Introduction

There are three types of epigenetic modifications seen as DNA methylation, histone modification and RNA-associated silencing (micro-RNA) [1,2]. It was found that with methylation, demethylation and acetylation, deacetylation or combination of these worked to either express or repress genes during replication. DNA methylation is one of the most broadly studied and well characterised epigenetic modification dating back to studies done by Griffith and Mahler in 1969 which suggested that DNA methylation may be important in long term memory function [3]. These processes triggered by environmental influences regulated cytokines such as IL1 and IL6 which were responsible for inflammatory tissue destruction. Susceptibility of individual to periodontitis depends on his immune response. Decrease in number of neutrophils also increases the susceptibility of infections.

Cytokines noted as inflammatory response influence the host response in periodontitis [4]. Cytokines are reported as powerful regulatory proteins released by the immune cells influence the behaviour of other cells [5]. SNPs of interleukin (IL-) 1a, IL-1B, IL-4, IL-6, IL-8, IL-18 located in different regions of the cytokine have been shown to affect the risk of the disease in several populations [6]. A study was carried on and seen the polymorphism in IL 1 due to major part that IL 1B played in the development of periodontitis. Studies suggested that individuals who had higher level of IL1 have a greater chance of periodontitis then those having less level [7].
can be seen in deficiency of folic acid and deficiencies of selenium, arsenic and polyphenols. These deficiencies can cause diseases like neural tube defect and malignancies. Wilson reported on epigenetic factor aging methylation and acetylation process were carried out in mono zygotic twins and very similar results were seen in early life and substantial differences in later life. Several Environmental influences contribute to the progression of periodontitis. Alterations in DNA methylation status as a result of environmental stressors have been reported to begin before birth though some epigenetic marks are potentially reversible, many epigenetic changes appear to persist throughout the cell lineage and life of the organism several factors responsible for epigenetic modification are race, gender, diabetes, BMI etc. One of the important environmental risk factors for progression of periodontitis is smoking. Smoking causes long term hypo and hypermethylation changes in the DNA. Haffajee and Socransky in a study found that smokers had more severe form of periodontitis with more attachment loss and deeper pockets compared with non-smokers.

Epigenetics and Susceptibility of Disease
The person is susceptible to disease due to the process of hyper/hypo methylation, histone acetylation or combination of any of these. Disease is generally inflammatory in nature like rheumatoid, arthritis, periodontitis. In chronic periodontitis hypermethylation of gene E cardherin and cyclo oxygenase 2 takes place which increases the susceptibility of individual to periodontitis. In a study from the journal of translational medicine few DNA were extracted from the gingival of 108 systematically healthy non periodontitis patient, blood samples from 110 periodontitis patients and neoplastic tissues from 106 breast cancer patient and then all are tested for the methylation of E-cardherin and cyclo-oxygenase 2. In periodontitis patients detection rate of hypermethylation of E cadherin and cyclooxygenase was 25 and 19% and nothing was found in systematically healthy non periodontitis patient [10]. These findings were obvious enough to establish the relation between hypermethylation and periodontitis.

Conclusion
Susceptibility of individuals to periodontitis is increased when there is increased level of IL1 and IL1 gene polymorphism. Epigenetics and environmental factors changed the expression of DNA transcription through these factors (diet, diabetes, aging, gender, BMI, race, smoking) an individual’s predisposition for inflammatory response is determined.

References
5 Nield-Gehrig JS, Willmann DE (2011) Foundation of periodontics for the dental hygienist (3rd edn.).