МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
1-я КАФЕДРА ТЕРАПЕВТИЧЕСКОЙ СТОМАТОЛОГИИ

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КАРИЕС: ЭТИОЛОГИЯ, ПАТОГЕНЕЗ, ПРОФИЛАКТИКА

DENTAL CARIES: ETIOLOGY, PATHOGENESIS, PREVENTION

Учебно-методическое пособие

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Рассматриваются вопросы этиологии, патогенеза, профилактики кариеса зубов. Материал базируется на имеющихся в отечественной и зарубежной литературе современных представлениях по данной проблеме.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке.

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1. DEFINITION. THEORIES OF CARIES

Caries is a chemical dissolution of the tooth with the subsequent destruction of the organic matrix, remaining after the calcium salts dissolution. G. V. Black, 1908.

Caries (lat. caries — bacterial decay) is gradual destruction of bone tissue or teeth due to the degenerative or infectious process in the bone or periosteum. SES, 1981.

Pathological process manifesting after tooth eruption with subsequent tooth demineralization and cavity formation. E. V. Borovskiy, 2003.

Caries is infectious disease with progressive tooth demineralization, starting on the outer surface of enamel or bare cement. Mosby's Dental Dictionary, 2004.

Theories of caries (history):

1. Nerve-trophic theory (D. A. Entin, 1928).
2. Physico-chemical theory (D. A. Entin, 1928).
3. Trophic theory (I. G. Lukomskiy, 1948).
4. Exchange theory (A. E. Scharpenak, 1949).
5. Chelation theory (Schatz, Martin, 1954).
6. Ecological theory of homeostasis (O. Fejerskov, E. Kidd, 2004).
7. The concept of counter effects on the dental pulp (A. I. Rybakov, 1971).

CHEMICO-PARASITIC CARIES THEORY (Miller, 1890) (fig.1)

Arguments:

The pH of plaque can be reduced to a critical level.

In cavity PH is significantly lower than in saliva.

Caries develops faster, when saliva contains more acid-forming microorganisms.

There are no changes in organic substance in the early stages of enamel caries.

Enamel decalcification is confirmed histologically.

Doubts:

In vitro all enamel surface dissolves, in case of caries it dissolves partially.

There are always different conditions in vitro and in the oral cavity.

In rats experimental caries develops at pH ≥ 7.0.

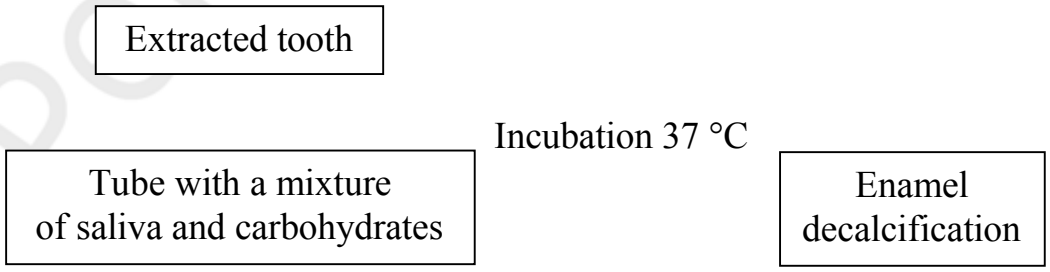


Fig. 1. Miller's experiment

2. ETIOLOGY AND PATHOGENESIS

Plaque is soft deposits, forming a biofilm on a tooth surface or other solid surfaces in the oral cavity, including fixed and removable restorations (Bowen, 1976).

Different strains of bacteria are capable of organizing associations for mutual survival.

This formed the basis of the "biofilm" concept, which is considered as a single active biological being, interacting in the complex state with the human body.

Biofilm — specialized bacterial ecosystem, which provide vitality, preservation of its species and an increase in the general population.

More than 95 % of bacteria are found in biofilms.

Biofilm formation is a constant dynamic process.

It proceeds in three phases:

1. Pellicula formation (precipitation of saliva glycoproteins and gingival fluid).

2. Bacterial colonization of the pellicula.

3. Biofilm maturation (intense reproduction and differentiation of bacterial strains).

Biofilm consists of 50–300 layers of different bacterial strains (15–20 % of the total substrate).

1 mg of plaque contains over 400 million of microorganisms. G. N. Jenkins, 1970.

