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Saliva Diagnostics in the Dental Practice

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Abstract

In medicine, therapeutic measures should only be provided once the diagnosis has been made. In the field of dental prevention, there is often a lack of profound diagnostics. The presence of plaque alone is usually sufficient as a diagnostic basis for the provision of preventive measures—usually professional cleaning measures. It is neglected here which parameters influence both the formation of plaque and the amount of plaque found. In addition to parameters to be determined clinically, such as domestic hygiene efficiency, fluoride supply, tooth morphology, nutritional history, compliance, knowledge, and motivation of the patient, this also includes bacterial and functional saliva parameters, which can only be determined as part of a saliva diagnosis. The parameters that are relevant and easy to determine for the dental practice are the number of cariogenic plaque-forming germs *Streptococcus mutans* and lactobacilli, the salivary pH, the secretion rate, and the buffer capacity. Plaque removal alone does not diagnose or treat these multiple factors. The mechanistic–localistic approach to professional tooth cleaning does not do justice to the complexity of the disease. Based on the extended ecological plaque hypothesis, this review article explains the indication, implementation, and therapeutic consequences of saliva diagnostics in dental practice.

Keywords: Extended ecological plaque hypothesis, saliva test, risk of caries, *Streptococcus mutans,* lactobacilli, buffering capacity, secretion rate, diagnosis-based individual prophylaxis, functional saliva parameters, bacterial saliva parameters, caries risk diagnostics, dental prophylaxis concept

INTRODUCTION

Preventive patient care is becoming increasingly important in practice. A paradigm shift is taking place away from the restorative orientation of dentistry toward a preventively oriented dentistry. This does not make the restoration superfluous—it just takes place under completely different framework conditions. The defect in the tooth is not the cause but the symptom of a profound disruption of the oral biome. Slowly, after more than 30 years, the sentence coined by Anderson[1] in 1993 in the *Journal of the American Dental Association* is being implemented: "The cutting edge is not the dental bur." However, preventive care not only includes professional tooth cleaning alone but also a determination of all risk parameters that influence the plaque formation rate. The present review article shows which possibilities exist in the dental practice to successfully implement a diagnosis-based individual prophylaxis.

Professional tooth cleaning has become an integral part of the prevention offer of many dental practices in recent years. Prevention is inconceivable without professional tooth cleaning. Unfortunately, however, this measure is often the sole content of a prevention session. This refers to the investigations of Axelsson and Lindhe[2,3] and overlooks the frequency with which the patients were summoned in the cited examinations.

- For the first 2 years every 2 months
- From the third–sixth year every 3 months
- Then, demand-oriented

It was obvious that this could not be achieved in a dental practice and would also overwhelm the financial resources of the patients. That is why Axelsson also spoke out in favor of risk diagnostics at an early stage. As an absolute diagnostic criterion, the presence of plaque alone should not suffice.

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This would also neglect which diverse parameters influence the formation and amount of the existing plaque. The sole reference to better home hygiene care is important, but not always expedient. Performing a professional tooth cleaning as the only measure does not do justice to the complexity of the problem.

The following parameters influence the amount of plaque found and should therefore be included in a risk diagnosis:

- Number of plaque-forming bacteria (*Streptococcus mutans* (*S. mutans*), Lactobacilli)
- Consume fermentable carbohydrates
- Nutrition (chewing activity)
- *Salivary secretion rate*
- *Buffer capacity of saliva*
- *General pH environment of the oral cavity*
- Fluoridation concept
- Morphology of teeth
- Tooth position
- Motivation and knowledge of the patient
- Oral hygiene (knowledge and implementation)

It can be seen that parameters to be determined clinically as well as clinically invisible—i.e., subclinical parameters—are written in italics in risk diagnostics. These can only be determined by saliva diagnostics.

A comprehensive diagnosis should therefore include not only the clinical parameters but also the subclinical parameters—written in italics in the table.

A decisive prerequisite for the interpretation of the subclinical bacterial and functional saliva parameters is the knowledge of the development of the oral biome from a homeostatic to a dysbiotic situation.

The scientifically recognized extended ecological plaque hypothesis postulates that the bacterial composition of the plaque is decisive for the development of caries and not the plaque per se.[4,5]

Lactobacilli and *S. mutans* have been identified as significant and most important germs.[6,7]

Scientific Concept

The first step in developing a non-invasive or minimally invasive treatment program is to identify tooth decay as a disease, both in the precavitary and cavitary stages.[8]

A continuous supply of fermentable carbohydrates leads to an increase in germs, which are able to metabolize the offered substrate and form acid from it. This proliferation of germs manifests itself clinically in increased plaque accumulation.[9]

The increasing plaque accumulation is therefore not only an expression of hygiene deficits of the patient but also the result of this increase in plaque bacteria due to the increased supply of substrates.

First, germs of the so-called non-mutans group multiply (Figure 1). These are weak acidifiers, but able to lower the pH level (acidogenic phase). As a result, strong acidifiers, *S. mutans* and *Lactobacillus* spp., find ideal growth conditions and can overgrow the whole system (aciduric phase). The biome has changed from a homeostatic to a dysbiotic state.[9]

The role of *S. mutans* is not limited to the strong acid-forming capacity: *S. mutans* is the main actor in the formation of extracellular polysaccharides and thus the guarantor of an undisturbed "quorum sensing" in the plaque since the matrix formed by it protects the tooth-lying biotope from the natural defense functions of the oral cavity—secretion rate and buffer capacity.[10,11]

This means that *S. mutans* continues to play a decisive role in the disease process and, together with lactobacilli, is an important parameter for disease risk.[12]

Streptococcus mutans is one of the decisive factors in maintaining the acidic environment that offers ideal growth conditions for lactobacilli.

