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Short Commentary

Omic Paradigms Enhance Interface between Periodontitis Pathogenesis and Metabolic Disorder

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Short Commentary

In spite of public health surveillance and its regulations, there are more disciplines needed those approach health related challenges that pose the greatest concern to the society's wellbeing in terms of disease development, progression monitoring, and protection. Consider the fields of environmental and occupational health where the natural verses built environment and the workplace settings can actually present factors those are deleterious to one's health due to exposure. Environmental health is mainly concerned with oxidative stress by which the environment influences human health and/or metabolic diseases [1-5]. The natural environment is inclusive of air, water, food, and soil, as well as all the physical, chemical, biological, psychological, and social features of our surroundings. The physical structures of our surroundings where people live and work and the community systems such as air contamination in roads or other means of transport, overspill of insecticide on land use along with waste chemical spills are considered aspects of the built-in macro-environment of metabolic disorders. There are also elements of the social environment (or micro-environment) such as lifestyle factors and socioeconomic status which can have an indirect effect on a person's health and their lifestyle. All of these macro- or micro-environmental components could interact in unique ways with the quality of life and can inevitably end up compromising one's health status.

The World Health Organization (2016) also indicates that environmental health "addresses all the physical, chemical, and biological factors external to a person and all the related factors impacting behaviors". Current challenges of environmental health are within the realm of many on going public health issues that include polluted air and water, poor waste management systems, as well as hazardous materials/toxic substances management, and food safety. Unfortunately the risks have potential to lead to more serious effects such as varying types of chronic diseases due to oxidative stress mediated metabolic disorder. Herein, dental health lays yet another public health burden. Biological and chemical toxins, contaminants, food ingredients, and even physical hazards within the environment driven stressors, include natural or occupational stressor those contributes towards the potential health risks. Inclusive of these diseases are some non-communicable health conditions such as the heart and respiratory diseases, cancer, obesity, and diabetes to name a few. In order to ensure the health of the population the only resolution for public health practitioners is to ascertain ways to prevent through early detection or onset of diseases. For these reasons, there has long been the use of molecule based detection methodology and imaging techniques used to evaluate, monitor, and assess risk of chronic periodontal disease in dental health as well as metabolic disorders.

Periodontal disease is a chronic inflammation of the peridontium involving the gingival tissue, the supporting collagen fibers and the compact alveolar bone of the mandible and the maxilla. An estimated 64.7 million or 47.2 percent American suffer from advanced form of periodontal disease (CDC, 2009). The etiology of periodontal disease is a collective interaction of the disease causing bacterial plaque with the predisposing gene factors of the host resulting in a destructive response of the supporting structures such as the alveolar bone and the collagen fibers. This can lead to poor prognosis of tooth loss. Periodontal disease can be acute or chronic mild, moderate or severe with bones loss which can begin at any age being juvenile or adult.
Periodontal disease is as prevalent in our nation as cardiovascular disease affecting half the population in the U.S. There have been numerous researches on the established association between periodontal disease and the increased risk of systemic conditions such as neutrophil dysfunction, atherosclerosis and cardiovascular disease in children and young adults [6-8]. Type II diabetes and preterm weight births are also linked with the chronic inflammation associated with periodontal disease [9,10].

Periodontal disease so far has been managed through control of the etiological factors such as dental treatment methods those minimize the bacterial colonies and the toxins those are produced and initiate the immune response of the host. This immune response results in self destruction of the supporting periodontium. Therefore, there is a need for advanced detection of risk assessment of the molecular basis of the inflammatory process through epigenetics intervention. Study of the alteration of histones, DNA methylation and micro-RNA role in translational repression can help develop medication such as deacetylase inhibitors and demethylation agents. In the previous studies, different type of microRNAs (i.e., let-7a, let-7c, miR-130a, miR301a, miR-520d, miR-548a and miR-146a) involved in the regulation of inflammation cause by infection of periodontitis gingivae in chronic patients and ApoE (-/-) mice as mouse model [11,12].

The mouth provides an ideal microbial flora to the various microbes. The collective characteristics of a moist, dark, consistent nutrients and the hard enamel altogether helps microbes thrive in a biofilm that is well attached to the teeth surfaces. The immune

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response to these thriving microbes is associated with different cytokine regulation such as IL-8 mRNA induction signaling from challenge bacteria such as P. gingivalis triggers human gingival epithelial cells (HGECs) through activation of MAPK kinase/ extracellular signal-regulated kinase (MEK/ERK) pathways, not mitogen-activated protein kinase (MAPK) p38 pathway. Other study of bacterial challenge to GECs-gingival epithelial cells using *Porphyromonas gingivalis* also suggest that *P. gingivalis* affects DNA methyltransferase and histone deacetylase by stimulate gingival cytokine secretion such as IL-6, CXCL1, and beta-defensin-2 [13,14].

