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The significance of supragingival plaque accumulation in periodontal disease

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I would like to speak about this very simple phrase: supragingival plaque. You know getting older sometimes makes life more difficult, but it's also sometimes more easy. Sometimes you start to realize that something in which you have believed for decades, may be interpreted differently, because you are raised in a way during your education which is dominated by the knowledge of that particular time. So I was not aware that when I was studying dentistry in the late 1960s that everything about dental plaque and the relationship between dental plaque and periodontal diseases was that new, it was only a recent finding. So when you look back into the literature, the epidemiology of periodontal diseases does not include plaque at all. So if you look to these studies, the very early studies in which they look for the epidemiology of diseases in the clinical way and in the radiographic way plaque was not included. But what you saw from all these studies that with increasing age there is an increasing prevalence and severity of periodontal disease. And of course there were numerous people who were advocating tooth brushing more than a century ago, but still that was not considered a very accepted field.

Gradually periodontology emerged more and more in science, and in 1956 the Russell Periodontal Index was introduced (1), but still it did not include dental plaque, and it was only in 1959 – that was 6 years before I started my studies – that Ramfjord (2) was the first to include dental plaque in his periodontal disease index system. Even the group in Norway headed by Jens Waerhaug, only talked in those days about poor, fair and good oral hygiene, not in terms of indexes and strict criteria. So what you see here is that it was only in the late 1950s and the first half of the 1960s that plaque indexes were developed, so 1964 – that was only just before I started my dental training – that studies showed that with increasing age and increasing oral

hygiene index you have increasing periodontal disease. That's shown throughout the decades; it has been shown in the 1950s, in the 1960s, in the 1970s, in the 1980s and I think if you would just do a study like this once again you will find the same results.

Then our famous studies on experimental gingivitis were published. They were the keystone for the relationship between dental plaque and periodontal disease. They made people very much realize that yes there is a cause-and-effect relationship between plaque and gingivitis and what we must do is to get rid of the dental plaque. So all the studies were relating plaque and periodontal disease, plaque and gingivitis, and it ended up I think in this beautiful scheme of Aubrey Sheiham in 1972 (3) in which he showed that if you have low plaque scores, you have almost no periodontal tissue loss when you are 20 and you will keep your teeth, but if you already have high plaque scores when you are 20 years of age and there is already some loss of periodontal tissue, by 50 you will have lost your teeth due to periodontal disease. I think that this is the accumulation of all the knowledge which has been obtained during those decades, and this, I think also forms the basis on which we developed our dental hygiene schools in Holland. Aubrey Sheiham also said in those days that if you are able to reduce the mean plaque score in the population you will reduce periodontal disease.

Based on this view, there came a number of studies from Scandinavia which showed that for successful treatment, the control of plaque is essential, and if you want to have good results after initial periodontal therapy or after surgery, you must have a proper plaque control. So, being raised in the era of supragingival plaque and experimental gingivitis that dominated in the 1970s, especially all the results from the Gothenburg Group, in my mind dental plaque is the key issue of aetiology and treatment of periodontal diseases, but sometimes you start to doubt is it true, is it really true? I remember that in a conference in the late 1970s that Sture Nyman and Jan Lindhe were presenting a course and then all of a sudden there was a French dentist who told us that for several years he did what they were advocating and in the majority of his patients it worked, but he had a number of cases where it did not work at all.

So is it really true what we are saying, does plaque cause periodontal disease or is it the other way round? Is periodontal disease causing the presence of plaque? And the first time I started to doubt this was a study published in 1984 by Burmeister and coworkers (4), where they were studying the clinical features of localized and generalized juvenile periodontitis. I was also brought up with the idea that in juvenile periodontitis there is a discrepancy between the amount of plaque and irritants and the amount of destruction, but what they showed was that if you look into the affected and unaffected sites in these patients that at the affected sites there is a lot of plaque, there is a lot of inflammation and there is a lot of bleeding. So you may be misled because there are only three or four sites in the mouth which have severe destruction but the other sites are healthy, and you look into the mouth and it just looks very healthy but if you look very carefully at the local sites it may not be true.

