Dr. Huan-Xin Meng, female, graduated from the School of Stomatology, Shanghai Second Medical College with her D.D.S. degree in 1976. She gained her M.S. and Ph.D. degrees at the Graduate School of Beijing Medical University in 1984 and 1987 respectively. From April to June 1989, Dr. Meng was a visiting research fellow at the Faculty of Dentistry, Osaka University, Japan. She then became a postdoctoral research fellow at the MRC Dental Research Unit of London Hospital Medical College, the UK from July 1989 to April 1992. From February to July 1998, Dr. Meng was appointed as Visiting Professor in the Department of Periodontology and Fixed Prosthodontics, School of Dental Medicine, Bern University, Switzerland.

Dr. Huanxin Meng has been a professor, a dental director and a postdoctoral mentor in Peking University for many years. She is professor of Department of Periodontology, Peking University School of Stomatology, and currently President of the Chinese Society of Periodontology. Dr. Meng is the chief editor of National Text of Periodontology, 3rd edition, and serves on the editorial boards of leading journals including Journal of Periodontal Research, Journal of Periodontology, Chinese Journal of Stomatology and Journal of Shanghai Stomatology, et al.

Dr. Meng’s key areas of research are: Engaged in periodontal research more than 29 years, and focused on the pathogenesis of periodontal diseases. Recent years, main work has been concerning on the pathogens and host susceptibility of periodontitis; the relationship between periodontitis and general health (systemic diseases, including diabetes mellitus, heart diseases, gastric diseases, et al.) and periodontal tissues engineering.

Dr. Meng won the Outstanding Thesis Award of Chinese Medical Association (Beijing Division) twice in 1987 and 1995. Her research on gum diseases and periodontitis received many accolades including the Science and Technology Award of Ministry of Health (second runner-up prize) in 1989, and the Science and Technology Award of Chinese Medical Association (second runner-up prize) in 2004.

Dr. Meng has published more than 160 academic articles, with 24 being published in the SCI. She has participated in the national coordination and editing of five dentistry textbooks. Furthermore, Dr. Meng is the mentor of 25 doctoral postgraduates, 16 master postgraduates and one postdoctoral research fellow. She has directed 23 national and provincial scientific research projects, and 6 international and national collaborative studies.
Part 1 Relationships between 25 hydroxy vitamin D3, bone-related biomarkers regulated by vitamin D and periodontitis

Sixty-six patients with aggressive periodontitis (AgP), 52 patients with chronic periodontitis (CP) and 60 healthy controls were included in this study. It was demonstrated that plasma 25OHD3 levels in patients with AgP were higher than those of healthy controls and were statistically significantly correlated with bleeding index, which indicated that 25OHD3 might be associated with periodontal inflammation.

Part 2 Influence of initial periodontal therapy on systemic and local levels of 25 hydroxy vitamin D3 and elements regulated by vitamin D in patients with aggressive periodontitis

Nineteen patients with AgP were enrolled and received initial periodontal therapy. It was found that the respective systemic 25OHD3 and interleukin-1β levels significantly dropped from baseline to 2 months after therapy. The respective local 25OHD3 and interleukin-1β levels significantly decreased from baseline to 2 and 6 months after therapy. Systemic and local 25OHD3 concentrations were positively correlated at baseline. The data indicated that 25OHD3 might be involved in periodontal inflammation.

Part 3 Expression and regulation of 25 hydroxylase in human periodontal fibroblasts

In HGF and HPDLC, CYP27A1 mRNA expression was higher than CYP2R1 mRNA expression and only CYP27A1 protein expression could be detected. HGF and HPDLC generated detectable 25OHD3 in response to vitamin D3 and the amount of 25OHD3 was positively correlated with the concentration of vitamin D3. The generation of 25OHD3 after incubation with vitamin D3 significantly dropped if CYP27A1 was knocked down using specific siRNA but was not influenced by knockdown of CYP2R1. The regulators of CYP27A1 transforming growth factor β1 and dexamethasone could also up-regulate CYP27A1 expression in HGF and HPDLC. In addition, interleukin-1β and Porphyromonas gingivalis lipopolysaccharide strongly induced CYP27A1 expression. Conclusively, this study showed expression, activity and functionality of 25 hydroxylase in HGF and HPDLC. CYP27A1 might be the dominant 25 hydroxylase and could be induced by inflammatory factors (interleukin-1β and Porphyromonas gingivalis lipopolysaccharide), transforming growth factor β1 and dexamethasone in HGF and HPDLC.

Part 4 Expression and regulation of 1,α hydroxylase in human periodontal fibroblasts

HGF and HPDLC expressed CYP27B1. HGF and HPDLC generated detectable 1,25OHD3 in response to 25OHD3 and the amount of 1,25OHD3 was positively correlated with incubation time and the concentration of 25OHD3. The generation of 1,25OHD3 after incubation with 25OHD3 significantly dropped if CYP27B1 was knocked down using specific siRNA. Exposure to 25OHD3 also resulted in up-regulated transcription of the 1,25OHD3 responsive genes CYP24A1 and RANKL in HGF and HPDLC. The up-regulation effect of 1000nM 25OHD3 was significantly stronger than that of 10nM 1,25OHD3 and specific knockdown of CYP27B1 using siRNA resulted in significant reduction in the transcription of CYP24A1 and RANKL. The classical renal regulators of 1,α hydroxylase (parathyroid hormone, calcium and 1,25OHD3) and Porphyromonas gingivalis lipopolysaccharide did not regulate CYP27B1 expression in HGF and HPDLC. In contrast, interleukin-1β and sodium butyrate strongly induced CYP27B1 expression. This study showed expression, activity and functionality of 1,α hydroxylase in HGF and HPDLC and 1,α hydroxylase was induced by inflammatory factors (interleukin-1β and sodium butyrate) in these cells. Vitamin D could act in an auto/paracrine manner in HGF and HPDLC.