Caries risk diagnostics

BASIS OF INDIVIDUAL PROPHYLAXIS

Plaque leads to caries; the common consequence is regular professional plaque removal. The factors that influence the development of plaque are neglected. Only when the dentist asks: "Why is plaque present in this patient?" can their oral health be maintained/restored in the long term.

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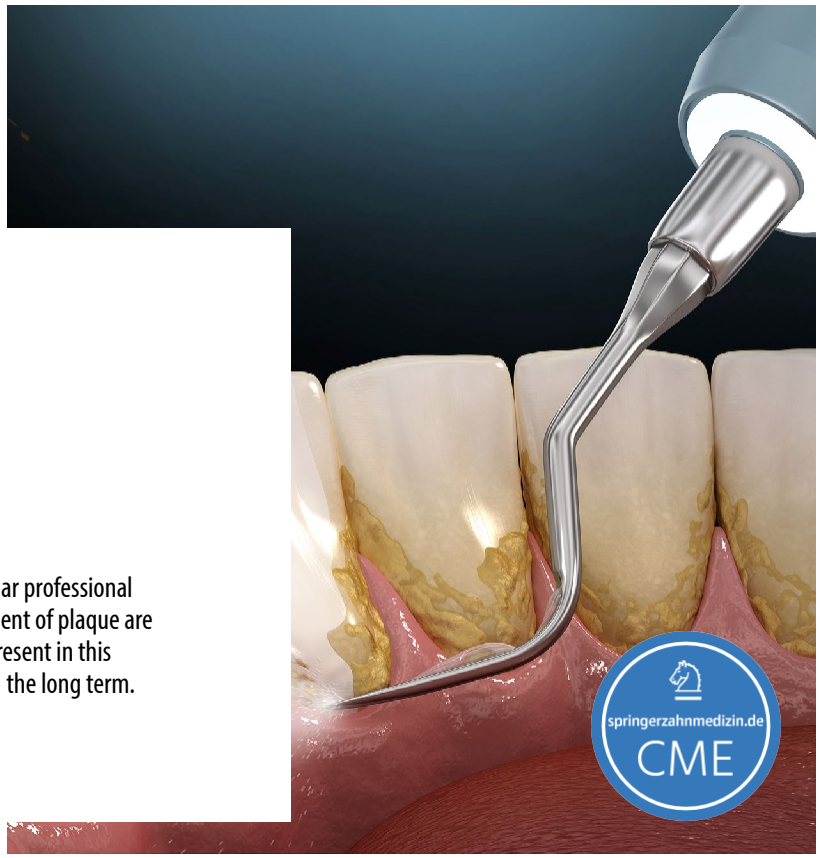

SUMMARY

In the field of preventive dentistry, there is often a lack of profound diagnostics. Thus, the mere presence of plaque is used as a diagnostic basis for the provision of preventive measures - professional tooth cleaning. However, this approach is no longer in keeping with the times, because the way caries aetiology is viewed has changed dramatically over the course of time. Today, caries is regarded as a multifactorial process and thus allows for preventive measures beyond professional tooth cleaning. This article provides the basic principles of diagnosis-based individual prophylaxis and shows ways to implement it successfully in dental practice. The focus is on the consistent care concept that results from the diagnostic parameters determined.

LEARNING OBJECTIVES

After reading this article, you will be able to analyse, reliably interact and assess the following essential conditions of multifactorial plaque development in your patient:

- The amount of plaque found on the teeth is the result of multiple individual risk parameters that are not treated by "professional tooth cleaning" alone.
- Not every supragingival plaque is classified as pathogenic per se.
- The changes in the homeostatic situation of the plaque are triggered by an unfavourable nutritional situation combined with suboptimal oral hygiene.
- Streptococcus mutans and lactobacilli are strong acid producers and are responsible for maintaining a dysbiotic situation in the plaque.

Keywords
Dental plaque -- Caries -- *Streptococcus mutans* -- Lactobacillus -- Patient care

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- Streptococcus mutans, through its ability to form extracellular polysaccharides, contributes decisively to quorum sensing and thus to the maintenance of caries activity in plaque.

INTRODUCTION

In medicine, therapeutic measures should always be provided only after a diagnosis has been made. In the field of dental prevention, there is often a lack of profound diagnostics. The mere presence of plaque is often used as a diagnostic basis for the provision of preventive measures - professional tooth cleaning. This is a continuation of the basic principle of the non-specific plaque theory, as already described in detail by Miller in his textbook of con- servational dentistry in 1896 [1].

However, the approach to caries aetiology has changed dramatically since then - caries is now seen as a multi-factorial event and thus also enables preventive measures beyond professional tooth cleaning.

Notice:

- Caries is based on a multifactorial process

INITIAL SITUATION

Historically, individual prophylaxis is distinct from group prophylaxis. Since the latter was first established in dentistry, many elements of group prophylaxis were adopted in dental practice with the introduction of individual prophylaxis. First of all, the regular use of preventive dental examinations (twice a year to the dentist) was postulated.

However, publications showed that there is insufficient evidence to support the common practice of encouraging patients to visit the dentist every six months to maintain their oral health [2]. Controlled randomised trials also showed that oral health is not measurably improved by increasing the number of examinations per year [2, 3].

