

Dr Jörg Strate invited discussion about the presented papers:

Dr Philip Preshaw started by stating that he had seen a lot of patients with rampant caries and lots of plaque but not much periodontal disease, and conversely, a lot of patients with aggressive or juvenile periodontal disease who did not have much in the way of caries – bearing in mind that the caries bacteria favour a more acidic environment compared with periodontal bacteria – so he wondered if caries could sometimes be somehow protective against periodontal disease by altering the microenvironment so that the periodontal species don't flourish. Professor Philip Marsh replied that it was an interesting observation and suggested that clearly at a very superficial level, the type of environment that favours caries bacteria is very inhibitory to many of the Gram-negative anaerobic pathogens, so there could well be kind of a lifestyle change which is making the plaque much more full of cariogenic organisms which would exclude or reduce the numbers of periodontal ones. Superficial, because there's obviously a lot of other risk factors that might determine whether one suffers from rampant periodontal disease and there's a lot of dietary issues as well, but he thought that that this sort of observation is an interesting one and could well be explained by the way suggested by Dr Preshaw.

Professor Hubert Newman stated 'half tongue in cheek', that as it is now well over 30 years since the work began on the true nature of the ecological relationship between plaque and caries, and plaque and chronic inflammatory periodontal diseases, so he wondered whether 'our transatlantic cousins, the Europeans and others' will now begin to listen to these basic ecological things. Professor Newman explained that when he has attended ecological conferences with true microbial ecologists, they are very surprised that we have not taken these lessons on board which are common in agriculture, in plant research, in all kinds of other fields.

Professor Marsh said that the simple answer to Professor Newman's question was that there has been a change. He explained that one sees far more papers from North America, where the term ecological is used in the title, so he believes there is a recognition and hope that the field might have come

up with some simple answers in terms of critical organisms that may be responsible for disease vanishing as fast as the discovery of new organisms increase the complexity. Professor Marsh also sees that we are looking at a much more complex environment and the bacteria seen in plaque are a consequence of other factors which could encompass in ecological thoughts. He believed that the papers Professor Newman wrote in the seventies where he was proposing ecology were at the wrong time – were too early for people – who were hoping for a specific plaque hypothesis, but that the concept of ecology is gaining a lot of ground. Professor Michael Noak added a question in the same field, and asked how Professor Marsh explained the clinical occurrence of subgingival caries in cases with inflamed periodontal sites without taking the immunological aspect into the model. Professor Marsh explained that he did not have a simple answer to that question but reiterated that at the end of his talk he did focus on taking an holistic approach and that the organisms that are found are a consequence of all sorts of factors, including the host defences, and that the host is critical in deciding whether there will be disease or not. In certain organisms, in certain situations these won't cause disease, but in a weakened host they may well cause disease. So he thought that one does have to take into consideration both the hosts' immunity and other lifestyle factors to understand this relationship, it's not just the bacteria.

Dr Peter Floyd was very interested to see Ian Chapple's last slide from the *Daily Mail*, because he felt that when one reads newspapers they sell us products and ideas, and the *Daily Mail* as well as other newspapers, will promote the idea, for example that when people have gastrointestinal upsets they are offered probiotics to rebalance the gut flora. He asked therefore if there is any evidence that probiotics may have a role in the treatment of periodontal disease.

Professor Ian Chapple deferred to Professor Marsh on this but suggested that his understanding was no, he did not believe there is any evidence whatsoever, but quipped that Philip would probably just say the opposite.

