

UNIVERSITY OF LATVIA
FACULTY OF MEDICINE



**DMFT INDEX AND CARIES PREVALENCE OF THE
PATIENTS ATTENDING STUDENTS DENTAL CLINIC
AT UNIVERSITY OF LATVIA**

DIPLOMA THESIS

Author: **Kristina Kovalenko**

Student ID: **KK16050**

Thesis supervisor: **Lecturer Baiba Krauze, DDS**

RIGA 2022

ABSTRACT

Oral health plays a very important role in the well-being of a population. Poor oral health can lead to pain, tooth loss, loss of function and reduced self-esteem. Dental caries is one of the two most common oral diseases and reasons for tooth loss. The global burden of untreated caries in permanent teeth is estimated to be 2.3 billion people and in primary teeth – 532 million children as showed in epidemiological studies using different internationally accepted indices to determine caries prevalence in different populations. But there are far fewer studies in adults than in children, which justifies the aim of the thesis - to analyze the DMFT index and caries prevalence of adult patients attending students dental clinic at University of Latvia.

Keywords: Dental Caries Epidemiology; DMFT Index; Caries Prevalence in Adults; Caries Etiology.

ABSTRAKTS

Mutes veselībai ir ļoti nozīmīgs iedzīvotāju labklājības faktors. Slikta mutes dobuma veselība var būt par iemeslu sāpēm, zobu zaudējumam, funkciju zudumam un pazeminātai pašcieņai. Zobu kariess ir viena no divām visbiežāk sastopamajām mutes dobuma saslimšanām un zobu zaudējuma iemesliem. Epidemioloģiskie pētījumi, izmantojot dažādus starptautiski pieņemtus indeksus, lēš, ka globāli neārstēts kariess pastāvīgo zobos ir 2,3 miljardiem cilvēku un neārstēts kariess piena zobos - 532 miljoniem bērnu. Taču pētījumu pieaugušajiem ir daudz mazāk nekā bērniem, kas arī pamato darba mērķi - analizēt DMFT indeksu un kariesa izplatību pieaugušiem pacientiem, kuri apmeklē Latvijas Universitātes Studentu zobārstniecības klīniku.

Atslēgvārdi: Zobu kariesa epidemioloģija; DMFT indekss; kariesa izplatība pieaugušajiem; Kariesa etioloģija.

TABLE OF CONTENT

LIST OF SYMBOLS.....	6
1 INTRODUCTION.....	7 – 9
2 MATERIALS AND METHODS.....	10 – 15
2.1. Literature review.....	10 – 11
2.2. Quantitative research.....	11 – 15
3 LITERATURE REVIEW.....	16 – 50
3.1 Caries definition and characteristics	16 – 17
3.2 Historical aspects of dentistry development	18 – 23
3.2.1. Ancient origins.....	18
3.2.2. The beginning of a profession – middle ages.....	18
3.2.3. The development of a profession – 18 th century.....	19
3.2.4. Advances in science and education – 19 th century.....	19 – 20
3.2.5. Innovations in techniques and technology – the 21 st century.....	20 – 21
3.2.6. Preventive dentistry and change in caries treatment approaches.....	21 – 23
3.2.7. Importance of epidemiological studies.....	23
3.3. Epidemiology of dental caries	24 – 25
3.4. Etiopathogenesis of dental caries	26 – 36
3.4.1. Aetiology of dental caries	26 – 28
3.4.2. Mechanism of dental caries development.....	28 – 29
3.4.3. Etiological factors of dental caries.....	29 – 33
3.4.3.1. Primary etiological factors.....	29 – 30
3.4.3.2. Secondary etiological factors.....	30 – 33
3.4.4. Pathogenesis of dental caries.....	33 – 36
3.4.4.1. Dental plaque and oral microflora.....	33 – 35
3.4.4.2. Cariogenic diet.....	35 – 36
3.5. Classification of dental caries	37 – 41
3.6. Epidemiological studies of dental caries	42 – 44
3.6.1. Definition of dental caries as a disease.....	42
3.6.2. Definition of terms used in dental caries epidemiology.....	42 – 44
3.7. Dental indices in cariology	45 – 46
3.8. Prevention and non-invasive treatment of dental caries	47
3.9. Sociodemographic aspects of dental caries	48 – 49

Literature review: conclusion.....	50
4 RESULTS.....	51 – 63
5 DISCUSSION.....	64 – 65
6 CONCLUSION.....	66
ACKNOWLEDGEMENTS.....	67
BIBLOGRAPHY.....	68 – 78
DOCUMENTATION PAGE.....	79

LIST OF SYMBOLS

DMFT - decayed, missing and filled permanent teeth

FDI - World Dental Federation

GBD - global burden of disease

NSPH - nonspecific plaque hypothesis

SPH - specific plaque hypothesis

WHO - world health organization

1 INTRODUCTION

Dental caries is a biofilm-mediated, diet modulated, multifactorial, non-communicable, dynamic disease resulting in net mineral loss of dental hard tissues (Fejerskov 1997; Pitts et al., 2017). It is determined by biological, behavioural, psychosocial, and environmental factors. As a consequence of this process, a caries lesion develops. This oral disease is already known for thousands of years - 5000 BC in a Sumerian text “tooth worms” are described as the cause of dental decay.

Oral disorders, including dental caries, periodontitis, and edentulism are the most prevalent diseases of mankind, affecting more than 44.5% of the global population in 2019, according to the last report from the GBD-2019, with caries of permanent teeth being the most common condition. The GBD-2019 is currently the most comprehensive epidemiological study of disease burden.

In 2010, untreated caries in permanent teeth was the most prevalent condition worldwide, affecting 2.4 billion people, and untreated caries in deciduous teeth was the 10th-most prevalent condition, affecting 621 million children worldwide. The global age-standardized prevalence and incidence of untreated caries remained static between 1990 and 2010. There is evidence that the burden of untreated caries is shifting from children to adults, with 3 peaks in prevalence at ages 6, 25, and 70 y. (Kassebaum et al., 2015). Also, there is considerable variations in prevalence and incidence between regions and countries.

Dental caries is a globally occurring disease, which has exhibited some signs of decreasing but burden of dental caries remains a global public health challenge.

Caries is dynamic process, which is hidden before the damage also becomes visible for people without special education and culminates at the end of negative scenario with the formation of a cavity. It is prolonged process, requiring a couple of years, and therefore, needs long-term caries prevention methods.

Dental caries constitutes the signs and symptoms of the chemical dissolution of the tooth surface. This process is caused by metabolic events in the biofilm covering the tooth. Dental caries can affect every surface of a tooth where dental plaque is allowed to accumulate and mature in a biofilm over time (Fejerskov et al., 2013). Generally, it is accepted that *Streptococcus* spp. is considered as initial colonizers of dental biofilm and that *Streptococcus mutans* (*S. mutans*) is a common etiologic agent in cariology due to its virulence and ability to metabolize and process sugar carbohydrates (Ito, 2019). This dental plaque and biofilm are a prerequisite for caries lesions to develop, but the mere presence of biofilms is not enough for

caries to develop, as additional factors are involved (Bowen, 1999; Bratthall et al., 1996; Fejerskov, 2004; Lingström et al., 2003). Dental caries is therefore known as a multifactorial disease, in which behavioural, environmental and genetic risk factors interact (Fejerskov, 2004).

Environmental factors such as addiction to sugary snacks and drinks, poor oral hygiene, high levels of cariogenic bacteria, salivary dysfunction, and insufficient fluoride exposure are critical to its development (Fisher-Owens et al., 2007; Giugliano et al., 2018). Evidences also found in studies estimated that 26-64% of caries susceptibility is genetically determined (Nibali et al., 2017). Genes involved in enamel formation, the immune response, saliva proteins and food preferences have been considered to be involved in the aetiology of dental caries (Chapple et al., 2017).

Over the past decades a large number of research reports have shown that dental caries is linked to social and behavioural factors (Chen, 1995; Cohen et al., 1984; Richards et al., 1971).

Across countries and oral health systems, the existence of a social gradient in dental caries prevalence was found as measured by the association in dental caries indicators and socioeconomic status. The effect of educational background on measures of dental caries was observed for all countries but was found to be particularly strong when the disease prevalence was high. (Petersen, 2005)

Numerous studies conducted in different countries showed that applying preventive measures and improving the social environment significantly reduce the frequency of dental caries (Anil et al., 2017; D'Cruz et al., 2013). Socio-economic status is directly reflected in the eating habits and the lifestyle patterns which affect the prevalence of dental caries (Fernández-Alvira et al., 2015; Wu et al., 2020). Moreover, gender differences in dietary behavior have been reported where boys tend to eat sweets and drink soft drinks more often than girls do (Okeyo et al., 2020). Many epidemiological studies have observed that the rate of dental caries was effectively controlled by enhancing the optimum oral hygiene preventive protocol (Buldur, 2020; Chen et al. 2020; Kelly et al., 2005; Kumar et al., 2017; Štefanová et al., 2020).

The above leads to the conclusion that the prevention of dental caries in childhood could reduce the prevalence of caries in adulthood.

In most countries, untreated dental caries is a major challenge for public health, due to the high cost of treatment and the fact that in many countries dental treatment is not included in health insurance programmes.

The analysis leads to the conclusion that the most effective way to reduce the prevalence of caries is early, effective and responsible prevention of the disease, public education and affordable access to treatment in the community.

Even considering the large number of scientific evidence from several epidemiological studies about caries prevalence worldwide, the majority are regional studies. In addition, the information is too outdated for some countries, which does not make easy international comparison. The index that measures the number of permanent teeth decayed, missing and filled teeth (DMFT) is the common outcome for such studies. But those studies highlight the existing problem and allows conclusions to be drawn about the effectiveness of prevention and the need of prevention improvement.

A lot of epidemiological studies worldwide are done in children population, but rather few – in adult population, which justifies the topicality and choice of the aim of the thesis.

The Decayed (D), Missing (M), Filled (F) teeth index has been used since 1938 (Petersen, 2005) and is well established as the leading measure of caries in dental epidemiology. It has been developed by the WHO (WHO, 1997) to be recommended as a framework of reference to index dental caries. The index expresses the number of decayed, missing and filled teeth in a group of individuals. In dentistry, the DMFT index calculates different clusters per individual; the number one demonstrates the existence of caries from screening teeth, in a given environment that might be affected by other factors such as oral hygiene, public health awareness, genetic factors and the accessibility to the oral health services (Khamis, 2016). The WHO, in setting the average goal for determining dental caries suggested that there should not be more than 3 DMFT (decayed missing filled teeth) at 12 years of age and not more than 14 DMFT for adults aged 35 to 44 years of age (Khan, 2011; Petersen et al., 2007).

In order to analyze the prevalence of caries in the population group, the population of adult patients visiting the student dental clinic at the University of Latvia were selected and the aim of the theses - to analyze the DMFT index and caries prevalence of adult patients attending Students Dental Clinic at University of Latvia - was formulated. As well as a hypothesis for this epidemiological pilot study was generated based on the analysis of the literature. The null hypothesis generated is that an increase in the DMFT index may be observed with increasing of patient age.

2 MATERIALS AND METHODS

This is epidemiological pilot study, cross-sectional, descriptive and quantitative hypothesis testing study.

The aim of the study is to analyze the DMFT index and caries prevalence of adult patients attending students dental clinic at University of Latvia.

Null hypothesis what should be tested – increase in the DMFT index may be observed with increasing of patient age.

Ethical approval was obtained from Research Ethics Committee of the University of Latvia, Faculty of Medicine.

2.1 Literature review

Objective

The aim of the literature review part was to collate evidence-based information about:

1. The history and etiopathogenesis of caries;
2. Modifiable and non-modifiable caries risk factors;
3. Clinical classification of caries and protocol of clinical examination;
4. Methods of determination of caries prevalence and its advantages and disadvantages
5. Worldwide epidemiological studies about caries prevalence in adults.

Methodology

To meet the criteria for inclusion the studies had to have:

- 1) a specific focus on caries as oral disease in adults;
- 2) to involve epidemiological studies about caries prevalence in adults;
- 3) to analyze international indices for detection of caries prevalence, including DMFT index.

A literature search was undertaken during the period between September 2021 and March 2022. Keywords used for literature search - Dental Caries Epidemiology; DMFT Index; Caries Prevalence in Adults; Caries Etiology. Combination of keywords were used as well.

A search of the English literature using keywords was systematically conducted using database and web-based searches. The used databases were: National Center for Biotechnology Information – NCBI (2000-2019), PubMed (2000-2020), ReasearchGate (1972-2019),

ScienceDirect (1998-2015), Pocket Dentistry (2015), WebMed (2018). Websites of official organizations that were used – World Health Organisation, Global Burden of Disease, The remaining sources were applied using Google Search Engine.

Potential papers that resulted from this search were screened for relevancy, which was done together with the supervisor.

Results

In total 63 164 results with key words combinations were found.

Approximately 180 were relevant, from articles and books published from 1972 till 2022 and 142 were included as references.

Out of the 80 relevant articles, “Dental caries”, “Etiology and pathogenesis of dental caries”, “Epidemiology of dental caries”, “Caries classification and caries indices”, ”Dental caries prevention and management”, “Caries diagnosis and risk assessment” were some of the articles that had a fair amount of information that corresponded to this research project.

6 books were assessed. Three of the books that contained a lot of relevant material was “Dental caries. The diseases and its clinical management” 1st and 2nd edition, “Dental caries. Principle and management”.

From all relevant articles and books 142 were used for the references.

2.2 Quantitative research

The study population was selected from patients attending the Students Dental Clinic (Dentistry program at Medical Faculty of University of Latvia).

Medical cards of all patients of both genders in age 18-80 identified in the patient journal system in the period of time from January 1, 2017 to December 31, 2018 were selected for the further screening. Data from 2019 to date were not included as the number of patients decreased in a Covid 19 pandemic.

158 patients medical cards were found for this diploma work research. Regarding exclusion criteria 8 patients cards were excluded - 4 patients were pregnant women and 4 were edentulous.

Inclusion criteria:

- Both genders were included in 18-80 years of age.
- Patients medical cards should be properly filled (according to Regulation No. 265 of Republic of Latvia Adopted April 4, 2006 “Procedures for Keeping Medical Documents”).

Exclusion criteria:

- Edentulous patient.
- Any discrepancies in patient’s medical card.
- Pregnant patient as no radiographical examination could be done and dental status information could be incomplete.

No analysis was done for systemic diseases, additional caries risk factors, use of fluoridated toothpaste or medications used.

The results of the dental formula from the patient cards can be used safely, because the examination in the student clinic is performed according to a uniform protocol.

Intraoral examination protocol of dental hard tissue:

1. Visual or visual-tactile examination.

It is identifying caries according to their visual appearance, aided by a dental mirror and probe, on clean and dry teeth. The fundamental step in the detection of caries, but limited in the diagnosis of early lesions. All patients presenting to students dental clinician receive a visual

examination. At first visual examination is done by student. Then the supervisor is asked to recheck findings, and patient is referred to radiological examination.

2. Radiography

Bite-wing radiology is the most commonly used method. 2-4 bite-wing x-rays usually are done (1 or 2 on each side, covering the molar and premolar region). If needed periapical x-rays should be done for frontal teeth (incisors and canines). Radiographs aid the detection of caries and are shown to be more sensitive than visual examination on approximal and occlusal lesions (Wenzel, 2004). All radiographical examinations are done by student and supervised. X-ray what used in dental clinic, model Kodak CS 2200, 50/60 Hz.

Radiological findings are discussed and approved by supervisor and all additional information gained is added to patients dental formula.

DMFT index was used to gain information from patients dental formulas.

Primary caries in patients cards is coloured red.

Incipient caries is marked with red dot and Ci.

Fillings and any other – prosthetic - coronal restorations are coloured blue.

If any coronal restoration has secondary caries, this restoration is circumscribed with red.

If tooth is extracted, it is marked in the card as crossed and “EX”. If patient has hypodontia it is written in words and tooth is not marked as extracted.

Impacted teeth are market with “I” and this clinical situation is diagnosed radiographically.

If some prosthetic restorations are performed in the oral cavity then it is marked in patient’s card and described in words. (Fig.2.1)

11. Klīniskā karte

Izvārds, vārds _____ Datums _____

Zobu formula	Plc	C	C	C	Plc	ATR	ATR	ATR	ATR	ATR	ATR	ATR	Plc	Plc	C	Plc	not erupted
obakmens	G												ENDO				
Aplikums																	
Zobu formula	HALF ERUPTED	Plc	Plc	Plc	G	ATR	ATR	ATR	ATR	ATR	ATR	G	Plc	PLA	Plc	Plc	C
Zobakmens				ENDO		+	+	+	+	+	+		C				
Aplikums						+	+	+	+	+	+						

Apzīmējumi Kariess - C (sarkans); Plomba - Pl (zils); Pl_A - amalgāmas plomba; Pl_K - kompozīta plomba (ķīmiski cementēta, gaismas cementēta, jonocermentā); Ex - ekstrahēts zobš; K - apvalka kronis; starpdāja ☺

Fig. 2.1 Example of dental status

Quantitative research was started by collecting the data from the dental status.

