

## PROBLEM STATEMENT

Diabetes is a metabolic disorder resulting in chronic hyperglycemia and hyperlipidemia that ultimately induces diverse multiple system pathologies; increasing the risk for atherosclerosis coronary heart disease, stroke, myocardial infarction, renal disease and periodontitis.<sup>1-5</sup> Diabetes has become the fifth leading cause of death.<sup>5</sup> It affects approximately 17 million individuals or 6.2% of the population in the U.S.,<sup>6</sup> resulting in chronic hyperglycemia and hyperlipidemia that ultimately induces diverse multiple system pathologies.<sup>3</sup>

Periodontitis is an infection caused by the gram-negative organisms in the plaque biofilm that affects 7-15% of the adult population.<sup>7,8</sup> An abnormal inflammatory response, that is a hyper-inflammatory trait,<sup>9,10</sup> has been linked to diabetes where there is an increased susceptibility to infections such as periodontal disease. The hyperinflammatory trait is associated with an exaggerated secretion of inflammatory mediators (TNF $\alpha$  and IL-6) and systemic markers of inflammation. It is suggested that this process mechanistically contributes to the pathology associated with this chronic disease processes.<sup>2-4, 7, 11-15</sup>

Diabetes and adult periodontitis are both common chronic diseases observed in a significant proportion of the adult U.S. population.<sup>3,4</sup> It is well established that diabetes is a risk factor for poor periodontal health.<sup>1-5</sup> However, recent studies have shown that periodontal disease adversely affects glycemic control in diabetes.<sup>2-4,7,11</sup> Diabetic complications have been attributed to the hyperglycemic state, which over time results in the irreversible covalent modification (glycosylation) of structural proteins and lipids that comprise the extracellular matrix and connective tissues, as well as the vascular tissues.<sup>16</sup>

<sup>18</sup> These structural changes result in impaired capillary function, poor blood perfusion of tissues and organs and the release of reactive oxygen species (oxidative stress) triggering a systemic inflammatory process.<sup>11,28</sup> The activation of inflammation at a systemic level results in the chronic elevation of inflammatory mediators (IL-1, TNF $\alpha$ , IL-6, and PGE<sub>2</sub>) and acute phase reactants such as C-reactive protein, elevated fibrinogen and lowered albumin all hallmarks of the acute phase reaction (APR) observed in diabetes and periodontitis.<sup>28,36,42</sup> Thus, a hyper-inflammatory trait may predispose an individual to a more severe systemic disease that may occur as a result of over expression of inflammatory mediators and may ultimately lead to metabolic dysregulation. The purpose of this study is to determine if periodontitis serves as a stimulus for systemic-based inflammatory response that may represent a previously underestimated metabolic stressor enhancing insulin resistance and impairing insulin secretion. Further, looking at the effect of non-surgical periodontal therapy, scaling and root planing (S&RP), in type 2 diabetics on the inflammatory mediators TNF $\alpha$  and IL-6 and the relationship of these mediators to markers of insulin resistance.

### **RESEARCH HYPOTHEIS/RESEARCH QUESTION**

What role do the inflammatory mediators, TNF $\alpha$  and IL-6, have in destroying alveolar bone and periodontal tissues in a type 2 diabetic population?

Ho1. Inflammatory mediators cause bone destruction or inflammation in the oral cavity.

RQ2. Can scaling and root planing decrease the levels of systemic inflammatory mediators TNF $\alpha$  and IL-6 in a type 2 diabetic population?

Periodontitis patients receiving scaling and root planing will show reduction in the inflammatory mediators IL-6 and TNF $\alpha$  in a type 2 diabetic population.