Researchers find new proteins

Researchers have found two new proteins that may lead to more effective treatment of endocarditis and infections associated with implants.

Endocarditis is an inflammation of the heart valves that can be life threatening and streptococcus gordonii, a bacterium that exists in the mouth, is one of the bacteria that cause the disease.

To survive in the oral cavity, the bacteria must be able to attach to a surface such as the mucous membrane.

This is done with the help of proteins.

In the mid-1990s, one of these proteins from S. gordonii was identified by a research team in England.

Most antibiotics initially work extremely well, killing more than 99.9 per cent of microbes they target.

But through mutation and the selection pressure exerted by the antibiotic, a few bacterial cells inevitably manage to survive, repopulate the bacterial community, and flourish as antibiotic-resistant strains.

Professor Vern L Schramm and Ruth Merns, chair of Biochemistry at Einstein and senior author of Transition State Analogs of 5’-Methylthioadenosine Nucleosidase Disrupt Quorum Sensing’ tested three transition state analogs against the quorum-sensing pathway.

All three compounds were highly potent in disrupting quorum sensing in both V cholerae and E. coli 0157:H7.

‘In our lab, we call these agents everlasting antibiotics,’ said Dr Schramm.

This study involved three compounds, but Dr Schramm said that his team has now developed more than 20 potent MTAN inhibitors, all of which are expected to be safe for human use.

Once the bacteria are encapsulated, it is extremely difficult to get rid of them.

But with enhanced knowledge of how bacteria fasten to surfaces, it will be easier to find effective strategies to treat biofilm-induced diseases.

‘If we can block this binding with the help of drugs, treatment will be more effective,’ said Professor Davies.

Previously, researchers knew that bacteria that grow in biofilms alter their properties when they settle on a surface.

For example, they become more resistant to antibiotics and antibacterial compounds. These researchers are now investigating how this resistance arises.

Oral osteoporosis drugs

Even short-term use of common oral osteoporosis drugs may leave the jaw vulnerable to devastating necrosis, claim researchers.

Researchers at the University Of Southern California, School Of Dentistry have released results of clinical data that links oral bisphosphonates to increased jaw necrosis.

The study is among the first to acknowledge that even short-term use of common oral osteoporosis drugs may leave the jaw vulnerable to devastating necrosis, according to the report in the Journal of the American Dental Association (JADA).

‘Oral Bisphosphonate Use and the Prevalence of Osteonecrosis of the Jaw: An Institutional Inquiry’ is the first large institutional study in America to investigate the relationship between oral bisphosphonate use and jaw bone death, said principal investigator Parish Sedghizadeh, assistant professor of clinical dentistry with the USC School of Dentistry.

After controlling for referral bias, nine of 208 healthy School of Dentistry patients who take or have taken Fosamax, the most widely prescribed oral bisphosphonate, for any length of time were diagnosed with osteonecrosis of the jaw (ONJ).

The study’s results are in contrast to drug makers’ prior assertions that bisphosphonate-related ONJ risk is only noticeable with intravenous use of the drugs, not oral usage.

Professor Sedghizadeh said. ‘We’ve been told that the risk with oral bisphosphonates is negligible, but four percent is not negligible.’

He hopes that other researchers will confirm his findings and thus encourage more doctors and dentists to talk with patients about the oral health risks associated with the widely used drugs.

The results confirm the suspicions of many in the oral health field.

He said: ‘Here at the School of Dentistry we’re getting two or three new patients a week that have bisphosphonate-related ONJ and I know we’re not the only ones seeing it.’

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Thinking outside the box

Cardiovascular disease represents one of the leading causes of death in the Western world. With this in mind, it is interesting to note that the American Academy of Periodontology has just informed its members of its new clinical recommendations developed in regards to arteriosclerotic cardiovascular diseases, which has been published in the American Journal of Cardiology.

The American periodontologists presented scientific evidence which convinced the cardiologists that they needed to alert their fellow colleagues that they were able to help reduce the risk of cardiovascular diseases in patients suffering from periodontitis.