In fact, Lactobacilli (LB) are able to produce acid up to the pH of 3, while *S. mutans* stops acid production at a pH between 4 and 5. Lactobacilli—itself not actively involved in plaque build-up—thus use the pH environment created by *S. mutans* to promote caries progression.[13-15]

If *S. mutans* and Lactobacillil are detectable in high numbers, then the oral microbiome seems to be in a dysbiotic situation and thus represents a risk of disease.[9]

The therapeutic approach is important because the lactobacilli numbers give us indications of the carbohydrate content of the food and sugar consumption of the patient and thus might be an early indication of an unfavorable nutritional situation.[16,17]

Dysbiotic situations are also often associated with unfavorable functional saliva parameters. Decreased chewing activity due to the intake of soft and/or sticky foods and repeated intake of acidic drinks lead to changes in saliva pH, buffer capacity, or secretion rate.[18]

A reduced secretion rate also means a lower concentration of sodium bicarbonate in the oral cavity, which is expressed by a decrease in buffer capacity. This reduces a natural defense function against dietary and plaque acids in the oral cavity.[19]

In summary, the plaque consists of potentially pathogenic and apathogenic germs, as well as great diversification. For the development of a pathogenic plaque, it is not primarily the genotype of the bacteria alone that is decisive, but the phenotype. This develops from the individual circumstances that can turn a potentially cariogenic germ into an actual cariogenic germ. The acid-forming capacity of the biofilm increases, which leads to a further pH lowering in the oral environment.[20]

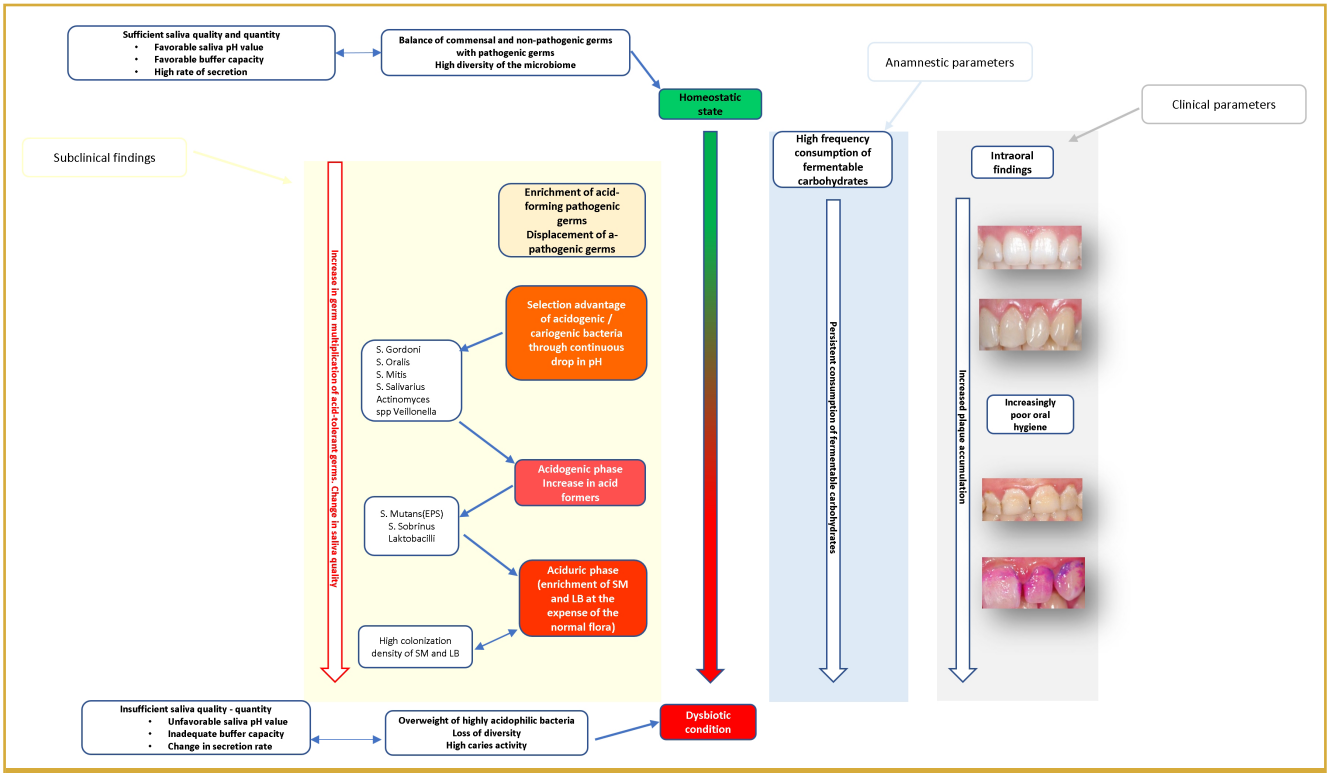

Figure 1. Caries model according to the extended ecological plaque hypothesis.

Recent studies have already been able to prove that it is possible to determine this acid formation potential by a simple detection method. This could give an early indication that the milieu in the oral biome is changing to a dysbiotic situation.[20]

This process is important for the preventive concepts of the dental practice: reversibility is possible if the decisive factors responsible for the changes can be influenced.

With saliva diagnostics, we can determine both bacterial (*S. mutans*, Lactobacilli) and functional saliva parameters (secretion rate, buffer capacity, and saliva pH). This not only creates a diagnostic basis for the therapy but at the same time opens up the possibility of an objectifiable follow-up.[21]

In summary, the health-associated bacterial species are suppressed in the phases of dysbiosis but are not completely lost. This makes the reversibility of the change in the oral biome possible[22,23] (Figure 2).