The decayed, missing and filled teeth (DMFT) index was used to describe the prevalence of dental decay in a study population.

From 150 patients cards information from dental status was gained by recording the DMFT index, where D is decayed, M is missed, F is filled and T is tooth, what means that index is recorded for tooth, not for surface.

Third molars, or wisdom teeth were included (recommended in basic methods for oral health surveys).

The rules and principles used in recording DMFT are:

- a single tooth may have several restorations but it is counted as one tooth - F,
- fillings include tooth-coloured fillings, silver-coloured (amalgam) fillings, crowns veneers, etc.,
- a tooth may have restoration on one surface and caries on the other, it should be counted as decayed - D,
- if a tooth has both - a caries lesion and a filling - it is calculated as D only.
- no tooth must be counted more than once.

Following questions should be answered recording DMFT index:

- How many teeth have caries lesions (incipient caries not included)?
- How many teeth have been extracted?

- How many teeth have fillings or crowns?

Thus, including third molars, patient could have maximum 32 teeth.

DMFT obtained was recorded as follows – D-M-F. Intact teeth = 32- (D+M+F).

For example, patient has 5 decayed, 2 missing and 7 filled teeth. D-M-F = 5-2-7. Intact teeth = $32 - (5+2+7) = 18$.

Calculation of the DMFT index:

- For individual DMF= D + M + F
- Percent (%) needing care =
$$\frac{\text{total number of decayed tooth}}{\text{total number examined}}$$
- Percentage (%) of teeth lost =
$$\frac{\text{total number of missing teeth}}{\text{total number of examined}}$$
- Percent (%) of filled teeth =
$$\frac{\text{total number of filled teeth}}{\text{total DMFT}}$$
- Group average =
$$\frac{\text{total DMFT}}{\text{total number of the subjects examined}}$$
- Maximum score is DMFT = 32 and minimum score is 0
- For the measurement of prevalence of dental caries, the formula below is used

$$\text{Prevalence of caries} = \frac{\text{number of persons with dental caries}}{\text{total amount of examined patients}} * 100 \%$$

Data were organized using Excel. Nominal, ordinal and quantitative variables were used for descriptive statistics. SPSS program was used with Mann – Whitnet test, One-Sample Kolmogorov – Smirnow test and t-test.

3 LITERATURE REVIEW

3.1 Caries definition and characteristics

Dental caries may be defined as a bacterial disease of calcified tissues of teeth and is characterized by demineralization of the inorganic and destruction of the organic substance of the tooth.

Dental caries is dynamic, multifactorial disease process what begins within bacterial biofilm (Fig. 3.1) and is affected by multiple host factors (e.g., tooth minerals, saliva, fluoride exposure, dietary sugar intake, preventive behaviours). (Selwitz et al., 2007)

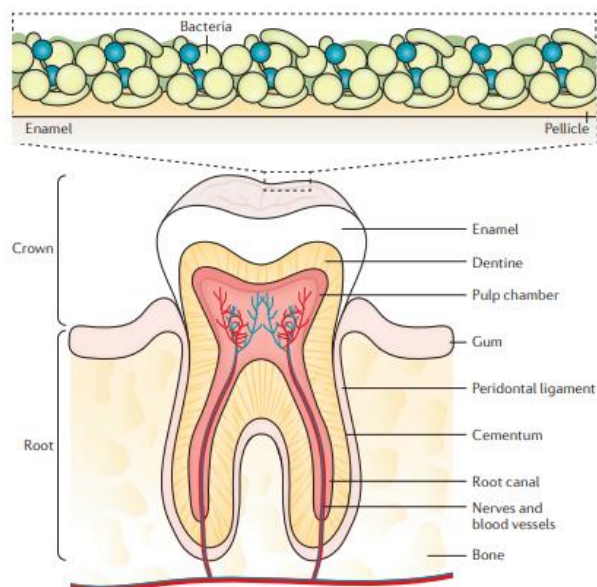


Figure 1 | Normal tooth anatomy and developing dental biofilm. The hard tissue of the tooth consists of enamel, dentine and cementum. Enamel is a hard material composed almost exclusively of mineral — which is mainly composed of hydroxyapatite ($\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$) — and covers the dentine on the crown of the tooth. Cementum is a bone-matrix-like substance, composed of mineral and collagen; it covers the root of the tooth. The dental pulp forms the central part and contains connective tissue, blood vessels and nerves. Teeth are covered by a salivary pellicle layer, consisting of proteins and glycoproteins, which facilitates binding of the oral microbiota to the teeth; this structure is called the dental biofilm (also known as dental plaque). The biofilm shuts off the surface enamel from the saliva and oral cavity and produces a protected microenvironment at the tooth surface. Gums (also known as gingiva) surround the teeth. In humans, primary teeth erupt around 6 months of age; these are gradually replaced by permanent teeth from ~6 years of age.

Fig. 3.1 Normal tooth anatomy and developing dental biofilm (Pitts et al., 2017)

The dynamic caries process consists of rapidly alternating periods of tooth demineralization and remineralization, which, if net demineralization occurs over sufficient time, results in the initiation of specific caries lesions at certain anatomical predilection sites on the teeth. It is important to balance the pathological and protective factors that influence the initiation and progression of dental caries. Protective factors promote remineralization and lesion arrest,

whereas pathological factors shift the balance in the direction of dental caries and disease progression (Pitts et al., 2016) (Fig. 3.2). The daily use of fluoride toothpaste is seen by many authorities as the main reason for the overall decline of caries worldwide over recent decades; the mode of action of such toothpastes is concerned with shifting the balance of the oral biofilm towards health. (Pitts et al., 2017)

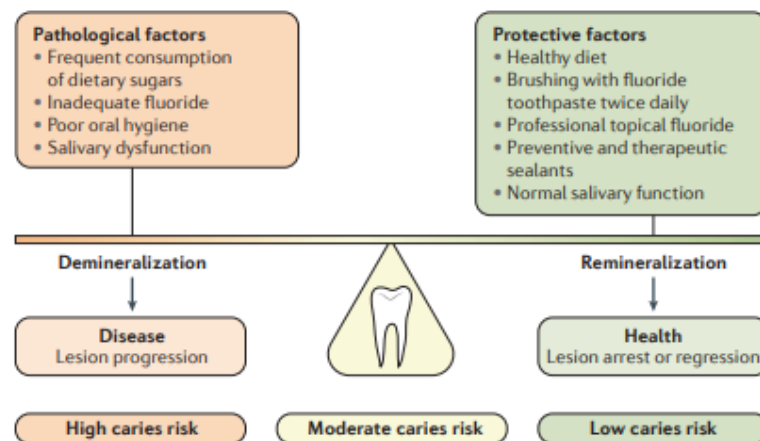


Figure 2 | **Balancing pathological and protective factors in dental caries.** A focus on optimizing the protective factors (those favouring healthy teeth) will promote remineralization and shift the dynamic balance of the caries process in the direction of health and lesion arrest. A failure to mitigate the effects of the pathological factors will promote demineralization and shift the dynamic balance in the direction of disease initiation and disease progression.

Fig. 3.2 **Balancing pathological and protective factors in dental caries (Pitts et al., 2017)**

Caries may refer to both the disease process and the lesions that develop as a result of that process.

Early disease (demineralization) is marked by visible changes on the surface enamel of the tooth and is asymptomatic. Disease is initially reversible; destructive process may be halted even in the presence of cavitation. (Selwitz et al., 2007)

3.2 Historical aspects of dentistry development

3.2.1 Ancient origins

Personal hygiene may not have been a normal daily practice thousands of years ago, but there are evidences that ancient civilizations did some oral care procedures and dental treatment. While the exact dental practices used at that time are not mentioned, early ancient texts revealed that dental health was of interest.

Human remains from 7000 B.C. show holes what were made in teeth to rid tooth decay. In Sumerian texts from 5000 B.C. the “tooth worms” were described as the cause of dental decay. Hesy-Re, an Egyptian scribe, who lived around 2600 B.C. is often called the first “dentist.” An inscription on his tomb includes the title “the greatest of those who deal with teeth, and of physicians.” This is the earliest known reference to a person identified as a dental practitioner. (Coppa et al., 2006) Ebers Papyrus in 1700-1550 B.C. described diseases of the teeth and various toothache remedies in Egyptian texts. Hippocrates and Aristotle in 500-300 B.C. wrote about dentistry, including the eruption pattern of teeth, treating decayed teeth and gum disease, extracting teeth with forceps, and using wires to stabilize loose teeth and fractured jaws.

3.2.2 The beginnings of a profession -middle ages

During the Early Middle Ages – 500 – 1000, medicine and surgery, including dentistry, in Europe is generally practiced by monks, the most educated people of the period. A medical text from 700 in China mentions the use of “silver paste,” a type of amalgam for treatment of tooth decay.

The main manipulation of that time in the oral cavity were extraction of decayed teeth. After monks were forbidden to do any oral treatment, barbers took their place. The Little Medicinal Book for All Kinds of Diseases and Infirmities of the Teeth by Artzney Buchlein was published in 1530. It is the first book devoted entirely to dentistry and published in Germany. It was written for barbers and surgeons who treat the mouth, it covered practical topics such as oral hygiene, tooth extraction, drilling teeth, and placement of gold fillings. Ambrose Pare, known as the Father of Surgery, publishes his Complete Works in France in 1575. This included practical information about dentistry such as tooth extraction and the treatment of tooth decay and jaw fractures.

3.2.3 The development of a profession- 18th century

Pierre Fauchard, a French surgeon published *The Surgeon Dentist, A Treatise on Teeth (Le Chirurgien Dentiste)* in 1723. Fauchard is credited as being the Father of Modern Dentistry because his book was the first to describe a comprehensive system for the practice of dentistry including basic oral anatomy and function, operative and restorative techniques, and denture construction. In late 1700 dentistry was proposed as the branch of medicine and services were offered to people.

John Greenwood in 1790 constructed the first known dental foot engine. He adapted his mother's foot treadle spinning wheel to rotate a drill. He was one of George Washington's dentists.

Josiah Flagg, a prominent American dentist, in 1790 constructed the first chair made specifically for dental patients.

3.2.4 Advances in science and education- 19th century

Richard C. Skinner wrote the *Treatise on the Human Teeth*, the first dental book published in America in 1801. As dentistry was developing as independent branch of medicine the need for dental education appeared. So Horace Hayden and Chapin Harris founded the world's first dental school in 1840, - the Baltimore College of Dental Surgery, and establish the Doctor of Dental Surgery (DDS) degree.

The first commercially manufactured foot-treadle dental engine was patented by James B. Morrison in 1871. Morrison's inexpensive, mechanized tool supplies dental burs with enough speed to cut enamel and dentin smoothly and quickly, revolutionizing the practice of dentistry. The collapsible metal tube revolutionized toothpaste manufacturing and marketing started in 1880s. Dentifrice had been available only in liquid or powder form, usually made by individual dentists, and sold in bottles, porcelain pots, or paper boxes before. Tube toothpaste, in contrast, became mass-produced in factories, mass-marketed, and sold nation-wide. In twenty years, it became the norm.

Willoughby Miller an American dentist in Germany in 1890 noted the microbial basis of dental decay in his book *Micro-Organisms of the Human Mouth*. This generated an unprecedented interest in oral hygiene and started a world- wide movement to promote regular toothbrushing and flossing. So we can assume that 1800s could be announced as the beginning of prevention of dental caries.

Prominent New Orleans dentist C. Edmond Kells in 1896 took the first dental x-ray of a living person in the U.S. and it started the era of radiological examinations for diagnosing dental diseases.

3.2.5 Innovations in techniques and technology- the 21st century

Greene Vardiman Black, the leading reformer and educator of American dentistry, in 1908 published his monumental two-volume treatise *Operative Dentistry*, which remained the essential clinical dental text for fifty years. Black later developed techniques for filling teeth, standardizes operative procedures and instrumentation, developed an improved amalgam, and was the pioneer in use of visual aids for teaching dentistry. Alfred C. Fones opened the Fones Clinic For Dental Hygienists in Bridgeport in 1913, Connecticut, the world's first oral hygiene school. It was done following preventive dentistry tendencies of that time.

Frederick S. McKay, a Colorado dentist, in 1930-1943 was convinced that brown stains (mottling) on his patients' teeth are related to their water supply. McKay's research verified that drinking water with high levels of naturally occurring fluoride is associated with low dental caries and a high degree of mottled enamel. By the early 1940s, H. Trendley Dean determines the ideal level of fluoride in drinking water to substantially reduce decay without mottling. The water fluoridation era begun when the cities of Newburgh, New York, and Grand Rapids, Michigan, add sodium fluoride to their public water systems in 1947.

Oral hygiene became more and more important not only for dental professionals, but for patients as well, and new approaches were found to improve oral hygiene. For example, the nylon toothbrush what first was made and marketed with synthetic bristles in 1938.

But not only prevention of oral diseases and mainly dental decay – caries – was of importance. Populations still has problems with caries prevalence and needed treatment. But treatment should be improved as populations showed increased beauty tendencies. Following professional and social requirements Oskar Hagger, a Swiss chemist, in 1949 developed the first system of bonding acrylic resin to dentin.

The effectiveness of fluoride already was studied and proved as well, so the first fluoride toothpastes were marketed in 1950s.

John Borden introduces a high-speed air-driven contra-angle handpiece in 1957. The Airtor obtains speeds up to 300,000 rotations per minute and is an immediate commercial success, launching a new era of high-speed dentistry.

Efforts to find more effective dental care methods continued as well and the first commercial electric toothbrush was developed in Switzerland in 1960s and introduced in the United States. A cordless, rechargeable model followed in 1961.

To improve use of resin filling materials Rafael Bowen in 1962 developed Bis-GMA- the thermoset resin complex used in most modern composite resin restorative materials.

One of the most exciting recent developments for the field of dentistry is the use of Silver Diamine Fluoride, or SDF. This topical treatment developed in 1972 in Japan, has the amazing property of being able to stop caries from developing and strengthening already affected tooth structure. (Yamaga et al., 1972) It has been used around the world for decades.

Aesthetics being one of driving forces for the development of new filling materials, new tooth-colored restorative materials appeared in the market in 1990s inaugurating an era of esthetic dentistry.

In 1997 FDA approves the erbium YAG laser, the first for use on dentin, to treat tooth decay. The digital transformation in dental medicine, based on electronic health data information, is recognized as one of the major game-changers of the 21st century to tackle present and upcoming challenges in dental and oral healthcare. The advent of modern group practices has changed the business structure and allowed practitioners to focus more on treatment than running a business, facilitating interdisciplinary collaborative treatment.

3.2.6 Preventive dentistry and change in caries treatment approaches

After many decades of extensive treatment intervention in case of dental caries, the significant progress in delivering more effective prevention was achieved. Since the 20th century, numerous advances in research and technology have improved the status of oral health for most populations (Benjamin, 2010). With the development of fluoridated drinking water and dental sealants, less experience of tooth loss and gingivitis by middle age were observed in different populations. In addition, the development and the refinement of dental materials and treatment techniques have improved our ability to restore function due to dental tissue loss.

It is the ethical responsibility of the dental profession to deliver dental care in the best interest of the patient by using the best available evidence to achieve and maintain oral health.

Caries treatment approaches have evolved over the extended timeframe that the profession and practice of dentistry has developed across the world. There are inevitable country and regional variations in the evolution of care, but generally three phases have been described (Pitts, 2004).

These are the:

- 1) extractive,
- 2) restorative, and
- 3) preventive phases of caries management.

As would be expected, countries have progressed through these phases at variable rates over recent decades.

The extractive phase of caries control involves the tooth extraction as the first line of treatment to stop pain and remove the threat of infection spreading from the consequences of caries which has progressed to involve the dental pulp and compromise its vitality. This phase was normal treatment method historically. Now extracting the offending tooth could be good and appropriate practice when the alternative is continuing pain and the risk of sepsis and infection spreading to involve the orofacial region and beyond. So our days this practice could be approved if patient has some serious medical or financial restrictions which do not allow the treatment of the tooth, or tooth is not restorable because of extensive carious lesion.