With the findings of Axelsson and Lindhe, the importance of dental plaque was recognised. Thus, professional plaque removal became the focus of preventive dental efforts [4]. Therefore, it is not the examination that is decisive for oral health, but rather the resulting professional measures that are carried out in the individual sessions. These measures are mainly limited to finding the plaque and removing it professionally. The studies by Axelsson and Lindhe showed that only the pre-treatment of the plaque

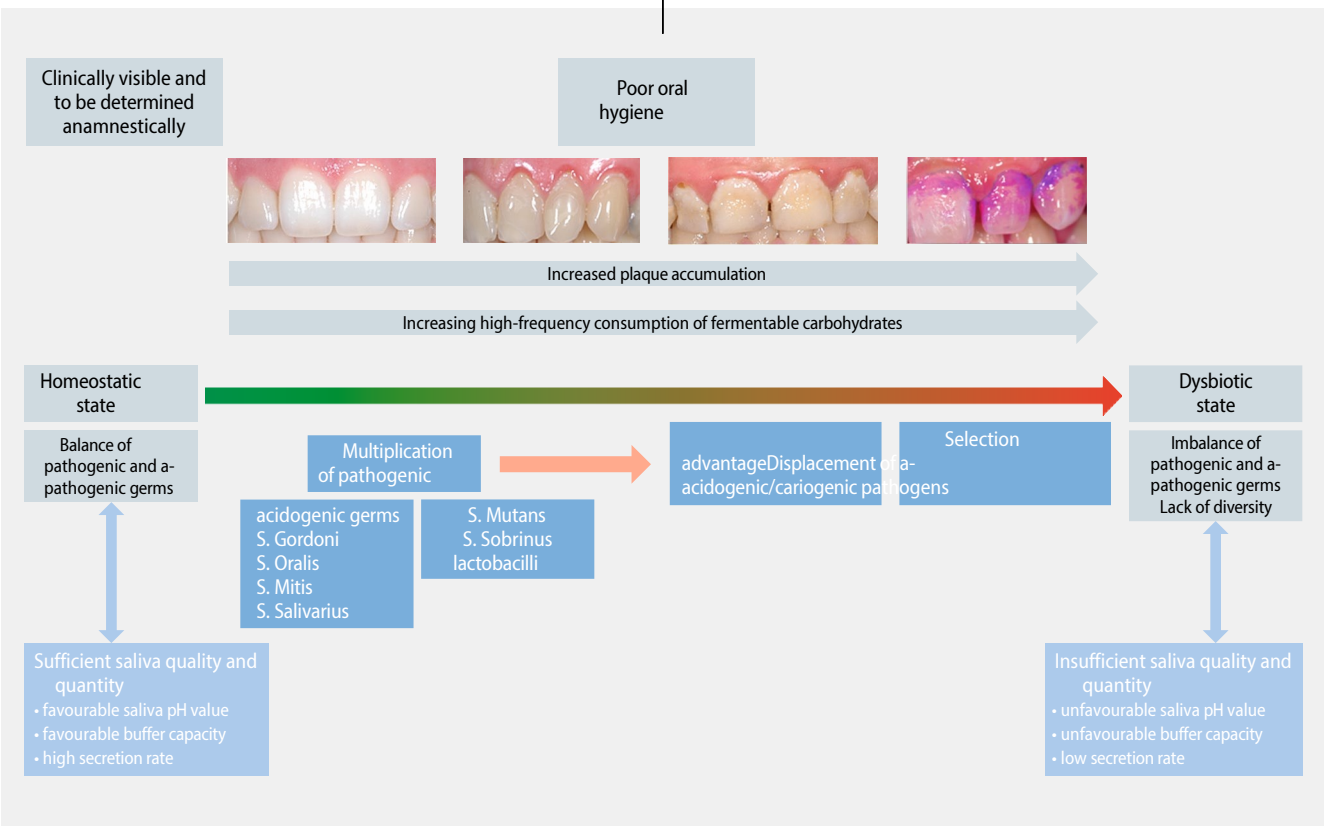


Fig. 1 Illustration of the extended ecological plaque hypothesis. Clinically visible parameters are grey and clinically invisible changes are highlighted in blue (courtesy of the author).

Tab. 1 "No caries risk" versus "high caries risk". (Axelsson [29])

Patient-specific factors	No caries risk	High caries risk
Streptococcus mutans content in saliva	negative	>500,000 ml/min
Oral hygiene habits	excellent	very bad
Lactobacilli levels	low	>100,000 ml/min
DMF/DMF-T index	Very low DMF	very high DMF-T, with buccal/lingual DFS
initial caries	No active	a great deal
Salivary secretion rate	Sufficient	<0.7 ml/min
Consumption of sticky, sugary products	low	high
Buffer capacity: pH value	>5,5	<pH 4.5

DFS ###, DMF-T: D "decayed", M "missing", F "filled", T "tooth".

The presence of plaque and the amount of plaque were indicative of the need for therapeutic intervention. Plaque is a threat to dental health, so regular professional removal is indicated.
What is neglected here are the factors that influence the development of plaque in the first place. These multiple factors are neither diagnosed nor treated by plaque removal alone. Plaque is the result of multiple parameters, and professional plaque removal alone does not do justice to the complexity of the disease. The question: "Why is plaque present?" is not asked in this concept or dismissed with a lack of domestic hygiene. However, only by asking "why" does it succeed in identifying the multiple parameters responsible for both plaque per se and the amount of plaque. These parameters include:

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

If all these factors are not diagnostically assessed and taken into account, there is a possibility that professional cleaning will have to be repeated regularly and frequently, and that no real change in the situation will occur.
Determining the possible diagnostic parameters within the framework of diagnosis-based individual prophylaxis (DIP) enables a more differentiated care concept. This provides an answer beyond the mere presence of plaque to the question of "why" [5]. A diagnosis of the individual caries risk can only be based on the generally accepted caries aetiology [6, 7, 8, 9].