An important relationship
Here I will make a few points on the interrelation between periodontal and cardiovascular diseases.

Dental plaque may become colonised by periodontal pathogens such as:
- Porphyromonas gingivalis;
- Campylobacter rectus;
- Fusobacterium nucleatum;
- Bacteroides forsythus;
- Prevotella intermedia;
- Actinobacillus actinomycetemcomitans, for example.

Bacteria and its products reaching the gingival tissue stimulate inflammatory response leading to infiltration of neutrophils, lymphocytes, macrophages and mast cells. Inflammation is a well-established determinant for cardiovascular and periodontal disease.

Beside the inflammatory response an immune response of the body will be as well induced.

Interleukin-1 gene polymorphisms has been identified as a ‘candidate that influences inflammation’.

Genetics today describes three IL-1 genes located on human chromosome 2q13. Polymorphism in these genes are described in different levels in patients suffering from periodontitis.

In their paper called, Elevation of systemic Markers Related to Cardiovascular Diseases in the Peripherial Blood of Periodontitis Patients, Loos et al. (Jifp 2000) proved that: ‘Periodontitis results in higher systemic levels of CRP (C-Reactive Protein), IL-6, and neutrophils.’

In his reappraisal on the topic of inflammation and periodontal diseases, Van Dyke draws the public’s attention to the following:

‘Inflammatory mechanisms appear to be critical factors in the development and progression of most of the chronic diseases in aging.’

‘Diet and genetic variations interact to control differences in inflammation among individuals. Inflammation is actively resolved by specific mechanisms that help to restore homeostasis, and through which treatments may be designed.’

‘Although our genes do not change, the control of how our genes are expressed in specific tissues can change substantially throughout our lives by factors such as diet, stress, and bacterial accumulation.’

‘Visceral fat accumulations around one’s waist substantially increase the inflammatory burden on the body.’

‘Over expression of inflammation may be one of the key aspects that influences and links different diseases in different individuals.’

Discussion
Patient education as well as inter-professional communication (‘thinking outside the box’) may help save our patients lives, and if they came trustfully into our dental practices it is our responsibility to help, advise and protect their health and life.

Conclusion
Continuing professional education today should contain understanding for infection, inflammation and its consequences for the human body.

Dentistry can help and prevent dramatic cardiovascular diseases.
Perio disease – risk factors

It’s more likely a combination of factors than one single one that makes a person susceptible to periodontal disease. Fiona Clarke explains

Effective periodontal treatment in practice involves a series of stages; firstly understanding the aetiology and pathogenesis of the disease, early and accurate diagnosis, followed by effective treatment with due consideration of all the associated risk factors. This needs to be followed by timely reassessment and continued periodontal maintenance and monitoring.

When considering the aetiology of a complex disease like periodontitis, we need to consider both the direct cause and factors associated with cause. Within this, we can define two components; risk factors and susceptibility factors. A risk factor is defined as any environmental or behavioural characteristic of an individual that increases the probability of developing a disease. Risk factors such as smoking and poor oral hygiene are modifiable. In contrast, susceptibility factors (often called risk determinants) such as age, gender and genetic make-up cannot be modified.

It is now accepted that bacteria are essential, but insufficient for the development of periodontitis. A susceptible host is a prerequisite. Periodontitis is primarily driven by a bacterial challenge, but a complex interplay exists between the oral bacteria and host which is influenced by a range of risk factors.

Periodontitis is thus described today as a complex disease having a multifactorial aetiology and although bacteria are still believed to be the primary aetiological agents in periodontitis, often the amount of plaque present does not fully explain the observed severity of the disease. Some patients experience severe periodontal breakdown despite low levels of plaque and conversely other patients have little destructive disease despite widespread build-up of plaque.

So if poor oral hygiene alone cannot account for severe destructive periodontal disease and everyone is not equally prone to the disease, the question then becomes what makes some individuals more susceptible than others. Risk factors which have been reported to be associated with increased susceptibility to periodontitis include the specific bacteria within the microflora, smoking, systemic disease, behavioural and psychosocial factors such as negative life events and stress (figure 1).