The same reaction pattern can also be seen in periodontal diseases: the health-associated types of bacteria are suppressed in the phases of dysbiosis but are not completely lost.

Saliva tests detect lactobacilli, *S. mutans*, and yeast fungi. These germs are located in the plaque attached to the tooth. By chewing paraffin or a sugar-free flavorless gum, these germs organized in the plaque are released into the oral cavity and can be detected here by examining the total saliva. The amount of these planktonic germs correlates with the number of germs organized in the plaque.[24-27]

In addition, it is possible to directly examine the number of germs organized in the plaque by means of a plaque smear. There is a statistical correlation between caries experience and germs in dental plaque and saliva.[28]

WAYS TO OBTAIN A SALIVA SAMPLE

Plaque Smear

This method determines the caries activity on a specific tooth surface. Detection of *S. mutans* and/or Lactobacilli in the plaque is associated with high caries activity at this site, with caries-active and caries-inactive sites being quite close to each other.[24] The plaque smear captures the S.mutans (SM) and LB organized in the plaque (Figure 3-6).

The Spatula Method

The spatula method is used to transfer oral cavity germs to an agar. Due to the use of a wooden spatula, it is also referred to as the "copying technique," as the germs are applied to the culture medium by clapping (Figure 7-9).

This method is primarily used in small and very small children to detect colonization of the oral cavity with *S. mutans*.[25]

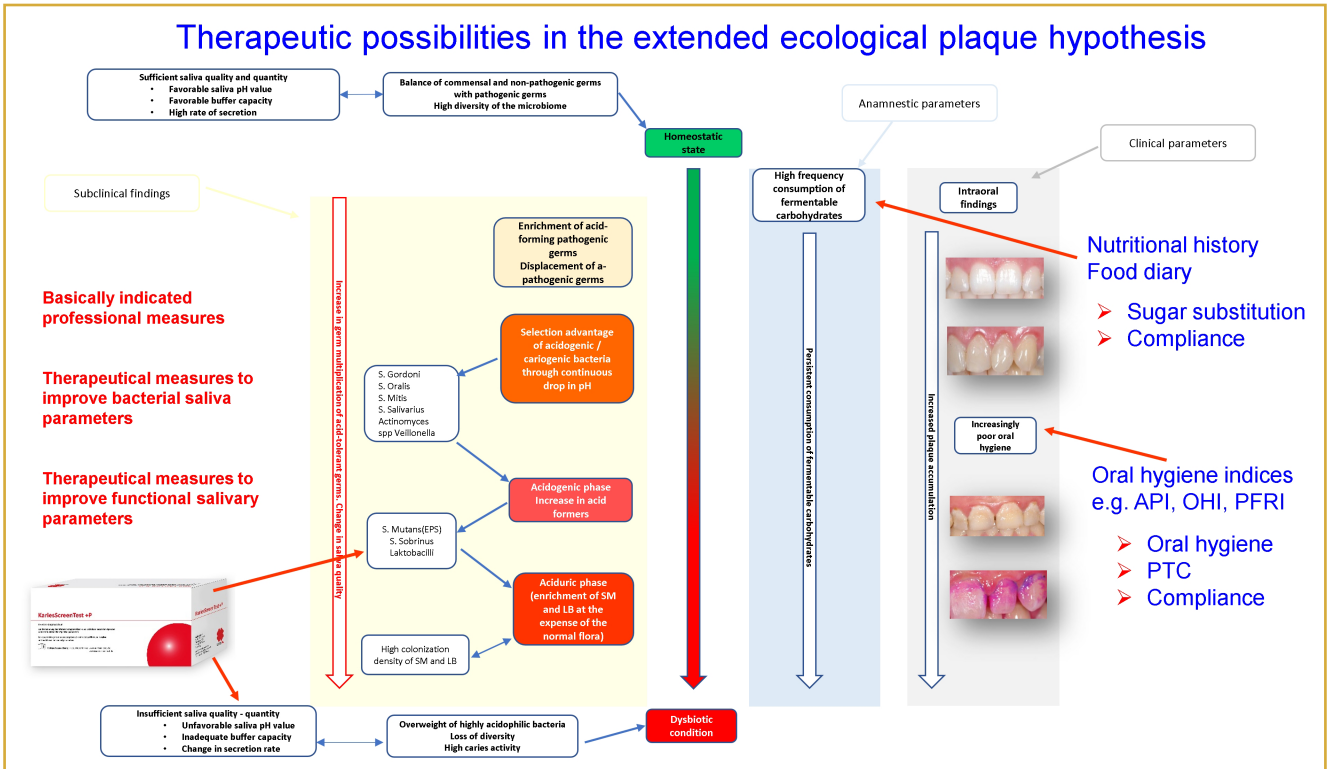

Figure 2. Subclinical diagnostic possibilities and the resulting therapeutic possibilities (see below). Saliva diagnostics with the KariesScreenTest+P from AUROSAN, Essen, Germany (formerly CRT bacteria and CRT buffer (IvoclarVivadent).

INVESTIGATION OF THE TOTAL SALIVA

Detection of large numbers of germs in the saliva does not necessarily correlate with caries activity on certain tooth surfaces. Plaque-free tooth surfaces can be found next to areas with massive plaque colonization, which are responsible for and maintain the increased number of germs relevant to dental caries.[24]

On the other hand, individual risk points in which microbiotopes can form, such as chewing surfaces, rotation and tilting tooth positions or crown edges, etc., are insufficiently represented in an examination of the total saliva with otherwise inconspicuous clinical findings. Here, a plaque smear may be necessary as a diagnostic supplement for validation (see earlier).

Figure 3. Smear technique smooth surface.

Figure 4. Smear technique in the approximate space.