Shifts from the extractive to **restorative phase** historically happened with the development of the air turbine dental hand piece. It dramatically increased the rate of cutting of tooth structure that could be achieved and, therefore, the dentists' ability to restore many teeth economically in a reasonable time frame. Across the developed and large parts of the developing world, this technology changed the way in which dental care could be delivered. This approach was a step forward at the time and saved many teeth which would otherwise have been extracted and has been followed in good faith by generations of dentists. (FDI, 2013; Glick et al., 2012; Selwitz et al., 2007)

Shifts from the restorative to **preventive phase** is so-called "repeat restorative cycle" (Petersen, 2008; Selwitz et al., 2007). It is a process in which small fillings lead, over relatively modest periods of time, to larger fillings involving more surfaces, which in turn are replaced repeatedly, until the dental pulp becomes involved, which then requires the provision of endodontic treatment (or extractions), then crowns and then dentures (and more recently, also involves the provision of implants). The expectation among many in dentistry was that, as these dangers were recognized ever more widely, a more preventive strategy would be promoted and adopted. Tooth structure should be preserved, but little has changed in many countries over decades and restorative-orientated systems of caries care and payment persist to this day.

This lack of change persists despite the widespread understanding gleaned from the decades of evidence of effectiveness of fluorides, sealants and other methods of sugar control and preventive care being translated into evidence based guidelines. (FDI, 2013)

In addition, there has been a shift in opinion amongst many in the dental profession moving towards a preventive philosophy where, at both population and individual patient levels, prevention is optimized and restoration is a last resort (Ismail et al., 2013; Petersen, 2008; Pitts et al., 2013).

There have been parallel initiatives to update undergraduate dental education in cariology in order to ensure that in the future the profession is well equipped to deliver evidence-based caries care (Pitts et al., 2011; Schulte et al., 2013).

In many countries the transition from the restorative to the preventive phase has been slow, or delayed, or not there yet, despite the accumulating evidence and professional recommendations over decades (Ismail et al., 2013).

Evidence about the caries continuum - from initial-stage disease through moderate and extensive stages - and the trajectories of caries experience acquired throughout life, demonstrate that for the majority of those with caries in childhood, new caries will be likely to continue to develop into adulthood (Broadbent et al., 2008).

This means that risk-based caries prevention and management is needed across all age groups what shows the importance and need to evaluate caries prevalence in different populations worldwide.

3.2.7 Importance of epidemiological studies

Epidemiological studies of caries have been undertaken for many decades, and some of the data available through the WHO and other organizations give an impression that we have plentiful comparable global data. However, to evaluate and plan policy, epidemiology should provide data that meet the following specification: timely, accurate and understandable data for key age groups on the total amount of disease present (prevalence), the rate of disease progression (incidence) and disease trends over time. In addition, information on variations in disease levels between and within countries, including the estimates and trends in health inequalities (that is, differences in health status between groups within populations) are needed. (Pitts et al., 2017)

3.3 Epidemiology of dental caries

Although there has been notable national and international epidemiological research for some years, the study of dental epidemiology is a relatively new field in dentistry that has been stimulated by its increasing concern in public health. Epidemiology is fundamental to understanding the clinical and the public health importance of the disease, as well as providing insights for devising and assessing methods of caries control (Antunes et al., 2006; Pereira et al., 2003).

To understand the disease process and how caries affect the different groups of society, one needs to know the distribution of the disease in various communities. The average levels of disease seen in the inherently atypical groups of self-selected and/or referred patients attending dental clinics frequently give a false picture compared with the rest of the population. The mean levels and distribution of disease seen in representative samples of the total population will usually be different. The existence of this inherent and explainable difference is a key issue that dental students, dentists and public health planners must understand (Fejeskov et al., 2003).

Dental caries is still a neglected topic, despite the acknowledgment of the WHO that is still a major health problem in most industrialized countries, in which 60–90% of children and the vast majority of adults are affected by dental caries. (Petersen et al., 2005) Although caries has been considered a childhood disease, in reality, it continues into adulthood (Broadbent JM et al., 2008). Health inequalities exist in the burden of dental caries in both children and adults (Pitts et al., 2011).

Emphasis should be placed on the fact that there are few studies on the prevalence of caries in the adult population. An analysis of the epidemiological studies shows the following picture.

In Denmark, a study on oral health studied a national sample of 762 adults 35-44 years of age (Krustrup & Petersen, 2006; Krustrup, 2004). Results regarding DMFS and DMFT are presented in table. Significant differences were found for missing surfaces/teeth: women had more missing surfaces and teeth than men did (Krustrup, 2004). (Tab. 3.1)

Table 3.1 Average caries experience according to gender. P-values for gender differences (Krustrup, 2004)

	DS	MS	FS	DMFS	DT	MT	FT	DMFT
<i>35-44-year-olds</i>								
Men (n=343)	1.2	17.5	27.0	45.7	0.6	3.5	12.6	16.7
Women (n=419)	0.8	20.7***	25.8	47.3	0.4	4.1**	12.1	16.6
Total (n=762)	0.9	19.3	26.4	46.6	0.5	3.9	12.3	16.7

** p<0.01; *** p<0.001

The Finnish Health 2000 Health Examination Survey reports on average number of sound, filled, decayed and chipped teeth in relation to gender and age group, from a national sample of 3,027 subjects (Vehkalahti, Varsio & Hausen, 2004) (Tab. 3.2). In a baseline report from The Health 2000 Health Examination Survey, caries prevalence was examined in age groups (Nordblad et al., 2004b). Here, caries prevalence distribution is only presented for 30-34 and 45-54-year olds. Among 30-34-year old men, 29% had caries activity. Among women the same age, caries activity was found in 17.3%. In the male 45-54-year-olds, 31% had caries, and 20% of the women. Collapsing all the age groups (30-64-years old) to study gender differences, a significant difference was found among men and women. Among men 30-64-years old, 30% had caries prevalence, compared to 20% of the women (p<0.001) (Nordblad et al 2004b).

Tab. 3.2 Average number of sound, filled, decayed and chipped teeth in relation to gender and age group. Total n=3,027, but results for adults 65 years or over are not presented (Vehkalahti, Varsio & Hausen, 2004)

	Sound	Filled, no decay	Decay	Chipped, no	Nr. of teeth, total
<i>30-34-year-olds</i>					
Men	16.9	10.5	1.0	0.2	28.6
Women	16.9	10.8	0.3	0.1	28.1
<i>45-54-year-olds</i>					
Men	8.9	12.0	1.2	0.3	22.4
Women	7.5	14.6	0.5	0.3	22.9

As to our knowledge, there are no recent national clinical data on caries among adults in Norway and Sweden. There are regional studies (e.g. Skudutyte-Rysstad & Eriksen, 2007; Holst et al., 2004), but since they are geographically limited, the results should not be generalized to depict the oral health of adults in the entire country. The lack of national clinical data on adults has been pointed out in national reports about dental care in Norway (Sosial- og helsedirektoratet 2007, NOU 2005:11) and dental care in Sweden (SOU 2007:19).

Data about caries prevalence in adult population in Latvia are not available as epidemiological studies mainly are done on caries prevalence in children.

3.4 Etiopathogenesis of dental caries

3.4.1 Etiology

Dental caries is a complex illness, that depend upon varied factors principally on the presence of fermentable sugar, host factors, presence of cariogenic microorganism flora, and different associated environmental factors. Researchers have proposed numerous theories on dental caries. One of theory that has been accepted almost worldwide, but with modifications, is the ‘chemo-parasitic’ theory. This theory proposed by W. D. Miller in 1881 elucidated a combined effort of the acids (chemo) and the oral microorganisms (parasites) in tooth decalcification. (Ismail et al., 2001). Considering this concept because the backbone , J.L. Williams mind of plaque-inflicting dental caries. Keyes and Fitzgerald version to explain the relationship provide an explanation for of presence of microorganism in dental plaque and prevalence of dental caries.

According to Louis Pasteur and Robert Koch theory, the microorganisms in the oral cavity metabolize the dietary starch and produce organic acid that dissolves tooth minerals. Miller's theory, in fact, had inadequacies within the clarification of feat of dental caries, however it became an inevitable backbone for future studies within the discipline of cariology.

Tooth becomes an ideal place for the adherence for many of species. This colonization occurs as a string of methodical adhesion, succession, and progression. (Roberson et al., 2006) Organisms that are capable of adhesion adhere to the salivary pellicle on the tooth and form arena for the aggregation of other organisms that are incapable of initial adhesion. These are all external infectious agent and not endogenous/ host microorganisms. For many years, either all plaque flora were collectively considered as being pathogenic (nonspecific plaque hypothesis- NSPH) or certain specific organisms were considered pathogenic (specific Plaque hypothesis - SPH). (Loesch, 1979) A new hypothesis was proposed called the “ecological plaque hypothesis”. (Marsh PD, 1994) According to this hypothesis, pathogenicity to specific species that produce the disease only at specific sites caused by a certain change in the environment of the residential plaque flora. The ecological plaque hypothesis targets the factors that resulted in the environmental change of the plaque.

On the other hand the etiology of dental caries can be described by a simple Venn’s diagram, which consists of three circles and the interaction of these circles. Two circles depict diet, dental plaque, or microorganism load, and therefore the third one depicts the host. The intersection of all three circles shows caries (Fig. 3.3).

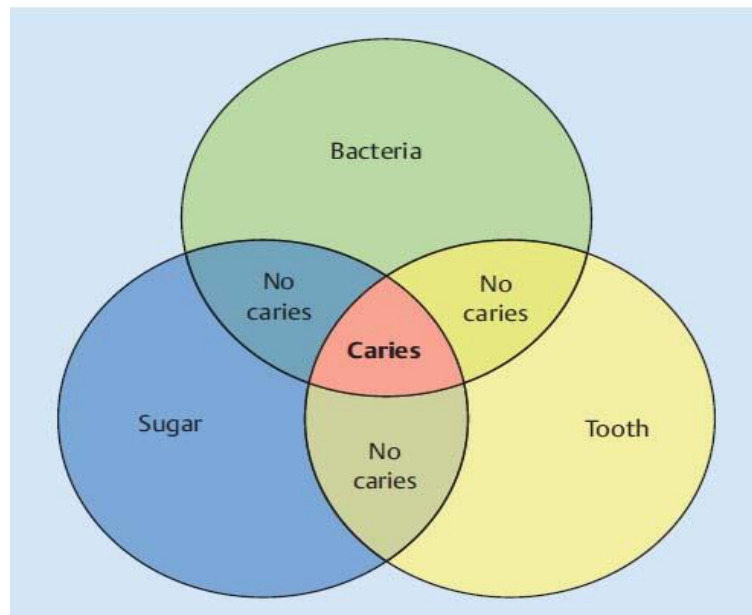


Fig. 3.3 Venn diagram summarizing the etiology of caries. The diagram demonstrates that caries requires both the presence of acidogenic bacteria and availability of a diet from which the bacteria can produce acid, in conjunction with acid-susceptible dental tissues (Adapted from Pocket Dentistry)

Recently, a fourth circle “time” has been added to the above circle, which describes the duration of the interaction (Fig. 3.4).

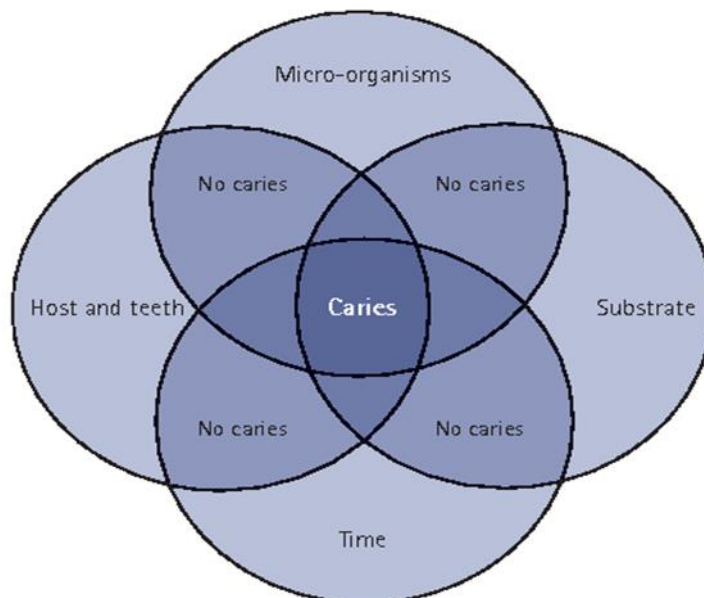


Fig. 3.4 The Venn diagram emphasizes that all four factors must be present and acting together for caries to occur and to progress (Yipp et al., 2012)

Plaque and dietary factors are mutualist upon one another in the progression of dental caries. (Usha, 2009) Specific microorganisms such as *Streptococcus mutans* (*S. mutans*) primarily deals with dental caries initiation, and *Lactobacilli* deals with the progression of dental caries. The substrates for these microorganism are possible supermolecules and therefore the bacterial-generated carbohydrate reserve within the biofilm. These bacteria metabolize these substrates, and results in the formation of lactic and other acids. Lactic acid, along with host factors, lowers down the oxygen coefficient and results in dental caries. Acid generation result in the dissolution of calcified tissue in tooth and eventually into cavitation. Enamel demineralization occurs at a pH of 5.5 and below.

3.4.2 Mechanism of dental caries development

Dental caries are clinically characterized by a large polymorphism and a very complex aetiology. Dental caries begins when there is a favourable interaction between multiple etiological factors that create an imbalance in the oral cavity which allows the development of the disease (Lima et al., 2007).

A carious lesion initiates with the production of organic acids by the microorganisms of the oral cavity, namely *S. mutans* and *Lactobacillus*, that metabolize the extracellular carbohydrates of the individual's diet (Cortelli et al., 2004; Lima et al., 2007). The presence of the organic acids produced will decrease the pH in the interface between the tooth surface and the bacterial plaque, allowing the development of the demineralization process on the tooth enamel (Pereira, 2003). In the mouth, these changes over time are known as Stephan responses or Stephan curves (Harris et al., 2004). The pH of dental plaque under resting conditions (when no food or drink has been consumed), is fairly constant. The response after exposure of dental plaque to a fermentable carbohydrate is that pH decreases rapidly, reaching a minimum in approximately 5 to 20 minutes. This is followed by a gradual recovery to its starting value, usually over 30 to 60 minutes, although this can be longer in some individuals. When the oral cavity has a pH below 5.5 (considered the critical pH), the saturation of the dental tissues initiates causing demineralization. If this process is frequent and constant a initial lesion will initiate and it may become the precursor of a dental caries (Axelsson, 2004; Fejeskov, 2003).

Prevention methods have as a main goal to decrease the time of exposure of tooth tissues to the low values of pH and, therefore, it is strictly necessary frequently remove the bacterial plaque, avoiding its increase contact with tooth surfaces(Daniel et al., 2008). The buffering action of saliva must also be considered. Saliva is supersaturated with various ions that act as a buffer. In the physiological pH range (6.5-7.4) for saliva, a high saliva buffer

capacity, mostly due to HCO_3^- concentration (nearly 10mg% are present in the oral cavity), is protective against dental caries, possibly reducing the rate of tooth demineralization caused by a lower pH value (Fejerskov et al., 2003). Therefore, saliva has an important role in the prevention of dental caries, maintaining low levels of acidity in the oral cavity. (Fig. 3.5)

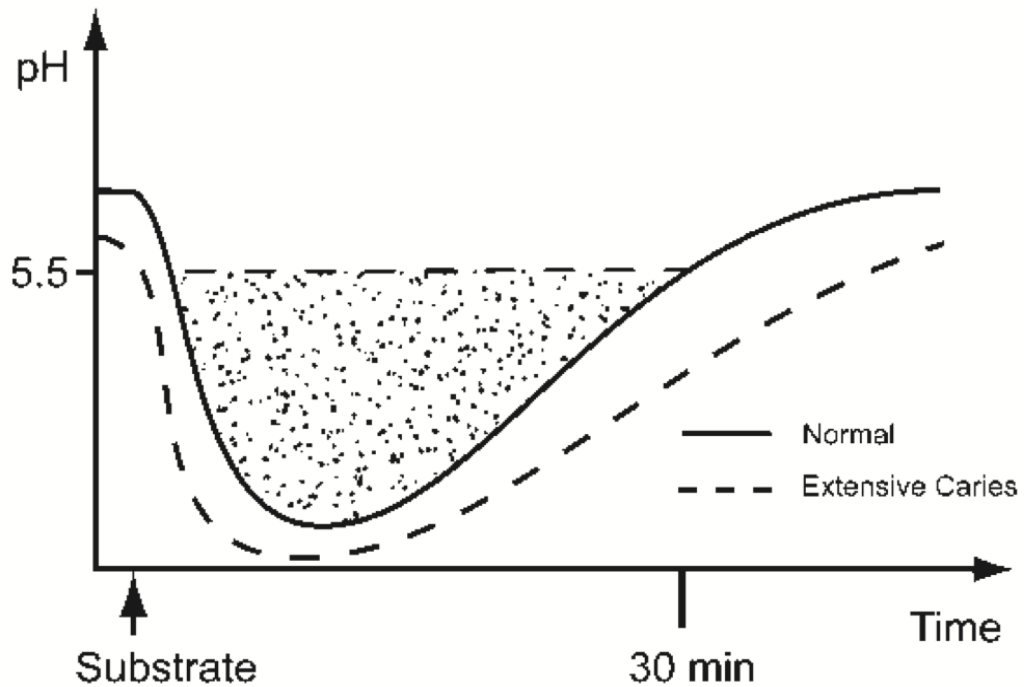


Fig. 3.5 Illustration of the Stephan curve. Demonstration of the decrease of pH according to the time after sucrose rinse. The dotted zone corresponds to the critical pH (below 5.5) that takes place in the oral cavity and causes demineralization of tooth enamel (Qualtrough et al., 2005)

3.4.3 Etiological factors of dental caries

3.4.3.1 Primary etiological factors

The researcher Paul Keyes developed a diagram that describes the multifactorial aetiology of dental caries. In this diagram, we can observe that there are three main etiological factors that are essential for the initiation and development of the disease:

- Susceptible host;
- Cariogenic oral microflora;
- Substrate that depends on the host's diet, which is then metabolized by the microorganisms that constitutes that bacterial plaque (Fig. 3.6). (Lima et al., 2007).

