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Contributions of phosphodiesterases to type-2 diabetes induced cardiac dysfunction

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Phosphodiesterases (PDEs) play an important role in modulation of cardiac contractility through the degradation of cyclic nucleotides. It has been shown that altered PDE activity induces obesity, insulin resistance, hypertension as well as cardiovascular diseases. PDE inhibitors have inotropic and lusiotropic effects on cardiomyocytes by modulating L-type Ca2+ channels (LTCC), ryanodine receptors (RyR2), sarcoplasmic reticulum Ca2+ load and myo-filament Ca2+sensitivity. The proposed mechanism for the effects is increased activity of protein kinase-A mediated protein phosphorylation. While PDEs-inhibitors have acute inotropic effects on the heart, chronic use of the drugs leads to heart failure and arrhythmias. Studies have been designed to understand the mechanisms underlying these effects. Recent studies show that PDEs are specifically located nearby key contractility modulators such as RyR2, LTCC and SERCA. This micro-domain specific function has to be taken into consideration in evaluating the effects of PDEs-inhibitors on the heart. Although several studies have explored cardiac PDE activity, only few studies investigated the role of cardiac PDE activity in obesity. Therefore, we investigated the role of PDE activity in the heart from high sucrose-induced metabolic syndrome (Met S) developed overweight-rats. Our data showed important levels of increased protein expression and activity in both PDE3 and PDE4, which seem to play an important role in the development of cardiac dysfunction in insulin resistant overweight-rats. In conclusion, the use of PDEs-inhibitors seems to have beneficial actions in the treatment of Met S-associated heart diseases, while it needs further studies on selectively target PDE subtypes or micro-domains under these types of pathological conditions.

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Green tea modulates cytokine expression in the periodontium and attenuates alveolar bone resorption in type 1 diabetic rats

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Introduction: Diabetes mellitus comprises a group of disorders with the main feature of hyperglycemia. Chronic hyperglycemia increases the severity of periodontal disease via an exacerbated inflammatory response. In particular, green tea has been shown to possess anti-inflammatory properties mediated by its poly-phenol content.

Objectives: This study investigated the mechanisms by which green tea attenuates the spontaneous onset of diabetes-induced periodontitis.

Methods: Diabetes was induced in rats via intra-peritoneal injection of streptozotocin. Diabetic and control animals were divided into water-treated and green tea-treated subgroups and were analyzed at 15, 30, 60 and 90 days after diabetes induction. Immunohistochemistry was performed to quantitatively evaluate TNF-α, RANKL, OPG, IL-10 and RUNX-2 expression in serial sections of each hemi-maxilla. Morphometric measurements of the distance from the cementum-enamel junction of the superior distal root of the first molar to the alveolar bone crest were performed to assess bone loss.

Results: Diabetes resulted in significant bone loss and positive cells for inflammatory mediators. In the diabetic rats treated with green tea, we observed a decreased number of cells expressing RANKL and TNF-α compared with that observed in the diabetic rats treated with water. Additionally, green tea increased the numbers of cells that stained positive for OPG, RUNX-2 and IL-10 in the diabetic rats.

Conclusion: Green tea intake reduces expression of TNF-α and RANKL to normal levels while increasing expression of IL-10, RUNX-2 and OPG. Therefore, green tea represents a potential therapeutic agent for the treatment of diabetes-related periodontal disease.

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