CARIES AETIOLOGY BASED ON THE EXTENDED ECOLOGICAL PLAQUE HYPOTHESIS

The basis of a caries risk diagnosis is knowledge of the aetiological parameters in the extended ecological plaque hypothesis ([10, 11, 12, 13, 14, 15]; **Fig. 1**).
The homeostatic biofilm changes due to unfavourable patient-specific parameters such as high-frequency consumption of fermentable carbohydrates, insufficient hygiene, unfavourable host defence (e.g. diabetes), unfavourable saliva qualities and parameters. These clinically visible and anamnestic parameters (**fig. 1** grey) trigger clinically undetectable changes in the biofilm (**fig. 1** blue).
Only the increase in plaque is impressive, which, according to experience, is attributed to insufficient hygiene efforts at home. Unfavourable individual parameters (diet, hygiene) lead to an increase in the total number of cariogenic and acid-tolerant microorganisms. Acid tolerance and the subsequent selection of non-Streptococcus mutans bacteria with low pH values seem to play a decisive role in destabilising plaque homeostasis.
In this environment, Streptococcus mutans and Lac- tobacilli can promote lesion development through excessive growth by maintaining an environment characterised by a persistently low pH. Therefore, high levels of Streptococcus mutans and Lactobacilli can be considered biomarkers for sites with particularly rapid caries progression [16, 17, 18, 19].
This leads to a shift in the biofilm from commensal and apathogenic germs to pathogenic, aciduric and acid-forming germs. The diversification of the biome is lost [17, 18]. Due to the increased acid-forming potential in the oral cavity, the saliva pH can decrease and the buffer capacity is reduced. Through

patient-specific parameters (e.g. extensive medication intake, chewing behaviour not conducive to saliva secretion) simultaneously reduce the saliva secretion rate and the clearance rate with corresponding consequences (e.g. reduced remineralisation phenomena, loss of the rinsing function, bad breath).

A caries-pathogenic biofilm has developed; this causes a disturbance in the homeostatic state of the oral system and is characterised by a strong increase in pronounced acid producers [20].

The role of Streptococcus mutans is not limited to acid formation alone: Streptococcus mutans is the main producer of extracellular polysaccharides. This matrix acts as a diffusion barrier that plays a major role in preventing the acid formed in the plaque from escaping into the oral cavity. At the same time, this matrix protects the biome on the tooth from the natural defence functions of saliva [21, 22, 23]. In addition, Strepto- coccus mutans maintains the acidic environment in which the lactobacilli can overgrow the system.

Lactobacilli, themselves not actively involved in plaque build-up, use the pH environment created by Streptococcus mutans to drive caries progression [24, 25]. Therefore, they are also correspondingly present in active carious lesions [26, 27].

In summary, this means that a determination of the number of Streptococcus mutans and Lactobacilli can be an indicator for the condition of the biome. The detection of these germs in adequate numbers, possibly in conjunction with the functional saliva parameters, can be used as a diagnostic basis for the preventive measures to be taken (DIP).

This targeted prevention can bring about a reversal of the pathological condition. The measures available for this are manifold and include certain therapeutic aids, medications, probiotic substances, fluorides, antibacterial varnishes or gels, hygienic intensification, sealing and others. They do not cause the plaque to disappear completely - but they do reduce the pathogenicity of the plaque on its way back to a homeostatic state. "In this respect, it is no longer appropriate to speak of "bad" plaque in general and to demand its rigorous removal from the oral cavity" [28].

Axelsson [29] and König [30] have considered some of these factors to determine caries risk: For example, in 1990 Axelsson defined "no caries risk" and a "no caries risk".

"high caries risk", as can be seen in **Tab. 1** [29]. Even in 1990, components of a risk definition were already included, which could only be determined by analysing subclinical risk parameters.

