سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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سامية محمد مصطفى

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

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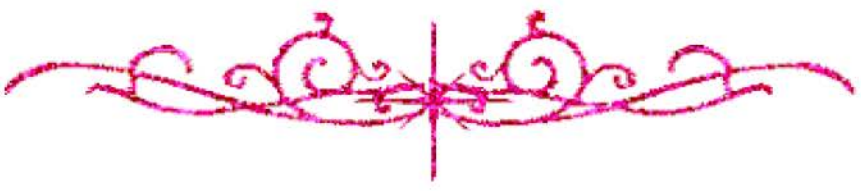

سامية محمد مصطفى

شبكة المعلومات الجامعية

بالرسالة صفحات لم ترد بالأصل

Evaluation of Gingival Crevicular Fluid Interleukin-10 in Diabetic Patients Suffering from Periodontitis

Thesis

Submitted in Partial Fulfilment For The Requirement of The Master Degree in

Oral Medicine and Periodontology

By

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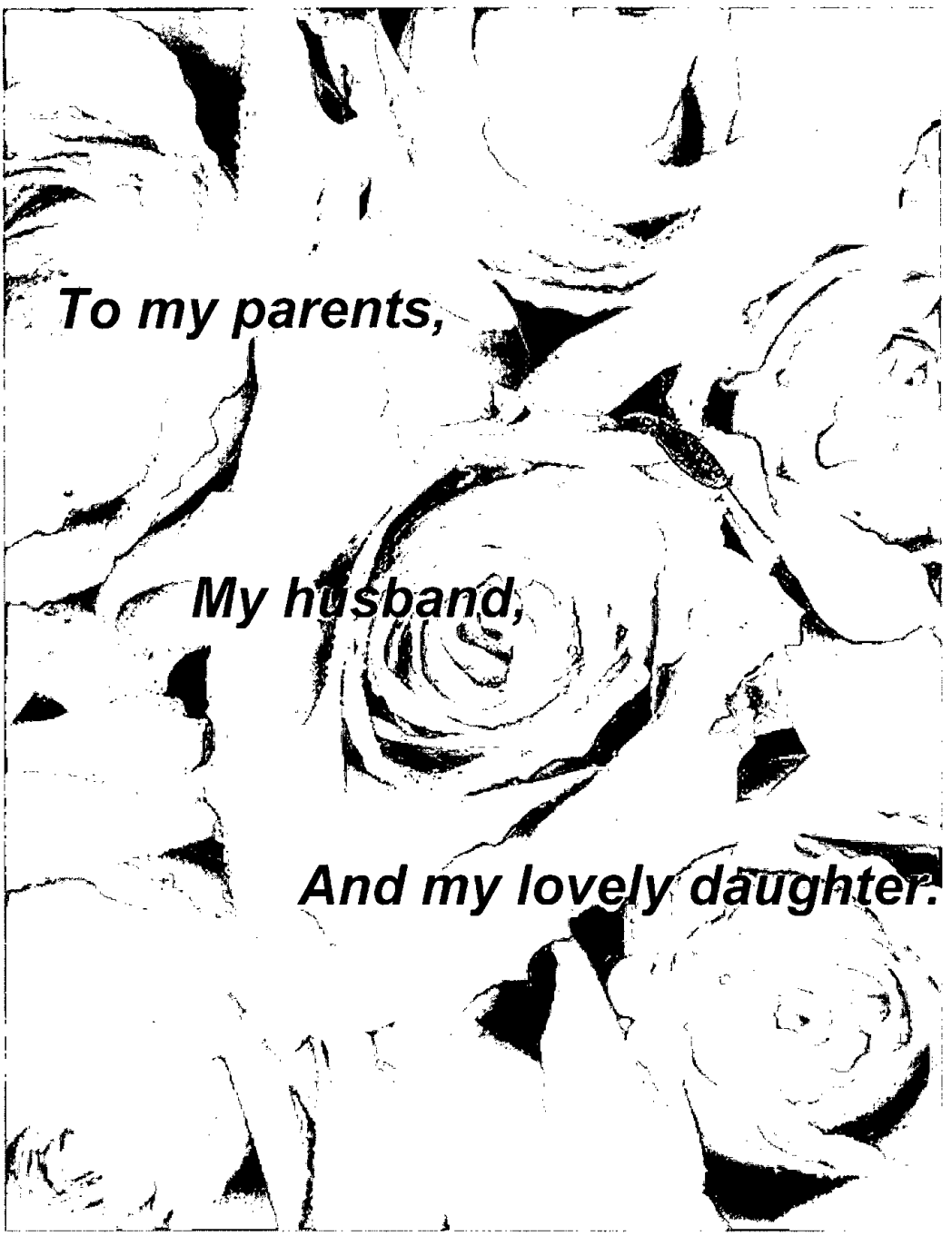
To my parents,
My husband,
And my lovely daughter.

