The effect of antioxidant (vit) C on the total oxidant capacity in gingival crevicular fluid of smoker patients with chronic periodontitis after phase I periodontal therapy

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List of Content

List of Tables

List of Figures

List of Abbreviations

A group	Antioxidant group
A.a	Aggregatibacter actinomycetemcomitans
AA	Ascorbic acid
AA2P	Ascorbic acid 2-phosphate
AAP	American Academy of Periodontology
AO	Antioxidants
AP-1	Activating protien -1
ATP	Adenosine triphosphate
ATS	Arterial tortuosity syndrome
BOP	Bleeding on probing
CAL	Clinical attachment level
CAT	Catalase
CD	Cluster of designation
CpG	Cytosine-phosphate-Guanine
CRP	C reactive protein
CTRL	Control
Cu^{3}+	Cupper
DHA	Dehydroascorbic acid
DKG	Diketo-L-gulonic acid
DNA	Deoxy ribonucleic acid
ELISA	Enzyme linked immuno-sorbent assay
ENZ	Enzyme
EDTA	Ethylene Diamine Tetraacetic Acid
ER	Endoplasmic reticulum
Fe^{3}+	Ferric
FR	Free Radicals
F.N	Fusobacterium Nucleatum
GCF	Gingival crevicular fluid
GI	Gingival index
GLUT-10	Glutamine - 10
GM-CSF	Granulocyte Monocyte-colony Stimulating Factor
H_2O_2	Hydrogen peroxide
HGF	human gingival fibroblast
HIF-1	Hypoxia – inducible factor
HIV	Human immunodeficiency virus
IL	Interleukin

Ig	Imuunoglobulin
IFN	Interferon
LPS	Lipopolysaccharide
LTA	lipoteichoic acid
MMPs	Matrix metalloproteinases
µmol/L	Micro mol/Litre
µl	Microleter
NA group	Non Antioxidant
N- FMLP	N-Formylmethionine Leucyl – phenylalanine
NADPH	Nicotin amide adenine dinucleotide phosphate
NF-Ƙβ	Nuclear factor of Kappa Beta
NHANES	National Health and Nutrition Examination Survey
O2	Superoxide anion
OPG	Osteoprotegerin
OD	Optical density
PAMPs	Pathogene – associated molecular pattern
PCR	Polymerase chain reaction
PD	Probing Depth
P. gingivalis	Porphyromonas gingivalis
P.micra	Peptostreptococcus-micros
PGE_2	Prostaglandin E2
PI	Plaque index
P.I	Prevotella Intermedia
PMNL	Polymorphonuclear leukocyte
PRRs	Pattern recognition receptors
RDA	Recommended Dietary Allowance
RIA	Radio-Imuuno Assay
RNA	Ribonucleic Acid
RNS	Reactive Nitrous Species
RER	Rough Endoplasmic Reticulum
READBUF	Reagent buffer
RESCOL	Recontitution solution

ROS	Reactive oxygen species
SD	Standard deviation
SODs	Superoxide dismutases
TF	Tannerella forsythia
Th	T helper
TLR	Toll-like receptor
TMB	Tetramethyl Benzidine
TNF-α	Tumor necrosis factor-alpha
TOC	Total oxidant capacity
TOS	Total oxidative status
TAOC	Total Antioxidant capacity
8-OHdG	8-hydroxydeoxyguanosine

Introduction

Chronic periodontitis is an infectious disease associated with specific bacteria and characterized by inflammation of the supporting tissues of teeth and progressive destruction of the alveolar bone and connective tissues **(American academy of Periodontology, 2000).**

Although chronic periodontitis is initiated by the sub-gingival bacteria **(Madianos et al., 2005)**, the progression of periodontitis appears to be dependent upon an abnormal host response to those organisms **(Page and Kornman, 1997)**. The periodontitis phenotype is characterized by hyper-inflammatory immune response involving excess free radical release by neutrophilic polymorpho-nuclear leucocytes (PMNL) via the respiratory burst as part of the host response to infection **(Gustafsson and Asman, 1996).**