Professor Marsh stated that he had not seen any evidence in periodontal disease. There are attempts to produce probiotics

for dental caries, so people in Scandinavia have looked at lactobacillus which is found in dairy products and introduced it, and there is some evidence that shows benefit and a very weak effect. In the United States, people are looking at having genetically modified mutans streptococci, which could exclude natural mutans streptococci strains. These bacteria have been modified so they don't make lactic acid, and they also produce a natural inhibitor that would inhibit wild-type mutans streptococci strains, but Professor Marsh has not seen the equivalent in periodontal disease. In that area, a more likely approach would be prebiotics, where one can introduce a substrate that would favour the growth of a favourable bacteria. So in gut microbiology there are various complex carbohydrates with which one can supplement your diet that are supposed to promote the growth of bifido bacteria and lactobacilli, which are considered to be favourable. He believes that there is a huge opportunity to identify products that would favour the growth of beneficial organisms at the expense of the so-called pathogens. Something that isn't really a probiotic, but there is data to show that some of our natural bacteria, like the *Streptococcus sanguinis* and related organisms, produce hydrogen peroxide naturally which can inhibit the periodontal anaerobes, and some people have shown an inverse relationship between higher levels of these bacteria and lower levels of the anaerobes, and vice versa. There are some possibilities in that field, but it may be more in the idea of prebiotics rather than probiotics. Dr Jörg Strate posed a question to Professor Ubele van der Velden about the 'beautiful parameter' in his presentation which apparently no longer holds true anymore. He questioned – not even thinking about the commercial side of that at this moment, but in the clinical environment – what would be the kind of parameter and risk assessment on which he would base his individual recommendation for an oral hygiene regime for specific patients? He asked if dentists have to do this risk assessment, because as was shown in the experimental gingivitis example, different patients react in different ways, which could eventually result in a very individual regime, because a certain patient will only necessitate a certain kind of oral hygiene to stay healthy. Professor van der Velden agreed that that was true and he thought that the degree of gingival inflammation was presently the most useful parameter. His problem with this was that nowadays dental professionals have shifted from the gingival indices towards the bleeding problem to the bottom of the pocket, and that may not necessarily be the same, so he believes we need to re-investigate which type should be used to determine the quality, because the study using the gingival indices showed a predicted value. He used bleeding on probing to the bottom of the pocket in his Indo-

nesian study, and there it could not be shown that the bleeding on probing was a risk factor. So it may be a combination which would help, but he thought the gingival indices are very crude indices, and that a more detailed look into these evaluations is needed.

Dr Strate invited questions from the audience after the second set of presentations:

Marjolijn Hovius questioned Dr Arthur Hefti about preterm births or miscarried babies due to mothers with periodontitis, but stated that the figures do not take into account access to prenatal care. Ms Hovius described a scenario in the United States where many women have no access to prenatal care either because they live in rural areas or they cannot afford it, because there are a large number of very poor women in the United States. In addition, there are fewer gynaecologists because most medical students do not want to take up gynaecology due to an increase in lawsuits. Yet nobody takes this into account when compiling statistics and one could imagine that a lot of problems exist due to other circumstances which nobody looks at.

Dr Hefti agreed with Ms Hovius that lack of access to care is one of the most important reasons for preterm births. The particular study which was referenced in the presentation was not executed in an area of low or no access to care, and that was one of the reasons why it was so difficult to enrol subjects into the study. Philips Oral Healthcare is currently supporting a health programme in the Appalachians area which is also investigating that question amongst women with very, very low access to any care, and hopefully in about 4 years from now the results will be produced. But many, many studies up until now have actually just ignored this very important issue. Professor van der Velden also referred to studies about preterm low birth and stated that he thought it would be important to give the data on the extent of each subjects' periodontal disease. He questioned that because selection criteria were minimal, what was the percentage of really severe periodontitis cases in this study? For instance how many had at least at two sites of 5 mm pocket depths or more, in conjunction with 6 mm attachment loss at two non-related teeth, because if too many subjects are included who have minimal disease, then one can fade away all the possibilities for the effect in the others? Dr Hefti responded that it is true that this particular population really didn't show a lot of periodontal disease, maybe a form of periodontal disease called gingivitis, but there is an inherent difficulty in executing such studies in a study population aged between 18 and maybe 29, 30, 35 years old, where there is not a lot of periodontitis to find,

so one has to reduce the enrolment to a quantity that can be absolutely neglected. He agreed that in future better plans need to be developed.