Epigenetics is the study of the changes in the gene function that is not the result expected DNA sequence. Environmental factors in any form induce epigenetic changes those lead to increased susceptibility to disease. The three gene expressions are the DNA methylation, micro-RNA and modification of histones. Patients those are given similar clinical treatment respond differently thus raising the question of understanding the underlying mechanism involved in periodontal disease. This can be achieved by the advanced methodology of epigenetics. For examples, Zhang et al (2013) reported that change in promoter methylation status of Tumor necrosis factor-α (TNF- α) regulate periodontal disease and its pathogenesis in human chronic gingival biopsies and cell lines (THP.1 cells and RAW294.7 cells) [15]. In additions, it indicates that inflammation of periodontitis correlate methylation status of Toll-Like Receptor (TLR) 2 gene as innate immune response marker in periodontopathic bacteria using the Methyl Profiler DNA Methylation qPCR assay [16].

Increasing body of evidences support that different micro-RNA species regulate gingival mediated inflammation process in periodontitis as compared to healthy gingival tissue studied using quantitative real-time PCR (QPCR) and micro-array techniques. Experimental studies have supported the premise such as engagement of microRNA between lipid metabolism and inflammation by altering epigenetic change and immune dysfunction result in pathological events such as glucose tolerance, hypertriglyceridemia, central obesity, rheumatoid arthritis and coronary heart disease [17]. Furthermore, other study for a role of micro-RNA on stem cell driven differentiation indicate that risk factor such as cigarette smoking attributes to delay on regenerative healing process by stimulation of two nicotine specific miR species (i.e.,hsa-miR-1305 and hsa-miR-18b) in human Periodontal Ligament-Derived Stem Cells (PDLSC) [18] and deregulation of miRNAs is associated with metabolic disorders [19]. Of note, there is evidence of a connection between the effects of changes by maternal periodontal infection as the oral micro flora with alterations on the placenta and/or the fetal environment result in preterm low birth weight through a case-control studyusing124 pregnant or postpartum mothers led by Offenbacher et al. [20].To confirm the correlation between Epigenetic and immune alteration in human population due to exposure to periodontal infection, clinical study using genomic tools led by Lavu V et al. have shown that chronic periodontitis susceptibility is associated with Single Nucleotide Polymorphisms (SNPs) in the IL1B gene and Variable Number of Tandem Repeats (VNTRs) polymorphism in the IL1RN gene after examining blood samples and genotyping in healthy gingiva and chronic periodontitis patients [21]. In furtherance of, other researcher also reported that interleukin (IL)-1β C-511T polymorphism is involved in Chinese chronic periodontitis using meta-analysis [22]. Therefore, these results shed light emphasizing that epigenetic modification by periodontopathic bacteria is pivotal genomics signature towards health complications from dysfunction of immune regulations as well as exacerbated inflammatory signaling circuit.

In conclusion, Comparative analysis of disease detection would reveal significant differences in methodologies between the conventional and the more innovative techniques of the omics approach such as Nutrigenomics, Metabolomics, Immunomics and Immuno-informatics to mitigate periodontal disease as a health burden as well as to explore its therapeutic value in the process of disease complications by Nutrition or Food ingredients. In addition, epigenetic have been able to provide a greater insight into understanding the mechanism of the periodontal disease. It has enabled us to understand that periodontal disease does not only have severe ramification in the mouth but also effects the gene expression in systemic environment and has associations with causing oxidative stress, DNA-methylation and alteration in the gene expression micro-RNA. It also has adverse effects on the T-cells which play an important role in the immune response which is not limited to clinical intervention or limitation of dental treatment since the immune response varies in different individuals. It is because of using the advanced methodology of epigenetics that we are able to understand this difference in immune response and why certain patients suffering from periodontitis are not able to respond to the current dental treatments in place. Hence, this can become a changing point in dentistry by providing them with the alternative tools to utilize advanced risk assessment before the disease progresses as tissue destruction and accompanying health complications, including Obesity as a metabolic disorder.

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