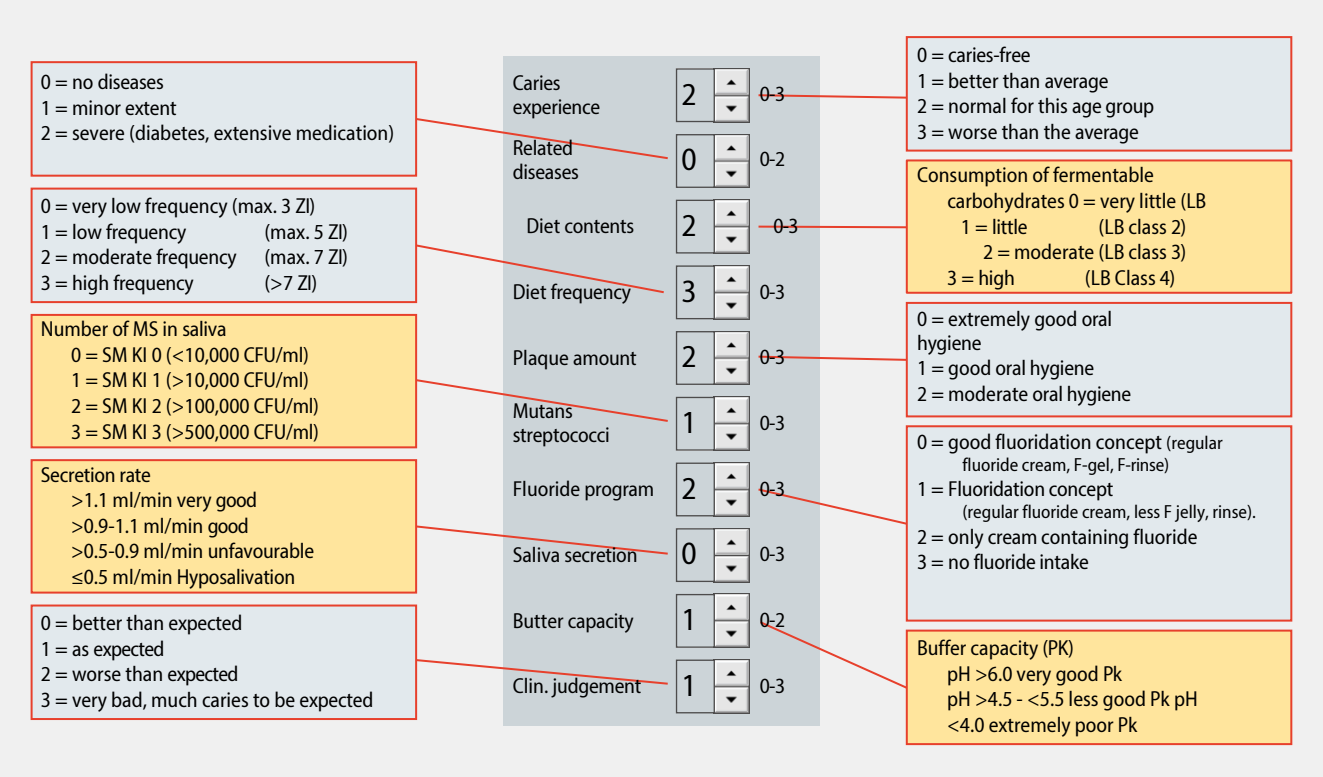


Fig. 2 The "Cariogram" calculates the probability of no caries occurring after entering at least seven determined risk parameters. Subclinical risk parameters to be determined are highlighted in yellow. In practical use, it has proven useful to modify or supplement some of the risk parameters. CFU "colony forming unit", F Fluoride, Kl Class

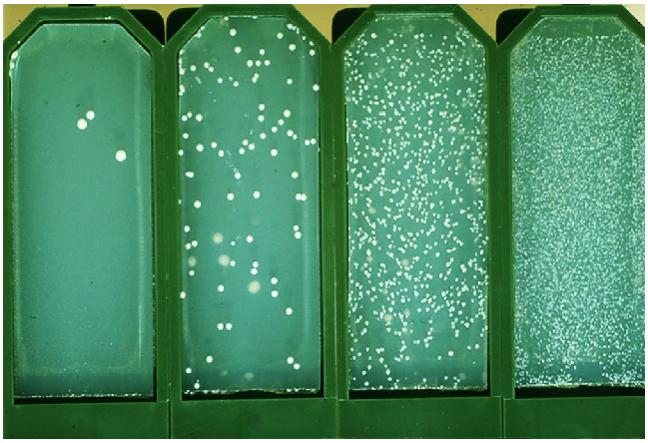


Fig. 3 Increasing sugar consumption by the patient means increasing numbers of lactobacilli (determined using the CariesScreenTest, formerly CRT bacteria, or the CariesScreenTest+P) (courtesy of Fa. IvoclarVivadent)

("saliva test") were to be determined. However, this definition was rather to be interpreted as a static description of the condition and did not necessarily do justice to the claim of the dynamic process. Therefore, in the future, we should not always speak of the caries risk, but of determining the current caries activity of the patient. This corresponds more to the dynamic process of the extended ecological plaque hypothesis and can only be achieved with the help of the "cariogram". In this, not only the risk parameters described by Axelsson are recorded, but also additional factors that have an influence on the assessment of the caries risk.

Notice:

- According to the dynamic process of the extended ecological plaque hypothesis, the instantaneous caries activity is to be determined

CARIOGRAM

An exact diagnosis of the caries risk - as the basis for further preventive treatment steps - requires the recording of all clinical and subclinical risk parameters. Shift

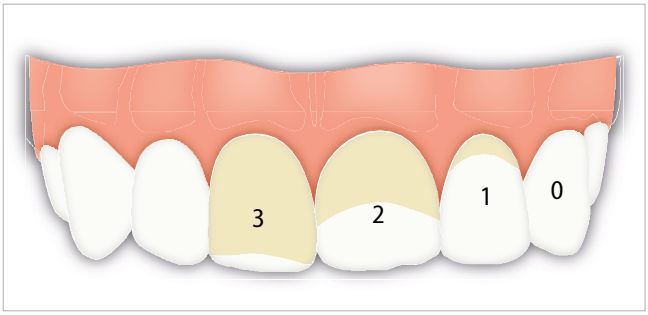


Fig. 4 Plaque classification according to the hygiene (HI) index

In recent decades, various authors, for example Axelsson, König and others, have formulated different concepts for assessing individual risk.

A process that had already been developed in the late 1990s by Bratthal

[31] and others, enables an interpretation and weighted analysis of the different factors involved in the development of caries. The cariogram offers the ideal prerequisites for a systematic approach; it graphically illustrates the caries risk or the chance of avoiding new caries in the future.

At the same time, it can be better integrated into the picture of the dynamic process of the current caries activity. The Cariogram can be downloaded from the internet, but only the 64-bit English version is available (Cariogram download: Malmö University (mau.se); the mobile version is also available as an app (**Fig. 2**).