Plaque is white or yellowish soft substance localized in the neck of the tooth or on its entire surface.

Plaque cannot be washed off with water.

Plaque is not erased during chewing food.

Plaque is easily removed by mechanical cleaning with toothbrush.

Intensity of forming depends on:

1) secretion rate, saliva composition and viscosity;
2) microflora;
3) the desquamation of the oral mucosa epithelium and the self-cleaning;
4) it has individual and age-specific peculiarities.

Str. mutans produces extracellular glucan from sucrose, which promotes the development of an adhesive cariogenic plaque on the tooth surface and initiates the beginning of enamel demineralization.

Str. mutans prevails in the plaque during microbial plaque retention in the fissures and enamel demineralization.

Lactobacilli join the development of the pathological process 12 months before the detection of primary caries.

Lactobacilli dominate during the stage of cavity forming.

Pathogenic properties of cariogenic bacteria:

1. Str. mutans has a unique transport system for the transfer of fermentable sugars (phosphoenolpyruvate, phosphotransferase).

2. Sugar is converted into acid.

3. Development of extra-and intracellular polysaccharides (glucans and fructans), promoting the deposition of plaque matrix.

4. Sugar metabolism maintenance in low pH (Str. mutans has tolerance to the acidic environment).

The worse oral hygiene, the harder carious disease. However, there are no convincing arguments of a direct relationship between hygiene and caries:

Plaque is not the direct cause of caries disease, but the risk of tooth decay reduces by reducing the amount of plaque.

The amount of plaque is less important than its prolonged retention.

Food cariogenicity — is a relative food substrate potential capable of inducing biochemical processes that initiate acid demineralization of tooth enamel.

Food cariogenicity depends on:

1) food adhesiveness;
2) amount of acid resulting from carbohydrate fermentation;
3) amount and microbial composition of plaque;
4) enamel resistance;
5) fluorine concentration in food and water;
6) amount and properties of saliva;
7) time of acid neutralization.

Frequent carbohydrates abuse contributes to:

1) the microbial composition of plaque;
2) the increase in the rate of plaque formation;
3) the increased caries intensity.

Milk sugar (lactose) has the lowest cariogenic potential among disaccharides. Other di-and monosaccharides are potentially “dangerous” for the teeth health, especially when present in the food.

Sucrose significance in caries etiology is confirmed by:

– a sharp decline in the caries intensity during the two world wars, when the consumption of sugar was significantly reduced due to the economic breakdown;

– caries incidence rate, coinciding with an increase in sucrose intake among population of industrialized countries;

– ability to artificially cause tooth decay in people (volunteers) during 23 days (people rinse their mouths with sucrose solution 9 times per day);

– caries is rarely detected in patients suffering from hereditary intolerance to sucrose;

– among confectioners multiple cervical caries of front teeth occurs 100 times more frequently.

– Japan: increased prevalence of caries occurred during the period when sucrose consumption exceeds 10 kg per year for 1 person;

– I. Tristan (Atlantic Ocean): the prevalence of caries due to the use of "civilized" foods increased from 5 % to 30 %.

Caries resistance and caries receptivity affect the caries course.

Resistance to caries is a complex of hard tissues and oral fluid (saliva) defining tooth resistance to caries.

Sensitivity to caries is absence of one or more caries resistance factors.

The course of caries depends on:

1) oral fluid composition and properties;
2) structural features of hard tissues;
3) biochemical composition of hard tissues;
4) functional state of organs and body systems during the formation and maturation of dental tissues.

Plaque PH reduction to a critical level is a reversible process: oral fluid (saliva) prevents possible enamel demineralization by neutralizing acid and plaque pH returning to the original level standards.

The presence of plaque and short-term presence of sugar do not cause caries: oral fluid supports structure of the tooth enamel due to the balance of enamel hydroxyapatite crystals dissolution and their formation.

Sensitivity to caries depends on their group property and morphological features: first molars, incisors of the lower jaw are more frequently affected by caries.

Resistance to caries is shown in an expressed spherical crown, smooth surface, thick enamel.

Caries sensitivity zones are fossae, sulculi, plicae, grooves, thin enamel.

The incisors, canines and premolars are affected mainly on the contact surfaces.

Caries occurs predominantly on the occlusal and contact surfaces of molars.