The relationship between plaque and gingivitis has also been studied in more experimental ways by looking at how new plaque develops. The results of these studies were that *de novo* plaque formation varies greatly between individuals; some people develop it very quickly and others develop it very slowly.

In the 1980s we carried out a number of experimental gingivitis studies where individuals developed gingivitis very slow or rapidly (5). For instance, I had the case of a very healthy individual who then stopped brushing for 18 days and developed lots of plaque and lots of bleeding. Another patient started out exactly the same, both healthy and clean – 18 days later they looked a little bit different – especially their level of bleeding. This was repeated by Trombelli *et al*. (6) and he found the same results. We also carried out experimental gingivitis studies in patients who had been treated by periodontal surgery, i.e. apical position flaps, and they developed in an experimental gingivitis model very rapidly plaque and gingival inflammation. We also started carrying out a study looking for subjects who were over 60 years of age, with no periodontal destruction and no recession. We found a few of them, but Edwin Winkel conducting the study had a nightmare finding such people (7). In the protocol it was said that we stopped the experimental gingivitis as soon as we have had two consecutive measurements of a bleeding site, so we said finally after 33 days of no oral hygiene let's stop the experiment although we did not had in all cases two consecutive bleeding sites.

During the years there have been numerous studies relating this *de novo* plaque formation to gingivitis and all confirm that there are people who develop more plaque and therefore more gingivitis and you have people develop less plaque and less gingivitis, but still what is the chicken and what is the egg? There's no clue for that yet.

Especially the study by Ramberg *et al*. is an interesting one (8). They also studied the *de novo* plaque formation and they looked on the one hand at the degree of inflammation and also into the saliva and the numbers of bacteria in the saliva. Now

what they show was in the first place that if you look at sites which have a gingival index score of zero, whether you look after 4 days of no oral hygiene or after 18 days of no oral hygiene, the plaque index is at the same low level, and if you look into sites which have a gingival index score higher than one after 4 days or after 18 days, the amount of plaque is at the same high level, so that suggests that the degree of inflammation determines the amount of plaque which develops. They found no influence of the number of microorganisms in the saliva, and I always had the impression that the numbers of microorganisms in the whole oral cavity should play a role, so I was very much surprised that they did not find a relationship between the numbers of microorganisms in the saliva and the rate of the *de novo* plaque formation.

So with one of our MSc students we carried out a study into the *de novo* plaque formation in periodontitis patients before and after periodontal therapy (9). We investigated the relationship between the numbers of bacteria in saliva and the amount of *de novo* plaque formation in periodontitis patients before and after non-surgical periodontal therapy. Before treatment as well as 3 months after treatment, we took an initial rinsing sample, we then measured the amount of plaque, pockets, bleeding and recession, and we did the same after 3 months. After these measurements we did a prophylaxis and cleaned it up completely, and then 24 h after no oral hygiene took another rinsing sample and looked to the amount of plaque which had developed during those 24 h. Now what did we see? The effect of treatment meant that the plaque index dropped from 1.53 to 1.45. Okay they were brushing a lot better. The bleeding decreased enormously, the probing depth decreased and the number of sites of 5 mm or more also decreased enormously from 23 to 6, so the patients indeed became healthier due to initial therapy consisting of supra- and subgingival scaling, rootplaning and oral hygiene instruction.