The following parameters are determined (the exact specification is shown in **Fig. 2**):

- previous caries experience ("caries experience").
- Individual health status of the patient ("related diseases"): Somatic diseases that could have an influence on the oral cavity biotope, such as diabetes or extensive medication, are determined here.
- Consumption of fermentable carbohydrates ("diet contents"): The amount can be estimated from the lactobacilli count. The content of lactobacilli in the saliva sample is not an original part of the cariogram. The addition of the lactobacilli count also allows an estimation of the amount of sugar ingested. In addition to the frequency of sugar intake, this is another independent risk parameter according to the guideline on caries prophylaxis from 2016 (www.dgzmk. de). Knowledge of the lactobacilli count also opens up an objectifiable possibility to check the success of the preventive efforts and the patient's compliance during the follow-up. Thus, a dynamic component can be added to the cariogram (**Fig. 3**).
- Frequency of sugar intake ("diet frequency"): The amount of fermentable carbohydrates and the sugar they contain determine the cariogenic potency. Only the way of sugar intake - i.e. the frequency of sugar intake - determines whether cariogenic potency also becomes cariogenic efficacy. Under this aspect, the frequency is recorded and weighted accordingly. While in the Cariogram the actual number of meals/day, including inter-meal periods, is recorded, here the number of sugar impulses (ZI) is also determined. In this way, the counselling discussion is directed from the outset to the most important parameter for preventive care: the avoidance or restriction of an unnecessary intake of fermentable carbohydrates.

- The amount of plaque found ("plaque amount"): The patient's oral hygiene is assessed under this item. This can be individually classified by the practice according to different indices (**Fig. 4**).
- Content of Streptococcus mutans in saliva ("mutans streptococci"): This value provides information about the general level of colonisation of the oral cavity with the caries pathogen Streptococcus mutans. A high content in the saliva indicates the risk of persistent caries activity [32].
- Of course, only the bacterial counts of Streptococcus mutans are determined, and not those of other acid-forming germs. Streptococcus mutans still has a key position, as it is the only one capable of forming extracellular polysaccharides, whereas most other acid-forming organisms lack this property ([21, 23], **Fig. 5**).
- Fluoridation concept ("fluoride programme"): Here, the individual fluoridation concept of the patient is recorded.
- Saliva secretion rate ("saliva secretion") and buffer capacity ("buffer capacity"): The functional saliva parameters are recorded. These can be determined with the help of the CariesScreenTest+P (**Fig. 7**).
  By determining the saliva flow rate, it is possible to determine whether there is sufficient saliva. The natural protective function of saliva, the rinsing function, the thinning effect during sugar intake, the removal port and the availability of minerals for remineralisation as well as the clearance rate depend on the amount of saliva available. The secretion rate should be at least about 1.0 ml/min. Values below this reduce the clearance rate and the remineralisation potential and are thus favourable to caries [7, 30, 33,
  34, 35].
  Buffer capacity is a crucial protective mechanism of the oral cavity against food and plaque acids. Very good buffer capacities are associated with pH values above 6 and acceptable ones with pH values between 4.5 and 5.5. Unfavourable buffer capacities are present at pH values <5 and poor buffer capacities at pH values <4. The buffer capacity is related to the saliva flow rate. Thus, reduced buffer capacities were also found in connection with a corresponding caries finding in the case of a reduced saliva flow rate; high saliva flow rates result in good buffer capacities due to the increased sodium bicarbonate content [36].
- Individual assessment ("clinical judgement"): The dentist's individual caries risk assessment can be entered here, based on the findings and his or her experience.

The cariogram weights the individual parameters according to their importance and assigns them different areas of a circle according to their respective effect on the caries risk. The determined individual

Fig. 5 Increasing content of Streptococcus mutans (determined using the Caries Screen Test (formerly CRT bacteria) and the CariesScreenTest+P). (Courtesy of IvoclarVivadent)

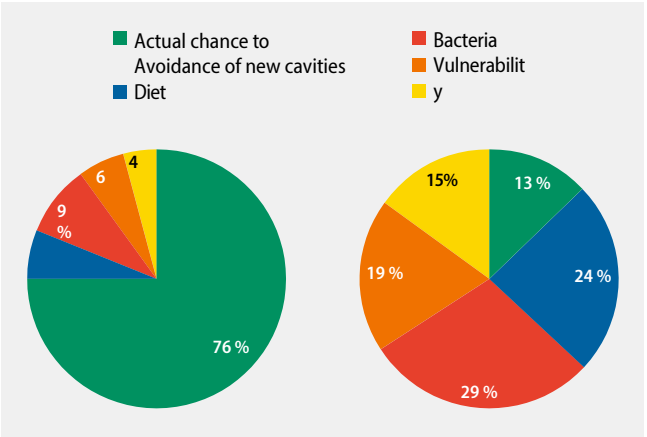


Fig. 6 Result in the "Cariogram": The green area indicates the probability of no caries. On the left a low caries risk, on the right a high caries risk.

Risk areas are highlighted in the presentation of the results. summarised in the Cariogram into the following 4 groups ([31], **Fig. 6**):

- Nutrition: Frequency of food intake and composition of food (the area is shown in dark blue in the cariogram),
- Bacteria: Plaque quantity and qualitative composition of the plaque (number of Streptococcus mutans/lactobacilli; the area is shown in red in the cariogram),
- Susceptibility: resistance of the tooth substance (fluoridation) and quality of the saliva (secretion rate, buffer capacity, pH value; the area is shown in turquoise in the cariogram),
- Circumstances: carious tooth damage in the past and general state of health (the area is shown in yellow in the cariogram).

The graphical representation of the result looks as follows. The green area corresponds to the probability that no new caries is to be expected. It can be seen that the determined subclinical risk parameters (shown in red) have a decisive influence in determining the individual caries risk.
Therefore, it is obvious that the predictive value of the results of the Cariogram software is higher if saliva parameters and the Streptococcus mutans count are determined and included in the evaluation [37].