At the age of 40–50 years, caries on the contact surfaces occurs in about 4 times more often than on the occlusal surfaces.

The low concentration of Ca, P and a high concentration of carbonate in the enamel promote rapid development of caries.

Plaque trace substances (fluorine, molybdenum, vanadium, strontium) reduce tooth sensitivity to caries; selenium increases tooth sensitivity to caries.

Fluorine is the main component of tooth resistance to caries.

Features of plaque microorganisms:

1. Firmly hold on the tooth surface, dissolve the protective and organic tooth layer (pellicula).

2. Predetermine the direct contact of the acids formed in plaque with enamel minerals.

Under certain conditions (frequent use of carbohydrates, poor oral hygiene) on a limited enamel surface with plaque pH decreases to a critical level.

The pathogenesis of caries disease is based on:

1) factors directly affecting the teeth surface and involved in the biochemical processes that lead to caries;

2) status, conditions or circumstances that are indirectly associated with biochemical processes, or influencing them, or reflecting the degree of their activity.

Of paramount importance are :

1) plaque (cariogenic microorganisms, for example, Streptococcus mutans);
2) food carbohydrates;
3) saliva properties;
4) fluoride impact.

Of indirect importance are:

1) social and economic factors;
2) factors associated with general health;
3) epidemiological factors or circumstances;
4) clinical signs.

ENAMEL CARIES

H + ions in the area of long-existing source of acid production cause:

1. **Uniform enamel dissolution** if acid production occurs actively.
2. **Uneven dissolution** if acid is partially neutralized by saliva components.

Pathogenesis:

1. Superficial enamel layer is difficult to dissolve (it has more fluorapatite).
2. H + ions enter the subsurface layer, causing demineralization at the least resistant sections (prism striation, Retzius strips).
3. In clinic this corresponds to the initial caries (the stage of "white" spot).
4. Demineralization area extends parallel to the tooth surface, the concentration of H + is maintained by the acid production on the enamel surface, covered with dental plaque.
5. Deep penetration of H + ions in the process of early enamel demineralization is impossible due to their partial neutralization.
6. Enamel microspace is gradually increased. Microorganisms penetrate these microspaces transferring source of acid production into enamel.
7. Demineralization extends both along the tooth surface and inside, forming a cone-shaped lesion.

8. Superficial enamel layer lesions over an area of destruction dissolves slower due to the greater resistance to dissolution due to the presence of fluorapatite and more pronounced remineralization processes.

9. Micropores in the superficial enamel layer expands due to the continuation of acid production.

10. Enamel becomes thinner, breaches. A defect visible to the naked eye formes.

11. In clinic this corresponds to the enamel caries (superficial caries).

DENTINE CARIES

Pathogenesis:

1. Caries extends along the enamel-dentine border.

2. Microorganisms can invade dentinal tubules leading to dentine demineralization.

3. Dentine organic matrix is dissolved by proteolytic enzymes.

4. In invasion sites dentinal tubules expand and join forming microscopic oval cavities which contain bacteria, destroyed organic matrix ("dissolution focuses", Miller). With the lapse of time the foci expand, compressing and curving the adjacent dentinal tubules.

5. Simultaneously, dentine disintegration occurs (formation of transverse fissures closer to the enamel-dentine border).

6. Microorganisms covers dentine.

7. Dentine site from its surface to microcavities melts. Clinical carious cavity forms.

8. With the lapse of time its size increases, which corresponds to dentin caries (average, if the process is localized in the mantle dentine, deep — in the parapulpar dentin).

9. The process extends into the pulp, periodontium, root cement.

Epidemiology is a science that studies the relationship between different factors that determine the frequency and distribution of diseases in human society.

Prevalence of diseases is the percentage of sick people to the total number of examined patients. The prevalence of major dental diseases (caries, periodontal disease) is expressed in %.

Intensity of diseases is a numeric expression of one disease sign or sum of the disease signs to an average of one person among the total number of examined patients.

CARIES INTENSITY — INDEX DMFT

WHO recommends the following key (basic, index) age-related groups on the caries morbidity:

– 5–6 years: investigator determines the number of children free of caries (caries prevalence);

– 12 years: the group of global caries monitoring on the international level (investigator determines the intensity of caries, DMFT);

– 18 years: investigator determines the component «M» in the structure of DMFT index;

– 35–44 years and 65–74 years (investigator determines DMFT, the number of functioning teeth and percentage of toothless population).