Now what are the results in relation to the amount of the *de novo* plaque formation at sites with no bleeding? Before treatment you have before a plaque score of 0.44, and after periodontal therapy, a plaque score of 0.3, and there is no difference. Also at sites which had bleeding before treatment, of course there was more plaque than at the sites which had no bleeding, but after therapy the bleeding sites had the same amount of the *de novo* plaque as before treatment. And then we looked at sites which were healthy before treatment and the same healthy sites after treatment – no bleeding on probing and pockets less than 4 mm. They had 0.49 plaque score before treatment and this dropped to 0.22 after treatment. So in my view this suggests that the major cause for the *de novo* plaque formation is the gingival fluid, but the saliva plays also a role, but in my opinion it is not as big as that of the subgingival area itself.

This is also supported by our rinsing samples. Before treatment we had in the saliva 5.2×10^8 bacteria, and after 24 h of no oral hygiene it was raised to 6.0, and after treatment we saw a drop in the bacteria in the saliva and an increase after 24 h but both were significantly different, so indeed we have less microorganisms in the saliva.

So the conclusions were that plaque grows more rapidly in the presence of inflammation, periodontal treatment results in the reduced number of bacteria in the saliva, and raised number of bacteria in the saliva may contribute to increased the *de novo* plaque formation.

Recently, there was this study published by Rudiger *et al.* and they looked into what they now call biofilms (10). I think it's more or less semantics whether you'd call it dental plaque or biofilms. In the 5-day experimental gingivitis study they looked at 4-h-old *de novo* biofilm formation on day 0 and on day 5 of their experimental gingivitis study, and they took a pooled sample of three teeth per quadrant in eight subjects. That means that they had 32 samples on day 0 and 32 samples on day 5, and they looked into the distribution of the microorganisms in this 4-h-old *de novo* biofilm or plaque. The results showed that in the presence of inflammation on day 5 the percentage of samples positive for *Prevotella* spp., *Prevotella nigrescens*, *Fusobacterium nucleatum* and *Capnocytophaga* spp. were higher as compared to day 0. This means that if you have a healthy site, different microorganisms start to colonize the surface, compared with an inflamed site, so it's not only the quantity which is different but also the quality of this biofilm which is different and that may have major consequences.

In summary, what is the significance of supragingival plaque? You can say it harbours putative periodontal pathogens, secondly plaque removal prevents subgingival colonization of putative periodontal pathogens, and the rate of the nova supragingival plaque accumulation may indicate the susceptibility of the host for periodontal inflammation.

Now these previously mentioned studies are all short-term studies – the relation of gingivitis to plaque – but is supragingival plaque predictive for *future* periodontal breakdown. Well there are very few studies which can answer that question. I selected a few and this is a study of Albandar and coworkers in the States, where they studied the gingival inflammation and sub gingival calculus as determinants of the disease progression in early onset periodontitis (11). They had a large patient pool of about 14 000 people in the age of 13–20 years with early onset of baseline defined as the presence of 3 mm

attachment loss at, at least four teeth, and they found 156 subjects who showed this characteristic and they followed them up for 6 years to see what happened in these subjects. They studied teeth which had no attachment loss at baseline and 3 mm loss of attachment 6 years later. Now if you look to the sites which had no gingivitis at baseline, then only 4.8% developed 3 mm attachment loss in 6 years, but if gingivitis is present it is 9.3%, and in their analysis plaque didn't come out, calculus did. If you have no calculus at baseline then there's only 3.6% of the sites showing further progression, but if you do have it then it's 14.4%.

If you look into other studies like the study from Burt and coworkers (12) or the more recent study in Oslo of Schätzle and coworkers (13) they show that gingivitis maybe a parameter which may predict future periodontal breakdown in tooth loss, but not dental plaque. And when carrying out a longitudinal study in Indonesia, we also found that the amount of dental plaque which was present at baseline did not predict the future of the periodontal breakdown in these subjects. So how do we explain the relationship between supragingival plaque and periodontal destruction as found in epidemiological studies on one hand and on the other the finding that dental plaque not a risk factor for onset and progression of periodontitis? In opinion the explanation is that the amount of supragingival plaque is a secondary phenomenon, people with lots of inflammation develop lots of plaque.

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