to the LPS group. Improvement of intestinal mucosa occured in LPS-Zinc group.

Conclusion
Administration of zinc in sepsis improves the condition of sepsis by decreasing pro-inflammatory cytokine (TNF-α and IL-6), increasing anti-inflammatory cytokine (TGF-β and IL-10) and improving intestinal mucosa (villi structure of jejunum).

Keyword: zinc, sepsis, cytokine, intestinal mucosal