Figure 5. Smear from the chewing surface.

Figure 6. Incubated smear (here Lactobacilli).

Figure 7. Spatula technique in the mouth.

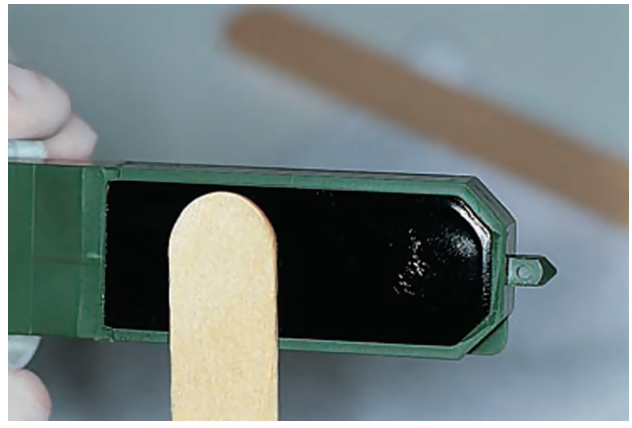

Figure 8. Spatula technique: Application to the culture medium.

Figure 9. Incubated test (spatula method and smear).

In addition to the bacteriological parameters, it is also the functional parameters that can be used for risk diagnostics (Figure 10, 11).[29,30]

THE FUNCTIONAL SALIVA PARAMETERS

pH

The resting pH of the saliva can be determined with indicator test paper (e.g., in Saliva-Check Buffer or in KariesScreenTest+P). The resting pH should be higher or equal to pH 7, especially on exposed root surfaces where demineralization already starts at a pH of 6.7.

Salivary Flow Rate

By determining the saliva flow rate, it becomes apparent whether sufficient saliva is present. The natural protective function of saliva, the rinsing function, clearance, buffer capacity, as well as the availability of minerals for remineralization depend on the amount of saliva available. The secretion rate should be around 1 mL/min. Values below this reduce the clearance rate and the remineralization potential and are therefore caries-promoting.[30,31]

Figure 10. Lactobacilli findings (>10^5 KariesScreenTest, AUROSAN).

Buffering Capacity

There are 3 buffer systems in saliva: the sodium bicarbonate buffer and the phosphate and protein buffers. The most important buffer for saliva diagnostics is the bicarbonate buffer.[32,33]

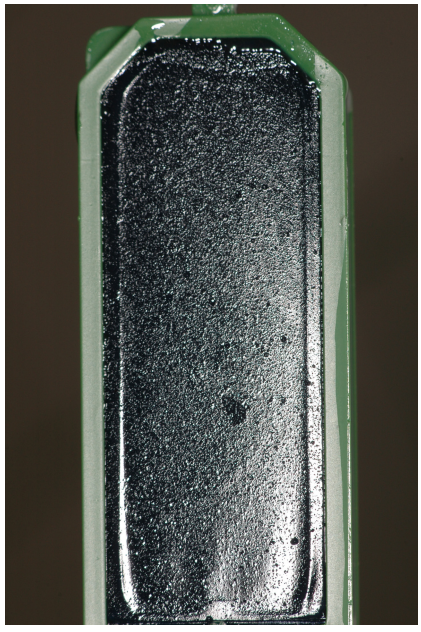

Figure 11. *S. mutans* findings (10^6 KariesScreenTest, AUROSAN).

The buffer capacity represents a crucial protective mechanism of the oral cavity against dietary and plaque acids and is related to the saliva flow rate. High saliva flow rates cause good buffer capacities due to the increased sodium bicarbonate content. There is therefore often a correlation between the secretion rate and the buffer capacity found.[34]

A good buffering capacity can neutralize dietary and plaque acids in the oral cavity. The buffer capacity therefore has an important function in the stability of the pH environment in the oral cavity. However, if there is a more frequent intake of acidic foods or drinks, this leads to a greater drop in pH in the oral cavity. If the buffer capacity of saliva is no longer sufficient for the neutralization of this larger amount of acid, demineralization or erosion may develop.[35]

This constitutes a mandatory indication for extended diagnostic measures and for the implementation of individually designed prevention and disease management measures.[36]

Very good buffer capacities have a pH value of >6, good buffer capacities are between 5 and 6, and poor buffer capacities are referred to as a pH value of <5.

As already mentioned earlier, the sodium bicarbonate content of saliva is also dependent on the rate of secretion. High secretion rates are mostly accompanied by good buffer capacities.[37]

Therapeutic Consequences of Saliva Diagnostics

Knowing the identified risk parameters, therapeutic measures can be defined differently (Figure 2). If one assumes only clinically and anamnestically determined patient parameters, then almost always central components of a therapy proposal are improved hygiene, nutritional care, a fluoridation concept, and regular prophylaxis sessions with a professional tooth cleaning. Further therapy proposals are usually based on clinical findings and therefore often only allow damage limitation.[30,38]

THERAPEUTIC CONCEPTS CAN NOW BE DEFINED DIFFERENTLY

Basically indicated professional measures:

- X-ray control for the detection of clinically invisible cavitations
- Intensification of professional support
- Intensification of home oral hygiene measures
- Adequate home and professional fluoridation concept

Measures to improve bacterial saliva parameters:

- Measures to reduce the number of germs with antibacterial rinses or gels. However, these measures are only successful if they are provided in an overall concept of preventive care (see further points later). These further measures prevent premature recolonization of the oral cavity with cariogenic germs. Chlorhexidines are

still suitable for antibacterial therapy. These influence the growth of *S. mutans* under planktonic and biofilm conditions.[39]
- Reduction of sugar impulses
- Reduction of the amount of sugar
- Recommendation of sugar substitutes or sugar-free sweets (xylitol, etc.)
- Recommendation of sweeteners (e.g., Stevia)
- Professional tooth cleaning with antibacterial cleaning pastes
- Application of antibacterial varnishes to problem areas with microbiotopes
- Elimination of retention niches
- Application of fluoride-containing varnishes to problem areas and demineralizations
- Use of application aids (professional and domestic)