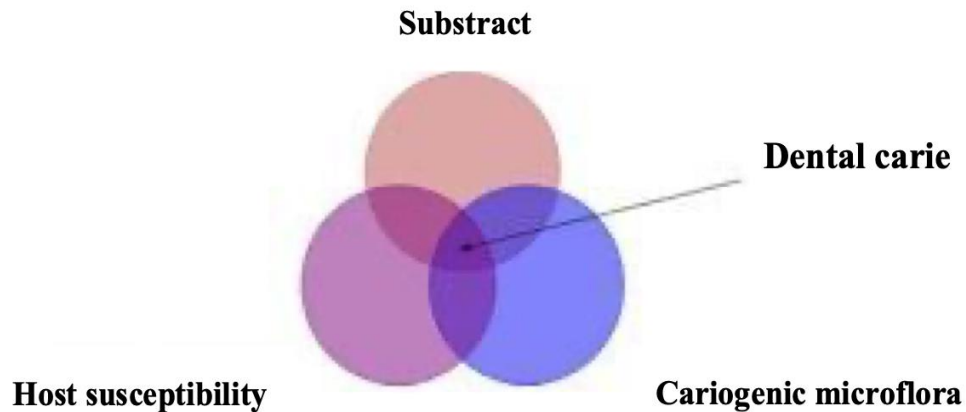


Fig. 3.6 Diagram proposed by Keyes about the multifactorial character of dental caries (Lima et al., 2007)

Host susceptibility refers to the characteristics that the individual's dental tissues may influence on the susceptibility to acid dissolution developed by the microorganisms in contact with the substrat (Pereira, 2003). The presence of dental caries in dental morphology is influenced by the type of oral microflora, saliva composition and quantification in the oral cavity, by food intake such as high consumption of sugary foods and the application of fluorides or a combination of these and other individual and genetic factors.

We must also take into account the susceptibility of the individual, which is directly related to socioeconomical and cultural factors and that translates into specific individual behaviours, namely related with oral health (Lima et al., 2007).

3.4.3.2 Secondary etiological factors

As the most important secondary etiological factors the following should be mentioned:

- Time;
- Use of fluorides;
- Saliva;
- Oral hygiene.

Time

The time factor has an important role in the manifestation of clinical signs of the development of carious lesions (Pereira, 2003). This factor was added by Newbrun to the primary etiological factors identified by Keyes, since these need to be present for a certain period of time, so that

the progressive demineralization of enamel may develop (Axelsson, 2004; Newbrun et al., 1999).

Fluorides

Research has shown that fluoride is most effective in dental caries prevention when a low level of fluoride is constantly maintained in the oral cavity. The goal of community-based public health programmes, therefore, should be to implement the most appropriate means of maintaining a constant low level of fluoride in the oral cavity (Aoba T, 2004) Fluorides can be obtained from fluoridated drinking-water, salt, milk, mouth rinse or toothpaste as well as professionally applied fluorides, or from combinations of fluoridated toothpaste with some of other sources. Fluoride is being widely used on a global scale, with much benefit. Millions of people worldwide use fluoridated toothpaste. Recent local studies have shown that affordable fluoridated toothpaste is effective in caries prevention and should be made available for use by health authorities in developing countries. The WHO Global Oral Health Programme is currently undertaking further demonstration projects in Africa, Asia and Europe in order to assess the relevance of affordable fluoridated toothpaste, milk fluoridation and salt fluoridation (WHO 2003; 2014). There is clear evidence that long-term exposure to an optimal level of fluoride results in diminishing levels of caries in both child and adult populations. However, populations in many developing countries do not have access to fluorides for prevention of dental caries for practical or economic reasons (Pereira, 2003). There are some undesirable side-effects with excessive fluoride intake. Experience has shown that it may not be possible to achieve effective fluoride-based caries prevention without some degree of dental fluorosis, regardless of which methods are chosen to maintain a low level of fluoride in the mouth. The public health administrators must seek to maximize caries reduction while minimizing dental fluorosis (Peres et al., 2003).

Saliva

The mixed fluid in the mouth in contact with the teeth and oral mucosa, referred to as whole saliva, is derived predominantly from three paired major salivary glands: the parotid, submandibular and sublingual glands, but also from the minor salivary glands in the oral mucosa (Fejerskov et al., 2003; Pereira, 2003). Normally, the daily production of saliva ranges between 0.5 and 1.0 litre and is composed of more than 99% water and less than 1% solids, mostly proteins and electrolytes. The multiple functions of saliva relate to both its fluid characteristics and specific components (Axelsson, 2004).

Examples of the former are:

- Rinsing effect;
- Solubilization of food taste-substances;
- Bolus formation;
- Food and bacterial clearance;
- Dilution of detritus;
- Lubrification of oral soft tissues;
- Chewing capacity;
- Swallowing;
- Speech.

Other important functions of saliva consists in the protection of the teeth by neutralization of acids by buffering actions, the saliva maintains supersaturated calcium phosphate concentration with regard to hydroxyapatite, and also by participating in enamel pellicle formation. Furthermore, saliva components participate in mucosal coating and antimicrobial defence as well as digestive actions. Thus, saliva plays a major role in oral health and changes affecting salivary function, it may also compromise hard and soft oral tissues structure and functions (Fejerskov et al., 2003).

The oral cavity is constantly exposed to many different kinds of substances, some of which influence the caries process to a great extent. An important function of saliva is therefore the dilution and elimination of substances introduced into the oral cavity, through a physiological process usually referred to as salivary clearance or oral clearance (Axelsson, 2004; Fejerskov et al., 2003). In patients with reduced quantity of saliva the mechanistic and cleaning properties of this fluid in the mouth are impaired. With regard to prolonged oral clearance, a low oral sugar clearance inevitably increases the risk of caries development. Concerning this relation, the unstimulated flow rate has been found to be diagnostically more important than the stimulated one (Fejerskov et al., 2003).

Oral hygiene

There is a strong correlation between oral hygiene and the prevalence of dental caries (Vanobbergen et al., 2001). Good oral hygiene habits help to prevent the development of caries by reducing the build-up of dental plaque (Harris et al., 2004). The composition of the dental plaque varies not only from individual to individual, but also upon the location of the oral cavity and tooth surface. Control of bacterial plaque through proper hygiene, performed by each individual and complemented with the intervention of a dental professional are key preventive

primary measures for the improvement of oral health and disease prevention, including dental caries (Pereira, 2003).

3.4.4 Pathogenesis of dental caries

The pathogenesis is discussed in the following section as disturbances in homeostasis / physiological equilibrium. They are categorized as follows:

1. Disruption of microbial homeostasis in the 'biofilm'.
2. Disruption of mineral homeostasis that is seen between the tooth and the 'oral fluid'.

An aggregate of microorganisms in which cells adhere to each other and/or to a surface called as dental biofilm. This aggregate of cells is inside a self-produced organic matrix of polysaccharides, proteins, and DNA. There are soft tissue, teeth, saliva, and others; each of these is a separate ecological niche. Colonization is dependent upon the specific organism and microbiological niche. The saliva act as a medium for free- floating or planktonic bacteria. A closer study of plaque reveals a much more organized of plaque depicts. Various microbial species are found to occupy their respective microcosms. These channels act like a two- way transport for the nutrition and by-products of the microbes. (Fejerskov, 2004) Oral fluid encompasses the saliva and the gingival crevicular fluid.

3.4.4.1 Dental plaque and oral microflora

The oral cavity is inhabited by hundreds of bacterial species that play vital roles in maintaining oral health or in shifting to a diseased state such as dental caries and periodontal disease (Kleinberg, 2002; Reyes et al., 2012).

The term biofilm is used to describe communities of microorganisms attached to a surface and can be formed very easily if the formation process is not interrupted, mainly by regular toothbrushing. Such organisms are spatially organized into a three-dimensional structure enclosed in a matrix of extracellular material derived from both the cells themselves and the environment. Dental plaque is considered as a microbial biofilm and its development can be divided into several stages:

1. Pellicle formation;
2. Attachment of single bacterial cells (0-4 hours);
3. Growth of attached bacteria leading to the formation of distinct microcolonies (4-24 hours);

4. Microbial succession and co-aggregation leading to increased species diversity concomitant with continued growth of microcolonies (1-14 days);
5. Climax community/mature plaque (2 weeks or more).

It should be noticed that plaque formation is a highly dynamic process, and that attachment, growth, removal and reattachment of bacterium may occur at the same time (Fejerskov et al., 2003).

Dental caries, as a infectious disease, with bacterial aetiology, correlates directly with bacterial strains that co-exist in the oral cavity, like *S. mutans* and *Lactobacillus* (Fejerskov et al., 2003). The cariogenic properties of *S. mutans* and *Lactobacillus* are widely recognised and, as significant oral pathological agents, the former group is linked to enamel lesion formation while the latter is associated with cavity progression (Brambilla et al., 1999).

A study developed by Hart et al. identifies the top 10 bacterial species or groups according to the score that have been definitely and/or that could be possibly implicated in caries onset and progression which included *S. mutans* and *Lactobacillus* (Hart et al., 2011) (Fig. 3.7).

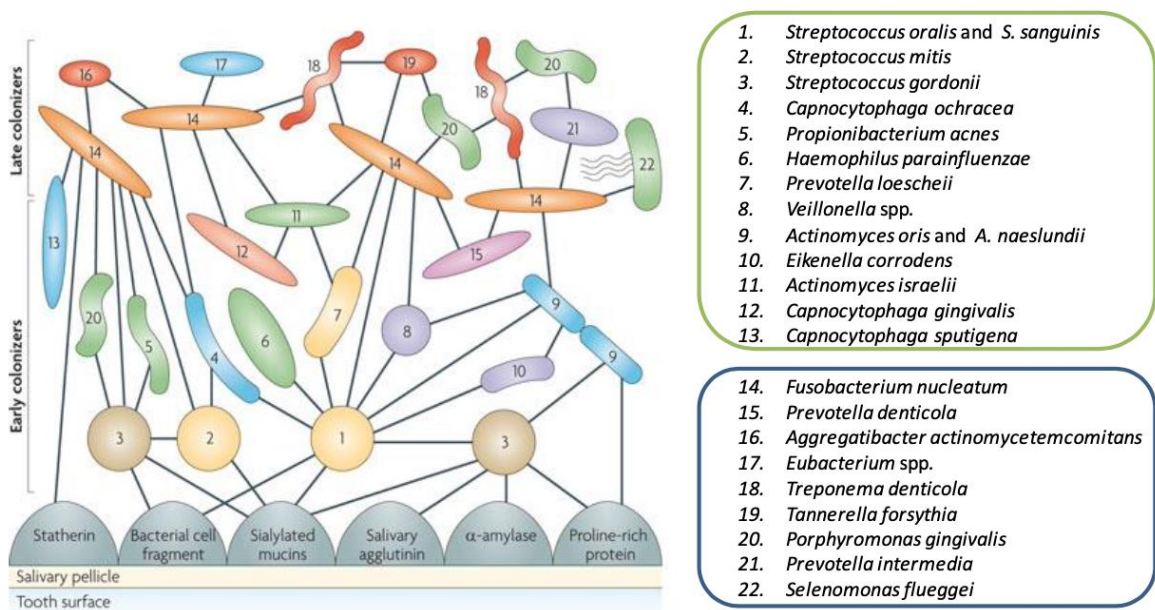


Fig. 3.7 - Oral biofilm model proposed by Kolenbrander et al. (2010). Early colonizers (framed in green) bind to the salivary pellicle on the tooth surface and coaggregate with other bacterial species in a sequential manner. Fusobacterium appears to have multiple interactions and it has been proposed as a “bridge” between early and late colonizers (framed in blue), which are mainly anaerobic and include several pathogenic organisms (Kolenbrander PE et al., 2010)

Aas et al., 2015 confirmed in their study that there is a distinctive predominant bacterial flora of the healthy oral cavity that is highly diverse and can be site and subject-specific. It is

important to fully define the human microflora of the healthy oral cavity before we can understand the role of bacteria in oral disease.

It has concurrently been clearly established that social, economic, cultural, ethnic, and environmental factors also play an important role in the progression of dental caries and also influences the individual oral microflora highly related with oral health behaviours (Steinberg et al., 2014).

The bacteria present in the oral cavity are: other *Staphylococcus* spp. strains, which forms part of the oral normal microbial population; *Actinomyces* spp. which exists in large quantity in the dental plaque; *Neisseria* spp. that are early colonizers of the oral cavity; the *Veillonella* spp. which is isolated from all surfaces of the oral cavity and there is a large number present in the tongue and dental plaque; *Haemophilus* spp. that is often present in saliva, dental plaque and epithelial surface. (Brook I., 2011).

3.4.4.2 Cariogenic diet

The level of cariogenic diet is determined by the presence of carbohydrates or refined sugars, especially sucrose, which serves as a substrate for microorganisms of the oral cavity (Aoba, 2004). The oral microflora synthesizes extracellular polysaccharides that play a key role in dental plaque formation and also in the production of organic acids that promote and facilitate enamel demineralization and, thereby, the development of dental caries (Cortelli et al., 2004). Thus, for the genesis of the disease there is required in addition to cariogenic diet microorganisms to metabolize the substrate, and a host susceptible to the damaging effects of metabolism (Touger-Decker et al., 2003).

Today the world faces two kinds of malnutrition, one associated with hunger or nutritional deficiency and the other with dietary excess. Urbanization and economic development result in rapid changes in diets and lifestyles, which may be reflected by a higher risk of dental caries development (Levin et al., 2015). Market globalization has a significant and worldwide impact on dietary excess leading to chronic diseases such as obesity, diabetes, cardiovascular diseases, cancer, osteoporosis and oral diseases. Diet and nutrition affects oral health in many ways. Nutrition, for example, influences cranio-facial development, oral cancer and oral infectious diseases. Dental diseases related to diet include dental caries, developmental defects of enamel, dental erosion and periodontal disease.

The nutrition transition is a relevant example on how common risks influence public health, including oral health. The public health community involved with oral health should gain an

understanding of the health effects of these complex developments in order to prevent or control oral diseases (WHO, 2014).

3.5 Classification of dental caries

Dental caries consists in a oral disease that is the main responsible for most of the consumption of biomaterials and human resources used in dental medicine, therefore dental caries have serious economic repercussions related to the high costs of their treatment and also with professional and school absenteeism (Pereira, 2003; WHO, 2004;2013)

The absence of oral treatments invariably leads to an increase in lesion size progressing towards the dental pulp, resulting in a progressive development of pulpal inflammation accompanied by pain symptomatology and possible infection.

The knowledge and ability to diagnose and classify dental caries will determine the right method of prevention and treatment.

Cariou lesions can be classified as cavitated and non-cavitated.

Non-cavitated carious lesions

Surfaces appear macroscopically intact and without clinical evidence of cavitation.

Also called incipient, initial, early, or white-spot lesions (although can be white or brown).

Can potentially be reversed or arrested by chemical or mechanical interventions. (Slayton et al., 2018).

Cavitated carious lesions

Surfaces are not macroscopically intact and with a distinct discontinuity or break in the surface integrity that can be discerned at clinical examination

Less likely to reverse or arrest without intervention. (Slayton et al., 2018)

Other classification systems for dental caries include:

International Caries Detection and Assessment System score (ICDAS), where

- 0: sound
- 1: first visual change in enamel
- 2: distinct visual change in enamel
- 3: localized enamel breakdown
- 4: underlying dark shadow from dentin
- 5: distinct cavity with visible dentin
- 6: extensive cavity with visible dentin. (Pitts et al. 2017) (Fig. 3.8).