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List of Abbreviations

Aa	Actinobacillus actinomycetemcomitans
AGEs	Advanced glycated end products
AL	Attachment level
APC	Antigen presenting cells
bFGF	basic fibroblast growth factor
Bf	Bacteroid forsythus
CD	Cluster of differentiation
Cr	Campylobacter rectus
Conc.	Concentration
DC	Dendritic cells
DM	Diabetes Mellitus
DM1	Diabetes Mellitus type 1(formely IDDM)
DM2	Diabetes Mellitus type 2 (formely NIDDM)
EBV	Epstein Barr Virus
ELISA	Enzyme linked immunosorbent assay
FPG	Fasting plasma glucose
GAD	Glutamic acid decarboxylase
GCF	Gingival crevicular fluid
GI	Gingival index
GM-CSF	Granulocyte Macrophage Colony Stimulating Factor
H2S	Hydrogen sulphide
HbAlc	Hemoglobin A1C
ICAM	Intercellular adhesion molecules
IDDM	Insulin dependant diabetes mellitus
Ig	Immunoglobulin
IL-1ra	Interleukin 1 receptor antagonist
IL-10	Interleukin 10

INF	Interferon
LDL	Low density lipoprotein
LPS	Lipopolysaccharides
MHC	Major histocompatibility
MMPs	Matrix metalloproteinases
NIDDM	Non insulin dependant diabetes mellitus
NK	Natural killer cells
NO	Nitrogen monoxide
O2	Oxygen
PCR	Polymerase chain reaction
PD	Pocket depth
PDGF	Platelet derived growth factor
Pg	Porphyromonas gingivalis
PG	Prostaglandin
PMN	Polymorphnuclear leucocytes
PPG	Post prandial plasma glucose level
RAGEs	Cell surface receptors for advanced glycated end products
SD	Standard deviation
TCR	T cell receptor
TGF	Transforming Growth factor
Th1	T helper 1
TIMPs	Tissue inhibitors of matrix metalloproteinase
TNF-α	Tumor necrosis factor alpha
TRG	Triglyceride
VCAM	Vascular cell adhesion molecule

INTRODUCTION

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Periodontal diseases are inflammatory disorders that give rise to tissue damage and loss, as a result of the complex interactions between pathogenic bacteria and the host's immune response **(Chapple, 1997).**

Chronic periodontitis is the most common form of periodontal diseases, occurring in human. It is initiated and sustained by bacterial plaque, but host defence mechanisms play an integral role in the pathogenesis **(Pershaw et al., 1999 and American Academy of periodontology, 2000).**

Bacterial components and products have been suggested as being responsible for the characteristic pathology of periodontal disease. These can be divided into those that adversely affect the host tissue directly by virtue of their antigenicity, enzyme liberation, end products of it's metabolism, and those that stimulate the release of inflammatory mediators from host cells resulting in auto-destructive process **(Wilson et al., 1996 and Kinane, 2001).**

Although periodontitis is mainly caused by bacteria, the progression of the disease is modulated by the components of the cellular and humoral immune systems, as well as, various soluble mediators produced by activated immunoregulatory cells **(Agarwal et al., 1995).**

Histologic observations revealed that the three primary leukocytes participating in the immune response involved in periodontal diseases are neutrophils, monocytes, tissue macrophages and lymphocytes **(Miyasaki, 1996)** these immunoinflammatory cells produce many different cytokines

which increase in GCF and gingival tissue of the inflamed sites **(Deschner et al., 1999).**

Studies have shown that pro and anti-inflammatory cytokines were expressed in diseased tissues taken from pockets of chronic periodontitis **(Jin et al., 2002).**

Lappin et al., 2001, found that in periodontitis' granulation tissue, the number of inflammatory leucocytes (Th2) that express the anti-inflammatory cytokines IL-10 are much more widely distributed than those (Th1) that express pro-inflammatory cytokines IL-6 and TNF- α.

Interleukin -10 is a polypeptide of 18 Kilo dalton (KD) expressed as a non - covalent homodimer . IL-10 was shown to be pleiotropic and to act on different cell types **(Moore et al., 1993)** Interleukin -10 is produced by variety of cell types including cells of monocytes /macrophage, Keratinocytes, T and B lymphocytes and mast cells **(Mosmann, 1994).**

The most prevalent systemic disease that modify the tissue reaction to local irritating factor in periodontal disease is diabetes mellitus **(John Brooke, 2001).**

Diabetes mellitus (DM) is a heterogenous group of disorders with the common characteristic of altered glucose tolerance associated with impaired lipid and carbohydrate metabolism. DM can be divided into 2 main types: type 1 (formerly IDDM), type 2 (formerly NIDDM) **(Mealey, 2000).**