Measures to improve functional saliva parameters:

- General medical anamnesis/determination of medication
- Ensuring adequate chewing activity (dentures)
- Intensification of chewing activity (diet/sugar-free chewing gum)
- Gustatory stimulation
- Adequate fluid intake
- Recommendation of products for the treatment of dry mouth
- Control for halitosis with appropriate recommendations (e.g., Meridol-Halitosis)
- Use of application aids for Chlorhexidine/Fluoride-containing gels
- Use of sodium bicarbonate-containing cleaning pastes

All these measures do not cause the plaque to disappear completely—they cause a change in the pathogenicity of the plaque on the way back to a homeostatic state.

Repeated diagnosis of subclinical saliva parameters allows both a control of patient compliance and the effectiveness of the domestic and professionally performed measures and gives indications as to whether the treatment goal has been achieved.[21]

This is a decisive difference from the concept often practiced in the prevention of defining the type and scope of the preventive services to be carried out according to the available billing items.

AREAS OF APPLICATION OF SALIVA DIAGNOSTICS IN DENTAL PRACTICE

Pregnant Patients

In addition to the changes that take place in the oral biome of pregnant women, there are also risk factors that can affect the future oral health of the child.

Depending on the germ concentration of *S. mutans* in the mother, there is a risk of transmission of this germ to the child. The transmission of *S. mutans* from mother to child has been referred to since 1978. It could be verified again in 2019 using polymerase chain reaction (PCR) technology.[40,41]

Therefore, determination of the maternal levels of *S. mutans* using the current breakpoints is proposed as an efficient method to assess the risk of maternal transmission of *S. mutans* in childhood.[45]

A prerequisite for germ transmission is a proven concentration of at least 10^5 germs in the mother's saliva.[42]

The transmission of germs does not automatically lead to colonization of the child's oral cavity with *S. mutans*: this is only possible if there is a sufficient amount of fermentable carbohydrates in the child's oral cavity.[43]

For the child, the time of the first detection of *S. mutans* in the oral cavity is crucial for his dental health.

The general prevalence of caries of the child at the age of 4–5 years depends on the time of the first detection of *S. mutans* in the plaque of the child's oral cavity.[44] The earlier this point in time, the higher the prevalence of caries can be estimated.[45] Preventive measures should therefore be aimed at reducing the bacterial counts of cariogenic germs in pregnant women, as this can reduce the risk of transmission.

Since the colonization of the child's mouth with *S. mutans* requires sufficient availability of fermentable substrate, the nutritional situation of the mother should also receive our attention here.

The mother's eating behavior already prenatally influences the child's eating behavior through in-utero programming and shapes it for certain preferences. In a mother who prefers to eat sweet foods, this can also increase the craving for sweetness in the child—regardless of the already existing genetic disposition for sweet taste.[46] In addition, there is also the experience that the prenatal dietary behavior of the mother will not necessarily change postnatally.

To clarify the nutritional situation and thus as a basis for the educational discussion, the lactobacilli numbers of the mother can be used.

In addition to this goal of establishing optimal oral health of the child from the very beginning, other risk factors in pregnant women must also be taken into account, which can change the homeostatic situation of the oral biome:

- There is an increase in mutans streptococci, lactobacilli, and yeasts from the end of the second trimester. This can result not only from a change in dietary behavior but also from increasing hygiene deficits due to the hormonally induced gingival changes caused by pregnancy. Therefore, early preventive care is best indicated in the first trimester.[47]
- The saliva pH value can decrease up to the pH value of 5.9 and thus increase the risk of root caries.[48]

- The buffer capacity decreases with a simultaneous decrease in the calcium and phosphate content of saliva. Dietary and plaque acids are no longer sufficiently buffered. As a result, this also causes a change in the risk of caries as a result of lower remineralization.[49-51]

Studies have also shown that certain anamnestic and clinically easy-to-determine risk factors often correlate with high *S. mutans* and Lactobacilli numbers.

These were:

- The level of education of mothers,
- The frequency of daily brushing, and
- The prevalence of caries found[52]

Statistically significant correlations were also found in caries prevalence and bacterial saliva parameters in working and non-working mothers. In the working mothers, higher *S. mutans*/Lactobacilli values and a higher prevalence of caries were found in the children.[53]

Infants and Siblings

S. mutans is still a leading germ in the development of caries due to its ability to adhere to the enamel structure and form biofilms. The time of the first colonization with *S. mutans* in the oral cavity of the children is of particular importance for siblings.[54]

Colonization of *S. mutans* in the child's oral cavity is only possible with a continuously high substrate intake of easily fermentable carbohydrates.

When advising parents, it must therefore be clearly pointed out that it is the parent's duty of care to prevent the colonization of the oral cavity with cariogenic germs by ensuring that the child has a healthy diet.

Then, even after a transmission that may have occurred, *S. mutans* has no chance of permanently establishing itself in the child's oral cavity. So here the compliance of the parents is primarily important.[55]

In a systematic review in 2006, Thenisch et al[56] were able to show that detection of *S. mutans* in saliva at the age of 2 years means a doubling or the detection in the plaque (plaque smear) a quadrupling of the risk of caries.