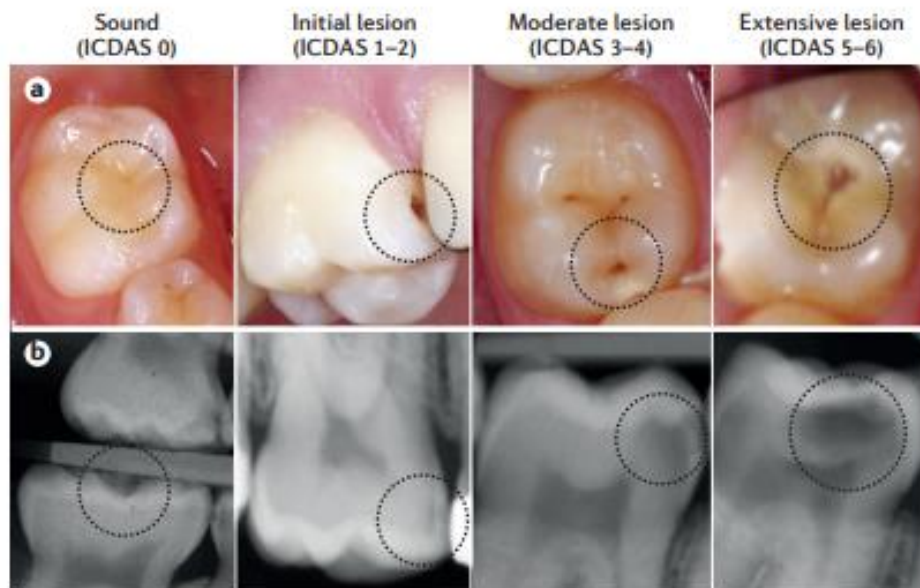


Figure 6 | Clinical and radiographic appearance of the stages of severity of tooth decay. Clinical appearance (part a) and bite-wing radiograph (part b) of the same tooth. Examples of sound and extensive caries surfaces are shown on the biting (or occlusal) surfaces, which contain developmental depressions and grooves (known as pits and fissures) that collect dental biofilm and are caries predilection sites. The initial-stage and moderate-stage lesion examples show approximal surfaces (where adjacent teeth are in contact). Caries also develops on the free smooth surfaces (adjacent to the cheeks, lips and tongue).

Fig. 3.8 Clinical and radiographic appearance of the stages of severity of tooth decay (Pitts et al., 2017)

American Dental Association Caries Classification System

- Sound tooth (International Caries Detection and Assessment System score 0): No clinically detectable lesion; dental hard tissue appears normal.
- Initial lesion (International Caries Detection and Assessment System score 1 to 2): Earliest clinically detectable lesion (mild demineralization). Limited to enamel or to shallow cementum/dentin layer; visually non-cavitated
- Moderate lesion (International Caries Detection and Assessment System score 3 to 4): Visible signs of enamel breakdown or of dentin being moderately demineralized; early, shallow cavitation
- Advanced lesion (International Caries Detection and Assessment System score 5 to 6): Full cavitation through the enamel and dentin exposed; deep/severe demineralization of dentin. (Young et al., 2015)

G. V. Black Caries Classification

- Class I- Cavity in pits or fissures on the occlusal surfaces of molars and premolars, facial and lingual surfaces of molars, or lingual surfaces of maxillary incisors.
- Class II- Cavity on proximal surfaces of premolars and molars.
- Class III- Cavity on proximal surfaces of incisors and canines that does not involve the incisal angle.
- Class IV- Cavity on proximal surfaces of incisors or canines that involves the incisal angle.
- Class VI- Cavity on incisal edges of anterior teeth and cusp tips of posterior teeth. (Macri et al.,2017, Rashid, 2007).

Primary caries

Primary caries is a caries lesion on previously sound tooth surface. (Machiulskiene et al., 2019). Primary caries is the term used to describe caries lesions developing on intact, natural tooth surfaces, as opposed to secondary or recurrent caries, which develops next to an existing restoration (Fig. 3.9) (Fejerskov and Kidd, 2015). Two regions have been described when considering the process of secondary caries; the surface lesion, which develops perpendicular to the tooth surface and can be considered a primary lesion developing next to a restoration, and the wall lesion, which develops perpendicular to the tooth/restoration interface (Hals & Nernaes, 1971).



Fig. 3.9 A-initial carious lesion on occlusal surface. B- histological section through the lesion. The acid resistant and fluoride rich superficial layer is clearly visible. C-dentinal occlusal caries with cavitation and shadow. D- histological section through the lesion (Diniz M., et al 2012)

Secondary caries

Secondary caries is a disease that occurs on the tooth after the filling has been used for a period of time. Secondary caries is also the main reason for the replacement of dental restorations. Regardless of the material used for fillings, secondary caries cannot be completely avoided. The proportion of secondary caries is very high after filling in permanent teeth or primary teeth. Secondary caries mainly occurs because of the formation of micro cracks after filling. When the micro crack width exceeds 50 microm, saliva will enter the micro cracks between the filling and tooth tissue. The cariogenic bacteria in the saliva will grow when the environment of micro cracks is appropriate, thereby producing secondary caries. The prevention of secondary caries includes micro crack control, fluoride use, teeth cleaning, tooth decay and gum disease treatment, and regular checkups. (Zhi et al., 2014)

The secondary carious lesion displayed histologically the same basic pattern (Fig. 3.10):

- 1) an outer lesion, which is caused by the a new primary attack on the outer surface of the tooth;
- 2) a wall lesion, might be the consequence of the diffusion of bacteria, fluids or hydrogen ions between the restorations and the cavity wall. (Hals, et al 1975a)

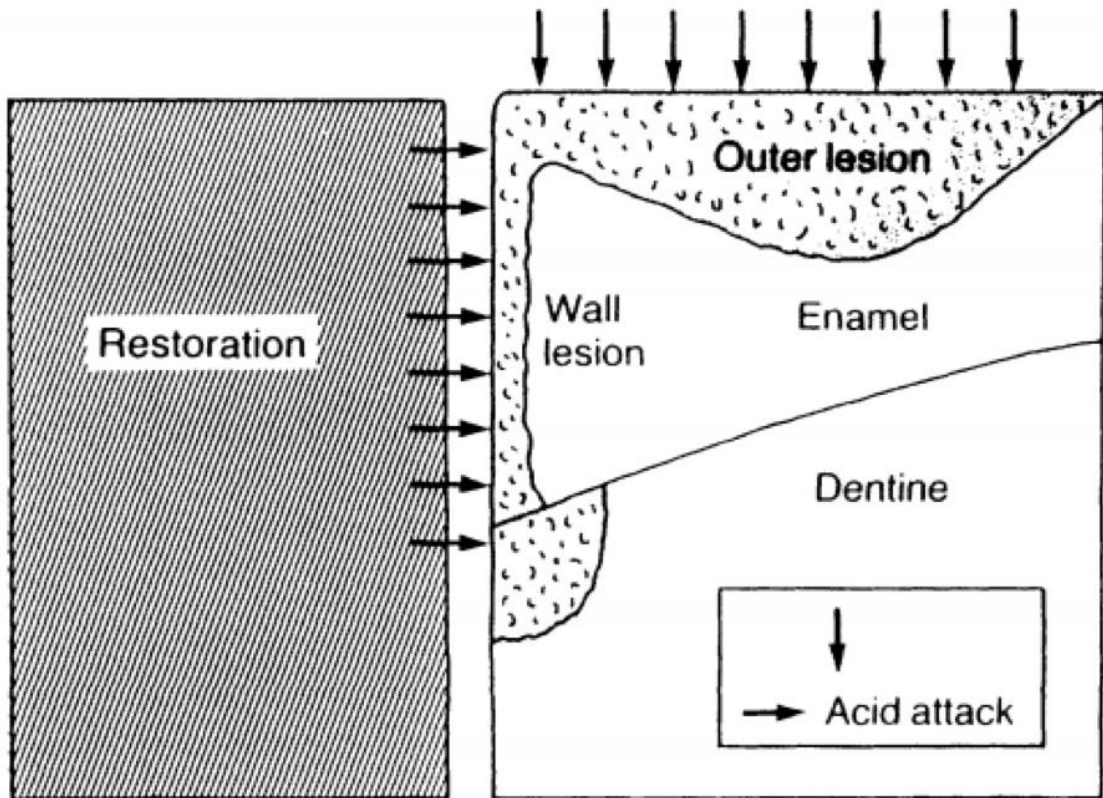


Fig. 3.10 A diagrammatic representation of secondary caries (Hals et al., 1975a)

3.6 Terminology of epidemiological studies about caries prevalence

This disease is also considered as being the most prevalent chronic disease in developed societies, being influenced by numerous genetic, cultural and social factors, which may explain, in part, large variations in the prevalence and incidence worldwide (Roberts et al., 2014).

In epidemiological studies the definitions of 59 terms related to dental caries and dental caries management were reviewed. (Machiulskiene et al., 2020)

Some of those terms should be revised to to understand the structure of epidemiological studies.

3.6.1 Definitions of Dental Caries as a Disease

Caries Activity

Caries activity is a concept that reflects the mineral balance, in terms of net mineral loss, net mineral gain, or stasis over time. Caries active implies caries initiation/ progression; caries inactive implies caries arrest/regression (Thylstrup et al., 1994).

Prognosis of Caries

Prognosis of caries is the likely or expected course of dental caries.

Caries Free

Caries free implies that there are no detectable signs of dental caries. It is a label that often leads to misunderstanding. This term should not be used without clearly indicating the threshold level. (Machiulskiene et al., 2020)

Cavity Free

Cavity free implies that there are no detected cavities in dentine. However, thorough clinical examination may reveal the presence of non-cavitated and/or micro-cavitated carious lesions. (Machiulskiene et al., 2020)

3.6.2. Definitions of Terms Used in Dental Caries Epidemiology

Caries Experience

Caries experience is the number of teeth/surfaces that have caries lesions (at a specified threshold), restorations, and/or are missing due to caries, accumulated by an individual, up to a designated point in time. Traditionally, it has been measured by means of DMFT/S (dmft/s) at

varying detection levels. New models and indices are being explored internationally. (Machiulskiene et al., 2020)

Caries Prevalence

Caries prevalence is, in a strict sense, the number/ proportion of individuals with caries in a given population at a specified threshold, at a designated point in time. Case definitions are often misunderstood and are necessary. In many studies, the prevalence of caries experience has been reported. Other specific examples of case definitions include reporting of total (untreated and treated) caries lesions in primary and permanent teeth or untreated caries, which includes lesions in primary and permanent teeth that have not received appropriate treatment (Fleming & Afful, 2018).

Caries Incidence

Caries incidence is, in a strict sense, the number/proportion of individuals with new or progressing caries at a specified threshold in a given population, detected during a given period. (Machiulskiene et al., 2020)

Caries Surveillance

Caries surveillance is the ongoing, systematic collection, analysis, and interpretation of caries data, essential to the planning, implementation, and evaluation of public health practice, and the timely dissemination of these data to those who need to know so that action can be taken. (Machiulskiene et al., 2020)

Caries Risk

Caries risk is the probability that caries lesions will appear or progress if conditions remain the same within a stated period of time. (Machiulskiene et al., 2020) Caries risk is a proxy for the true outcome (new caries lesions or progression), which can only be validated over time.

Caries Risk Factor/Determinant

Caries risk factor/determinant is an environmental, behavioral, or biological factor confirmed by temporal sequence, usually in longitudinal studies, which, if present, directly increases the probability of caries occurrence. The risk factor is part of the causal chain (Burt, 2001).

Modifiable Risk Factor

Modifiable risk factor is a determinant that can be modified by intervention, thereby reducing the probability of caries.

Caries Risk Indicator/Marker

Caries risk indicator/marker is a characteristic associated with increased probability of caries or increased occurrence of caries . (Machiulskiene et al., 2020) A risk indicator is not causally associated with the disease.

Caries Risk Management

Caries risk management is the measures taken to reduce the caries risk to which an individual or population is subject. (Machiulskiene et al., 2020)

3.7 Dental caries assessment tools

A variety of tools for the assessment of dental caries have been presented in the literature. The most widely known and used is the decayed, missing and filled teeth (DMFT) index, recommended by the World Health Organization (WHO, 1997). Although the DMFT index has advantages, these are outweighed by its weaknesses in relation to current thinking in caries epidemiology because it does not account for the consequences of untreated cavitated dentine lesions and do not record carious lesions in enamel. Subsequently, the index provides an underestimation of the prevalence and severity of caries. (Fejerskov et al., 2008)

In an attempt to overcome difficulties experienced with the DMFT index and to combine other caries assessment indices, a new visual and tactile dental caries detection system was developed for international use; this is designated the International Caries Detection and Assessment System (ICDAS) (Ismail et al., 2007; Pitts, 2004) This index was meant to be used in clinical practice and for education, research and epidemiological purposes. The ICDAS Coordinating Committee has since made a few changes to the original index and has named the latest version of the index ICDAS II. (Pitts, 2009)

However, the ICDAS II two-digit coding system is complicated for use in caries epidemiological research. The system encumbers data analysis and makes it difficult to present results in a meaningful and easy-to-read manner (Agustsdottir et al. 2010; Cadavid et al., 2010; de Amorim, 2012). Moreover, application of the system requires the use of an air compressor for drying each tooth surface, which necessitates additional financial resources and a source of electricity. The latter makes it difficult to consider ICDAS II an index for global use in epidemiological surveys. Furthermore, it is time-consuming to use (Braga et al., 2009; de Amorim et al 2012).

A special index for assessing the very advanced stages of the carious process was reported recently. This is termed the *pulpal involvement ulceration fistula abscess* (pufa/PUFA) index (Monse et al., 2010). The pufa/PUFA index provides information about the presence of oral conditions resulting from untreated dentine cavities. However, subsequent to its application in schoolchildren, it has been suggested that code 'u' should be excluded from the PUFA index and codes 'f' and 'a' combined (Figueiredo et al., 2011)

In view of the respective advantages and disadvantages of the ICDAS II, pufa and DMFT indices, a new index was proposed. The rationale and content of this index, termed the Caries Assessment Spectrum and Treatment (CAST), have been reported (Frencken et al., 2011). The CAST index describes, in a hierarchical way, the complete range of carious conditions, from the absence of carious lesions, to the presence of caries protection (sealant) and caries treatment

(restoration), lesions in enamel and dentine, lesions penetrating the pulp and tissue surrounding the tooth (abscess/fistulae), and loss of teeth. The hierarchical approach implies that a high CAST score is considered to represent more severe condition than a low CAST score. The index was developed to be used solely in epidemiological surveys globally. Assessment is performed visually and the use of compressed air is not required (Frencken et al., 2011).

DMF surfaces index (DMFS) which measures the severity of dental caries. Each tooth was recorded scored as 4 surfaces for anterior teeth and 5 surfaces for posterior teeth. Retained root was recorded as 4 D for anterior teeth, 5 D for posterior teeth. Missing tooth was recorded as 4 M for anterior teeth, 5 M for posterior teeth. Tooth with crown was recorded as 4 F for anterior teeth, 5 F for posterior teeth.

Minimum score = 0

Maximum score= 128

For DMFS, this is either 128, or 148, based on the inclusion of the third-molar surfaces. (Cappelli et al., 2007)

For over 70 years, the Decayed, Missing and Filled Teeth (DMFT) index has been globally used as the most important index for assessing the status of oral and dental health. Moreover, this index is the most important index used in epidemiological studies of the health status of the community (Broadbent et al., 2005). This index determines the number of decayed teeth, the number of treated teeth, and the number of teeth missed due to decay (Roland et al., 1994). This index is used to evaluate and monitor oral health interventions in the community by developing policies and programs related to this area (Marthaler, 2004; Nadanovsky et al., 1995).

3.8 Prevention and non-invasive treatments of dental caries

Primary prevention involves the adoption of measures to prevent disease or ill health. In contrast secondary prevention involves strategies to diagnose and treat an existing disease in its early stages before it results in significant morbidity. Tertiary prevention involves treatment of established disease, by restoring function and also reducing disease-related complications. (Harris et al., 2004)

Primary prevention in dentistry essentially covers the following methods: brushing, use of dental floss, regular dental appointments, fluoride administration, application of fissure sealants; balanced and non- cariogenic diet. The application of these methods are dependent of the knowledge about primary prevention and oral health and sociodemographic and cultural factors of the community (Daniel et al., 2008; Harris et al., 2004).

3.9 Sociodemographic aspects of dental caries

The increased risk of oral disease is associated with low socioeconomic status, low educational level and cultural factors that often determine a greater difficulty in access to information and health (Nicolau et al., 2005; Oliveira et al., 2008; Timis, 2005; Touger-Decker et al., 2003).

