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Review Article

FREE RADICALS AND ANTIOXIDANTS INTERACTIONS IN PERIODONTAL DISEASES- A REVIEW

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ABSTRACT

Periodontal disease is a multifactorial disease. Many mechanisms have been proposed for the pathogenesis of the disease. This article reviews the role of free radicals and reactive oxygen species in periodontal destruction. Also the significance of balance between free radicals and antioxidants is highlighted.

Keywords: Free Radicals, Reactive Oxygen Species, Antioxidants, Periodontal Disease

INTRODUCTION

Periodontal disease is an inflammatory lesion of the surrounding tissues of the teeth having a microbial etiology, leading to continuous loss of attachment and alveolar bone. Bacteria initiate the lesion directly through their toxins or indirectly by means of activating the host defence mechanism as in inflammation. Different inflammatory products are generated and among them are the free radicals (FR) and reactive oxygen species (ROS). These are mostly involved in the pathogenesis of different chronic degenerative diseases.

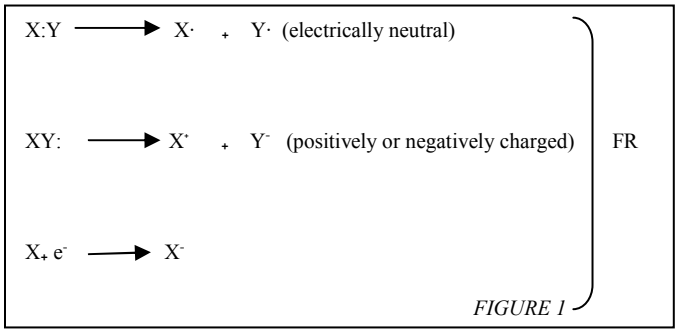

FIGURE 1

FREE RADICALS AND ROS

Free radical is an atomic or molecular species with one or more unpaired electrons in its structure. It can be positively or negatively charged or electrically neutral. These radicals are formed by either homolytic cleavage of a normal molecule or by loss or addition of single electron from a normal molecule (Fig 1). Heterolytic fission of a molecule will never result in free radical formation.

Reactive oxygen species are highly reactive compounds which include oxygen derived radicals and non radical oxygen derivatives (Table 1). ROS causes damage to the tissues through lipid peroxidation, DNA damage, and release of inflammatory mediators, protein damage and oxidation of certain enzymes[1].

Table 1: Types of Reactive Oxygen Species

Radicals	Non radicals
superoxide	Singlet oxygen
Hydroxyl	Ozone
Hydroperoxyl	Hypochlorous acid
Alkoxyl	Hydrogen peroxide
Aryloxyl	
Peroxyl	
Acyperoxyl	

FREE RADICALS, ROS AND PERIODONTAL DESTRUCTION

Free radicals and reactive oxygen species may be directly or indirectly involved in periodontal destruction (Fig 2). The periodontal pathogens mainly the Gram Negative bacteria initiate the inflammatory reaction causing the release of ROS from the neutrophils as a result of respiratory burst. These ROS may directly cause damage to the host tissue by

damaging DNA, proteins and lipid peroxidation intracellulary. Also ROS may directly cause damage extracellularly by destruction of host tissue proteoglycans, glycose aminoglycans and collagen.

Indirect damage of the host tissue by the ROS is mediated by enhancing the release of inflammatory cytokines like tumor necrosis factor, interleukins. There is also increased secretion of chemokines and cellular adhesion molecules. Increase in apoptosis is seen as a result of DNA damage[2].

Reactive oxygen species possess two main roles: the redox regulation of cell signalling/functions and the detrimental effect on certain substrates, the link between these two distinct functions is the body's antioxidant defence systems, which evolved to limit free radicals in biological systems. The human body possesses a plethora of antioxidants to defend against free radical activities and in normal physiology there is a dynamic equilibrium between the two, the so-called redox balance. It is only when ROS activity exceeds antioxidant defence capabilities or antioxidant defences are reduced that the balance shifts in favour of the ROS, resulting in oxidative stress and possible tissue damage[2]. (Fig 3)

