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Influence of genetics on dental caries

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Abstract

Dental caries is a multifactorial disease due to interaction of several factor (bacteria, host, food debris, time), There is also many evidences for a genetic contribution in the etiology of the disease. This is clear in many dietary studies as certain environmental factors are potentially more cariogenic for some people than others.

Keywords: *Dental caries, gene, genetic, Syndromes, Heredity disease.*

1. Introduction

Dental caries is the medical term for tooth decay or cavities. Tooth decay is one of the most common of all disorders of teeth. It is not occur if the oral cavity is free of bacteria. Bacteria, those are normally present in the mouth, convert all food especially sugar and starch into acids. Bacteria, acid, food debris and saliva combine in the mouth to form a sticky substance called plaque that adheres to the teeth. Teeth decay begins if this plaque is not removed thoroughly and routinely. Plaque forming bacteria converts sugar and carbohydrates in food into acids, these acids dissolve minerals on the surface of the tooth (Hassell et al, 2010).

Multiple factors contribute to a persons risk for caries include (Townsend et al, 2012):-

- Environmental factors (Diet, oral hygiene, fluorid, exposure and the cariogenic bacteria).
- Host factors (Salivary flow, salivary buffering capacity, position of teeth relative to each other, composition of tooth enamel and host immune response).

There are individuals who still appear to be more susceptible to caries and those who are extremely resistant to dental caries regardless of the environmental risk factors to which they exposed. Similar to periodontitis and cleft palat and lip, dental caries is also said to be a multi-factorial (complex) disease, though dental caries are seemingly caused by interaction between environmental and genetic factors, yet the disease is to great extent influenced by environmental factors.

Genetic of dental caries

It is clear from many dietary studies (Dahlberg, 2011 and Mansbridge et al, 2010) that variation in susceptibility to dental caries exists even under identical, controlled conditions. This implies that, because of genetic difference, certain environmental factors are potential more cariogenic for some people than for others. This is not to say that dental caries is an inherited disease, rather, genetic influences may modify the overt expression of this disease in the individual.

Several investigators (Caufield PW et al, 2012 and Li, and Caufield PW, 2014) are indicated that children have caries experience similar to that of their parents when the susceptibility of both parents is the same (either high or low), but when caries

susceptibility of the two parents is dissimilar, however, the childrens susceptibility tends to be more like that of the mother than that of the father.

Li and Caulfield (2014) found that mothers are the principle source of mutans streptococci to their infants with a greater rate of transmission to female than male infants. However, most authors agree that this genetic component of dental caries is a minor one in comparison with the overall effect of environmental.

The conclusion from twins and family studies may be confounded by the transmission of cariogenic bacteria within the family, and the susceptibility to dental caries influenced to a significant but minor degree by heredity. All the similarity in twins would expectedly be due to common genes and all dissimilarity will be due to environmental factors. Environmental factor clearly have a greater influence but that genetic factor also contribute to the dental carries. This may be explained by the existence of genetic variability among individuals (Shuler, 2001).

The genetic influences modify the expression of disease in the individual. Different individuals respond in indifferent way to specific biochemical difference in oral environment depending on their genetic constitutions. For example there is significant difference between monozygotic and dizygotic twins in terms of salivary flow, ph, and salivary amylase activity when compared between two groups. On the contrary, both the monozygotic twins of pair will respond similarly (because of same genetic constitution) to these factors whereas such similarity may be lacking between the two individuals of a dizygotic pair (because of difference in their genetic constitutions) (Nariyama et al, 2004).

The morphology of teeth related to their shapes, sizes, pit and fissure morphology, enamel structure and composition, arch forms, dental spacing and order of the teeth are some of important factors that regulate the washing effects of saliva and thereby may influence the production of caries, these factors are in fact largely determined by heredity factors. Many genes are known which are active in the formation of enamel which Amelogenin X- linked gene (AMELX), Enamelin gene (ENAM), are: kallikrein 4 gene (KLK-4) and Matrix metalloproteinase 20 gene (MMP20) which encode various proteins like (ameloblastine, amelogenin, enamelin, tuftelin-1 and tuftelin interacting

protein 11), certain variations in some of these enamel matrix genes may be associated with enhanced caries susceptibility (Klineberg et al, 2007; K. Deeley et al, 2008).

Low caries susceptibility genes were found on chromosome number 5,14 and x. The high caries susceptible genes were identified in chromosome number 13 and 14 (Vieira et al, 2008). The presence of gene for caries on X-chromosome may account for the gender difference observed in the incidence of caries (Lukacs and Largaespada,2006). There is no significant association concluded between single candidate genes and caries susceptibility. A significant interaction between tuftelin and S.mutans was however observed (Slayton et al, 2005).

Ihe Osteopontin (OPN) gene found on chromosome number 4 and plays an important in mineralization, (OPN) gene was found to be associated with incidences of enamel hypoplasia in primary dentition, So there is association between the OPN gene and caries in the primary dentition (Pal GP and Mahato NK, 2010).

2. Descution :

Heredity diseases or syndromes associated with dental caries

Dental caries is either syndromic syndromic or non Syndromic

A.Syndromic dental caries:- means it is associated with syndrome or disease.