Overtime, and despite the continuing high prevalence of dental caries worldwide, the prevalence of the disease has declined from high to low and it is moderate for the majority of the developed countries (Axelsson, 2004; Petersen et al., 2000). There is a persistence of oral problems in many communities, particularly among the less privileged with worse socioeconomic status, both in developed and in developing countries (Bastos et al., 2007; Timis, 2005).

In some industrialized countries, the decrease of the prevalence of dental caries and other oral diseases is explained largely by the interventions achieved through oral health promotion programs (WHO 2003; 2014). This pattern of evolution of the prevalence of dental caries is directly related to the many preventive measures implemented in recent years, as the fluoridation of public water supply, administration of fluoride mouthwashes and fluoride toothpastes and the improvement in standards of oral hygiene. In addition to these factors, some authors described the improvements in the provision of oral health services as responsible for the decline of dental caries (Hobdell, 2009).

As an example, the large decrease in the prevalence of dental caries in the Scandinavian countries (Sweden and Finland), Australia and New Zealand, was achieved through the implementation of oral health promotion programs aimed at the prevention of dental caries (Hattne et al., 2007; WHO 2003;).

However, in many developing countries whose populations do not have access to oral healthcare the health system implemented give very insufficient attention to the need for primary prevention towards oral diseases (Oliveira et al., 2008; WHO 2007). In some of these countries there has been an increase in the prevalence of dental caries justified by the direct influence of the existing socioeconomic conditions in these populations. The exceptions were noticed for the countries where programs to promote oral health and disease prevention have been implemented (Cortelli et al., 2004; Sgan-Kohen, 2009).

As an example, the large decrease in the prevalence of dental caries in the Scandinavian countries (Sweden and Finland), Australia and New Zealand, was achieved through the implementation of oral health promotion programs aimed at the prevention of dental caries (Hattne et al., 2007; WHO 2003).

However, in many developing countries whose populations do not have access to oral healthcare the health system implemented give very insufficient attention to the need for primary prevention towards oral diseases (Oliveira L et al., 2008; WHO, 2007). In some of these countries there has been an increase in the prevalence of dental caries justified by the direct influence of the existing socioeconomic conditions in these populations. The exceptions were noticed for the countries where programs to promote oral health and disease prevention have been implemented (Cortelli et al., 2004; Sgan-Cohen, 2009).

The impact of oral diseases both at individual and community level, in terms of pain and suffering, disability and reduced quality of life is quite considerable. This impact is also higher in developed countries (WHO 2007; 2012).

Reducing oral health conditions calls for a reform of oral health systems to shift the focus from invasive dental treatment to prevention and more minor treatment.

WHO has identified key strategies for improving oral health, with a focus on low-income and marginalized populations where access to oral health care is most limited. These include strengthening both cost-effective population-wide prevention and patient-centred primary health care.

This work is being implemented through a three-year roadmap (2019-2021) that comprises a mix of normative work and practical support to countries. A top priority is the development of a global oral health report, which will provide information about the status of oral health globally. The report will serve as the evidence base for the development of a global oral health action plan.

Reducing oral health conditions calls for a reform of oral health systems to shift the focus from invasive dental treatment to prevention and more minor treatment.

WHO has identified key strategies for improving oral health, with a focus on low-income and marginalized populations where access to oral health care is most limited. These include strengthening both cost-effective population-wide prevention and patient-centred primary health care.

This work is being implemented through a three-year roadmap (2019-2021) that comprises a mix of normative work and practical support to countries. A top priority is the development of a global oral health report, which will provide information about the status of oral health globally. The report will serve as the evidence base for the development of a global oral health action plan.

Literature review: conclusion

In order to understand any disease, including dental caries, the different natures of it should be taken into consideration. The most effective way to prevent any disease is the primary prevention. In case of dental caries it is a good oral hygiene, which means regular and technically correct brushing of teeth at least 2 times a day for 2-3 minutes using fluoride-containing toothpaste and tooth flossing. In addition to good oral hygiene, it is requested to reduce the factors that could harm oral health – unhealthy eating habits, snacking, excessive alcohol consumption and smoking. The paramount importance is oral health education in family, as the child will grow into an adult who could have adopted the family model and would not always be ready to change it if it does not work. It is also very important to understand and analyse the barriers that discourage an individual from receiving dental treatment - limited access to dental services, lack of knowledge about the importance of oral health, costs and fear of the dentist.

The purpose of epidemiology is to understand what risk factors are associated with a specific disease, and how disease can be prevented in groups of individuals; due to the observational nature of epidemiology, it cannot provide answers to what caused a disease to a specific individual.

Knowledge of prevalence of dental caries in population could allow analysis of public education on oral health, as well as to make comparisons between different age groups and genders. The results of epidemiological research may raise the question of the financial availability of dental services to the public and the level of public motivation to go to the dentist. As well as the comparison of epidemiological data over different time periods could help to assess the level of effectiveness of preventive measures. Last but not least, epidemiological studies that contain internationally recognized methods are comparable, which allows us to draw conclusions about the highest possible effectiveness of other countries' approaches.

Therefore, the conducting of this study is aimed to not only to assess possible epidemiological situation regarding caries prevalence in selected adult population of Latvia, but also to reveal the need for further research in this and derived areas.

4 RESULTS

Overall, there were 150 patients examined and treated by dental students (44 males and 106 females). The result are described and showed with diagram and tables (Fig. 4.1). The results are ascribed to the different subgroups. Total of examined teeth are 4800.

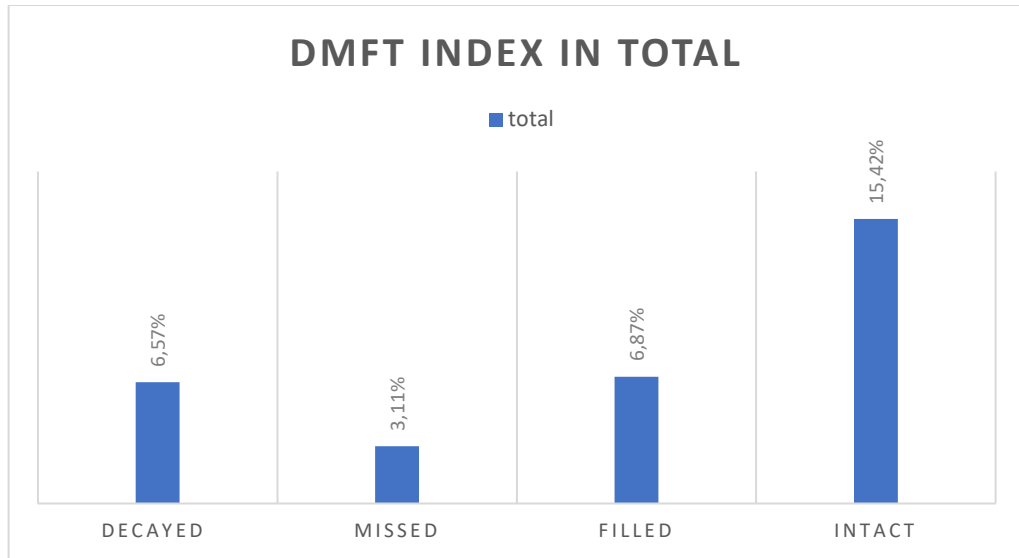


Fig. 4.1 DMFT mean value in total

The mean DMFT index value in both genders was analyzed as well.

The results can be evaluated and compared between 2 different group of the participants. In female group DMFT index is higher than it is in the male group (Fig. 4.2).

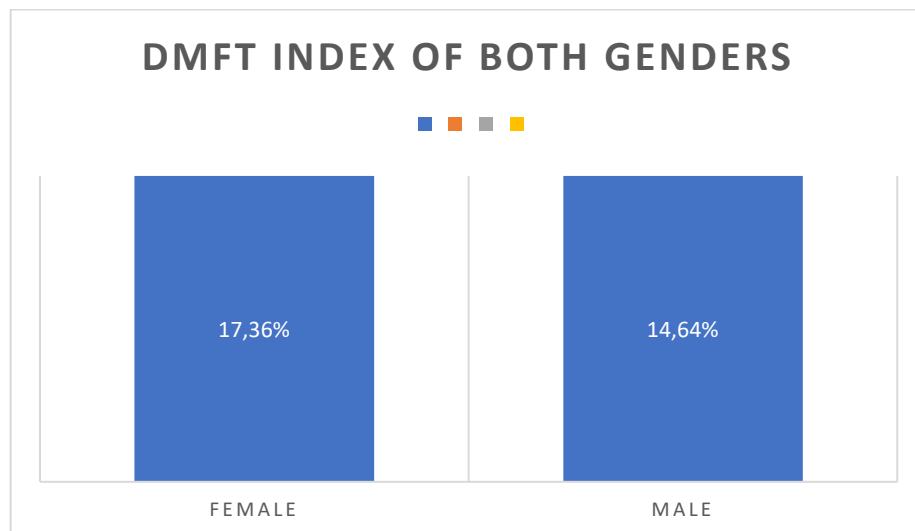


Fig. 4.2 DMFT index of both genders

Prevalence of decayed teeth what need dentist help higher in male group, but the difference is not that high. Prevalence of missed teeth is higher of female than in male, what could show us that the hormonal level of woman could play significant role in health of oral cavity (Fig. 4.3).

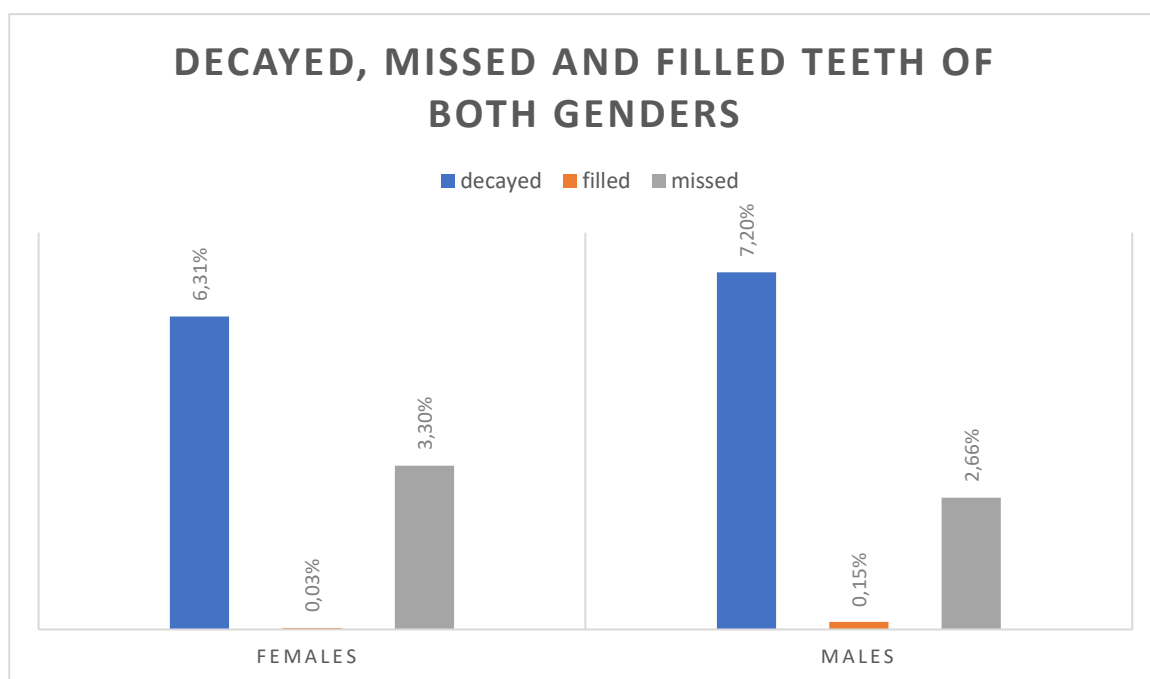


Fig. 4.3 Decayed, missed and filled teeth of both genders

Prevalence of filled teeth is higher in male than in female than could show us that prevalence of caries in male is higher than in female possible due to everyday oral hygiene and preventive measures (Tab.4.1).

Table 4.1 DMFT index of the participants

Value	Total nr of teeth in all participants	Total nr of teeth in all participants in %	Total N of teeth in men	Total N of teeth in men in %	Total N of the teeth in female	Total N of teeth in female in %
Total nr of teeth	4800	100%	1408	100%	3392	100
DT	986	6,57%	317	7,2	669	6,31
MT	467	3,11%	117	2,66	350	3,3
FT	1031	0,21%	210	0,15%	821	0,031%
DMFT	4800	16,56%	1408	14,64%	3392	17,36%
Intact teeth	2313	15,42%	764	17,36%	1539	14,52%

The mean DMFT values for various age groups and genders were analyzed and showed that the females who were participating in this research were slightly more in number than males (Fig.4.4).

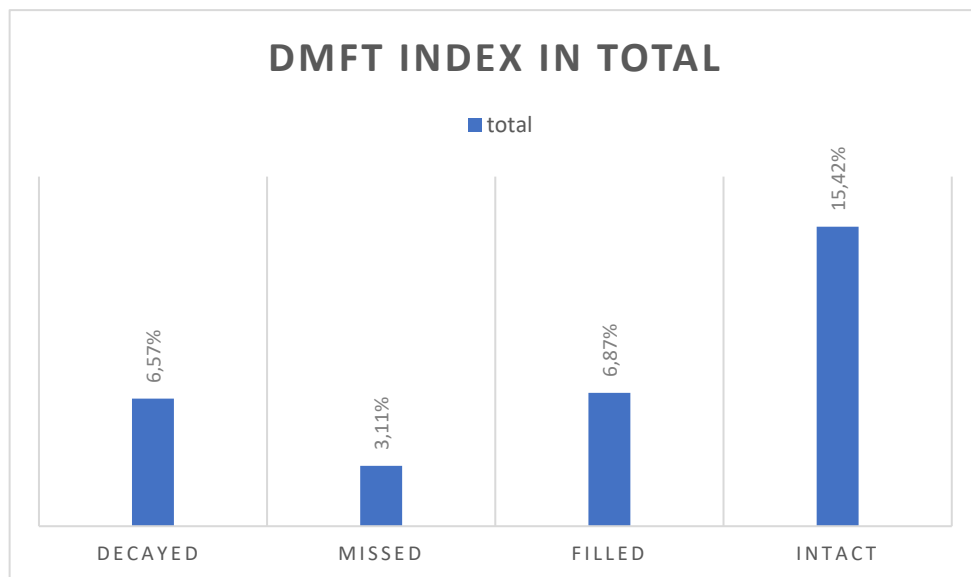


Fig. 4.4- DMFT index in total

The group of 18-25 years old was reported the highest result in female group what was 14,5 % and male 10,5%. The group of 26-35 years old females have the highest percent the males (F-14,32%; M-11,27%). The group of 36-45 years old females have the higher results than the male group (M-17,09%; F-19%). The group of 46-60 years old have higher result in male population then in female. And in group of more than 60 years old, (the age described in literature as old adult age) the male result was more higher than in the female group. (Fig. 4.5; 4.6; 4.7)