Subsequently another questioner referred Dr Hefti to the same 'excellent study', but was curious to learn about the study designer's arguments about the selection of the two treatment groups which had the treatment group undergoing scaling and root planing together with the Sonicare homecare treatment whilst a second group had a delayed periodontal treatment plus a manual toothbrush. The questioner did not understand this design as he assumed that if you really would like to know the influence of the Sonicare then you provide scaling and root planing for both groups and just change the brushes. If one is interested in the intervention of the periodontal therapy then one would assume that you maintain or keep the same brush.

Dr Hefti commented that the study was not designed to look at the effect of the Sonicare toothbrush, it was truly an interventional study conducted as a pilot study for a National Institutes of Health grant application, and it is currently ongoing as a multicentre study. Male Questioner: But am I wrong if I want to know the influence of the toothbrush, then I would make the scaling and root planing for both groups immediately and then just follow up the brushes? Dr Hefti: Well you're absolutely right that would be the appropriate design, however, it's predictable that the effect of mere tooth brushing versus scaling and root planing is probably hard to discover.

Another questioner referred to Professor Jörg Meyle's 'thrilling closing talk' which he believed was excellent and really tied together the 'Sense and Simplicity' presentation at the opening of the Symposium. However, though Professor Meyle showed the probe, the questioner thought that it really made no sense to use a graded steel stick to measure inflammatory disease however simple, and suggested that this shows the need for collaboration with industries like Philips which could move us forward.

Dr Strate thanked all the speakers and closed this year's 'Emerging trends in oral healthcare'. He added that he was really impressed with what the symposium achieved over a period of one and a half days, though it certainly has not made life easier for Philips Oral Healthcare. He mused that the speakers and questioners had destroyed the 'beautiful world a little bit', as they did not really come to straightforward conclusions, adding that 'my beautiful digital world of right or wrong doesn't exist anymore'.

Dr Strate told delegates that they would be given a new product which Philips Oral Healthcare was launching within a few weeks

of the symposium. The e9000 is a sophisticated oral care device, which has been proven to show superior plaque removal and it has been shown to help prevent and reverse gingivitis. After what was heard during the meeting, Dr Strate questioned whether Philips Oral Healthcare was really addressing the right questions, adding, 'Is it the plaque removal that we have to prove for all and every population we are talking about, or as we have learnt from Professor Ubele van der Velden do we have to look a lot more specifically at what kind of need and clinical situation we should be addressing, in which specific way depending on the individual we are talking about?' So when Professor Meyle raised the provocative question of 'Is periodontal disease life threatening?' Dr Strate had hoped the answer would be yes it is life threatening, because then he could have told delegates that Philips Oral Healthcare could provide a life-saving device, as long as it was agreed that gingivitis is a periodontal disease. Because as such agreement was not reached, this argument does not work anymore, so yes Philips Oral Healthcare is currently able to provide products that, given our current knowledge and our ethical and scientific ambitions as a business, do provide the benefits to patients and help them to maintain oral health, and after these discussions at least it can be assumed that these products are capable of supporting and maintaining overall health to whichever extent we will get more evidence that gives even stronger support to this hypothesis. There is a lot to do in the future however judging by the strong attendance from Philips during the meeting one can see that the Company is very serious about taking this message home and working on what we have heard here from tomorrow. Our team at the symposium includes the Head of Clinical Research, Senior Management from Marketing – so both business people and technical people – all of whom were listening closely because the Company really does try to develop a better understanding what the oral care device of the future has to look like. Which will then, perhaps not only address the oral care situation of individuals better, but will take into consideration all the knowledge that we already have and will gain on the systemic links and the oral situation.

Dr Strate concluded 'We did not expect an easy discussion because if you invite such a high-level panel of speakers and this kind of audience you are going to end up with the type of discussion we have had, however, I believe we all deserve this kind of discussion, because that's the kind of interaction and communication that moves us forward'. He finished by thanking everyone for being there and hoped to see them again next year 'when the challenge will be to bring together a group like this again and to come up with a stream of topics which actually fits as neatly as I believe we all experienced here today'.

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