

TLR4 Polymorphism and Periodontitis **Alina Smalinskiene***

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Periodontitis is a kind of chronic disease affected by multiple factors such as microorganism, host and environment. Periodontitis was identified into 3 types: chronic periodontitis (CP), aggressive periodontitis (AP), and periodontitis as a manifestation of systemic disease. Kassebaum et al predicted an increasing global burden of severe periodontitis on account of growing life expectancy resulted in decreased tooth loss and increasing world population during 1990 to 2010. Susan et al summarized the features of epidemiology and demographics in AP which clarified the prevalence of AP varied significantly in different regions and races. Thus, periodontitis has become one of the hot research fields all over the world.

The traditional method for dealing with periodontitis mainly focused on removing pathogenic bacteria, which resulted in bacteria resistance and disease recurrence. Besides, the host inflammatory response plays a critical role in the destruction of periodontal tissue. In the recent decades, the development of sequencing technology enabled us to discuss whether the variations of host's immune-related Deoxyribose Nucleic Acid molecules affected the occurrence and development of diseases. Thus, there is a great significance to discuss the gene variants of immune-related molecules for the prevention and treatment of periodontitis. Luigi elucidated host genetic variants may work in the occurrence and development of AP through selectively participating in the symbiotic process. Hajishengallis and Sahingur reported a polymorphic site in the TLR9 gene promoter region differentially expressed and TLR9 gene and protein expression increased in CP. Toll-like receptor 4 (TLR4) was a pattern-recognition receptor, which played an important part in innate immunity by realizing lipid-based structures of bacteria and mediating intracellular signaling [7,8]. Furthermore, Many studies reported the association between TLR4 polymorphism and periodontitis susceptibility, and they mainly focused on TLR4-299A>G or TLR4-399C>T of Caucasian but conducted different conclusions. Therefore, this meta-analysis and subgroup analyses were carried out to further illuminate the relationship between TLR4 polymorphism and periodontitis susceptibility based on the

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currently available studies. Bacterial deposits, smoking, and host genetic factors play a major role in an individual's predisposition to periodontitis. Bacterial components are recognized by CD14 and toll-like receptor 4 (TLR4), resulting in a NF-kappa-based inflammatory response. We hypothesized that functional CD14 and TLR4 polymorphisms contribute to periodontitis susceptibility. We aimed to investigate the occurrence of CD14-260C>T, TLR4 299Asp>Gly, and 399Thr>Ile gene polymorphisms in adult periodontitis. DNA was collected from 100 patients with severe periodontitis and from 99 periodontal healthy controls. The gene polymorphisms were determined by the PCR technique. The presence of the periodontal pathogens *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans*, and whether the subjects smoked, was included in the analyses. The CD14-260T/T genotype was found in 34.0% of periodontitis patients and in 20.2% of controls. Logistic regression analysis adjusted for gender, age, smoking, and prevalence of *P. gingivalis* and *A. actinomycetemcomitans* showed an association between the CD14-260T/T genotype and periodontitis ($P = 0.004$, OR 3.0, 95% CI 1.4-6.9). We conclude that the CD14-260T/T genotype contributes to the susceptibility to severe periodontitis in Dutch Caucasians.