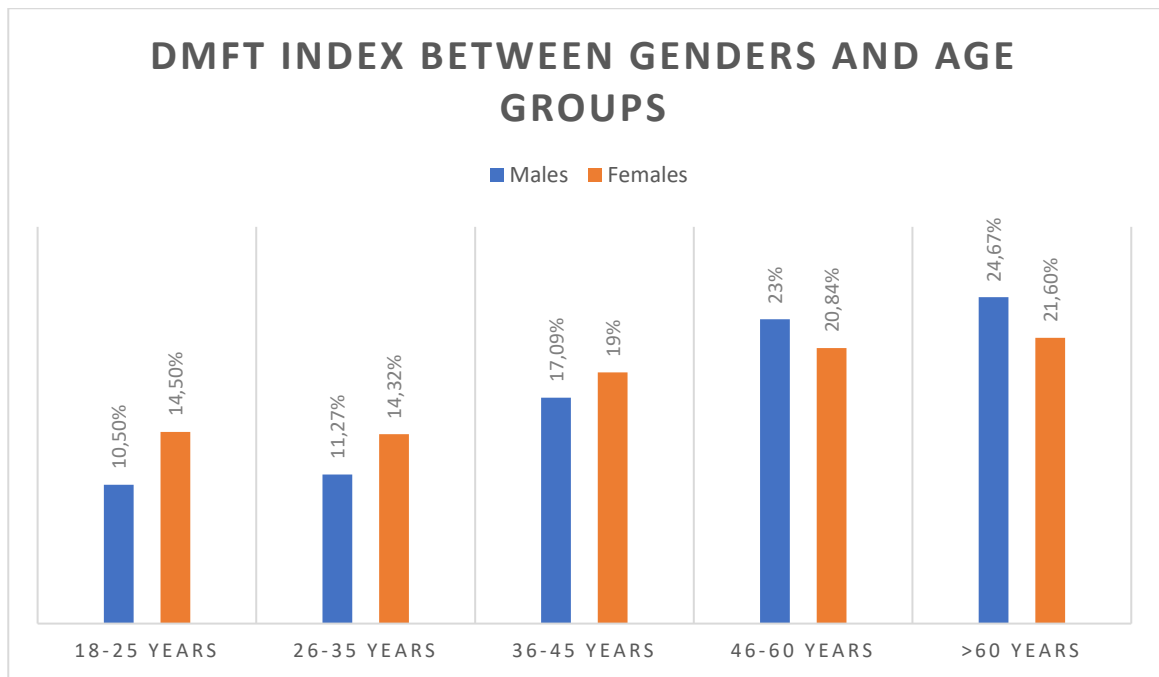


Fig. 4.5 DMFT index between genders and age groups

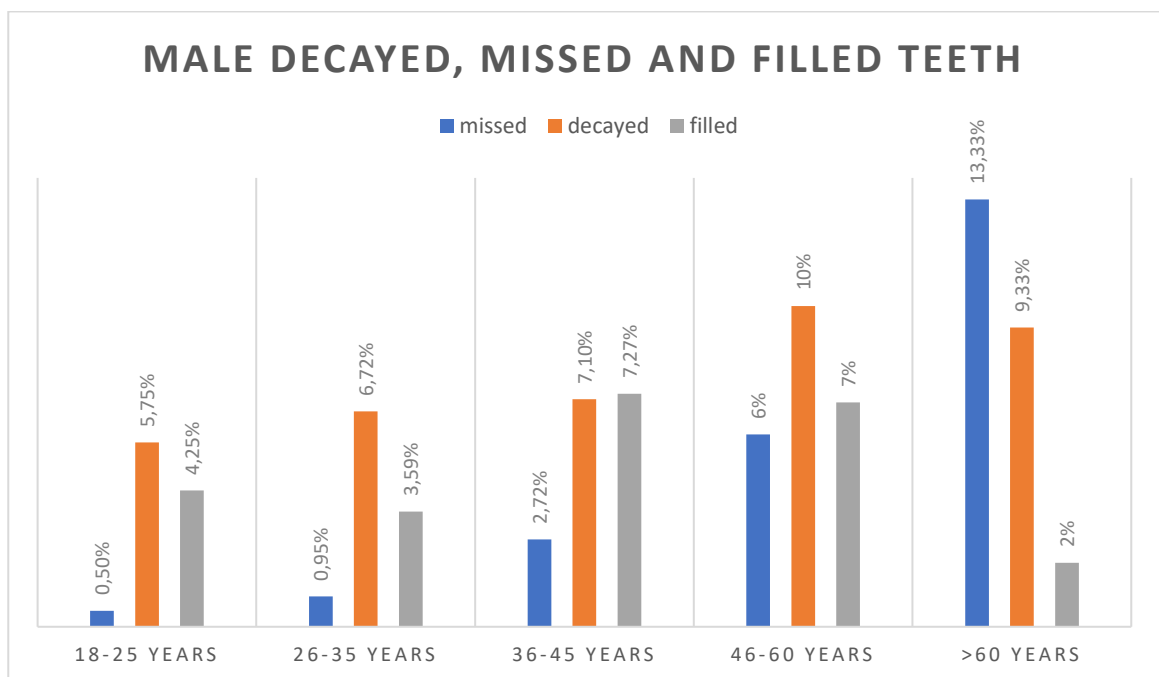


Fig. 4.6 Male decayed, missed and filled teeth

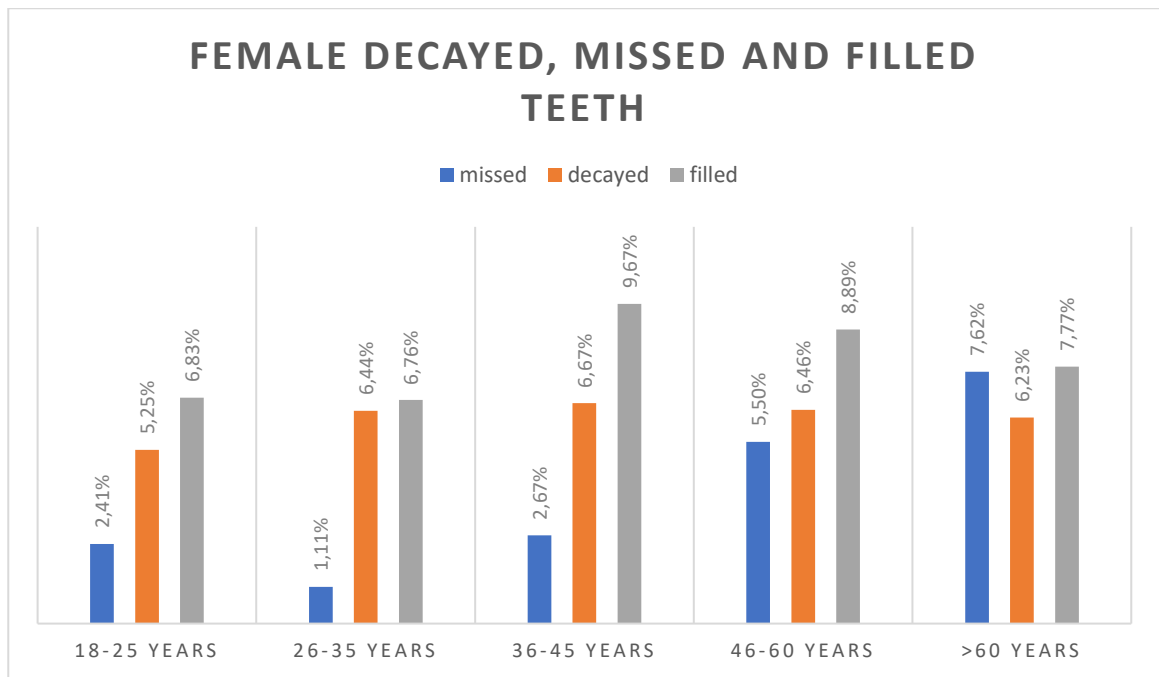


Fig. 4.7 Female decayed, missed and filled teeth

Nominal, ordinal and quantitative variables were used for descriptive statistics. Nominal variables used were – sex and dental status. Ordinal variable what was used- age. Quantitative variables used were decayed teeth; filled teeth; missed teeth; healthy teeth; age; DMFT. The relationship between age and sex and unhealthy teeth was determined.

Descriptive statistics of study variables

Age median for all participants was determined as 33.00 with SD = 14.206 (Fig. 4.8).

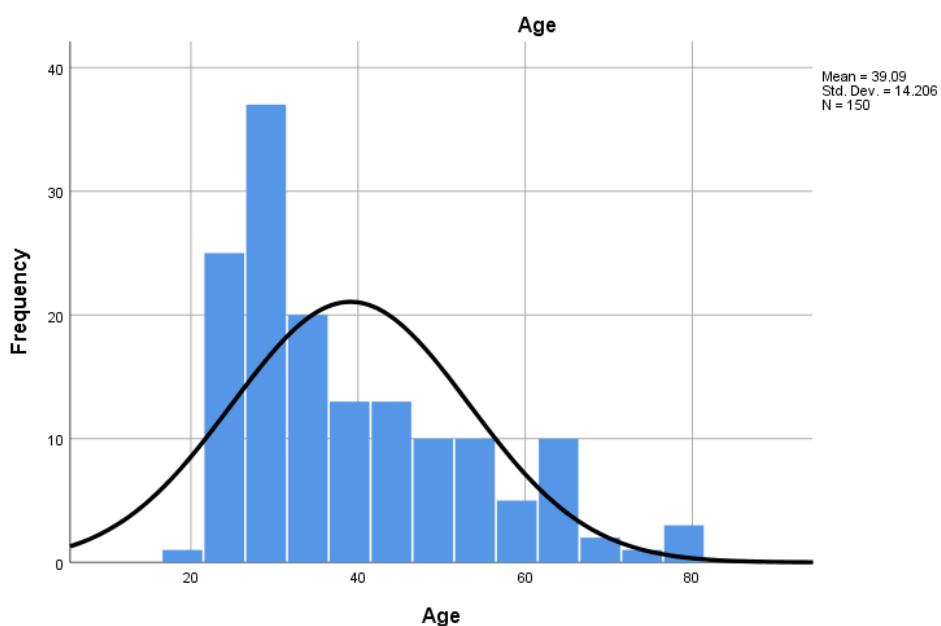


Fig. 4.8 Age median for all participants

Median for extracted teeth was 2.00 with SD = 4.144 (Fig. 4.9).

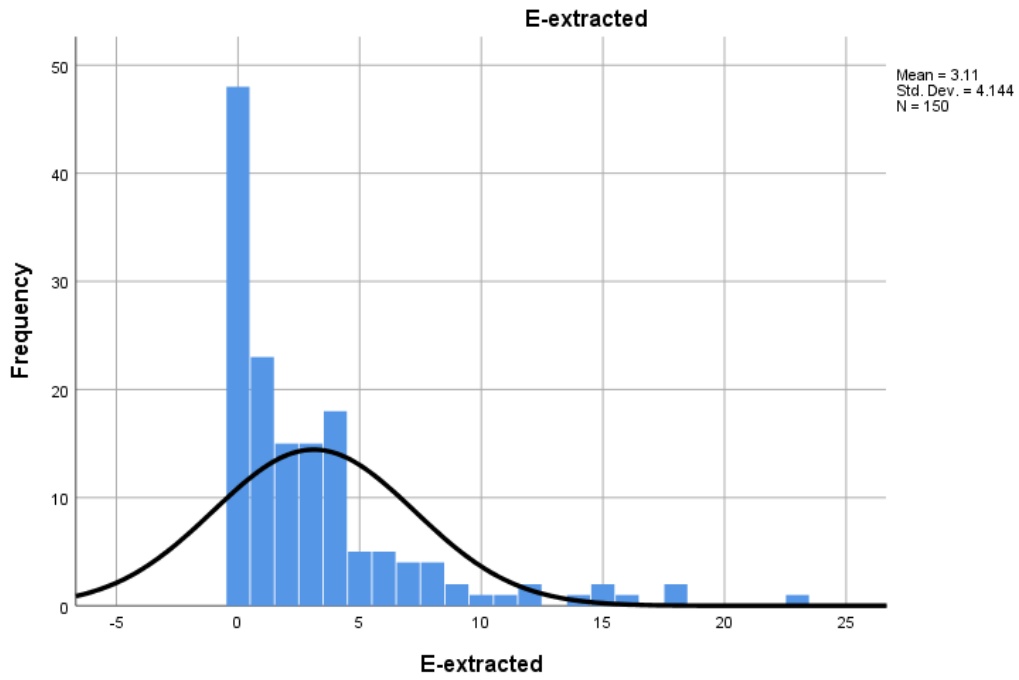


Fig. 4.9 Median for extracted teeth

Median for decayed teeth was 6.00 with SD = 3.943 (Fig. 4.10).

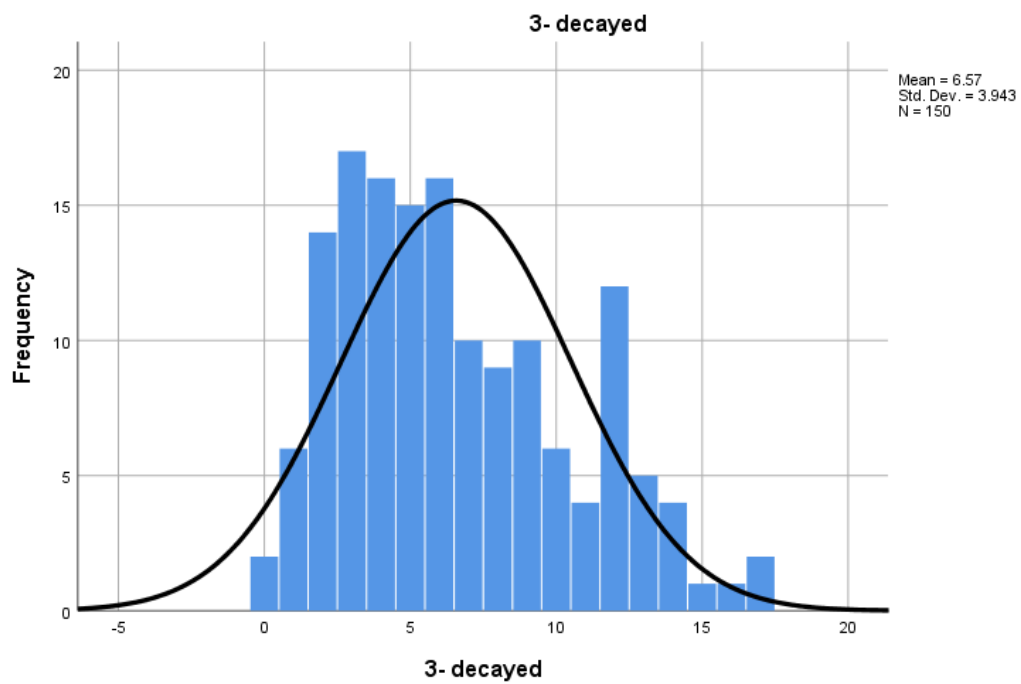


Fig. 4.10 Median for decayed teeth

Median for filled teeth was 6.00 with SD =4.573 (Fig. 4.11).

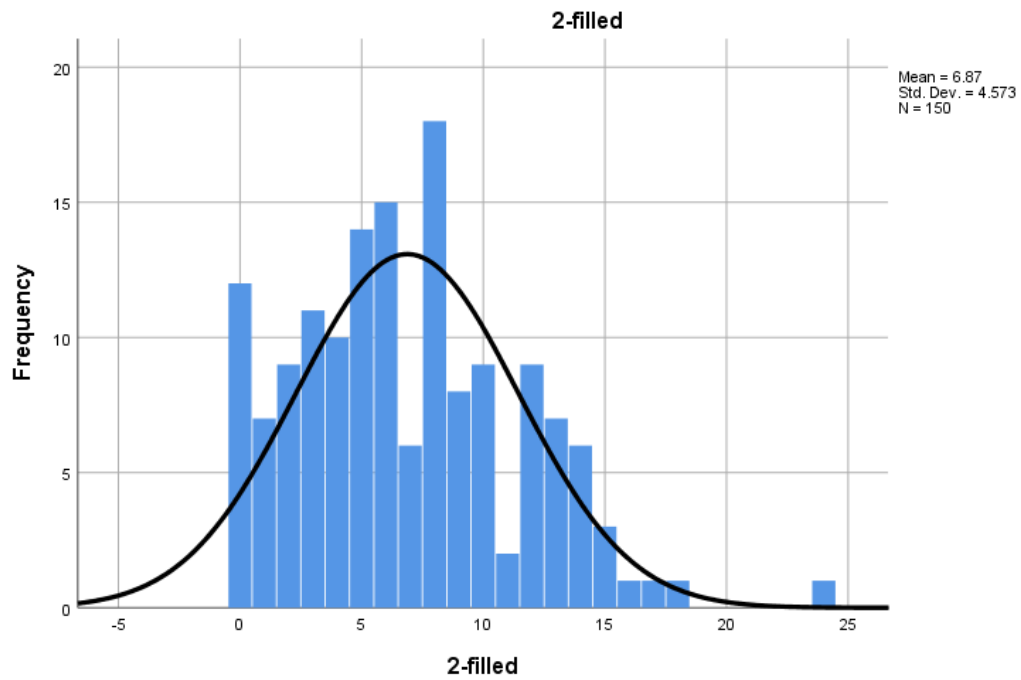


Fig. 4.11 Median for filled teeth

Mean value for healthy teeth in all participants was determined as 15.42. with SD = 6.257 (Fig. 4.12).