1. Celiac disease:- An autoimmune disorder of the small intestine that occurs in genetically predisposed people of all ages. (Symptoms include chronic diarrhea, weight loss and fatigue). The vast majority of celiac patients have one of the two types of human leukocytes associated antigens (HLA). Celiac disease patient exhibit an increased incidence of dental caries, this may be due to the fact that these patients have defective enamel that predisposes the tooth to dental caries this patient show significant positive correlation between their HLA type and presence of enamel defect. The association between HLA complex and caries has indicated that few genes in HLA complex are responsible for dental caries resulting from altered enamel development and also due to low dose response to cariogenic bacteria (Ravald et al, 1998; Ortega et al, 2008).

2. Epidermolysis Bullosa (EB):- A group of inherited disorders presenting with skin blisters that develop in response to minor injuries such as rubbing or scratching, there are three main types (Simplex EB, Junctional EB and Dystrophic EB) and various subtypes of Epidermolysis. Blisters may also occur in the oral mucosa. Junctional EB are always associated with generalized enamel hypoplasia, this is due to altered relationship ameloblasts cell and extracellular matrix of the developing enamel leading to primary defect in the enamel hard tissue, also enamel will suffers from greater porosity and thus it is imparted with an increased surface area for acids to work upon. EB has been shown to be involved with an alteration in the enamel and as well as with an increased incidence of caries. DMFS (decayed, missing, filled surface) scores always high in junctional EB and Dystrophic EB (Kirkham et al, 2000; Feijoo et al, 2011).

3. Siogren's syndrome:- An autoimmune disorder in which abnormally activated immune cells attack and destroy exocrine glands that produce saliva. The primary and secondary variants of these syndromes are found to be associated with increased caries risk this due to decreased flow rate of saliva (McCullough and Porter, 2002; Klineberg et al, 2007).

4. 22q11deletion syndrome (22g11 DS) or DiGeorge syndrome (DGS):- Caused by genetic deletions (loss of a small part of the genetic material) arm of one of the two 22nd chromosomes. Higher caries found on the long prevalence has been reported for individuals suffering from deletion at 22q11. It was observed that the patient with this syndrome had congenital heart malformations and immunological problems, impaired salivary secretion rates, higher numbers of cariogenic bacteria, increased salivary protein concentrations and reduced output of electrolytes in the saliva compared to the controls. This indicated that the salivary function is affected in 22q11DS explaining increased caries risk in these subjects (Wright et al, 1994).

5. Turner's syndrome:- A chromosomal abnormality with additional deficiency in gonadal and adrenocorticotropic secretions. The most frequent oral findings are high palatal vault, hypoplastic mandible and lower DMF-T mean values were found in patients with Turner's syndrome compared with the healthy controls (Pal GP and Mahato NK, 2010).

6. Cleft lip and palate:- Birth abnormalities of the lip and mouth. The lip and palate begin forming during just the first few weeks of gestation, often before a woman even knows she is pregnant. During this time, the sides of the mouth begin developing and eventually unite evenly in most infants. In rare cases, however, the sides of the mouth do not fuse together properly, creating a notch or cleft. There is high incidence of dental caries because it is associated with night-and nap-time nursing with bottle containing milk or juice or other fermentable liquid (Ravald et al, 1998).

B. Nonsyndromic dental caries: means dental caries is isolated without association with syndrome or disease.

Amelogenesis Imperfecta:- Genetic disease caused by mutation in gene which encode for enamel matrix proteins. Mutations in any of these genes alter the final structure of these proteins or completely prevent synthesis of any protein at all. As a result tooth enamel is abnormally thin or soft and has high a risk of developing dental caries (Pal GP and Mahato NK, 2009).

Dentinogenesis Imperfecta: Genetic disease, which leads in the formation defective dentine. The mineral content is less than normal dentine and contain more water (hence soft), which are weak and likely to decay and break (Zhang et al, 2001).

Association between human leukocytes associated antigens (HLA antigens) and susceptibility to dental caries

The plasma membrane surfaces of almost all the body cells (except RBCs) present "self antigens". Called the major histocompaatibility complex (MHC) antigens. These are also called human leukocyte associated (HLA) antigens because they were first detected on the white blood cells. These self antigens integrated membrane glycoproteins. The chance of rejection of graft are less if a large number of MHC antigens are similar in the donor and recipient. MHC (HLA) antigens help t-cell of the immune system to mount an immune response, there are three classes of HLA antigens (Class I, II, III). Current evidences support the relationship between immune complex genes (HLA) and dental caries and the association of different levels of cariogenic bacteria and the enamel defects. Many studies are now available which show the association between increased

risk for caries and immune complex (HLA) genes (Aguirre et al, 1997; Kirkham et al, 2000).

3. Conclusion

Dental caries is multifactorial disease due to interaction of several factor (bacteria, host, food debris, time), or a result of instruction of gene and environmental factors like (periodontitis and malocclusion). Low caries susceptibility gene were found on chromosome number 5, 14 and X, while high caries susceptibility genes were identified in chromosome number 13 and 14. The presence of gene for caries on X-chromosome may account for the sex difference observed in the incidence of caries. The susceptibility to dental caries depends to some degree upon the genetic background of person.

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