Thema: **Agressieve parodontitis**

8.30 – 9.00  ontvangst met koffie
9.00 – 9.15  opening

**Prof. Bruno Loos** Moderator

9.15 – 10.15  **Prof. Ubele van der Velden** Classificatie en epidemiologie

10.15 - 11.00  koffie

11.00 – 12.00  **Prof. Mogans Kilian** The etiology of aggressive periodontal disease reevaluated in view of the genetic polymorphism of bacteria

12.00 – 13.00  **Prof. Denis Kinane** Pathophysiology and genetics of aggressive periodontitis

13.00 – 14.15  lunch

14.15 – 15.15  **Prof. Inger Kjaer** Development and function of PDL

15.15 – 15.45  thee

15.45 – 16.45  **Prof. Gareth Griffiths** Treatment aspects of patients with aggressive periodontitis

16.45 – 17.15  **discussie + afsluiting**

17.15 – 18.00  borrel

Dit congres is voorzien van het Q-keurmerk.

Aan deelname aan dit congres kunnen 5 registratiepunten worden ontleend.
The recent explosion in genetic information about bacteria has revealed an unexpected degree of genetic polymorphism among members of the same species. Up to 25% of the bacterial genome consists of genes that are variably present in different strains. Added to the newly demonstrated high number of bacterial species that may be counted as belonging to the oral microflora (> 700), most of which are yet to be cultivated and characterized, this renders the complexity of the oral microflora extreme. On this background previous conclusions on the etiology of periodontal disease and dental caries may be too simplistic. Population genetic analysis of putative oral periodontal pathogens (e.g. P. gingivalis and A. actinomycetemcomitans) demonstrate that most Caucasian patients are colonized with distinct clones excluding that they may be viewed as traditional pathogens that disseminate from patient to patient. Surprisingly, a single clone of A. actinomycetemcomitans is strongly associated with aggressive periodontitis in Arabic and African individuals of North-West African descent. The clone is endemically present in Morocco and appears to explain an unusually high prevalence of aggressive periodontitis (15%) in Moroccan adolescents. Genetic analysis of members of this clone demonstrate a number of virulence-associated properties and suggest that the clone emerged as a distinct genotype in the Mediterranean area of Africa and subsequently spread with the trans-Atlantic slave trade and emigrations to other parts of the world.

Biosketch

Mogens Kilian is professor of medical microbiology at the University of Aarhus (since 1991). He has a DDS degree from 1968 and a D. Sc. degree from 1975. Trained in clinical microbiology 1970-74. Visiting professor at the Department of Microbiology, University of Alabama at Birmingham, USA 1977-79, professor and chairman of Oral Biology, Royal Dental College, Aarhus, from 1981-91. Member of the editorial board of 5 international scientific journals including Infection and Immunity and Journal of Clinical Microbiology, ad hoc referee for multiple periodicals in microbiology and immunology and for national research councils abroad. Member of the Danish Medical Research Council 1988-91. Member of the Danish Committee for Scientific Dishonesty. Member of the Senate of the University of Aarhus. Organiser and co-organiser of 9 international scientific conferences in microbiology and immunology, Research in molecular microbial evolution, ecology and pathogenesis. Interactions of pathogenic bacteria with the mucosal immune system. Bacterial taxonomy and identification. Has published more than 200 scientific papers. Invited speaker at more than 75 international scientific conferences.
Aggressive Periodontitis is a form of chronic periodontitis that has been linked with a variety of aetiological factors, few of which have been scientifically proven. In the past, neutrophil chemotactic defects were suggested as predisposing factors particularly for localized forms of AgP. This has been largely disproved. Actinobacillus actinomycetemcomitans, previously renamed Haemophilus actinomycetemcomitans and now named Aggregatobacter actinomycetemcomitans, has also been linked to AgP. This putative microbiological link has been the reason that antibiotic regimens for AgP have included tetracycline. The finding that A. actinomycetemcomitans are also cultivable from supragingival plaque and present in healthy mouths puts the onus on clinical microbiology to actually show causal association to justify antibiotic regime recommendations. Reports from the US associate AgP in African Americans with IgG2 antibody prevalence but this evidence again has to be studied in diverse and numerous populations before it can be generalizably utilized. The familial transmission evidence for AgP is strong and available literature supports an autosomal dominant mode of transmission. Having said this there are increasing reports on associations between specific pathophysiologically related single nucleotide polymorphisms and AgP but none are presently accepted as incontrovertibly linked although haplotype studies have suggested interesting possibilities. The familial studies using segregation analyses, support an autosomal dominant mode of transmission. Having said this there are increasingly frequent reports on associations between specific pathophysiologically related single nucleotide polymorphisms and AgP but none are presently accepted as incontrovertibly linked although haplotype studies have suggested interesting possibilities. The familial studies using segregation analysis, support an autosomal dominant mode of transmission and support the clinical message: ‘we should examine all siblings and offspring as there is a 50% chance of finding more affected patients this way’.

