THE NUMBER OF MACROPHAGES CELLS OF WISTAR RATS GINGIVAL AFTER PROBIOTIK ADMINISTRATION INDUCED BY *P. GINGIVALIS* AND LPS *E. COLI*

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Abstract

Background: Probiotics are microbes of lactic acid bacteria group that works to maintain health of the host. Probiotic bacteria can stimulate the immune system, among others, increase the function of macrophage phagocytosis, natural killer cells, monocytes and neutrophils, and is able to stimulate the secretion of IgM and IgA production with the end result increasing the production of antibodies locally and systemic. This study aims to determine the effect of probiotics against the macrophage cell number in gingival wistar rats induced *P.gingivalis* and LPS *E.coli*. **Methods**: This study using a sample of male wistar rats and divided into five groups. Group I: is the control without treatment; Group II: induced *P.gingivalis* ATCC 33277; Group III: induced LPS *E.coli*; Group IV: induced *P.gingivalis* ATCC 33 277 + an injection of probiotic (*Lactobacillus casei* ATCC 4224); group V: induced LPS *E. coli* + an injection Probiotics (*Lactobacillus casei* ATCC 4224). The treatment carried out for 5 days, then decapitation were done to take the gingival tissue, after that the histological observation were done to see the number of macrophage cells. Furthermore, the data were analyzed by Kruskal Wallis test, was continued by Mann Whitney test. **Results**: The result show that there's increased of average macrophage cells to all groups; statistically showed the significant difference ($p<0,05$. **Conclusion**: Administration of probiotics can increase the number of macrophage cells in wistar rats gingival after induced by *P.gingivalis* LPS *E.coli*.

Introduction

Periodontal diseases were common and well spread in community. They affect all ages from children, adults, and elderly. One form of periodontal diseases is the occurence of inflammation in periodontal tissue. It can be limited to gingiva (gingivitis) or affect a more extensive area of periodontal tissue (periodontal ligamen, cementum and alveolar bone) which recognized with the term periodontitis [1].

Major etiology of periodontitis is plaque bacteria [2]. The typical nature of this periodontal diseases lies in its lack of pain characteristic, except when acute complications arise. Therefore this condition often present only when it has achieved advance stage [1]. Bacteria most commonly found as the cause of periodontitis were Gram negative bacteria, such as *Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Prevotella intermedia*, and *Bacteriodes forsythus*. These anaerobic Gram negative bacteria exert products such as biologically active endotoxins or lipopolisaccharide (LPS) which trigger biological acitvities leading to inflammation [2,3].

In periodontitis, there is a direct relationship with pathogenic disease. There are certain pathogenic bacteria that have been strongly linked with periodontal diseases,such as *Actinobacillus actinomycetemcomitans, Spirochetes*, and *Porphyromonas gingivalis* [4]. *Porphyromonas gingivalis* is anaerob Gram negative bacterial involved in pathogenesis of periodontal diseases, and were found in subgingiva of patients with periodontitis [5].

Lipopolysaccharide (LPS) was also one cause of periodontal abnormality. This substance is the main structure of cell walls of anaerobic Gram negative bacteria which function for maintaining integroty of bacterial cells and protecting bacteria from host's immune response [6]. Lipopolysaccharide can stimulate