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Alterations of VEGF and CSF-1 in periodontal tissue remodeling following biophysical force loading in hyperglycemia

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Diabetic mellitus is a well-known systemic disease to affect periodontal tissues behavior. However, underlying mechanism of how this alters the alveolar bone tissue homeostasis in a physiological condition or even under a biophysical force loading such as orthodontic force application is unknown. This study investigated the effect of hyperglycaemia itself or glucose metabolites on biophysical force-induced periodontal tissue remodelling. Alterations of two key factors for the altered alveolar bone remodelling were hypothesized: vascular endothelial growth factor (VEGF) and colony stimulating factor-1 (CSF-1). The alteration mechanism was investigated by examining the effects of hyperglycaemia and advanced glycation end products (AGE) and their receptor machineries. *In vivo* tissue responses were evaluated by applying orthodontic appliances to molars in streptozotocin-induced hyperglycaemic rats. Morphological features were examined by light microscopy and immunofluorescence and the gene alteration was determined by real-time RT-PCR. Also, the *in vitro* effect of hyperglycaemia itself and biophysical forces in a hyperglycaemic condition were determined in human primary periodontal ligament (PDL) cells and mouse bone marrow stromal cells. *In vivo*: In diabetic rats, tissue responses were histologically characterized by augmented angiogenesis in the PDL and additional undermining (or indirect) osteoclastic bone resorption from bone marrow surface. By diabetes itself, CSF-1, VEGF, AGE and AGER mRNA levels were upregulated, whereas changes in expression of DDOST, a decoy receptor for AGE and AGE-detoxifying Glo1 were not significant. VEGF expression in the PDL was enhanced in diabetic rats. Biophysical force-induced tooth movement (BTM) at day 6 was augmented in diabetic rats, compared with normoglycemic rats. In vitro: A hyperglycaemic condition (25 mM) itself downregulated the VEGF and AGER transcription in human PDL cells, compared with a normoglycemic condition (5 mM), whereas (glucose transporter 1) Glut-1 and CSF-1 were not varied. Furthermore, this hyperglycaemic condition decreased RANKL/OPG ratio and inhibited osteoclast genesis in mouse bone marrow stromal cells. In contrast, N-acetyl glucosamine or PUGNAC, an OGA (β-D-N acetylglucosaminidase) inhibitor treatment stimulated osteoclast genesis. Advanced glycation end products and N-acetyl glucosamine upregulated the expression of VEGF, CSF-1, receptors for AGE (AGER) and Glut1 at specific time points. The VEGF and CSF-1 mRNA in PDL cells was upregulated by either compression or tension force and moreover, this upregulation was more altered at the high glucose or glucose metabolites-treated conditions, compared with a normoglycemic condition. This study suggested that diabetic hyperglycaemia–induced metabolic end products may alter periodontal tissue remodelling due to augmented angiogenesis and macrophage activation and this alteration can be further altered by biophysical forces including orthodontic force.

Biography

Sun Hun Kim completed his DDS and MS., PhD from Chonnam National University, School of Dentistry, Korea in the year 1980-1991. He is a Visiting professor in UCSF medical school, USA; He is a Dean in School of Dentistry, Chonnam National University, Korea in 2013. He is a Head of Dental Science Research Institute, School of Dentistry, Chonnam National University, Korea from 2013. And also a professor in the Department of Oral Anatomy, school of dentistry, Chonnam National University, Korea from 1989.

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