Much research has focused around trying to better understand the microbial aetiology of the disease. In the mid 20th century, it was believed that all bacterial species found in plaque were equally capable of causing disease and that periodontitis was the result of cumulative exposure to dental plaque.

This non-specific plaque hypothesis assumed it was the elaboration of noxious products by the entire plaque flora that resulted in destructive disease. Clinical trials have documented the importance of controlling the microbial plaque in the treatment of gingivitis and periodontitis. The association of specific bacterial species generally harmful to the host, but the host response to this attack may be protective or destructive. The varying balance between harmful and beneficial interactions of the host accounts for the wide variety of patterns of tissue changes in patients. It is believed that bacteria can however only account for about 20 % of this variance in disease and current evidence indicates that most destruction of the periodontium is host mediated.

Systemic disease

Systemic disease can adversely effect host defence systems and therefore act as a risk factor for both gingivitis and periodontitis. Depressed neutrophil number and function (as in neutropenia, Chédiak-Higashi syndrome, Down’s syndrome and Papillon-Lefèvre syndrome) are associated with severe periodontitis. Diabetes mellitus is one of the strongest systemic risk factors for periodontitis and studies show that diabetic patients are at increased risk of periodontitis, in particular those with poor oral hygiene or poor diabetic control.

Despite identification of many of the genes responsible for most of the syndromic forms of periodontitis this has not shed much light on improving understanding of the pathogenesis of the more common types of periodontal disease. Although there has been a great deal of interest in the association between the risk of periodontitis and systemic diseases it still remains unclear to what extent common systemic disease may affect the severity and progression of periodontitis.

Smoking

A positive association between smoking and periodontal disease has been reported in both cross sectional and longitudinal studies. In the past few years increased attention has been paid to this relationship, and smoking is believed to be a significant factor in the development and progression of the disease. In studies in which plaque levels were adjusted between smokers and non-smokers, greater probing depths, clinical attachment loss and bone loss have been reported in smokers.

It has been found that there is reduced gingival inflammation and bleeding in patients who smoke. This may be explained by the fact that nicotine exerts local vasoconstriction reducing blood flow, oedema and clinical signs of inflammation. Smokers are believed to be between two and six times more likely to have severe periodontitis than non-smokers. Several studies have demonstrated that the severity of periodontal disease appears to be related to the duration of tobacco use and amount of daily tobacco consumption.

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intake. Of clinical significance is the fact that although smokers benefit from periodontal therapy, clinical improvements are less than those for non-smoking patients.

It is encouraging to note that clinical studies demonstrate periodontal disease progression slows in patients who quit smoking and that these individuals have a similar response to periodontal therapy as non-smokers. Thus smoking cessation advice and support should be as important in our management of patients as our improvement in the patient oral hygiene and we should acknowledge the important role we have in highlighting this issue to our patients.

Psychosocial factors
There appears to be an association between periodontal disease and stress. Socially determined behaviours and responses to life circumstances are thought to affect the immune system and thereby health and periodontal disease. Psychosocial factors lead to changes in oral habits and in behavioral responses, such as poor oral hygiene and smoking, and the host’s response to environmental determinants such as stress. Studies suggest stress is a significant risk indicator for periodontal disease and that the impact of negative life events, number of negative life events and being unemployed are all significantly associated with periodontitis and should not be underestimated.

Genetics
The view that genetic factors influence periodontal disease is not a recent one. In 1950, after reviewing the periodontal status of several families, Denny concluded that susceptibility to periodontal disease is probably heritable. The present theory of disease susceptibility is that it depends upon the presence of a critical number of one or more pathogenic bacteria in a susceptible host. Studies have shown that the number and type of bacteria required to exceed an individual’s critical disease threshold defines host susceptibility, and that this susceptibility is influenced by a number of factors, including genetics.

Initial attempts to define risk factors for periodontal disease have focused primarily on bacteriological and immunological parameters while significantly less effort has been directed at defining host genetic factors. Studies on periodontal disease incidence in humans have shown that genetics do not explain population variance in the incidence of P. gingivalis or of P. intermedia, two oral Gram-negative bacteria associated with periodontal disease. We also know that P. gingivalis has been demonstrated in many studies to be correlated with periodontitis yet not all individuals are equally prone to bone resorption when they are infected with this bacteria.