3. CLASSIFICATION

International classification ICD-10

K02	Dental caries*
K02.0	Caries limited to enamel. White spot lesion [initial caries]
K02.1	Caries extending into dentine
K02.2	Caries of cementum
K02.3	Arrested caries
K02.4	Odontoclasia
K02.8	Other specified dental caries
K02.9	Dental caries, unspecified

* The original version of the International Classification ICD (ICD-DA, WHO Geneva, 1995).

Caries classification (E. V. Borovskiy, P. A. Leus, 1979)

I. CLINICAL FORMS	1 Spot step (carious demineralization) a) progressive (white or light yellow spots); b) intermittent (brown spots); c) inhibited (dark brown spots). 2. Carious defect (disintegration) A. Enamel caries (Superficial). Б. Dentine caries: a) of average depth: б) deep caries. B. Cementum caries
II. LOCATION	1) fissure caries. 2) contacting surfaces caries. 3) cervical region caries
III. COURSE	1) rapidly developing caries; 2) slowly developing caries; 3) stabilized process
IV. INTENSITY	1) single lesion; 2) multiple lesions; 3) systemic disturbance

4. DIAGNOSTIC METHODS

4.1. ENAMEL CARIES (ICD-10)

K02.0

Caries limited to enamel:

Enamel caries

White spot lesion

Initial caries

The main methods of diagnosis:

1. Questioning:

Complaints.

Patient history.

Medical history.

2. Examination:

Probing.

Drying.

Visual assessment:

The most important method of diagnosis.

Reveals enamel caries.

Determines lesion activity.

Color of the spot.

Homogeneity.

Form of the spot.

Surface.

Localization.

Amount.

Drying

Visual assessment	Histopathologic feature
Enamel optical properties does not change after drying over 5 seconds	Lack of enamel demineralization
Enamel opacity or discoloration are not visible on the wet surface, but clearly visible after drying	Demineralization of the upper third of enamel
Enamel opacity or discoloration are clearly visible without drying	Entire demineralization of enamel and upper third of dentine

Additional methods of diagnosis:

Vital staining.

Digital X-ray study.

Computerized tomography.

Fibreoptic transillumination (FOTI).

Laser fluorescence (DIAGNODENT).

Quantitative light fluorescence (QLF).

Electrometric method (ECM).

Electrical impedance measurement.

Ultrasonic caries detection.

Proximal caries X-ray study.

Reveals the "hidden" carious lesions on the contact surfaces of closely located teeth.

The clinical manifestation of enamel caries is a white or pigmented spot or enamel defect in the cervical region, fissures, fossa (in places with plaque).

Combination of visual assessment with one or more additional methods is the most accurate method of diagnosis.

4.2. DENTINE CARIES (ICD-10)

K02.0 Dentine caries

Caries extending into dentine

The main methods of diagnosis:

1. Questioning:

Complaints.

Patient history.

Medical history.

2. Examination:

Visual assessment (carious cavity, localization).

Probing.

Additional methods of diagnosis:

Temperature probe (heat probe).

Transillumination (transmission).

Pulp electroodontometry.

X-ray study.

Diagnostic preparation.

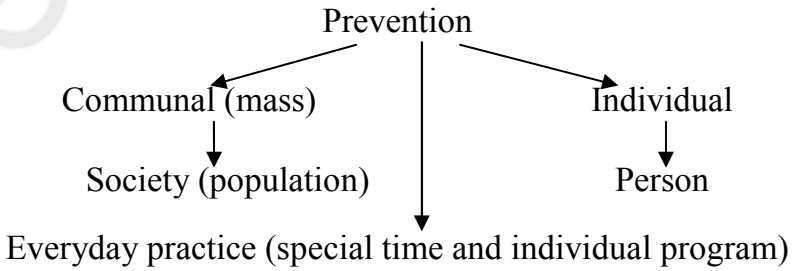
5. PREVENTION OF CARIES IN ADULTS

The system of social, medical, hygienic, educational activities

Primary prevention is aimed at the elimination of the causes and conditions of their appearance and development.

Secondary prevention is aimed at early detection of disease and prevention of recurrence, progression, appearance of possible complications.

The most important element of dental care- primary prevention!