Notice:
- The cariogram graphically illustrates the possibilities for preventing new caries

DETERMINATION OF THE SUBCLINICAL SALIVA PARAMETERS

All saliva parameters can be determined with the help of the CariesScreenTest+P (Aurosan, Essen). This test kit contains tools for determining the bacterial saliva parameters (formerly CRT bacteria, IvoclarViva dent) and the functional parameters ([38], **Fig. 7**).
Procedure:
- Collect saliva,
- Determine the pH value of the saliva,
- Determine secretion rate,
- Determine buffer capacity,
- inoculate microbiological culture medium with saliva.

In addition, the pH value of the saliva can be determined with the method. This is not recorded in the cariogram. However, its knowledge often opens up a more differentiated view of the condition of the oral biome. The resting pH value should be ≥7, especially for exposed root surfaces, where demineralisation already starts at a pH value of 6.7.

DIAGNOSTIC CONSEQUENCES FROM THE RISK ANALYSIS

The subsumption of the collected data and the associated graphical representation allow a clear weighting and delimitation of the cariogenic risk parameters.
At the same time, this enables the individually optimal therapeutic consequence.
The selection of preventive treatment steps and recommendations is thus based on a clearly comprehensible and objectifiable diagnosis. At the same time, however, this opens up the possibility of checking the efficiency of the measures used through a renewed analysis of the risk parameters found in the first examination. In this way, the DIP becomes feasible.
Some examples of preventive measures for the individual diagnostic areas are listed below.

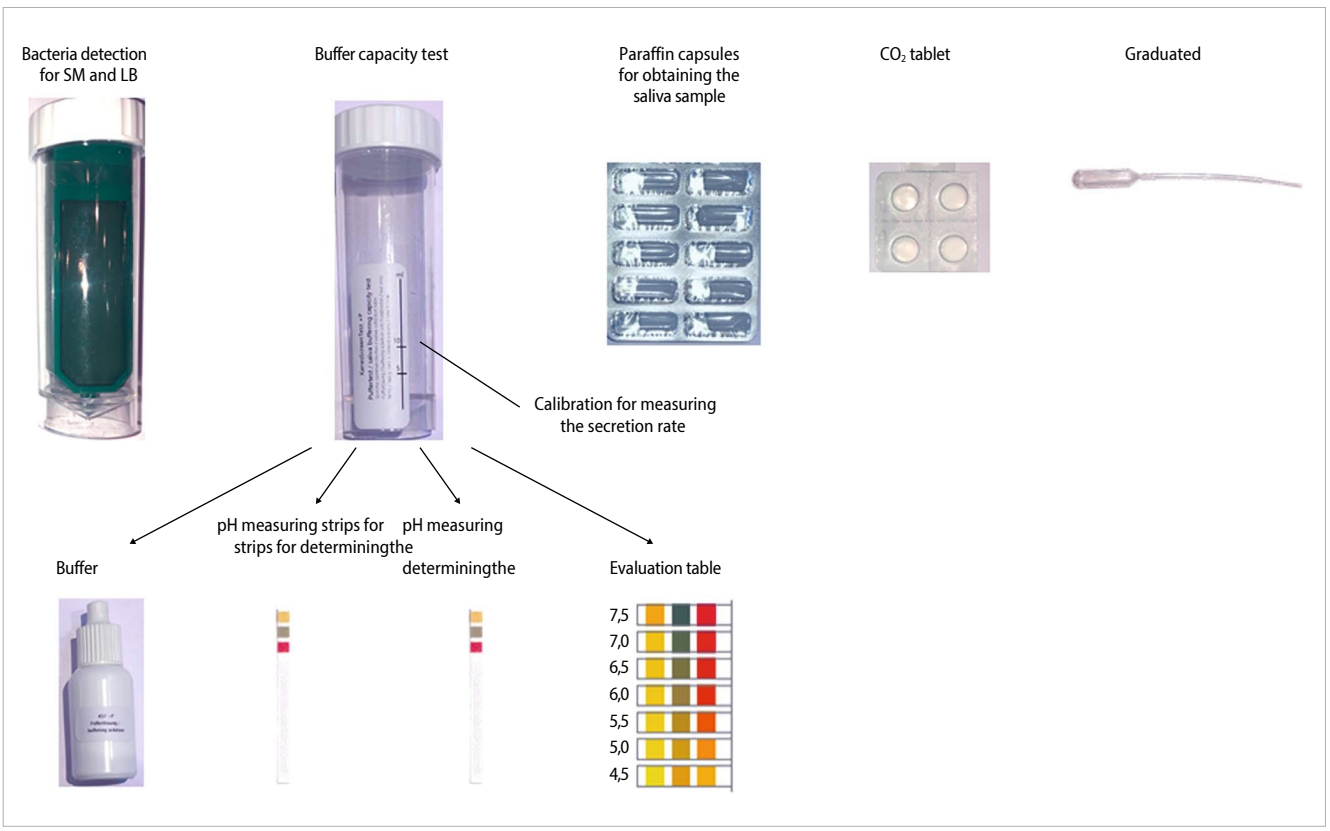


Fig. 7 Contents of the CariesScreenTest+P. LB Lactobacilli, SM Streptococcus mutans. (With kind permission of the author)

Notice:

- The selection of preventive treatment steps is based on clearly comprehensible and objectifiable diagnostics

Nutrition

The therapeutic consequence consists of informing the patient and, ideally, the resulting dietary care (cariogram, **fig. 6**, dark blue). Therapeutic recommendations are the use of sugar substitutes (e.g. xylitol) and, in the presence of a reduced salivary secretion rate, the indication of an appropriate chewing-active diet [39].

New studies indicate that the cariogenic efficacy of sugar intake should be seen in the context of the fluoridation concept. There is a high correlation between the amount and frequency of sugar intake, so both can only be assessed together.