Thus, it has been suggested that genetic factors may have more influence on host response to infection than on bacterial colonisation. In order to better understand, classify and ultimately manage the disease it would be useful if we understood how genetic variation in host response could explain the differences which can be observed in disease progression.

We are now beginning to understand that it is more likely a combination of risk factors that predispose a patient to periodontitis and not a single factor and so consideration of all associated risk factors involved in a particular patient’s condition should be considered before treatment commences.

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Dr Fiona Clarke
graduated from WITS Dental School, South Africa and completed an MSc and PhD at Barts and The London School of Medicine and Dentistry with a special interest in genetic risk factors for periodontal disease. She’s currently working as a clinical peri tutor at Guy’s Hospital and in private practice. Teaching interests include periodontology, local anaesthesia and the use of e-learning in dental education.
Keeping up appearances
Hygienist Leah Beckman looks at the long-term compliance and maintenance of dental implants

Today anyone can achieve permanent tooth replacement. Dental implants are the standard of care for patients presenting with lost or missing teeth. As dentistry moves forward in leaps and bounds, the dental care professional must provide the patient as well as other colleagues with the knowledge to be able to maintain these implants.

The most important factor is the regularity and frequency of homecare and the proper use of the appropriate hygiene aids. The patient must also be dedicated to excellent homecare and to maintain regular recare appointments with the dental hygienist at decided intervals usually every three months. At these recare visits, the dental hygienist must provide motivation for the long-term compliance and maintenance of the implants. Good dental health begins with superior dental hygiene.

Regular maintenance
A dental hygienist has the role of providing regular maintenance and professional care of dental implants. The area most prone to damage is the interproximal papilla. This mainly results from the fact that it is an area that is difficult to access and keep plaque free.

First, assess the area with a periodontal screening. The interproximal papilla around an implant should have the same characteristics as a papilla around a natural tooth. The tissue should be firm, pink in color and plaque free. There should be no swelling or bleeding. There should be no evidence of vertical bone loss upon radiographic examination and no mobility.

The dental hygienist must probe and scale the implant using only titanium or carbon fiber instruments. Metal curettes and probes can scratch the surface of the implant causing increased plaque retention. Today there are many options for using ultrasonic tips with a Teflon coating such as the piezoelectric tip from NSK. This tip is autoclavable and fits into the piezo headpiece.

Patient education
The role the dental hygienist plays in patient education is of the utmost importance. Without proper patient education, the proper daily homecare cannot be achieved. The dental hygienist must provide the patient with tangible results at each professional prophylaxis appointment that will motivate the patient’s compliance at home.

Homecare starts with the appropriate hygiene aids and the instruction on how to use them correctly. The use of a sonic toothbrush is highly recommended.

Nothing replaces floss
An implant patient should always use special floss with a thicker foam coating. Again, instructing the patient on how to adapt the floss around the implant and under the gingiva main priority.

Ask the patient if they have ever been instructed on how to properly adapt floss and especially teach them how to clean the interproximal areas around the implant.

Interdental brushes are a wonderful aid as well. However, only those with a plastic coating over the metal wire can be used.

About the author
Leah Beckman is a registered hygienist with 12 years’ experience in the dental field.

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Panoramic radiography changed the paradigm of diagnosis when introduced in the early 1960s. The limitations of two-dimensional radiography are:
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Due to this the use is and was limited.

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Cone Beam CT the change of paradigm in modern dentistry – clinical applications in endodontics and periodontology

By Prof. Dr. Liviu Steier

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Perio Tribune
DENTAL TRIBUNE United Kingdom Edition • August 7–13, 2009
The author has resumed this article for the purpose of demonstration how CBCT aided tremendous value to routine dental practice.