This early colonization of the child's oral cavity is not only crucial for the prevalence of caries at the age of 4-6 years. Even after 15 years, the mouth findings of the children differ significantly depending on the date of first colonization. The prevalence of caries is higher in children in whom *S. mutans* has been detected at an earlier age.[44,45,57]

The control for *S. mutans* in the 2- to 3-year-old child thus provides information about the preventive therapy to be carried out. Of course, this also means that a caries-free dentition at the age of 2-3 years is not to be equated with the absence of a caries risk, which may well be present without cavitation or even demineralization having occurred so far (Figure 12).

Checking for *S. mutans* gives us decisive indications of which preventive measures need to be applied in the present case. The saliva sample can mainly be carried out with the spatula method.

Extremely unfavorable nutritional situations usually lead to early detection of very high *S. mutans* numbers—this is

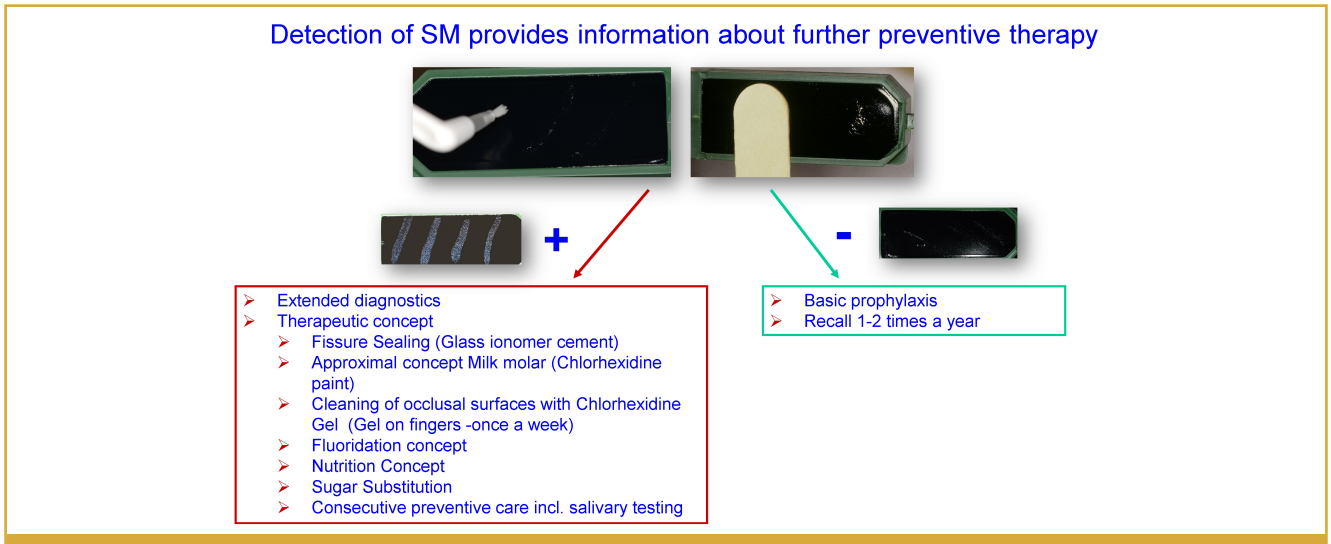

Figure 12. Therapeutic consequences of detection of *Streptococcus mutans* in 2- to 4-year-old children. The detection of SM at the age of 2-4 years gives indications of an existing risk of caries. Further risk factors should be reviewed (listed in the table as advanced diagnostics) and taken into account in therapy planning.

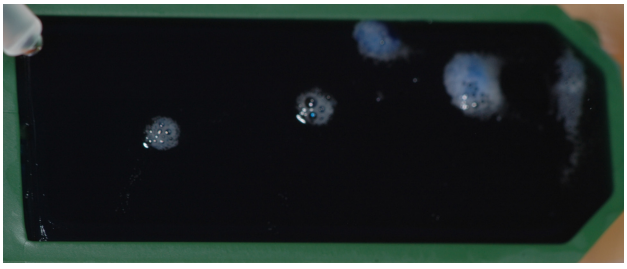

Figure 13. Positive catalase reaction (with H_2O_2) for the detection of yeast fungi here on the SM agar.

always a strong risk indicator for the occurrence of "Early childhood caries".[58,59]

At the same time, the growth of *Candida albicans* can also be promoted: this further increases the cariogenic potential in the plaque.[60]

C. albicans tolerate highly acidic environments and produce high amounts of organic acids, mainly acetic acid and pyruvic acid, which lower the pH of the environment more efficiently than the lactic acid secreted by *S. mutans*.[61]

These yeast fungi are also detected in saliva diagnostics, as the agar used for *S. mutans* as well as for Lactobacilli are not fungicidal. Yeast fungi can be distinguished from lactobacilli by a so-called positive catalase reaction (Figure 13).[62]

Of particular importance is the time of first colonization in siblings of different ages. In nutrition-conscious parents, the first-born child is often caries-free at the age of 4 with an inconspicuous subclinical finding. However, in this age group, it has increased access to or cravings for sweets. Due to this fact, experience has shown that the regular sugar intake of the younger sibling starts at an earlier stage. This means earlier colonization of the oral cavity with *S. mutans* and a completely different risk of tooth decay (Figure 14).

Based on the result of saliva diagnostics, the younger sibling now needs completely different preventive care than the older one. The younger one is—regardless of the currently still good clinical findings—a risk case and must also be treated as such (Figure 15).

