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**POCKET MEASUREMENT METHODS IN WISTAR RATS PERIODONTITIS INDUCED BY BACTERIA AND THE INSTALLATION OF SILK LIGATURE: AN EXPERIMENTAL STUDIES**

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**ABSTRACT**  
**Objective:** The purpose of this study was to develop an experimental model of induction of periodontal disease in Wistar rats, using a combination of bacterial induction and binding of silk ligature with respect to pocket periodontal depth.  
**Methods:** Experiments with a pre-post group design was applied. Five adult male Wistar rats from the Udayana University's Analytical Laboratory were included in the study. Measuring pocket depth in experimental animals using dental probe was previously administered. Then performed the installation of silk ligature and bacterial induction *Porphyromonas gingivalis*, on the mandibular anterior teeth. Release of silk ligature on day 7<sup>th</sup>, without the action of debondement of plaque or calculus on rat teeth. Observation of the development of the testing animals on the 3d, 7d, and 14d. On the 14d, re-measurement of pocket depth was conducted.  
**Results:** Periodontal tissue abnormalities with silk ligature placement and bacterial infiltration *Porphyromonas gingivalis* cause periodontal inflammation with periodontal pocket formation, with a mean depth of 3.2 mm, which was analyzed using Wilcoxon p<0.05.  
**Conclusion:** In this study combining bacterial induction and the installation of silk ligature can shorten the induction of periodontal tissue disease characterized by the formation of pocket periodontitis.  
**Keywords:** Wistar rat periodontitis, Periodontal pocket, Periodontal disease

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**INTRODUCTION**  
Periodontal disease is one of the oral and dental diseases that is most commonly found in humans caused by several factors, one of which is due to the accumulation of bacterial plaque [1]. The WHO report (2003) suggests that diseases have high prevalence rates worldwide. Periodontitis is damage caused by the host defense. Clinically periodontitis is characterized by plaque accumulation, calculus and pocket formation, periodontal tissue inflammation and alveolar bone loss, gum bleeding, accompanied by pain with untreated halitosis resulting in tooth loss. Pathogenic bacteria are suspected of causing an inflammatory response, gingival and periodontal damage [2]. To study the phenomenon of periodontal inflammation and the effects of periodontal treatment, several animal models have been used as animal studies of periodontitis [3].  
Periodontal disease can be divided into different phases and each can be studied separately depending on the animal model. This phase involves the colonization of biofilm-bacteria, invasion of bacterial products from epithelial tissue to connective tissue, destructive induction of host response to connective tissue and bone absorption. Improvement processes that follow tissue damage by selecting appropriate animal models in each of these phases can be analyzed individually, whereas in human research it is difficult to isolate specific steps, and in vitro studies are less complex to identify specific phases [4]. Rats in particular, are models relevant for experimental periodontal studies [5]. The structure of the rat tooth gingiva is similar to human gingival sulcus, superficial and the presence of junctional epithelium on the tooth surface, junctional epithelium is a pathway for the entry of foreign bodies and bacterial endotoxins, thereby causing the onset of inflammation [5].  
The incidence of periodontal disease in rats is certainly rare so it needs to be induced to cause periodontitis. Experimental results indicate a horizontal bone loss in rats infected with *Actinomycescummatus aggregatibacter* (*Actinobacillus*) or *P. gingivalis*. Periodontitis is induced in mice by placing a silk or cotton ligature, which results in retention of bacterial plaque on the gingival sulcus around the molar teeth [6]. The rat induction model was performed by Isac et al. by attaching a ligature to the mandibular anterior-mandibular tooth. After 14d of ligature insertion, histopathologic results show signs of inflammation with neutrophil infiltration and alveolar osteolysis [7]. The accumulation of plaque bacteria produces toxins that will irritate the gingiva, the tooth stimulates a chronic inflammatory response in which the body will react by itself then the tissues and bones supporting the tooth will be damaged. The gingiva will separate from the tooth, forming the infected pocket (the space between the tooth and gingiva). As the pocket progresses deeper and broader it damages the gingiva and alveolar bone. In some experiments, mice periodontitis is performed by injecting oral bacterial bacteria such as *Porphyromonas gingivalis* to induce periodontal abnormalities [8].  
The time required for the occurrence of chronic periodontitis in mice is generally 3 w. Inflammation begins at week 1 (acute inflammatory state), week 2 (severe inflammatory state), and week 3 (chronic inflammatory state). The period is almost equal to the time it takes for humans to produce the same disorder [6]. Research of induction of periodontitis by using ligature and bacterial induction has been done Utami et al. induced periodontitis with a combination of ligature and bacterial injection 1x weekly for 4 w [9]. Noting the length of time and repetition of injections in rats, the purpose of this study is to simplify the procedure and to shorten the time required for the induction of periodontitis in rats as well as obtained a method to measure the depth of pocket which is one of the clinical symptoms in periodontitis.

**MATERIALS AND METHODS**  
**Material**  
The research is experimental research with pre and post control group design. The study was conducted at Udayana University's