In this respect, a concept for reducing the frequency with a simultaneous continuous reduction of the amount of sugar while applying an individually adapted fluoridation concept is recommended.

Bacteria

As an early coloniser of the child's oral cavity, Streptococcus mutans (cariogram, **Fig. 6**, red) is of decisive importance for dental health in the first years of life [40, 41]. The colonisation of Streptococcus mutans in the child's oral cavity at the age of two years causes a high caries risk, with a 94% caries prediction for the age of four to six years. Detection of Streptococcus mutans in children's plaque increases the caries risk fourfold.

This finding - often in the absence of clinical caries - triggers extensive preventive measures to avoid caries prevalence in the future [42, 43, 44, 45]. In a systematic literature review, Harris et al. were able to show that the early detection of Streptococcus mutans in conjunction with a frequent intake of fermentable sugars and poor oral hygiene are the most significant risk factors for early childhood caries. However, the authors were also able to show that an unfavourable dietary behaviour, when paired with good oral hygiene, does not always have to be a risk factor [46].

Due to its ability to form extracellular polysaccharides, Streptococcus mutans has a decisive function in plaque build-up and acid formation in the plaque. In particular, when high Streptococcus mutans counts are combined with high Lactobacillus counts, there is a high risk of caries [40, 47].

The amount of bacteria (Streptococcus mutans/Lactobacilli) in the plaque or in the oral cavity therefore plays an important role in the risk of caries. In the case of caries pa-

INFOBOX 1 CARE OF THE PATIENT WITH PROBLEMATIC FUNCTIONAL SALIVARY PARAMETERS

Professional concept

► Control of domestic hygiene measures

Professional tooth cleaning (possibly with stannous fluoride), including cleaning of the base of the tongue.

► Regular control of subclinical parameters

Application of chlorhexidine-containing gels/varnishes in the approximal spaces (in conjunction with professional tooth cleaning).

► Deep-drawing foils for professional and home application of Elmex jelly (once a week, 5 minutes).

► Thermoforming sheets for the professional and domestic application of chlorhexidine gel

► Recommendation of saliva substitutes

Domestic concept

Gustatory stimulation of the salivary glands (dental care chewing gums, lozenges with microgranules)

► Possibly saliva substitute (Saliva®)

► Approximal hygiene and tongue cleaning

► Duraphat toothpaste for sensitive tooth necks

► Elmex Erosive Protection (for reflux)

► Meridol rinses for halitosis

► Use of an individually manufactured deep-drawing splint as a medicine carrier

A higher proportion of Streptococcus mutans is always found in patients than in caries-free individuals [48, 49]

Interventions to reduce pathogenic salivary germ counts are manifold. In many cases, germ count-reducing measures using intensive therapy with chlorhexidine (CHX) are indicated. The patient should brush his teeth with CHX gel three times a day for two days. Fissure sealants may also be considered depending on the occlusal morphology. Fissure sealants considerably reduce the ecological niche for germ colonisation. In addition to the local preventive effect of protecting the occlusal surface, this also has a positive influence on the biotope of the oral cavity by reducing the potential colonisation area [50, 51].

Receptivity

Functional saliva parameters and the established fluoridation measures are summarised here (Cario- gram, **Fig. 6**, turquoise).

Reduced saliva flow is not necessarily a question of age: side effects of systemic medication can occur in any age group.

A reduced saliva flow (hyposalivation) always poses a risk to the root surfaces, as these can be damaged.

can be demineralised from a pH value of 6.7. **Infobox 1** shows examples of therapeutic options that could be selected depending on the specific case.

Circumstances
Here, individual factors are determined that can strengthen or weaken the effect of risk factors (cariogram, **fig. 6**, yellow). For example, it is known that the glucose level in saliva is increased in people with diabetes. This is naturally a suitable nutritional basis for cariogenic germs [52].

RESUME

Knowledge of subclinical parameters provides important detailed information for assessing a patient's oral health or disease. The elimination of a risk parameter is more important for individual health prediction than the persistence of this parameter for disease prediction.
Targeted antibacterial and fluoride therapy based on microbial saliva findings and knowledge of the fluoride concentration in saliva positively changes the balance between pathological and protective caries risk factors [53]. Ultimately, this is an essential goal in prevention-oriented dental practice and can be successfully implemented in practice with the help of a DIP. For this purpose, it is necessary to know the current caries activity of the patient.
Thus, individual prophylactic concepts integrate very well into the "oral medicine" postulated by the German Society for Dental, Oral and Maxillofacial Medicine (DGZMK) [54]. For successful preventive care, it is not only important to collect all the risk factors mentioned above. It is much more important to understand and comprehend why these risk factors are present. This understanding alone helps to develop and implement individual preventive concepts and to check their long-term effectiveness. The frequently formulated position of an "individual assessment of the oral microbiome in combination with clinical parameters for the early detection of individuals with an increased risk of disease" is thus feasible [55].

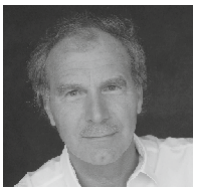
CONCLUSION FOR THE PRACTICE

- The homeostatic biofilm changes due to unfavourable patient-specific parameters such as high-frequency consumption of fermentable carbohydrates, insufficient hygiene, unfavourable host defence and unfavourable saliva qualities and parameters.
- These clinically visible and anamnestic parameters trigger clinically undetectable changes in the biofilm.
- The exact diagnosis of caries risk requires the recording of all clinical and subclinical risk parameters.
  The Cariogram offers the ideal prerequisites for this and shows the way to the individually optimal therapeutic consequence (diagnosis-based individual prophylaxis, DIP).
- A variety of measures are available for DIP, including certain therapeutic aids, medications, probiotic substances, fluorides, antibacterial varnishes or gels, hygiene intensification, sealants and others. They cause a change in the pathogenicity of the plaque back to a homeostatic state.

