

**UNIVERSITI TEKNOLOGI MARA**

**THE IMPACT OF  
VITAMIN D ON  
CLINICAL PARAMETERS AND  
THE BONE TURNOVER  
BIOMAKERS IN LIGATURE  
INDUCED PERIODONTITIS:  
EXPERIMENTAL STUDY  
IN RATS**

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of the requirements for the degree of  
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## **AUTHOR'S DECLARATION**

I declare that the work in this dissertation was carried out in accordance with the regulations of Universiti Teknologi MARA. It is original and is the results of my own work, unless otherwise indicated or acknowledged as referenced work. This thesis has not been submitted to any other academic institution or non-academic institution for any degree or qualification.

I, hereby, acknowledge that I have been supplied with the Academic Rules and Regulations for Post Graduate, Universiti Teknologi MARA, regulating the conduct of my study and research.

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## ABSTRACT

Periodontal disease is a multifactorial disease process involving the host and microbial challenges. Nutritional Vitamin D has been associated as a modifying factor to periodontal diseases. The aim of this study was to evaluate the effect of vitamin D on the clinical, radiographical and serum level changes of OPG, DKK1, SOST, and FGF23 in a ligature induced periodontitis, an experiment study in rats. A total of 28 rats were included in this study and divided into test groups of Vitamin D supplement, Vitamin D deficient and control. A ligature placed around the bilateral mandibular incisor for 2 weeks to induce periodontal tissue destruction. Clinical attachment and Radiographical changes were recorded as well as serum sample was tested for OPG, DKK1, SOST, and FGF23. Two weeks after ligature placement, gingival inflammations were induced. Groups that were made Vitamin D deficient exhibited greater amount of clinical attachment loss ( $1.05 \pm 0.50$  mm) as compared to control ( $0.83 \pm 0.14$  mm) and groups with supplemented Vitamin D at ( $0.60 \pm 0.13$  mm) showing significant differences ( $p < 0.05$ ). Amount of radiographic alveolar bone loss was greater were seen in Vitamin D deficient ( $0.61 \pm 0.06$ mm) as compared to control ( $0.45 \pm 0.04$  mm) and groups with supplemented Vitamin D at ( $0.26 \pm 0.03$  mm) showing significant differences ( $p < 0.05$ ). Vitamin D deficient groups also exhibited a statistically significant reduction in levels of OPG ( $421.65 \pm 29.49$  pg/ml) compared to Control Groups ( $584.84 \pm 137.96$  pg/ml), and Vitamin D Supplemented group ( $715.68 \pm 71.37$  pg/ml). Vitamin D deficient groups exhibited higher concentrations of DKK1 at ( $1336.56 \pm 272.71$  pg/ml) compared to Control Groups ( $1243.86 \pm 207.40$  pg/ml) and Vitamin D Supplemented group ( $909.42 \pm 149.06$  pg/ml). SOST concentration was higher in Vitamin D deficient groups ( $1684.12 \pm 41.92$  pg/ml) compared to Control Groups ( $1682.14 \pm 41.92$ ) and Vitamin D Supplemented group ( $1284.22 \pm 34.20$  pg/ml), and FGF23 concentration was higher in Vitamin D deficient groups ( $531.09 \pm 12.84$  pg/ml) compared to Control Groups ( $627.11 \pm 18.58$  pg/ml), Vitamin D Supplemented group ( $717.79 \pm 20.45$  pg/ml). The results revealed that vitamin D supplementation may have a role in periodontal disease progression. It shows bone turnover biomarkers related to resorption increase in concentration in group with vitamin D deficiency and reduced in vitamin D supplementation group.

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