Communal prevention methods (WHO):

1) limitation of sugar;
2) teaching oral hygiene;
3) systemic fluoridation;
4) local fluoride application;
5) secondary prevention;
6) education and ensuring healthy life-style (Republic of Belarus);
7) oral hygiene;
8) fluorides:
– fluorinated salt;
– fluorine-containing tooth pastes;
9) rational diet.

ORAL HYGIENE

The main purpose is caries prevention **(maximum reduction of the amount of soft microbial plaque and tartar)**:

– regular tooth brushing;
– removal of tartar.

Localization and the rate of plaque formation depend on:

– individual peculiarities;
– oral hygiene;
– diet;
– rate of saliva secretion;
– saliva composition.

The situation in the mouth	**Tooth brushing**
Food intake	
	Removal of plaque before food intake
Plaque	
5–10 minutes	After food intake
Acid	
48 hours (risk)	Prevention of enamel dissolution
Enamel dissolution	

Only in conjunction with a balanced diet and fluorides tooth brushing has a preventive value in reducing caries incidence.

FLUORIDES

The mechanism of fluoride action:

1) acceleration of remineralization;
2) glycolysis impairment;
3) bactericidal action;
4) increasing enamel resistance.

Methods of fluorine prevention:

1. System fluoridation:

– drinking water;
– food salt;
– milk;
– tablets / drops.

2. Local fluoridation

– toothpastes;
– gels;
– lacquers;
– solutions.

Combination of systemic and local use of fluorides is the most effective method of caries reduction.

System fluoridation:

1. Drinking water fluoridation.

Fluorine concentration — 0.5–1.0 mg/l (WHO, 1994).

Effectiveness — 40–50 % for deciduous teeth; 50–60 % for permanent teeth.

2. Food salt fluoridation.

Fluorine concentration — 200–350 mg/kg.

Effectiveness = Drinking water fluoridation.

3. Milk fluoridation.

Fluorine concentration — 5 mg/l; 200 ml of milk per day.

Effectiveness is satisfactory (research is ongoing).

Local fluoridation:

Professional procedures.

Individual (independent) procedures.

1. Rinsing the oral cavity with fluoride solutions.

Daily with 0,05 % Sodium fluoride solution (230 ppm).

Weekly with 0,2 % Sodium fluoride solution (900 ppm) (or 2 times a month).

! Not recommended for children under 6 years (WHO, 1994).

2. Fluoric gels.

F concentration:

– independent use 1000–5000 ppm;
– professional use 12 300 ppm.

Positive effect:

– professional treatments 2 times a year;
– home use 1 time weekly.

3. Fluorine-containing toothpastes.

A constant exposure of low fluoride concentration is preferable (WHO, STR № 846, 1994).

Tooth brushing is recommended for prevention of periodontal diseases, therefore it is most reasonable is to use fluoride toothpastes:

– used for hygiene, prevention and treatment of diseases of the oral cavity;

– toothpastes consist of abrasive, moisturizing, bindings, foaming substances, surfactants, preservatives, gustatory fillers, water and preventive additives;

– correlation of these components determines the properties, function, mechanism of action and efficacy of pastes.

Fluorine compounds in toothpastes are :

1. Inorganic.

Rapidly dissociating (sodium fluoride, stannous fluoride, potassium fluoride, ammonium fluoride, germanate fluorine, acidic phosphate fluoride).

Slowly dissociating (calcium fluoride, sodium monofluorophosphate).

2. Organic.

Aminofluorides, ftorinol.

Active substances:

– 2–4 % sodium fluoride (since 1940s);
– 8 % stannous fluoride (since 1950s);
– acidic phosphate fluoride (APF);
– aminofluorides (since 1950);

(N'-octadecyltrimethylendiamine-N,N,N'-tris(2-ethanol)-dihydrofluoride.)

Efficiency when brushing your teeth is 25 %.

Fluoride doses:

– ≤ 500 ppm — ineffective;
– 1500 ppm — optimum concentration;
– ≥ 2500 ppm — dangerous.

Nutrition

A set of processes associated with the consumption of nutrients and their assimilation in the body, thus ensuring the normal functioning of the body and health maintenance.

1) growth and development of microorganisms in the mouth;
2) proper development of teeth and supporting structures;
3) maintaining an optimal composition of saliva;
4) maintaining the oral mucosa in healthy condition;
5) influence on susceptibility to dental caries.