LITERATURE

You will find the bibliography in the article on www.springermedizin.de/der-freie-zahnarzt under "Supplementary content".

CORRESPONDENCE ADDRESS



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Curriculum vitae-- From 1989 to 1998, Dr Laurisch was vice-chairman of the Society for Paediatric Dentistry and Primary Prophylaxis in the German Society for Dental, Oral and Maxillofacial Medicine (DGZMK) and from 2001 to 2009 he was a member of the board of the DGZMK. In 2002, he received the Wrigley Prophylaxis Award for "many years of commitment and service to dental education in preventive dentistry" and in 2004 the Practitioner Award of the German Society for Paediatric Dentistry. Since 2003, Dr Laurisch has held a lectureship at Heinrich Hei- ne University, Düsseldorf. Since 2012, he has been Vice President of the German Society for Preventive Dentistry (DG- PZM) in the DGZ. In 2013, he was awarded the Golden Honorary Pin of the DGZMK for his services to the scientific development of concepts in prophylaxis and prevention of oral diseases as well as for his many years of involvement in the management committees of the DGZMK and the APW. Laurisch is the author of numerous publications on the topics of prevention, caries risk and practice management and is also the developer of treatment devices and detection methods.

Compliance with ethical guidelines-- Conflict of interest. The author is the developer of the CRT bacteria produced and distributed by IvoclarVivadent until 2018. In cooperation with Aurosan, he developed the successor product KariesScreen Test and KariesScreenTest+P.
The author did not conduct any studies on humans or animals for this article. For the studies listed, the ethical guidelines stated in each case apply.

CME questionnaire

- Free participation for FVDZ members and subscribers on CME.SpringerZahnmedizin.de
- The participation period is 12 months, you can find the participation deadline online at the CME course

How does a dysbiotic shift of the ecological balance in the plaque occur?

- ☐ Due to occasional insufficient domestic hygiene
- ☐ Through moderate intake of fermentable carbohydrates, the
- ☐ Massive intake of fermentable carbohydrates in combination with insufficient domestic hygiene
- ☐ There is no shift in equilibrium if the patient regularly takes the pro-
  professional dental cleaning in the dental practice.
- ☐ Through extensive fluoridation measures, especially in the dental practice

What role does Streptococcus mutans play in the ecological plaque hypothesis?

- ☐ It no longer has any significance at all, since many bacteria of the non-streptococcus mutans group can also produce acid.
- ☐ Streptococcus mutans is a major acid producer and, due to its ability to produce extracellular polysaccharides, has a
  essential in the maintenance of caries activity.
- ☐ The metabolic activity of Streptococcus mutans is inhibited by the presence of lactobacilli.
- ☐ Bacteriological evidence of Streptococcus mutans in the patient's oral cavity is of no significance.
- ☐ Streptococcus mutans is inhibited in its acid-forming capacity when the saliva pH drops.

According to the definition by Axelsson [29], at what level of lactobacilli is a high caries risk assumed?

- ☐ 10,000 ml/min
- ☐ 25,000 ml/min
- ☐ 50,000 ml/min
- ☐ 75,000 ml/min
- ☐ 100,000 ml/min

Axelsson [29] has defined risk factors for a high caries risk. Which of the risk factors listed is more likely to indicate no caries risk?

- ☐ Streptococcus mutans values >500,000 ml/min
- ☐ pH value <4.5
- ☐ Excellent oral hygiene habits
- ☐ High consumption of sticky, sugary products
- ☐ Very high DMF-T value

What is graphically illustrated by the cariogram?

- ☐ The likelihood that no caries will develop
- ☐ The likelihood that caries will develop
- ☐ The likelihood that sugar consumption alone can cause tooth decay
- ☐ The likelihood that insufficient oral hygiene alone will cause caries to develop
- ☐ That fluoridation measures alone can prevent caries

What is the minimum number of risk parameters that must be entered in order for the cario- gram to determine the chance of healthy teeth?

- ☐ Three parameters
- ☐ Four parameters
- ☐ Five parameters
- ☐ Six parameters
- ☐ Seven parameters

What should be the minimum secretion rate of the saliva in order not to reduce the clearance rate or the remineralisation potential and thus be conducive to caries?

- ☐ 0.1 ml/min
- ☐ 0.5 ml/min
- ☐ 1.0 ml/min
- ☐ 1.5 ml/min
- ☐ 2.0 ml/min

Which value is not recorded in the cariogram, but can be additionally determined by appropriate test kits to enable a more differentiated view of the state of the oral biome?

- ☐ Buffer capacity of the saliva
- ☐ Saliva pH value
- ☐ Fluoridation concept
- ☐ Frequency of sugar intake
- ☐ Amount of plaque found

By what factor is the caries risk of children increased when Streptococcus mutans is detected in plaque, even if the child is clinically caries-free at the time of detection?

- ☐ Two
- ☐ 2,5
- ☐ 3,5
- ☐ Four
- ☐ Six

Regardless of a patient's age, what is most likely to have a noticeable impact on salivary flow rate?

- ☐ Tooth colour and shape
- ☐ Use of dental floss/interdental space brushes
- ☐ Number of teeth treated with root canal therapy
- ☐ Taking systemic medication
- ☐ Use of fluoride toothpaste

CME certification

This training course was prepared in accordance with the guidelines of the German Dental Association, the German Society for Dental, Oral and Maxillofacial Medicine and the Federal Association of Statutory Health Insurance Dentists on further training in dentistry of 01.01.2006.

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