

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

«إِنَّمَا يَخْشَى اللَّهَ مِنْ  
عِبَادِهِ الْعُلَمَاءُ»

صدق الله العظيم

سورة فاطر: آية ٢٨

**THE ASSESSMENT OF OSTEOPONTIN LEVEL IN  
GINGIVAL CREVICULAR FLUID IN PATIENT  
WITH  
PERIODONTAL DISEASE**

*Thesis*

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# *Dedication*

*To*

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*And*

*Brothers*

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## List of abbreviations

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<b>ABL</b>	Alveolar bone loss
<b>ALP</b>	Alkaline phosphatase
<b>BD</b>	Bone density
<b>BSP</b>	Bone sialoprotein
<b>CAL</b>	Clinical attachment loss
<b>ECM</b>	Extracellular matrix
<b>Eta_1</b>	Early T-cell activation gene -1
<b>FMLP</b>	Formly –peptide
<b>GAP</b>	Generalized aggressive periodontitis
<b>GCF</b>	Gingival crevicular fluid
<b>ICTP</b>	Pyridinoline cross linked carboxy terminal telepeptide of type 1 collagen
<b>IL</b>	Interleukin
<b>INF<math>\gamma</math></b>	Interferon gamma
<b>INOS</b>	Inducible nitric oxide synthase
<b>LAP</b>	Localized aggressive periodontitis
<b>LPS</b>	lipopolysacchrides
<b>MMP</b>	Matrix metalloproteinase
<b>mRNA</b>	Messenger Ribonucleic acid
<b>NK</b>	Natural killer
<b>NO</b>	Nitric oxide
<b>OCN</b>	Osteocalcin
<b>ON</b>	Osteonectin
<b>OPG</b>	Osteoprotegrin
<b>OPN</b>	Osteopontin
<b>PBMCS</b>	Peripheral blood mononuclear cells
<b>PDGF</b>	platelets derived growth factor
<b>PGE2</b>	Prostaglandin E2
<b>PL</b>	Periodontal ligament
<b>PPD</b>	Probing pocket depth
<b>RGD</b>	Argnine-glycine-aspartic acid (one letter code for Arg-Gly-Asp)
<b>SIBLING</b>	Small integrin –binding ligand N-linked glycoproteins
<b>SVVYGLYR</b>	Serine-Valine –valine –tyrosin-glycine-leucine-arginine
<b>TGF<math>\beta</math></b>	Transforming growth factor_ beta
<b>TNF<math>\alpha</math></b>	Tumor necrosis factor – alpha
<b>TRAP</b>	tartrate-resistant acid phosphatase



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# Introduction

Periodontal disease which is marked by inflammation may result in subsequent loss and / or damage to tooth, supporting tissue including bone, cementum and periodontal ligament. (*polson and caton 1982*). It is widely accepted that periodontal pathogens are accused for the initiation and sustenance of the inflammatory process in periodontal disease which is crucial for the destruction of mineralized and non-mineralized extracellular matrices in periodontal tissues (*page 1991*). Bacterial plaque products induce the differentiation of bone progenitor cells into osteoclasts and several host factors released by inflammatory cells are capable of inducing bone resorption in vitro and can play a role in periodontal disease (*Schwartz et al., 1997*). These mediators are interleukin – 1, (1L-1) tumor necrosis factor – alpha, IL – 6 and prostaglandinE2 (PGE2) (*Munday 1991*) and some that are involved in inhibiting bone resorption like osteoprotegrin (*Mc Cauley and Nohuch 2002*).

The national institute of dental and craniofacial research performed concentrated research in salivary diagnostics, significant advancements have been achieved within the past 10 years using saliva, gingival crevicular fluid (GCF) and mucosal transudate as biological samples for the detection of oral and systemic illnesses (*Streckfus and Bigler 2002*). Salivary secretions are easily collected and containing local and systemic – derived biomarkers of periodontal disease (*Mandel 1993*).

Biomarkers of bone resorption or turnover include several biomarkers such as alkaline phosphatase (ALP) which is a catalyzing enzyme and its