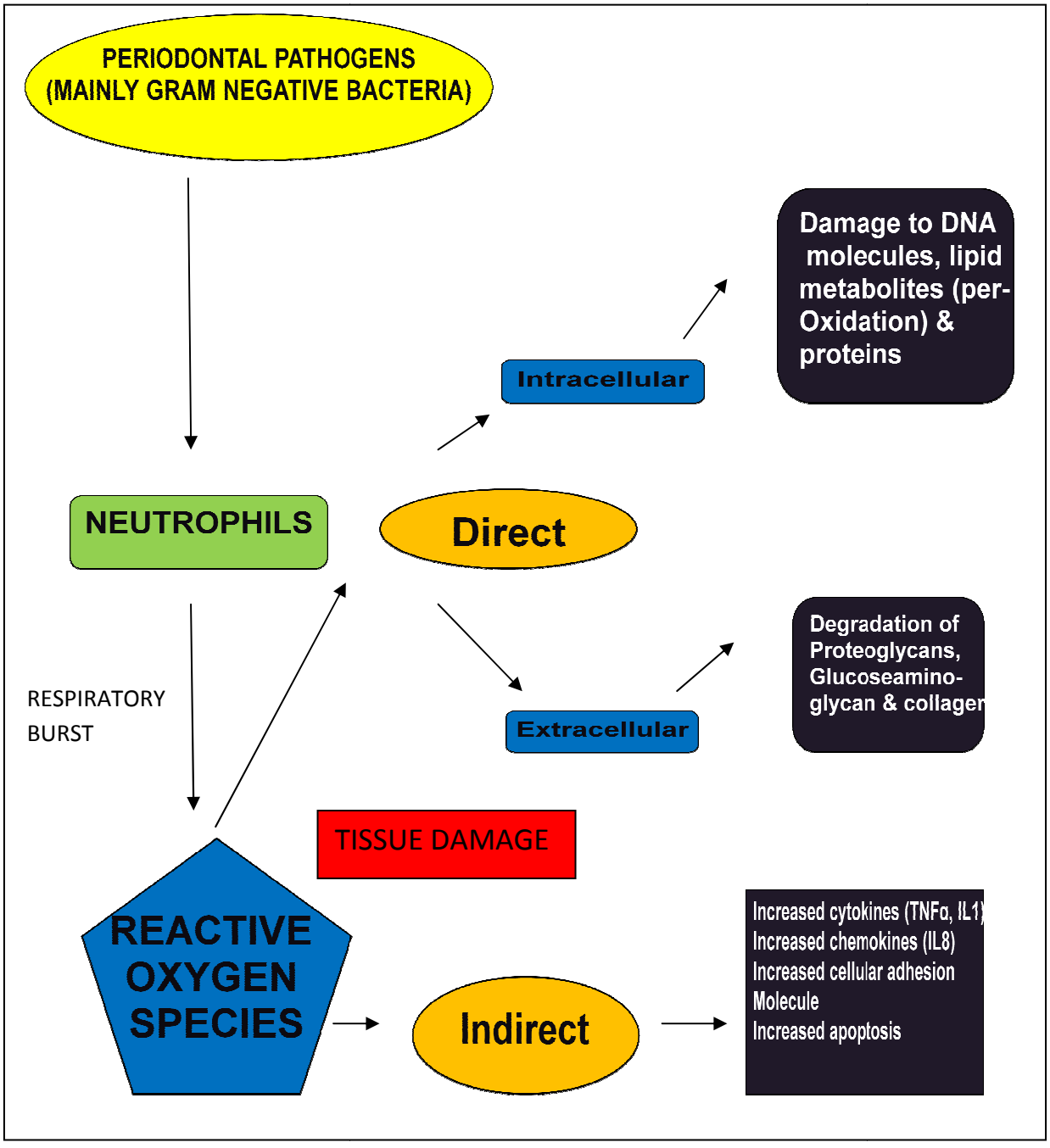

Figure 2: Ros Mediated Periodontal Destruction

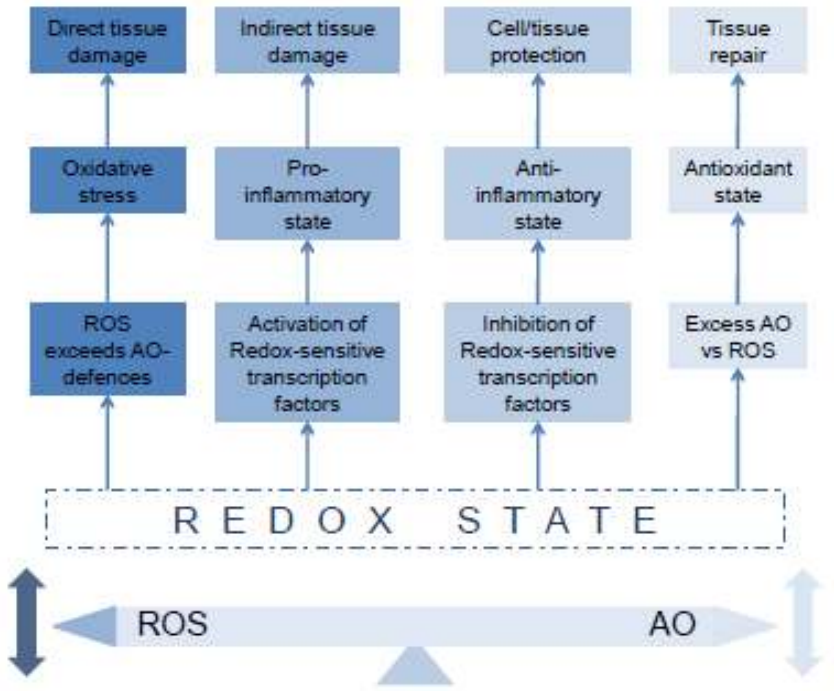

Figure 3: The biological effect of shifts in the balance of activity between reactive oxygen species (ROS) and antioxidant (AO) species.

TABLE 2 Classification of antioxidants on the basis of action[5]

Types of defense system	Mode of action	Examples
Preventive antioxidants	Suppress the formation of FR: A) Nonradical decomposition of LOOH and H2O2 B) Sequestration of metal by chelation C) Quenching of active O2	Catalase, GPX and serum-transfera Transferrin, ceruloplasmin, albumin, haptoglobin SOD, carotenoids
Radical scavenging antioxidants	Scavenge radicals to inhibit chain initiation and break chain propagation	Lipophilic: Ubiquinol, vitamin A, vitamin E, Carotenoids Hydrophilic: Uric acid, ascorbic acid, albumin, bilirubin
Repair and de novo enzymes	Repair the damage and reconstitute membranes	DNA repair enzymes, protease, transferase, lipase

TABLE 3 Antioxidants classified by structure they protect

Structure	Examples
DNA protective antioxidants	Superoxide dismutase 1 and 2, glutathione peroxidise, DNA repair enzymes e.g. poly(ADP-ribose) polymerase, reduced glutathione, cysteine Superoxide dismutase 1 and 2, glutathione peroxidise, DNA repair enzymes e.g. poly(ADP-ribose) polymerase, reduced glutathione, cysteine
Protein protective antioxidants	Sequestration of transition metals by preventative antioxidants Scavenging by competing substrates Antioxidant enzymes
Lipid protective antioxidants	α-tocopherol, ascorbate, carotenoids (including retinol), reduced ubiquinone, reduced glutathione, glutathione peroxidase, bilirubin

ATED PERIODONTAL DESTRUCTION

ANTIOXIDANTS

An antioxidant is any substance that when present at low concentrations compared to those of an oxidisable substrate significantly delays or prevents oxidation of that substrate[3,4]. Antioxidants may be classified in several ways: -

- according to their mode of function into either preventative or scavenging antioxidants (table 2)

- according to their location of action, intracellular, extracellular or membrane associated
- with regard to their solubility, lipid or water
- by their structural dependents
- by their source/origins, dietary or non-dietary