1. Use of CBCT in endodontics

2. CBCT in periodontics

2.1 CBCT and soft tissue

In 2008, Januario et al. published in the Journal of Esthetic & Restorative Dentistry (J Esthet Restor Dent 20: 566-574, 2008) a paper called: ‘Soft Tissue Cone Beam Computed Tomography: A Novel Method for the Measurement of Gingival Tissue and the Dimensions of the Dentogingival Unit’. In this paper, the authors described a simple method to diagnose the thickness of the gingiva specially in the anterior aesthetic zone. The scans were performed with an iCAT (Imaging Science International, Inc., Hatfield, PA, USA). The authors positioned the subject for the scan wearing a plastic lip retractor.

A 28-year-old female patient was referred to our practice for evaluation and treatment planning of the periodontal status. No special remarks regarding medical or dental history. The patient has undergone orthodontic over a couple of years.

The patient was referred for the completion of the diagnostic to take a CBCT at CTdent (2 Devonshire Place, W1G 6HJ, London, see also www.ct-dent.co.uk).

The CBCT confirmed the preliminary diagnosis.

A treatment plan has been elaborated.

2.2 CBCT and hard tissue

Vandenberghe and coworkers researched periodontal bone architecture using 2D CCD and 3D full-volume CBCT-based imaging modalities.

Their investigation concluded that CBCT offered a significant benefit over conventional radiography.

The authors concluded that CBCT can be used to diagnose the bony support as well as surrounding soft tissue and may reveal valuable informations for
example regarding furcation involvement.

A 53 year old human patient was referred to our practice for evaluation, treatment planning and execution. Of major concern was the first upper molars. After performing the routine diagnostic approaches such as BOP, periodontal probing, etc, the patient was referred to CTdent for a CBCT.

Summary

Information provided by this modern technology represents an invaluable milestone in diagnostic, treatment planning as well as evaluation of treatment outcome especially for periodontal applications, especially in the areas of intrabony defects, dehiscence and fenestration defects, and periodontal cysts, and in the diagnosis of furcation-involved molars.

Conclusion

1. For periodontology, CBCT proves to be superior to 2D imaging for the visualisation of bone topography and lesion architecture as well as for the covering soft tissue.

2. For endodontics CBCT seems to be the most promising applications for diagnosis, treatment planning and treatment evaluation.

CBCT images and 3D reconstructions allow for visualisation and exact measurement of dimensions.

Diagnosis built on the combination of clinics and CBCT are a reliable aid in planning and execution of simple as well as advanced dental procedures.

References are available on request.
Protecting the root

Prof. Dr. Liviu Steier outlines how best to prevent gingival recession

Root surface exposure as a consequence of gingival recession is a challenge for the dental practitioner. Over the past decade, many procedures have been introduced and presented to prevent and/or treat this complex phenomenon. Most of the treatment approaches consist of mucogingival graft techniques.

Variation in gingival thickness (GTH) has been related to different periodontal ‘biotypes’ (Seibert & Lindhe 1989):

- Thick – flat biotype (quadratic teeth with a broad zone of keratinised tissue)
- Thin – scalloped biotype (slender teeth with a narrow zone of keratinised tissue)

Gingival thickness not only interferes with dental procedures but can advance if left untreated. Among the most common found clinical manifestations are:

- Tooth sensitivity.
- Long tooth appearance

Gingival biotype and natural teeth

As a consequence of this thin biotype, gingiva can recede during life. It is not often that clinical situations like the one below can be seen in practice. Treatment which doesn’t address the primary disease was performed using adhesive filling material to reduce tooth sensitivity and mask esthetics. With time, restorations have to grow and an unpleasant image occurs.

Gingival biotype and prosthetics

Ericsson & Lindhe demonstrated in an experimental study (1984) performed on beagle dogs, that once metallic strips were inserted subgingivally, recession was more likely to appear in areas with thin gingival architecture.

Gingival biotype and surgical endodontics

It is a widely accepted clinical impression that a thin, highly-scalloped gingiva tends to recede from source of irritation, for example, an artificial crown. Margin or microbial irritants (Seibert & Lindhe 1989), and gingival recession often occurs following traumatic, or surgical injury (Claffey & Shanley 1986).

