Genetic Factors and Periodontal Disease

In the previous newsletter the genetic factor interleukin - 1 (IL -1) was mentioned as a causative basis for increased susceptibility to periodontal disease. Many studies have been carried out in the last 5-7 years implicating interleukin, however the exact nature and level of contribution has not been quantified. Other possible genetic factors have been studied recently and their influence upon clinical manifestations of periodontal disease will be discussed.

The scientific literature has witnessed an exponential increase in the amount of information linking genetic factors with a variety of medical diseases. This new era of information has been ushered in by the knowledge gained from human genome studies within the past ten years. Fundamental understanding of the structure and function of genes is providing a basis for the development of diagnostic and susceptibility testing for the presence of disease associated genes and eventually for better treatment options. There is now a significant amount of evidence that genetic factors are important determinants of periodontitis susceptibility and progression. Most human diseases have a genetic component. The extent of this genetic contribution varies greatly for different diseases.

Generally, genetic diseases are divided into two broad groups, "simple" and "complex". An example of a "simple" genetic disease is Amelogenesis imperfecta. All diseases in this category are characterized by having a diagnostic test available to identify individuals with the mutation for the disease, what level of probability the genetic mutation will be passed on to a child and to predict the clinical course of the disease. For "complex" diseases there is not a typical pattern of familial distribution or transmission. There are multiple genes that are involved and environmental factors are important in the disease process. Unlike "simple" genetic traits, which are often due to rare mutations at a single gene locus, the genetic variants (gene polymorphisms) for "complex" diseases are common in the population and can occur in unaffected as well as affected individuals. For this reason research is being focused on the clinical validity and clinical utility of genetic polymorphisms that have been reported to be associated with a disease.
In trying to estimate genetic influences as they relate to periodontitis studies have been done to help quantify the genetic component versus environmental (smoking, plaque, etc.). Studies on twins have been useful to help distinguish between environmental and genetic. Several studies have concluded that periodontitis is approximately 50% inheritable, however the exact nature of and what genetic factors are involved is the subject of ongoing research. In determining what are the actual genetic markers the IL - 1 (Interleukin - 1) factor has been studied extensively. Attempting to connect this factor with clinically measurable disease has been inconsistent. Whereas it has been found to be clinically predictive of disease in some populations in others it has not been positively correlated with disease. Other genetic markers have been studies such as Tumor necrosis factor (TNF), Interleukin - 10 genes, Fc -gamma receptor - all have been found to have some association with periodontitis. The problem is that none of these factors have been consistently linked with periodontal disease.

Overall reports on the genetic polymorphisms associated with periodontal disease are increasing, but the limitations of such studies have prevented their ability to be incorporated into meaningful clinical significance. In medicine, Alzheimer's disease has been studied extensively and has been shown to be associated with ten genetic polymorphisms of inflammation related molecules such as cytokines and protease inhibitors. It appears possible that a similar situation exists with periodontitis. In other words, there are probably many genetic factors that together are associated with periodontal disease. If this is the case then extensive future studies will have to be done to determine which factors are relevant and to what extent each influences clinical disease. If proven, this information would be very important for predictive value and have significant influence on therapeutic strategies.

Next Issue: Simple Muco-Gingival Problems

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