Evidence is emerging to implicate oxidative stress in the pathogenesis of periodontitis **(Chapple and Matthews, 2007)**.Oxidative stress is a state of altered physiological equilibrium within a cell or tissue/organ, defined as " a condition arising when there is a serious imbalance between the levels of free radicals in a cell and its antioxidant defense in favor of the former" **(Halliwell and gutteridge, 1989).**

Free radicals (FR) and reactive oxygen species (ROS) are essential to many normal biologic processes. Low levels of certain free radicals and ROS can stimulate the growth of fibroblasts and epithelial cells in culture, whereas higher levels may result in tissue injury **(Battino et al., 1999).**

Cigarette smoke destroys oral cavity homeostasis. It reduces salivary antioxidant status, initiates oral inflammatory diseases and promotes oral malignancies **(Greabu et al., 2008). Reznick et al., (2003)** have shown that cigarette smoke caused a significant decrease in the activity of numerous

salivary enzymes that are responsible for the protection against oxidative damage.

Antioxidants exist in all body fluids and tissues and protect the cell from harmful ROS by removing them or repairing the damage caused by ROS in vivo **(Halliwell, 2012)**. The scavenging or "chain breaking' ' antioxidants confer substantial protection on vital cell structures, because of their cellular and extracellular ubiquity and rapid rates of sacrificial oxidation **(Halliwell and gutteridge, 1990).**

Vitamin C is an important water-soluble vitamin that has antioxidant, anti-carcinogenic, and immune-modulatory actions **(Villacorta et al., 2007).**An epidemiological study had been done by **Amaliya et al., (2007)** indicated a negative association between plasma vitamin C level and severity of periodontitis**.**

Tomofuji et al., (2009) found that Vitamin C intake induced a 175% increase in plasma vitamin C level, resulting in a decrease in the gingival 8-hydroxydeoxyguanosine level and increase in the reduced oxidized glutathione ratio.

It seems that oxidative stress is a strong feature of periodontitis and the study of antioxidant defense systems therefore becomes important in elucidating mechanisms of tissue damage and potentially new therapeutic strategies **(Chapple et al., 2007)**

Review of Literature

Periodontal disease is a major public health issue because it is a source of social inequality. It reduces quality of life and chewing function and impairs aesthetics. Finally, it cause tooth loss and disability and responsible for a substantial proportion of edentulism and masticatory dysfunction. It has an impact on escalating dental costs and it is a chronic disease with possible impact on general health **(Baehni and Tonetti, 2010; Eke et al., 2012).**

There are two common diseases affecting the periodontium. The first is gingivitis, which is defined as inflammation of the gingiva in which the connective tissue attachment to the tooth remains at its original level. The disease is limited to the soft-tissue compartment of the gingival epithelium and connective tissue **(Beck and Arbes , 2006).**The second is periodontitis, which is an inflammation of the supporting tissues of the teeth with progressive attachment loss and bone destruction **(Flemmig, 1999).**

The etiology of periodontal diseases is plaque bacteria. The human oral cavity harbours a substantial and continuously evolving load of microbial species. The ecological interactions between the host and microbes determine the severity of the disease. Unlike many infectious diseases, periodontal diseases appear to be infections mediated by the overgrowth of commensal organisms, rather than by the acquisition of an exogenous pathogen **(Tonetti and Van Dyke, 2013).**

Specific Gram-negative, anaerobic, or microaerophilic bacteria were implicated in the causation of periodontitis**.** Subgingival microbial plaque adheres tightly to the tooth root surface and manifests all the characteristics expected of biofilms **(Slots, 1979). Nickel et al., (1986)** have defined biofilm as "matrix enclosed bacterial populations adherent to each other and/or to surfaces or interfaces" Microbial plaque is notoriously resistant to the normal host defence