بسم الله الرحمن الرحيم

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تم عمل المسح الضوئي لهذة الرسالة بواسطة / سامية زكى يوسف

بقسم التوثيق الإلكتروني بمركز الشبكات وتكنولوجيا المعلومات دون أدنى مسئولية عن محتوى هذه الرسالة.

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- بالرسالة صفحات لم ترد بالأصل
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- بالرسالة صفحات قد يكون بها خطأ ترقيم

CHEMOTHERAPEUTIC APPROACH IN TREATMENT OF PERIODONTITIS IN EGYPTIAN POPULATION

A THESIS

Submitted for The Partial Fulfillment of The Degree of
Philosophy Doctor in
Medical Sciences (Pharmacology)

By

Yahya Mohamed Ali Ghantawi

B. Dent. Sci. (1990); Master of Pharmacology Assiut (2002); A. Lecturer of Pharmacology; Department of Biological Sciences
Faculty of Dentistry
Teshreen University
Latakia, Syria

Supervised by

Prof. M. H. Abdel-Raheem
Professor and chairman of Pharmacology Department
Faculty of Medicine
Assiut University

Dr. Ehab S. Aldesoky
Assistant Professor of Pharmacology
Faculty of Medicine
Assiut University

Dr. Mohamed Saad Badary
Assistant Professor of Microbiology and Immunology
Faculty of Medicine
Assiut University

Dr. Fatma Abd El Aziz El-Nohmany
Assistant Professor of Periodontology
Faculty of Dentistry
Tanta University

DEPARTMENT OF PHARMACOLOGY
FACULTY OF MEDICINE
ASSIUT UNIVERSITY
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قَالُوا سُبْحَانَكَ لا عِلْمَ لَنَا إِلا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

(سورة البقرة الآية :٣٢)

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Actinobacillus actinomycetemcomitans	*Aa*
Aggressive periodontitis	AgP
Attachment level	AL
Black-pigmented bacteriods	BPB
Campylobacter rectus	*Cr*
Capnocytophaga sp.	*Ca sp.*
Eikenella corrodens	*Ec*
Gingival crevicular fluid	GCF
Gingival Index	GI
Minimal inhibitory concentration	MIC
Papillary bleeding index	PBI
Porphyromonas gingivalis	*Pg*
Prevotella intermedia	*Pi*
Probing Depth	PD
Reactive oxygen species	ROS
Standard error	S.E.
Thiobarbituric acid reactive substances	TBARS
Total antioxidant	TAO

DEDICATION

TO MY PARENTS,
WIFE AND
DAUGHTERS

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ACKNOWLEDGEMENT

In the name of **ALLAH** the beneficent, the merciful. First and above all praise be to **ALLAH** the almighty, the glorious, who has bestowed upon us the faculties of thinking, searching and learning.

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INTRODUCTION AND AIM OF THE WORK

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The term ''periodontal disease'' refers to both gingivitis and periodontitis. Gingivitis is an inflammatory condition of the soft tissues surrounding the teeth (the gingiva) and is a direct immune response to the dental microbial plaque building up on teeth. Periodontitis follows gingivitis and is also influenced by the individual's immune and inflammatory response. It is initiated by microbial plaque, but it occurs in only a subset of the population. Periodontitis involves the destruction of the supporting structures of the teeth including the periodontal ligament, bone and soft tissues (Kinane, 2001).

Aggressive periodontitis (AgP) generally affects systemically healthy individuals less than 30 years old, although patients may be older. Aggressive periodontitis may be universally distinguished from chronic periodontitis by the age of onset, the rapid rate of disease progression, the nature and composition of the associated subgingival microflora, alterations in the host's immune response, and a familial aggregation of diseased individuals (Loe and Brown, 1991)

According to American Academy of Periodontology, aggressive periodontitis was considered as high destructive periodontal diseases (AAP, 1999). Destructive periodontal diseases affect 10–15% of the world population and are a major cause of tooth loss (Brown and Loe, 1993). There is increasing evidence that the disease occurs in a predisposed group of the population that has an aberrant inflammatory/immune response to the microbial plaque that accumulates

around the gingival margin. This exaggerated response is known to result in inadvertent or collateral host tissue damage (Fredriksson et al., 1998).

Gingival crevicular fluid (GCF) is formed when fluid exudes from the vessels of the microcirculation into periodontal tissue and into the sulcus or pocket. As the fluid traverses the inflamed tissue, it is thought to pick up enzymes and other molecules that participate in the destructive process, as well as products of cell and tissue degradation (Perry et al., 1996). The fluid, therefore, offers great potential as a source of factors that may be associated with active tissue destruction. Efforts to develop diagnostic tests based on host factors have been focused almost entirely on analysis of GCF as malondialdehyde (degradation of lipids), total antioxidants (imbalance between free radicals and antioxidant) or iron (Mukherjee, 1985; Janero, 1990 Page, 1992; Chapple et al., 2002).

Collagen is the primary structural protein of all the periodontal tissues (comprising, for example, about 60% of total protein in gingiva and over 90% of the organic matrix of alveolar bone), and the destruction and loss of these fibers is the key event in the pathogenesis of the periodontal pocket (Narayanan and Page, 1983). A specific metalloprotease, collagenase, produced by several types of host cells which reside in or infiltrate the periodontal tissues (e.g., fibroblasts, polymorphonuclear leukocytes, macrophages) is largely responsible for this (Golub et al., 1976; Woolley and Davies, 1981). Also free radicals from host cells or microorganisms were responsible for the destruction of these structures leading to produce a specific compound was called malondialdehyde (Esterbauer and Cheeseman, 1990; Bax et al., 1992; Hall et al., 1995)

Gingival crevicular fluid (GCF) was analyzed to assess the inflammatory process in biochemical terms. into three general groups: 1)