The additional sugar substitution by xylitol can cause a further reduction of *S. mutans* in both saliva and biofilm.[64,65]

Orthodontic Cases

The introduction of orthodontic appliances always leads to an increased plaque accumulation and thus to a change in the oral microflora due to a large number of retention niches. This applies in particular to the caries-relevant germs *S. mutans* and lactobacilli.[66,67]

The changes are more pronounced with fixed treatments than with removable devices.[68]

Aligner treatments are also not exempt from this, although they do not cause such serious changes in the biome as fixed treatments.[69]

The longer the fixed treatment lasts, the more the risk of caries and the expected prevalence of caries changes.[70]

However, the change in the risk of caries is not at the same time as the occurrence of cavitation. This is only detected at a later date when the orthodontic treatment has long since

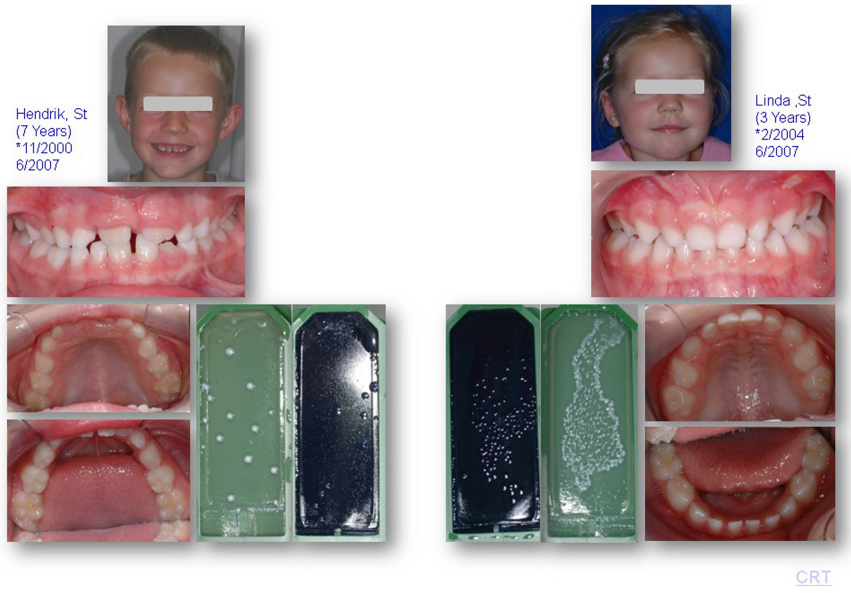

Figure 14. Different saliva findings and different caries risk in siblings (saliva test: CRT bacteria).

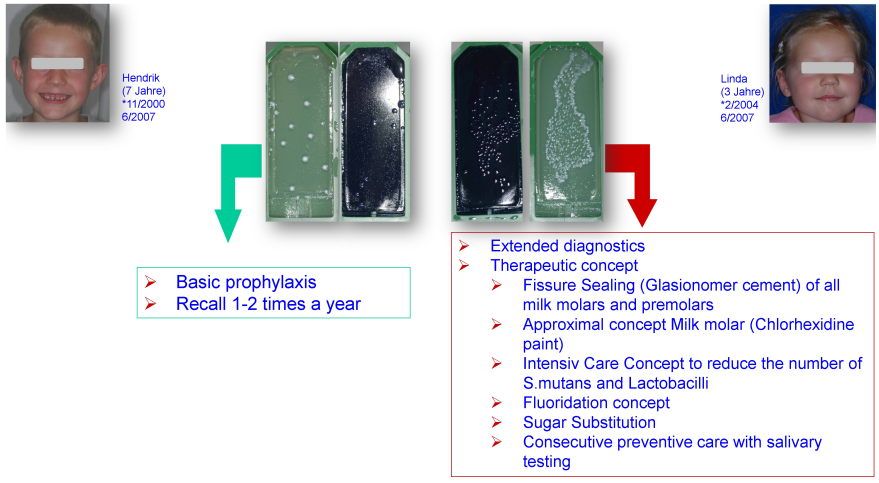

Figure 15. Therapeutic consequences in siblings due to the different risk assessment. Intensive therapeutic measures also include measures to reduce the number of germs of caries-relevant germs, in particular SM. It could be shown that it is possible to reduce the number of *Streptococcus mutans* in the saliva of children by using a chlorhexidine/thymol-containing and a fluoride-containing varnish.[63]

ended. This makes it all the more important to determine bacteriological and functional saliva parameters both during and at the end of orthodontic treatment, to compare them with the initial values, and to develop a therapy concept with the help of this information. This should be done both at the same time as the orthodontic treatment and after the completion of orthodontics.[70]

The unfavorable changes are caused not only by the insertion of the orthodontic appliances and the associated hygiene problems alone but also by potential behavioral changes of the patient. This is especially true if the treatment lasts until puberty or is only started in this period. In this age group, experience has shown that increased sugar intake takes place through snacks or other cariogenic snacks.[71]

Motivation for nutritional care and adequate home hygiene are not successful in all cases. In the case of fixed treatments, the effort at home is much more intensive, but even with treatments with removable appliances, there are often hardly seen any improvements over the years—despite intensive dental care.

This makes objectifiable information about the condition of the oral biome all the more important for the correct assessment of the risk of caries. Sudden and occasionally unexpected perfect toothbrushing results can thus be recognized as not congruent with the actual risk of caries (Figure 16, 17).[71]

Due to the increase of the acidogenic bacteria *S. mutans* and Lactobacilli, lactate production increases and the saliva pH value decreases. This in turn favors the growth of lactobacilli and *S. mutans*.[72]

Figure 16. Hygiene findings when integrating the removable orthodontic device.

Figure 17. Control 2 years later with even worsened hygiene findings despite regular motivation and control.

A continuous saliva diagnosis, which is carried out at regular intervals during therapy with orthodontic treatment, documents these changes in the biome in good time and thus enables a therapeutic intervention before damage occurs.

Studies have shown that self-ligating bracket types have lower plaque colonization. Probably because the plaque retentive fastening rubber is not present here. However, the effect of CHX was independent of the bracket type.[73]

Patients with Periodontal Diseases
Periodontal diseases usually lead to exposed tooth necks and exposed root surfaces. At the latest, however, after periodontal therapy for advanced periodontitis, root areas are exposed.