Biosketch

Denis F. Kinane, B.D.S., Ph.D., is a professor in the Department of Periodontics, Endodontics and Dental Hygiene in the School of Dentistry. He also serves as Associate Dean for Research and Enterprise and Director of the Oral Health and Systemic Disease Research Group. His research focuses on periodontal immune and inflammatory processes, mainly explaining the causes, development and susceptibility markers of periodontal disease. Kinane and his team also are examining the relationship between oral and systemic health and diseases such as diabetes, cardiovascular disease and periodontal disease. Kinane has received many awards for his work, among them Procter and Gamble’s International Animal Alternatives Award, the Restorative Dentistry Medal from the University of Edinburgh and the British Society of Periodontology’s Sir Wilfred Fish Research Prize. He has published more than 170 original articles in scientific journals and is the president of the Gordon conference in Periodontology. He is the councilor for the Periodontal Research Group of the IADR and chairs and organizes multiple symposia and international meetings. His research and publications admirably qualify him to present an overview of oral and systemic health interactions.

The development of the periodontal membrane (PDL) from prenatal life to puberty will demonstrate new histochemical results on epithelial occurrence in the membrane. This epithelial layer has developed from the epithelial sheeth of Hertwig (Becker et Kjaer, in press 2007). In relation to this epithelial layer, collagen fibres and innervation are demonstrated (Kjaer et al., submitted 2007). The structure and the function of the periodontal membrane during tooth eruption, anchylosis and resorption is illustrated (Bille et al., submitted 2007).

Tooth eruption and alveolar bone apposition are two interrelated processes dependent on the periodontal membrane. Accordingly, focus will also be given to alveolar bone growth and the chemical processes initiating this growth. Special focus will be given to protein gene product, PGP, marked in osteoblasts (Kjaer et Nolting, submitted 2007).

Alveolar bone has different origins
The embryological development of human jaws and dentition have shown that different jaw parts originate from different areas on the neural crest. The peripheral nervous system in the jaws and their innervation areas mark and delineate these different jaw components (Kjaer, 1997, 1998).

Juvenile Periodontitis
Initial alveolar bone “loss” in Juvenile Periodontitis is first registered in these developmental border regions between the jaw components located between the first molar and the second premolar, distally to the laterals in the upper jaw and mesially to the centrals in the lower jaw. These regions are also the most common agenesis regions.

It is suggested that the bone “loss” in Juvenile Periodontitis is not a “loss” but a failure in the normal alveolar bone apposition.

Biosketch

Education: Dentist and specialist in orthodontics from the School of Dentistry, Copenhagen, Denmark
Doctor Odont. 1982
Doctor Med. 1999
Current appointment: Since 2002 Professor in orthodontics, at the Department of Orthodontics, School of Dentistry, Copenhagen, Denmark
Research: Published 142 scientific papers in international peer reviewed journals.
Main scientific interest: Human prenatal normal and pathological development; The human dentition; Craniofacial development; Anthropology; Phenotype - genotype relations.
If we want to know more about the treatment of patients with aggressive periodontitis, several aspects have to be discussed:

1. Common features of Aggressive Periodontitis:
   Patient presenting complaints & clinical features.

2. Controlling the infection
   a. Response to initial therapy.
   b. Outcomes of a clinical trial using adjunctive antimicrobials
   c. Timing of the use of antibiotics

3. Repairing the damage
   a. Surgical approach to management
   b. Use of implants

Biosketch

Gareth Griffiths graduated from Cardiff in 1979 and completed his MScD there in 1984. He obtained his PhD in 1990 whilst working as a Clinical Research Scientist for the Medical Research Council. He became lecturer and then senior Lecturer in the Periodontology Department of the Eastman Dental Institute, where as Programme Co-ordinator he assumed responsibility for the programme of postgraduate studies in Periodontology. He moved to Sheffield in 2005 as Professor in Restorative Dentistry. He has published over 50 peer reviewed articles on Periodontology ranging from clinical trials, epidemiology, and diagnosis particularly with the use of crevicular fluid as a diagnostic tool to assess risk or to monitor the stages of the healing process. His current research focuses on patients with Aggressive forms of Peiodontitis and has two arms to it; 1) genetic susceptibility and host bacterial interactions. 2) therapeutic approaches aimed at controlling infection and repairing the resultant destruction.