The preventative antioxidants function by enzymatic elimination of superoxide and hydrogen peroxide or by sequestration of metal ions, preventing Fenton reactions and subsequent hydroxyl radical formation[6]. The scavenging/chain breaking antioxidants are the most important in extracellular fluids, inhibiting chain initiating and chain propagating radicals as they form[7]. The lipid soluble antioxidants act at the cell membrane and protect against lipid peroxidation, while water-soluble antioxidants are more important within extracellular tissue fluids. Several antioxidants have dual or triple actions such as ascorbate (vitamin C), which acts as a chain breaking/scavenging antioxidant as well as a preventative antioxidant by its ability recycles α-tocopherol (vitamin E) from its oxidised form[8] and to bind metal ions, thus making classification to some extent limited due to the multilayered defence systems that exist[2].

The improved understanding of the pathogenesis of disease provides opportunities for the development of novel antioxidant therapies, which could function not only as antioxidants but also as anti-inflammatory agents. There are few studies that have estimated antioxidant capacity in serum/plasma of periodontits patients. Studies by Diab Ladki et al[9], Stangley and Langley Evans[10] showed a decrease in the total antioxidant capacity of saliva in periodontits patients. Similar results were given by Moore et al[11] and Tsai et al[12].

To combat disproportionate ROS production the body possesses a variety of antioxidant defence mechanisms, which act in concert. Their role is to protect vital cell and tissue structures and bio-molecules from host-derived ROS as well as those of parasitic origin[13], by removing them as they form and repairing the damage they cause. A delicate balance exists between antioxidant defence and repair systems and pro-oxidant mechanisms of tissue destruction, and if the balance is tipped in favour of ROS activity, significant tissue damage ensues[14]

CONCLUSION

FR and ROS involvement in periodontal destruction enforces the role of antioxidants in preventive therapy. Further studies are needed to completely understand the role of free radical in periodontal disease pathogenesis

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