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بالرسالة صفحات
لم ترد بالأصل

Assessment of Intercellular Adhesion Molecule-1 in Smokers with Periodontal Disease

Thesis

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By

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Introduction

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Tobacco smoking has been identified as a potential risk factor for periodontal disease ***(Bergstrom & Preber, 1994).*** A relationship has been demonstrated between smoking exposure and the prevalence and the severity of periodontal disease ***(The American Academy of Periodontology 1996).*** An association has also been found between the prevalence of moderate to severe periodontal disease and the number of cigarettes smoked per day and to the number of years that the patient has smoked ***(Grossi et al., 1995).***

Since there seems to be a strong relationship between smoking and periodontal disease, smokers are expected to have increased number of deep pockets, increased calculus formation, greater bone loss ***(Haber et al., 1993, Grossi et al., 1995).*** Moreover, smokers exhibited significantly advanced gingival recession, clinical attachment loss, furcation involvement, and tooth mobility compared to non smokers ***(Kerdvongbundit & Wikesjo, 2000).***

Tobacco smoking has also been found to affect the normal functions of the host response particularly neutrophil functions. It has been shown to adversely affect neutrophil chemotaxis and phagocytosis ***(Barbour et al., 1997)*** as well as immunoglobulin production ***(Ah et al., 1994).*** In fact, many tobacco components may influence the host immune response by altering leucocyte function ***(Numabe et al., 1998, Bernzweig***

et al., 1998). Nicotine at low concentrations can stimulate neutrophil chemotaxis but at higher concentrations will impair phagocytosis ***(Totti et al., 1994, Ryder 1994).*** Smokers also appear to have depressed numbers of helper lymphocytes which are important components of the immune system ***(Costabel et al., 1986).***

It has been suggested that nicotine may also cause vasoconstriction in the gingival blood vessels impairing wound healing ***(Goultschin et al., 1990).*** Nicotine can impair the attachment of fibroblasts to root surfaces in vitro ***(Raulin et al., 1988)*** and may affect collagen synthesis and protein secretion thus interfering with the host's natural repair mechanisms ***(Chamson et al., 1982).*** It has been also shown that low dosages of nicotine can be stored in and released from periodontal fibroblasts ***(Hanes et al., 1991).*** Nicotine can also suppress the proliferation of cultured osteoblasts while stimulating osteoblast alkaline phosphatase activity ***(Fang et al., 1991).*** Moreover, it has been found to influence cytokine and prostaglandin release from monocytes ***(Payne et al., 1996).***

Adhesion molecules mediate cell–cell and cell–matrix communication, providing specific mechanisms which control the functional activities of all cells. Such interactions are of particular importance in the generation of immune responses, in which the modulation of adhesion molecule expression patterns may define both the nature of the infiltrating cells as well as their activity and the relationships between different cell populations ***(Imhof & Dunon 1995).*** Among the molecules that mediate adhesive interactions, is the intercellular adhesion

molecule–1 (ICAM–1) or CD54. ICAM–1 belongs to the immunoglobulin supergene family of recognition molecules and is composed of five immunoglobulin–like extracellular domains, a hydrophobic transmembrane domain, and a short cytoplasmic domain ***(Marlin et al., 1990).*** ICAM–1 is an 80–105 kDa transmembrane glycoprotein expressed by several leucocytic cell populations ***(Stople & Saag, 1996).***

ICAM–1 is widely expressed on cells of haemopoietic and non–haemopoeitic origin including lymphocytes, monocytes, fibroblasts, epithelial cells and endothelial cells. Its expression in vivo is low in normal tissues but high in inflamed tissues ***(Dustin et al., 1986).*** This expression can be dramatically increased by treatment with inflammatory mediators, interferon–γ, interleukin–1 (IL–1), and tumor necrosis factor–α ***(Pober et al., 1986).*** ICAM–1 is a ligand for at least three leucocyte cell surface molecules, LFA–1, MAC–1 and CD43 ***(Kishimoto et al., 1989b).***

In chronic adult periodontitis, ICAM–1 has been reported to be upregulated on endothelial cells, some keratinocytes and infiltrating lymphocytes at lesional sites ***(Takeuchi et al., 1995, Gemmell et al., 1995).*** Interactions between ICAM–1 and LFA–1 participate in adhesion and migration of leucocytes through endothelial cells in vitro ***(Warwyk et al., 1989).*** ICAM–1 is also expressed by junctional and pocket epithelium, which suggests that ICAM–1 and its ligands are not only involved in the migration of leucocytes through gingival endothelium, but also through junctional/pocket epithelium into the gingival crevice/periodontal pocket resulting in local defence of the periodontal tissues. ***(Crawford, 1992).***

A soluble form (sICAM–1) of the membrane bound ICAM–1 was identified in the sera of normal individuals ranging from 100–200 ng/ml ***(Rothlein et al., 1991).*** Soluble forms of ICAM–1 have been detected in biologic fluids and thought to result from proteolytic cleavage of the membrane–bound molecule. It could also be the result of an alternatively spliced messenger RNA with an effective deletion of the transmembrane and the cytoplasmic domains ***(Marlin et al., 1990, Rothlein et al., 1991).*** The soluble forms may be also shed from activated cells ***(Newman et al., 1993)*** and may have a physiological role in leucocyte function.

Rothlein et al.,1991 reported that the source of sICAM–1 is from mononuclear cells, while others thought that it might be the consequence of inflammation, tissue damage and non-specific proteolysis ***(Ballantyne et al., 1991).*** It has been shown that sICAM–1 retains its biological functions and is capable of binding to the ligand LFA–1 ***(Rothlein et al., 1991),*** thus sICAM–1 may compete with the membrane–bound ICAM–1 for its ligands found on leucocytes, thereby preventing their attachment and subsequent migration into the tissues ***(Boldt et al., 1995).***

Since the membrane bound form of ICAM–1 is higher in non smokers compared to smokers and the sICAM–1 exhibits biological activity and may represent "shedding" of the membrane bound form of ICAM–1, it is essential to estimate the level of sICAM–1 in smokers.

Review of Literature

Review of literature

Tobacco smoking is the most dangerous of all psychoactive drugs because of its deadly effects on the cardiovascular, respiratory and other body systems. Cigarette smoking is the most important environmental factor contributing to premature mortality. Although the health risk associated with smoking has been widely stressed, cigarette smoking still possess one of the greatest health problems.

Attention is being paid to the effect of passive smoking on health, as it has been associated with a variety of harmful effects. In recent years, many clinical and epidemiological studies have indicated that cigarette smoke significantly increases the risk factor for periodontal disease ***(Axelsson et al., 1998).***

Composition of cigarette smoke and its effect on general health

Tobacco leaves contain a complex mixture of several hundreds of chemical compounds. About 4,000 compounds are generated by burning of tobacco. The smoke can be separated into gaseous phase (Table I) and particulate phase (Table II) ***(Carter and Hasegwa, 1975).*** Among the gaseous phase components are carbon dioxide, carbon monoxide, ammonia, acrolein, acetone and benzene and among the particulate phase components are nicotine, phenol, cabozole and indole.

From the medical and physiological point of view, the constituents of tobacco smoke may be divided into 3 groups; gases, nicotine and carcinogens.