Gingival biotype and implant therapy in the esthetic zone

Subgingival elements covered by a thick – gingival biotype assure a predictable esthetical outcome lowering the treatment risks.

The thin – gingival thickness biotype is associated with the risk of recession and may lead to esthetical insufficiency.

Müller et al. (2007) demonstrated that subjects with a thin periodontal phenotype have also relatively thin palatal mucosa not very suitable for harvesting connective tissue grafts. As a consequence, the use of ‘acellular dermal matrix’ derived from donated human skin (AlloDerm – BioHorizons) may be the only treatment alternative in cases of thin gingival biotype.

Soft-tissue grafting

Soft-tissue grafting is performed for different reasons:

1. Changing of the natural gingival biotype by augmentation → a preventive approach.
2. Root coverage → a curative approach in ready installed disease.

Clinical picture showing a ‘dark margin’ of a PFM crown in a thin gingiva biotype patient having multiple recessions.

Clinical picture demonstrating gingival recession around an implant.

Post-operative pictures

Alloderm package

Swann Morton surgical blade used for the cervical incision

Tunnel periosteum used to raise the flap

Subperiosteal flap

Clinical picture demonstrating the ‘Treatment Algorithm for Gingival Recession’ (modification of the UCLA approach).
Alloderm is an acellular dermal matrix derived from donated human skin. The donor material is deprived of the epidermis and immunogenic cells. It undergoes a final freeze drying and an extensive panel of serology tests and a sterilisation process.

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It has been demonstrated that due to retained vascular channels the patient's blood infiltration is facilitated and accelerated - revascularisation can start as early as one week after implantation.

Clinical case
A 28-year-old male was referred for the management and prevention of recession. The medical history was uncomplicated with no special recording in the dental history.

The clinical dental examination proved:
• Thin gingiva biotype
• Temperature sensitivity
• Gingival recessions with tendency to expansion
• There were no signs of inflammation or ulceration.

The initial management was conservative and consisted in cleaning and hygiene instruction. Follow-up a few weeks later showed a clear improvement in oral hygiene. The patient was explained treatment goals and different available treatment options.

The selected and agreed treatment plan was two-fold:
• First to thicken the gingiva using graft technique (AlloDerm)
• At a later stage for complete root coverage a ‘coronally repositioned flap’ was obtained.

For more information, contact BioHorizons
Customer Care: 01344 752560
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Step-by-step treatment

Preparing Alloderm for use:
The package is opened and the graft is dropped into a sterile saline bath, where it is kept for 30 minutes. The paper back is removed from the graft. It is important to identify the two sides of the graft. The basement of the membrane is rough and does not absorb blood, while the dermal side does.

Preparation of the recipient side
Adequate anesthesia is administered – usually infiltration will suffice.

The surface of the root is scaled and planed. Papillary incisions are made using a Swann Morton blade to a depth of 3-4 mm apically. The papilla is left intact to prevent flap retraction and improve blood supply to the graft during the healing phase.

To lift the tunnelled papillae, a microsurgical papilla elevator is used and a mucoperiosteal pouch (past the mucogingival junction) is created.

The Alloderm graft is cut to fit from the distal of the canine to the mesial of the molar. The graft is inserted into the pouch preparation under the intact papilla using the micro papilla elevator. The graft is then positioned with the connective tissue side facing the bone.

The margins of the graft are fixed to the tooth with Histoacryl glue. No suture needed.

The patient was instructed not to brush the area for six weeks, to start rinsing the mouth with saline solution for seven days. After seven days, disinfection was performed with oral rinse with CHX solution. The patient was seen for postsurgical check up after two days, seven days and six weeks.

After six weeks, a thorough cleaning was performed and the patient instructed to restart regular hygiene. Healing was re-evaluated after three months and the next treatment step scheduled.

Conclusion
Early diagnosis and preventive regenerative periodontal treatment can avoid long-term gingival recession. Patients with a thin gingiva biotype will also lack adequate connective tissue in the palate for transplant. The use of donated human tissue represents a viable alternative technique.

References are available on request.

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