



Assessment of the Efficacy of the Host Modulating Agent Glucoseamine Sulphate in the Management Of Chronic And Aggressive Periodontitis

Thesis

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالَ

سُبْحَانَكَ لَا عِلْمَ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

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List of Abbreviations

Abb.	Full term
A.a.....	Aggregatibacter Actinomycetemcomitans
ADA	American Dental Association
Agp.....	Aggressive periodontitis
CAL	Clinical Attachment Loss
CMT.....	Chemically Modified Tetracycline
CP	Chronic Periodontitis
CpG.....	cytosine-phosphate-guanosine
D-CAL.....	Deepest site Clinical Attachment level
D-GI	Deepest Site Gingival Index
D-PD	Deepset Site Probing Depth
D-PI.....	Deepset Site Plaque Index
ELISA	Enzyme linked immunosorbent assay
FAW.....	Food and Agriculture Organization/World Health Organizatin.
FDA.....	Food and Drug Administration
GAgp	Generalized aggressive periodontitis
G-CAL	Generalized Clinical Attachment level
GCF.....	Gingival Crevicular Fluid
G-GI	Generalized Gingival Index
GI	Gingival Index
GluS	Glucosamine Sulfate
GP	Gingipans
G-PD	Generalized Probing Depth
G-PI.....	Generalized Plaque Index
GS	Glucosamine
HLA	Human Leucocytic Antigen
HMT.....	Host Modulation Therapy
IgG	Immunoglobulin G
IL.....	Interleukin

List of Abbreviations Cont...

Abb.	Full term
IL-1 α	Interleukin-1alpha
IL-1B.....	Interleukin-1beta
IL-1ra.....	Interleukin-1receptor anatgonist
IL-r1.....	Interleukin-1receptor
LA _g p	Localized aggressive periodontitis
LPS	Lipopolysaccharides
Lx.....	Lipoxins
MMPs	Matrix Metalloproteinases
NF κ B.....	Nuclear Factor Kappa Beta
NO	Nitric Oxide
NOS	Nitric Oxide Synthase
NSAIDs	Non-Steroidal Anti-Inflammatory Drugs
OA.....	Osteoarthritis
PD	Probing Depth
PDT.....	Photo Dynamic Therapy
PGE ₂	Prostaglandin E ₂
PI.....	Plaque Index
PMNs	Polymorhoneuclar leukocytes
RA	Rheumatoid Arthritis
RANK	Receptor–Activator of Nuclear Factor–KB
RANKL.....	Receptor–Activator of Nuclear Factor–KB Ligand
SDD.....	Sub–Antimicrobial Dose of Doxycycline
SRP	Scaling and Root Planing
TNF– α	Tumor Necrosis Factor Alpha

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Introduction

Periodontal disease is the result of the inflammatory processes that occur in the tissues surrounding the teeth in response to bacterial accumulation in dental plaque which is ultimately responsible for progressive loss of collagen attachment of the tooth and the underlying bone (*Loesche and Grossman, 2001*). It is a multifactorial chronic inflammatory disease in which the incidence and rate of progression of periodontal destruction involves a complex interaction between periodontopathic bacteria and cells of the immune system (*Kinane and Lappin, 2001*).

Periodontal pathogens elicit signals in resident gingival cells or in immune cells infiltrating the gingival tissues that result in immune responses; these responses lead to either the successful removal of the pathogens or to host mediated destruction of the periodontal tissues. In this respect, cytokines in inflamed periodontal tissues, which have been the focus of numerous studies, have been cited as being of major importance in periodontal disease progression (*Okada and Murakami, 1998*).

The most common form of periodontitis is chronic periodontitis either localized or generalized. Where clinical features included: (i) most prevalent in adults, but can occur in children and adolescents; (ii) the amount of destruction is consistent with the presence of local factors; (iii) sub gingival

calculus is a frequent finding; and (iv) slow-to-moderate rates of progression, but may have periods of rapid tissue destruction (*Armitage, 1999*).

While highly destructive forms of periodontitis, formerly considered under the umbrella term of early-onset periodontitis, were renamed Aggressive periodontitis (AgP), is defined as a specific type of periodontal disease that is characterized by rapid attachment loss and bone destruction, resulting in tooth loss at early onset (< 35 years of age) and familial aggregation (*Armitage, 1999*).

As the other chronic inflammatory diseases, cytokines are considered to play an important role in the initiation, progression and in host response to periodontal disease (*Bascones et al., 2005; Salvi and Lang, 2005*). Interleukin-1 β (IL-1 β) is an important pro-inflammatory cytokine involved in a variety of immunological processes in the host response to noxious challenges. Moreover, it is one of the most potent osteoclast-activating factors within the human organism and is thus believed to play an important role in periodontal tissue breakdown (*Nguyen et al., 1991; Quan et al., 2001*).

Numerous studies reported increased concentrations of IL-1 β in gingival crevicular fluid (GCF) at sites with gingivitis or periodontitis (*Rawlinson et al., 2000; Faizuddin et al., 2003*). Furthermore, the level of crevicular IL-1 β is found to increase during experimental gingivitis (*Deinzer et al., 2004; Deinzer et*

al., 2007) and to decrease after periodontal therapy (*Holmlund et al., 2004; Engebretson et al., 2002*). Since common feature of aggressive periodontitis is hyper-responsive macrophage phenotype, including elevated levels of PGE2 and IL-1B (*Tonetti and Mombelli, 1999*); and Some studies indicate that periodontal therapy of aggressive periodontitis reduces the GCF total amount of IL-1 β , suggesting a role for this cytokine in the disease process (*Toker et al., 2008; Rosalem et al., 2011; de Lima Oliveira et al., 2012*). Taken together, IL- 1 β has become a parameter of interest in periodontal research (*Bergmann and Deinzer, 2008*).

Mechanical debridement is the main strategy in periodontal treatment, which can eliminate microbial etiology of periodontal diseases. critical step in a complex chain of events leading to periodontal tissue destruction. However treatment strategies failed to block (or) inhibit the host response mediated tissue destruction to continued bacterial challenge (*Dyke et al., 1993*).

“Perioceutics” or the use of the pharmacological agents specifically developed to manage periodontitis is an interesting and emerging aid in the management of periodontal diseases along with mechanical debridement (*Ryan, 2002*). Host Modulation Therapy (HMT) is a treatment concept that reduces tissue destruction and stabilizes or even regenerates periodontal tissue by modifying host response factors. It has been used for treating osteoporosis and arthritis for several decades (*Shinwari et al., 2014*).