



## Dr. Hidefumi Maeda

Hidefumi Maeda is a Professor of Section of Endodontology & Operative Dentistry, Division of Oral Rehabilitation, Faculty of Dental Science, Kyushu University in Japan. With a D.D.S. from Kyushu University in Japan and a Ph.D. in Dental Science from Kyushu University in Japan. His background includes: Membership of 8 Professional Societies, 10 Distinguished Honors and Awards, 67 Publications in refereed journals, 14 Books and over 110 Conference Papers and Invited Lectures. He is an accomplished endodontist and an advising doctor for innovative therapies for endodontic diseases. The title of his research is “To research and develop the methods allowing for longer preservation of a tooth”.

### Abstract

#### **Title: Differentiation and Aging of Pulp Cells**

Pulp cells reside in pulp chamber and undergo normal metabolism throughout the life span of the landlord tooth unless it is subjected to pulpectomy. Although these cells are thought to differentiate into odontoblasts, pulp tissue does not mineralize under normal condition. Dentin, however gets thicker with aging, and the chamber space gets narrower. The biology of this curious cell population remains obscure. In this context, we have studied the odontoblastic differentiation and aging of these cells. We found that their differentiation is upregulated by the signal via their surface receptor, calcium sensing receptor (CaSR) and Semaphorin 3A (Sema3A), while downregulated by TGF-beta-induced protein ig-h3 ( $\beta$ ig-h3). Exposure of these cells to agonists of CaSR induced mineralization along with the increased expression of dentin-related molecules. We also found that Sema3A, an inducer of osteogenic differentiation of immature periodontal ligament cells in our recent report, promoted the odontoblastic differentiation of pulp cells *in vitro* and repair dentin formation *in vivo*. In contrary,  $\beta$ ig-h3 is expressed in pulp tissue in physiological condition whereas its expression is decreased under inflammation *in vivo*. This protein exerted an inhibitory effect on mineralization of pulp cells *in vitro*. On the other hand, aged pulp cells that expressed senescence-associated  $\beta$ -galactosidase and other senescent markers altered proliferation and mineralization ratios, compared with young cells. Taken together, the metabolism of pulp cells are regulated in an elaborate manner, and to manage these properties may allow to develop new materials and methods to protect pulp tissue by physiologically facilitating dentin formation.