In contrast to tooth enamel, which is demineralized at a pH value below 5.5, exposed root cement is already at risk at a pH value of 6.7, that is, just below the resting pH value of saliva (pH 7.0). In this way, discoloration or caries can occur on exposed tooth necks without changing the patient's behavior with regard to hygiene.

This underlines the importance of comprehensive risk diagnostics including the functional saliva parameters that are important for the disease.[74,75]

Although periodontal therapy leads to a significant decrease in anaerobic bacteria, it does not always lead to a reduction in *S. mutans*. Since these remain in constant numbers in the biotope, this leads to a proportional increase in this germ and to a change in the risk of caries.[75,76]

In addition to this proportional change, the reduction or elimination of the periodontal microflora creates a space for the reproduction of cocci, especially *S. mutans*.

There is therefore a risk that periodontal treatment will eliminate periodontal pathogenic germs, but the growth of cariogenic germs will be promoted and thus—especially in remaining residual pockets—the risk of root caries will increase.[77]

In chronic forms of periodontitis or in severe periodontitis cases, ecological niches can be created that create ideal conditions for the reproduction of *S. mutans*.[78]

These changes should always be expected in periodontal treatments. By appropriately determining bacterial and functional subclinical risk parameters, we can determine these changes before and after periodontal treatments. The same applies to the follow-up in the following years.

Patients with Medication/Hyposalivation
Reduced salivation is by no means a "sign of aging." Hyposalivation can occur in any age group. In addition to the well-known and often cited Sjögren syndrome, which we encounter relatively rarely in dental practice, it is often hyposalivation problems that are initiated by drug consumption and dietary changes.

Many drugs have an influence on the secretion rate as side effects, such as

- Antihypertensive agents
- Antidepressants
- Antipsychotics
- Contraceptives
- Muscle antispasmodic
- Drugs for Parkinson's disease
- Drugs for cardiac arrhythmia
- Anti-allergy medicines (antihistamines)
- Appetite suppressant
- Tranquilizers
- Anxiolytic
- Antispasmodic
- Water tablets (diuretics)
- Drugs for bronchial asthma

For example 1.4 billion drugs were purchased in Germany in 2019—about half prescription and half free for sale—it is not surprising that side effects can also be seen in the oral cavity.[79]

Clinical signs are often overlooked if the patient does not complain of dry mouth. An accumulation of saliva in the bottom of the mouth is also easy to miss but is often noticed in the prophylaxis session. Saliva diagnostics—in particular the determination of functional risk parameters—thus enable not only a more accurate diagnosis but also an objectifiable follow-up of the success of the preventive measures carried out or recommended.[21]

Lack of salivation also often leads to changes in dietary behavior. Since the food is no longer properly moistened when chewing, little chewable and sticky food is often consumed. This is especially true if an optimal chewing ability is not guaranteed by possibly existing dentures. Due to a lack of chewing activity as well as insufficient fluid intake, saliva secretion remains low.

Here, too, the determination of subclinical saliva parameters helps. For example, increased lactobacilli counts indicate an increased consumption of fermentable carbohydrates. In many cases, these are soft and sticky and therefore not very chewable. Accordingly, *S. mutans* also multiply.[80]

CONCLUSION

A practice concept based on prevention cannot be limited to the fact that professional tooth cleanings are carried out regularly. A preventive treatment concept in which the indication for preventive services is based solely on possible billing items does not meet the medical requirements of a dental practice.

As in other areas of dentistry, preventive services should be based on a diagnosis. This goes far beyond the diagnostic question "Plaque present or not?"

A comprehensive diagnosis of the clinical and subclinical risk parameters gives us important detailed information in the overall concept of the clinical assessment of a patient's health or disease. A clinically healthy finding does not always automatically mean the absence of a risk of disease.

For the assessment of the risk of caries, however, it is not only important to know which risk factors are present, but also why, and in particular what the subclinical parameters do.

In this overall concept of caries risk determination, the control of bacterial and functional saliva parameters has an important detailed function. They not only enable a more comprehensive assessment of the risk but also enable control after preventive treatment and patient compliance.

Regardless of scientific advances in the determination of saliva germs (PCR germs), classical methods of analysis (bacteria cultures) continue to determine the important parameters that have an influence on the risk of caries. This comprehensive diagnosis also improves communication with patients or parents through this interactive approach. The diagnostic inclusion of salivary parameters helps the patient to understand the disease process and can motivate a change in dietary habits.[81,82]

In addition, knowledge of salivation as well as the buffering capacity of stimulated saliva and knowledge of the number of *S. mutans* and lactobacilli are of great importance for deciding whether long-term remineralization therapy can be successfully applied to early-stage caries lesions.[82] Long-term studies in children have shown that the extent of oral colonization with *S. mutans* and Lactobacilli correlates positively with later caries prevalence. High bacterial counts predict a high prevalence of caries.

Saliva diagnostics alone can only ever be part of a comprehensive risk diagnosis. It provides us with more detailed knowledge of the patient's current health or disease findings than the clinical examination alone. It therefore represents a valuable addition to clinical findings. This is very nicely recorded and documented in the cariogram.[8,83]

This comprehensive diagnosis of both clinical and subclinical risk factors enables the implementation of comprehensible diagnosis-based individual prophylaxis.

For successful prevention, it is not only important to collect all the risk factors mentioned, but it is also much more important to understand why these risk factors are present. Here, saliva diagnostics is a valuable diagnostic aid. This understanding alone helps to develop and implement individual prevention concepts and to check their long-term effectiveness accordingly.

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