The cariogenicity of food — an ability of food substrate to cause biochemical processes that trigger acid demineralization of tooth enamel.

1. Frequency of use.
2. Duration of use.
3. Fluoride concentration in drinking water.
4. Plaque intensity.

5. The type and form of carbohydrate (adhesion).
6. Saliva excretion quantity.
7. Tooth susceptibility to acid attack.

Sweets without sugar

1. Intense sweeteners:

– saccharine;
– cyclomeitum;
– asparteim, allatinit;
– acesufatam.

2. Sugar substitutes:

– sorbitol;
– xylitol;
– lycasin.

Dentist's tactics:

1. To evaluate the adequacy of nutrition.
2. To determine the frequency of receiving carbohydrate food, especially sugars.
3. To explain the patient's caries risk factors associated with nutrition.
4. To help patients choose the right diet and non-cariogenic foods.

6. METHODS OF CARIES PREDICTION

Medical prognosis (Greek. prognosis) is a prediction of probability of a disease beginning or predicting a disease course and outcome, based on the knowledge of the patterns of pathological processes.

Prediction is the development of a prognosis of the object's state according to the information collected up to the current moment.

Stages of work with the patient:

1. Examination (basic and advanced techniques).
2. Diagnosis.
3. Prognosis.
4. Prevention and treatment planning.
5. Implementation of the plan.
6. Efficiency assessment and Monitoring.

Types of prognostic tasks:

1. Prediction of the risk of disease.
2. Prediction of the disease course.
3. Prediction of the disease outcome.

Risk factor — is a behavioral or biological and environmental factor revealed in long-term studies, the presence of which increases the possibility of disease onset, but the absence or elimination of this factor reduces the likeliness of disease occurrence.

Methods of forecasting:

1. Method of clinical prediction of dental caries.
2. Caries prediction method based on the definition of cariogenic microorganisms and buffer capacity of saliva.
3. Interactive computer program “Cariogram”.

Caries prediction method based on the definition of cariogenic microorganisms and buffer capacity of saliva:

– Predicting a carious disease course.
– Identifying patients with a very high intensity of dental caries.
– Determination of the Streptococcus mutans content in saliva of these patients and buffer capacity of saliva using rapid tests.

Original “Cariogram” (D. Brathall, 1997) is a computer online program for caries prognosis.

“Cariogram” includes ten risk factors. “Cariogram” is a circular diagram on the computer screen which is divided into five sectors. These sectors demonstrate the groups of caries risk factors. The significance of each factor is evaluated in points: from 0 to 2 or 0 to 3. “0” is a favourable indicator. 1–3 points are unfavourable indicators.

5 sectors:

1. Feasibility to avoid new cavities appearance (green zone) (A).
2. The diet (blue sector) (B).
3. Bacteria (red sector) (C).
4. Susceptibility (blue sector) (D).
5. Circumstances (yellow sector) (E).

10 risk factors:

1) history of caries;
2) comorbidities;
3) the diet;
4) food intake regimen;
5) plaque intensity (PI);
6) Streptococcus mutans (rapid test);
7) fluoridation program;
8) the secretion of saliva;
9) the buffer capacity of saliva ((rapid test);
10) clinical evaluation.

Investigator receives and summarizes all information about patient, makes a conclusion about caries activity and caries risk factors.

In the low level of caries risk factor:

– control the oral hygiene;
– system and local fluoridation;
– guidelines on diet and nutrition habits;
– there is no active treatment;

– if necessary-teeth restorations;
– caries monitoring once a year.

In the average level of caries risk factor:

– control the oral hygiene, home daily control of dental plaque;;
– guidelines on diet and nutrition habits;
– system and local fluoridation;
– professional oral hygiene twice a year;
– micro-restorations;
– caries monitoring twice a year.

In the high level of caries risk factor:

– control the oral hygiene every season;
– home use the toothpastes with high fluoride content;
– professional and home applications of fluorine-containing drugs;
– delayed teeth restorations (GIC);
– caries monitoring three or four times a year.

Interactive computer program «Cariogram»:

– provides a better understanding of the multifactorial nature of dental caries;
– illustrates the possible interactions between factors;
– explains the need to take preventive measures before the appearance of new carious cavities.

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