Dr. Thomas E. Van Dyke

略歴

Thomas Van Dyke, D.D.S., Ph.D., is Vice President for Clinical and Translational Research, and Director of the Center for Clinical and Translational Research at the Forsyth Institute in Cambridge, MA and Professor of Oral; Medicine, Infection and Immunity at Harvard School of Dental Medicine; D.D.S. (1973), Case Western Reserve University; M.S. (1979), Periodontics Certificate (1980), PhD (1982) SUNY at Buffalo. Balint Orban Memorial Prize for Research in Periodontology (1981), Diplomate of the American Board of Periodontology (1989), IADR Award for Basic Research in Periodontology (2001), Norton Ross Award for Excellence in Clinical Research (2002), William J. Gies Periodontology Award (2008). President of the International Association of Periodontology (1997-1999). 330+ original articles, numerous abstracts and book chapters published. Research interests are the structural and functional relationship of abnormalities of the inflammatory process in the etiology and pathogenesis of periodontal and other infectious inflammatory diseases.

Inflammation, Periodontitis and the link to Systemic Diseases

Forsyth Institute Cambridge
Thomas E. Van Dyke

Periodontitis is an infectious inflammatory disease; that is, a disease initiated by bacteria that has inflammatory destruction of tissues at the heart of its pathogenesis. Uncontrolled inflammation in infectious inflammatory diseases results from over-active stimulation and a failure to resolve inflammation. Resolution of inflammation is an active process mediated by endogenously produced lipid molecules that orchestrate a return to tissue homeostasis. These resolving molecules that actively regulate the resolution of acute inflammation, called lipoxins and resolvins, are eicosanoids; the same class of molecule as prostaglandins and leukotrienes. These small lipid molecules act through specific receptors on inflammatory cells. Lipoxins and resolvins have been shown to have significant impact in inflammatory diseases in addition to periodontitis, including type 2 diabetes and cardiovascular diseases. The new understanding of active resolution of inflammation has the potential to completely change how we approach inflammatory disease and periodontal therapy. Lipoxins and resolvins have been demonstrated to promote regeneration of bone lost to disease and to prevent inflammatory bone loss in animals. In addition to the emergence of new surgical and non-surgical, medical therapies for periodontal diseases, recent studies have suggested a relationship between oral infection, in particular periodontal disease, and systemic diseases. Epidemiologic studies have implicated periodontal disease as a risk factor for the development of cardiovascular disease and stroke, and a risk factor for pre-term low birth weight babies in pregnant women. In addition, studies in diabetics have revealed that untreated periodontal disease can lead to diabetic complications and have a direct impact on glycemic control. As our understanding of pathways of inflammation has matured, a better understanding of the molecular basis of inflammation has emerged. The potential for modification of resolution pathways for the prevention and treatment of periodontal diseases will be discussed in detail. More importantly, the potential, as well as real, associations between periodontal diseases and systemic diseases underline the importance of proper diagnosis and treatment of these extremely common oral diseases. The rationale for additional periodontal medical approaches will be presented and the potential benefits to systemic health will be explored.

Dr. Gregory J. Seymour

略歴

Professor Greg Seymour is currently an Emeritus Professor at the University of Queensland and an Honorary Professor at Griffith University in Australia. He was formerly Dean of the Faculty of Dentistry and Professor of Periodontology at the University of Otago in New Zealand. He graduated from the University of Sydney in 1971, did his specialist training in Periodontics also at the University of Sydney in Australia and his PhD in basic Immunology at the University of London in the UK. He has authored over 370 papers and book chapters and has over 16,000 citations. He has received numerous Fellowships and awards including a Distinguished Scientist Award of the IADR in 1997, Honorary Life Membership of the British Society for Periodontology in 2003 and Fellowship of the Royal Society of New Zealand in 2008.

Back to the Future: Periodontics Through the Looking Glass

Sir John Walsh Research Institute, Faculty of Dentistry, University of Otago
Gregory J. Seymour

While it is virtually impossible to predict the future of periodontics, there is no doubt that it will be determined by the need for the profession to provide safe and reliable periodontal care which, in turn, will be shaped by current research. Over the past two decades clinical periodontics has been dominated by implants. However, it is now becoming apparent that the initial promise of an almost 100% success rate is not being fulfilled and that some 15-20% of implants are failing due to varying degrees of peri-implant mucositis and peri-implantitis. This failure is often as a result of inappropriate placement in patients with existing untreated periodontitis.

Worldwide the population is aging and due to advances in dentistry over the past 50 years, these people will expect greater personalised care to ensure that they keep their teeth (and implants) as they get older and move into aged care facilities. Hence, in the future, a greater focus on more effective treatment of periodontitis and maintenance of periodontal health will be required, not only to maintain a healthy functioning dentition in the aging population, but also to ensure greater success of implants.

In this context, future periodontal care will need to be more tailored to the individual. Current research using next generation sequencing (NGS) is showing that plaques vary from individual to individual and that it is likely that a dysbiosis in an individual's plaque rather than specific organisms per se results in disease progression. And, while plaque control will remain the backbone of periodontal care, the use of specific oral probiotics to restore the balance in the oral microbiota is likely to assume greater importance. It is now firmly established that the progressive periodontal lesion (and probably the peri-implantitis lesion) is a B cell / plasma cell lesion and that anti-B cell therapies are effective in controlling its progression. In the future, the use of targeted, anti-inflammatory therapies locally, in conjunction with plaque based therapies, are also likely to be integral treatment modalities. Here pharmacogenomics will become essential in informing the clinician as to which host modulating agent is most suited to an individual patient. Epigenetic factors, such as smoking and stress, are also recognised to have a significant influence on disease progression and cannot be ignored by the treating clinician. Unravelling the complexities of bacterial, host and epigenetic interactions will lead to enhanced individual risk profiling and more targeted treatment in the future.

Equally, future periodontal care cannot ignore the young as recent data on the natural history of untreated periodontal disease has highlighted the importance of treating early disease in the young in order to ensure a functioning dentition over the age of sixty.

Finally, the association between periodontal disease and overall health is increasingly being recognised such that a strong working relationship with medical practitioners will further define periodontics in the future.