Periodontal Inflammation and HSP60

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The oral cavity has the potential to harbor at least 600 different bacterial species, and in any given patient, more than 150 species may be present, surfaces of tooth can have as many as billion bacteria in its attached bacterial plaque and good oral hygiene is the fundamental for oral integrity as it greatly affects the quality of life [1]. Periodontitis is a destructive inflammatory disease of the supporting tissues of the teeth and is caused by specific microorganisms or group of specific microorganisms resulting in progressive destruction of periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession or both. The host responds to the periodontal infections with an array of events involving both innate and adaptive immunity [2]. Periodontal diseases are recognized as infectious processes that require bacterial presence and a host response and are further affected and modified by other local, environmental, and genetic factors. The key organisms that cause periodontal disease were anaerobes including Aggregatibacteractinomycetemcomitans, Porphyromonasgingivalis, Prevotellaintermedia, Tannerella forsythia, Fusobacteriumnucleatum, Peptostreptococcus micros, and Campylobacter rectus [3].

Cells produce heat shock proteins (HSPs) upon encountering physiological stress such as heat stress or chemical stimulation [4]. HSPs play a major role in a cell’s fight against stress and in guarding the cell from its harmful effects. HSPs, also called stress proteins, perform many cellular functions, including cell protection, intracellular assembly, and the folding and translocation of oligomeric proteins [5]. HSPs can be categorized into 5 family groups, based on their size, structure, and function, which are HSP110, HSP90, HSP70, HSP60, and HSP27 and other small-molecule HSPs [6]. They are divided into high- and small-molecular weight molecules, based on their molecular weights.

Researchers hypothesized that pathogenic bacteria stimulate periodontal cells to increase Hsp60 expression that could in turn initiate macrophages, to start producing pro-inflammatory cytokines. Due to their high conservation among various microbial pathogens and their ability to induce very strong cellular and humoral immune responses, Hsp60s are thought to play a role as candidate antigens in periodontal disease [7]. Some of the landmark studies are illustrated in (table 1).

Heat shock proteins play an vital role in gingivitis and periodontitis and there will be an ongoing consumption of heat shock proteins in the inflamed periodontal tissue due to the release of reactive oxygen species and proteases. There is presently much conversation regarding the relationship between periodontitis and cardiovascular...
disease, since heat shock proteins are expressed on the cell surface on the periodontitis pathogen \textit{P. gingivalis} and it has been suggested that the expression of heat shock proteins and immunity to heat shock proteins is involved in the establishment and progression of cardiovascular disease. Furthermore, it has also been suggested that persons with periodontitis might have a deficiency in the ability to mount humoral responses to heat shock proteins.

**References**