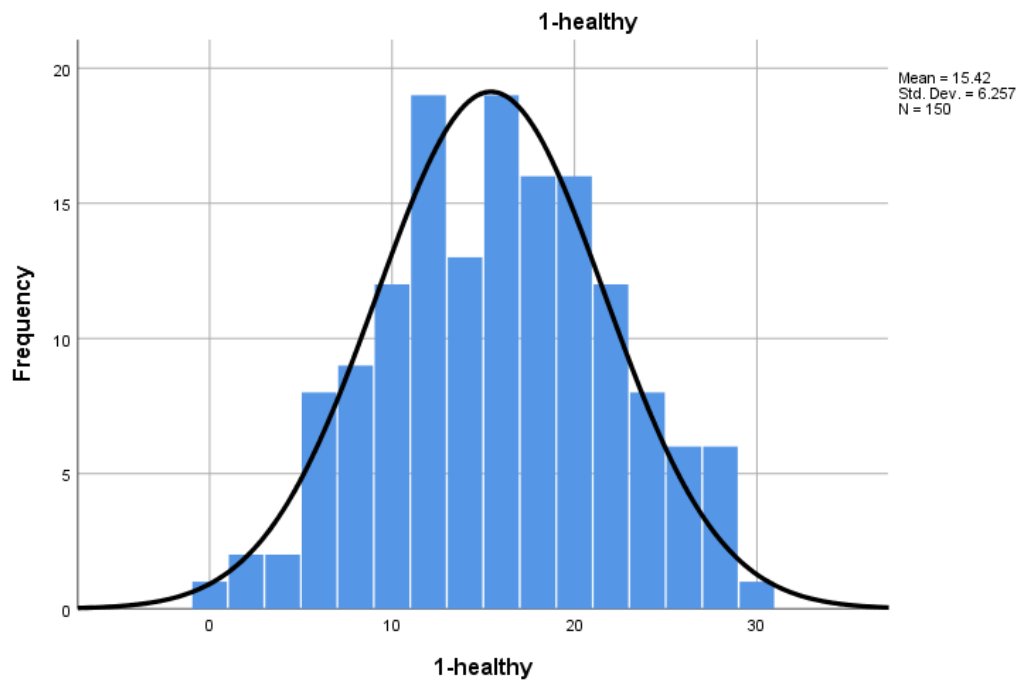


Fig. 4.12 - Mean value for healthy teeth in all participants

A percentile (Fig. 4.13) is a term that describes how a score compares to other scores from the same set:

$$P_x = \frac{x(n + 1)}{100}$$

P_x = The value at which x percentage of data lie below that value

n = Total number of observations

Fig. 4.13 Formula for calculation percentiles in statistics

Analysing data obtained, multiple modes exists.

Mean value of non-healthy teeth was showed 16.5600 with SD=6.26108 with proportion of 51.7500 with SD=19.56586 (Fig. 4.14).

25 percentiles showed value of 28.00 in age, 0.00 value in extracted teeth value, 3.00 value in decayed teeth value and 3.00 value in filled teeth value.

75 percentiles showed value of 49.25 in age, 4.00 value in extracted teeth value, 9.00 value in decayed teeth value and 10.00 value in filled teeth value.

One-Sample Kolmogorov-Smirnov Test showed the lower bound of true significance.

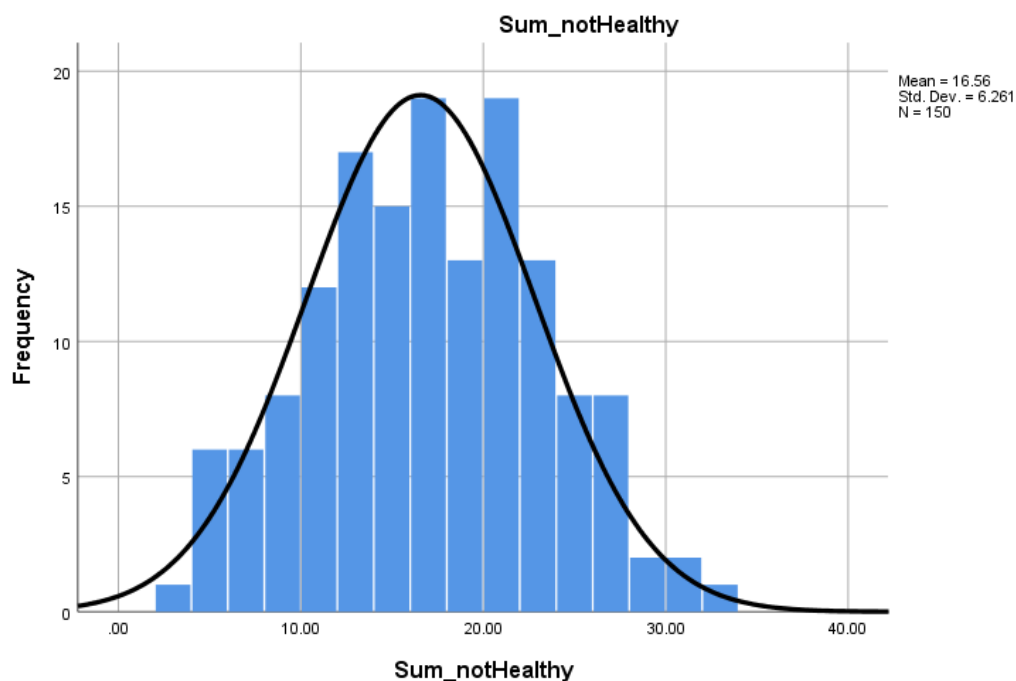


Fig. 4.14 Mean value of non-healthy teeth

One-Sample Kolmogorov-Smirnov test showed the lower bound of true significance.

Descriptive statistics according to the gender (each gender individually)

The mean value of extracted teeth in males was 2.66 (median – 1.00, SD = 4.226) (Fig. 4.15), decayed teeth – 7.20 (median – 6.50, SD = 3.951) (Fig. 4.16), filled teeth – 4.77 (median – 5.00, SD=3.697) (Fig. 4.17).

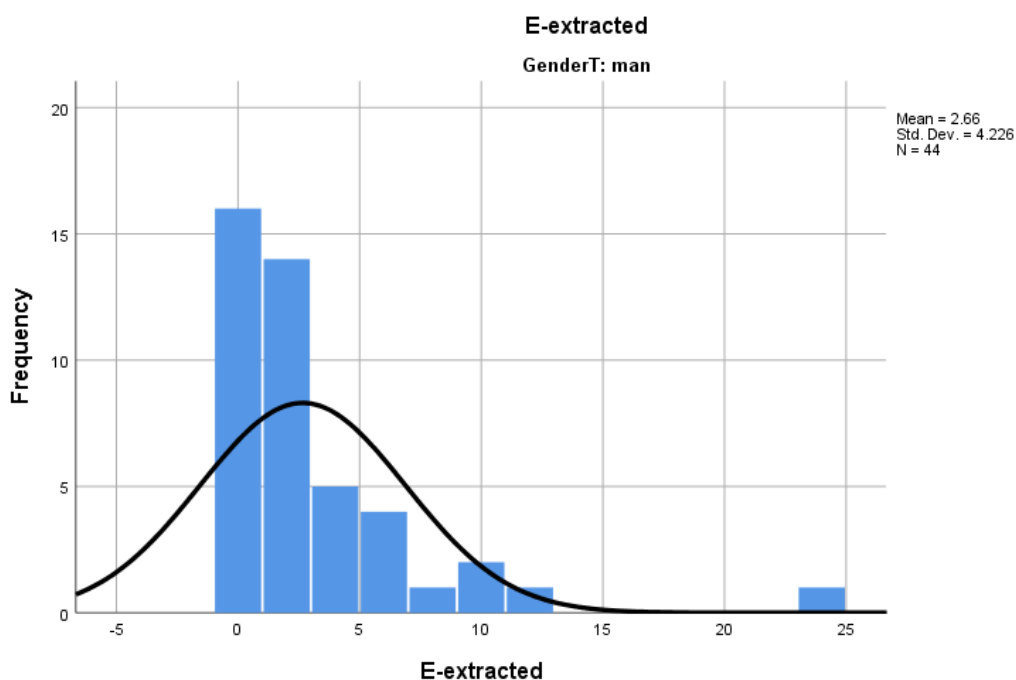


Fig. 4.15 Extracted teeth in males

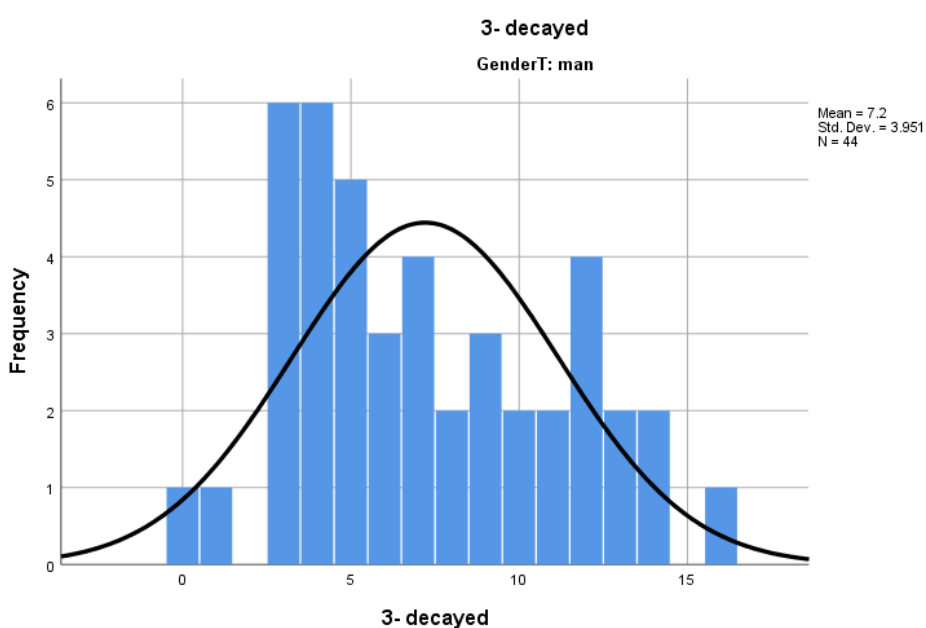


Fig. 4.16 Decayed teeth in males

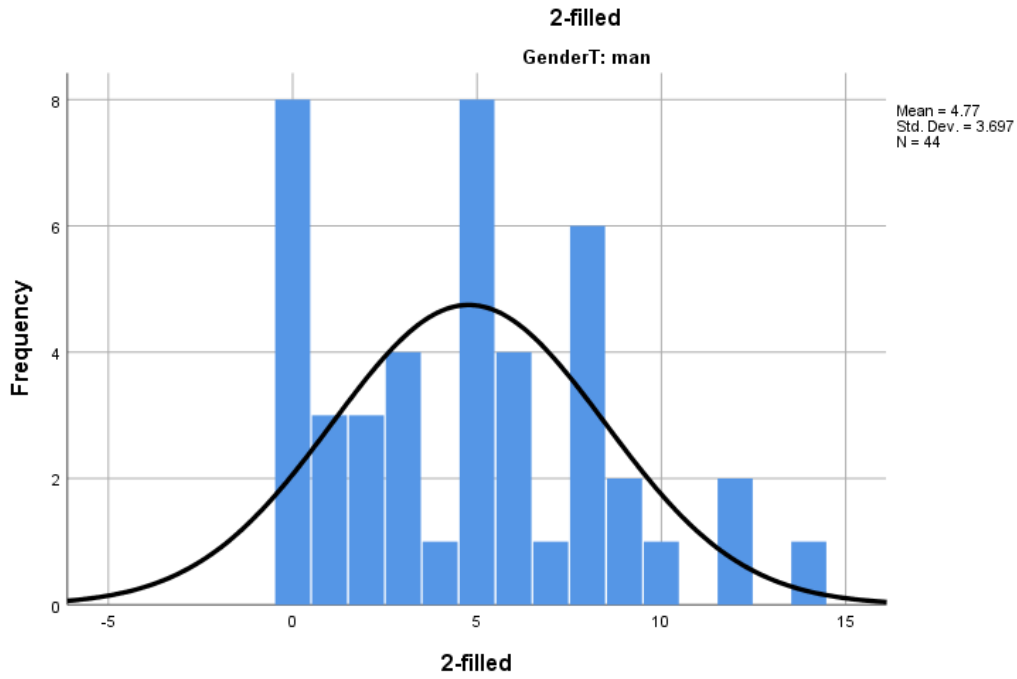


Fig. 4.17 Filled teeth in males

The mean value of extracted teeth in female was 3.30 (median 2.00, SD= 4.115) (Fig. 4.18), decayed teeth – 6,31 (median- 6.00, SD=3,929) (Fig. 4.19), filled teeth- 7.75 (median- 8.00, SD= 4.633) (Fig. 4.20).

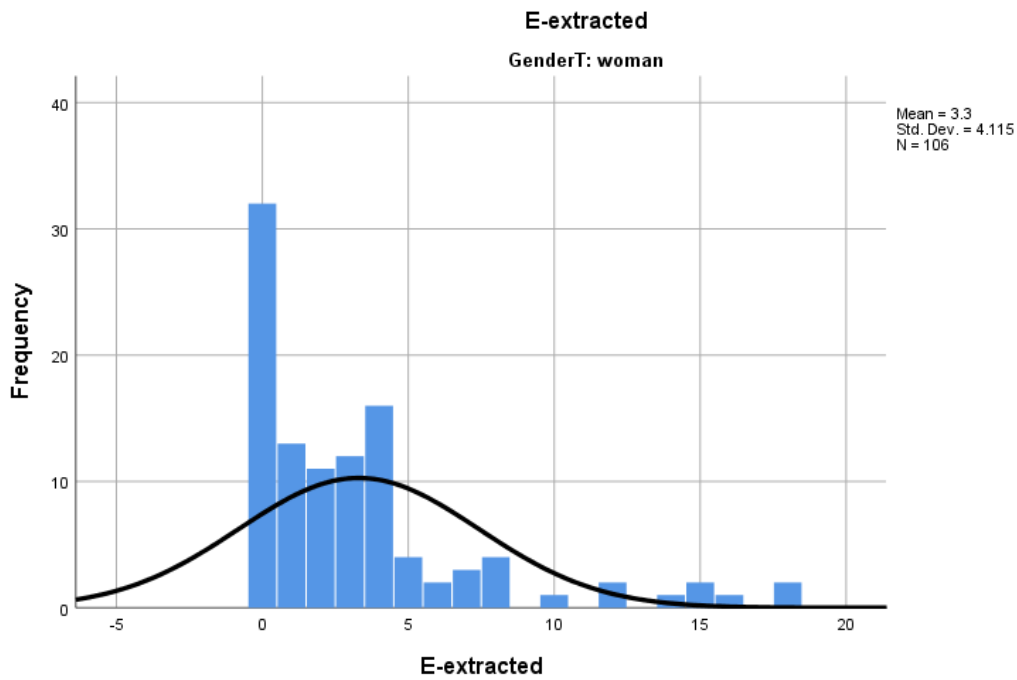


Fig. 4.18 Extracted teeth in females

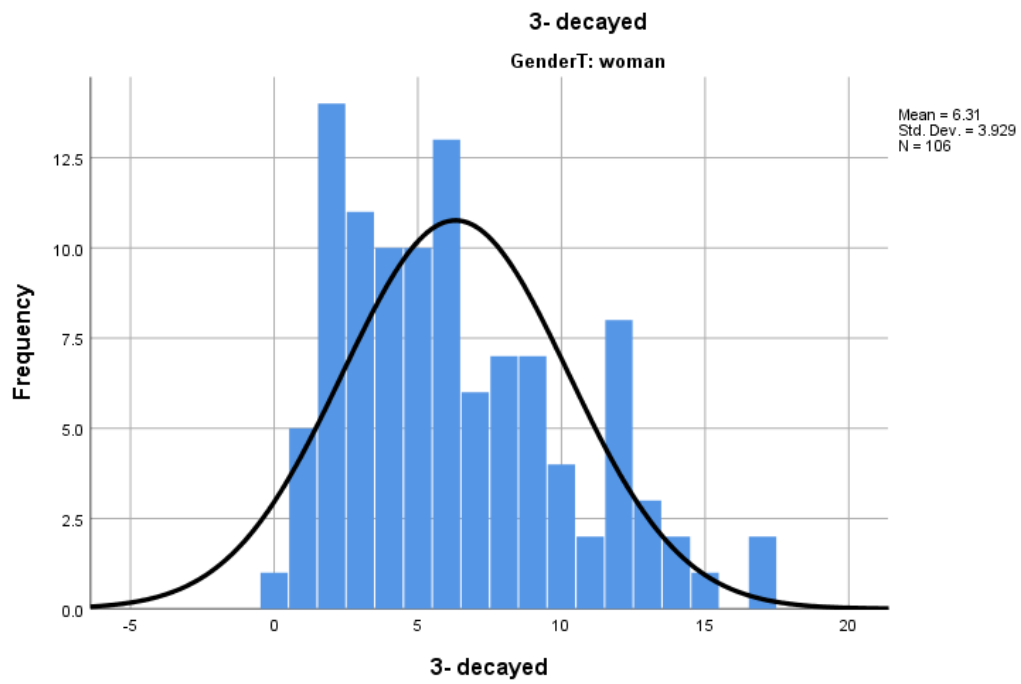


Fig. 4.19 Decayed teeth in females

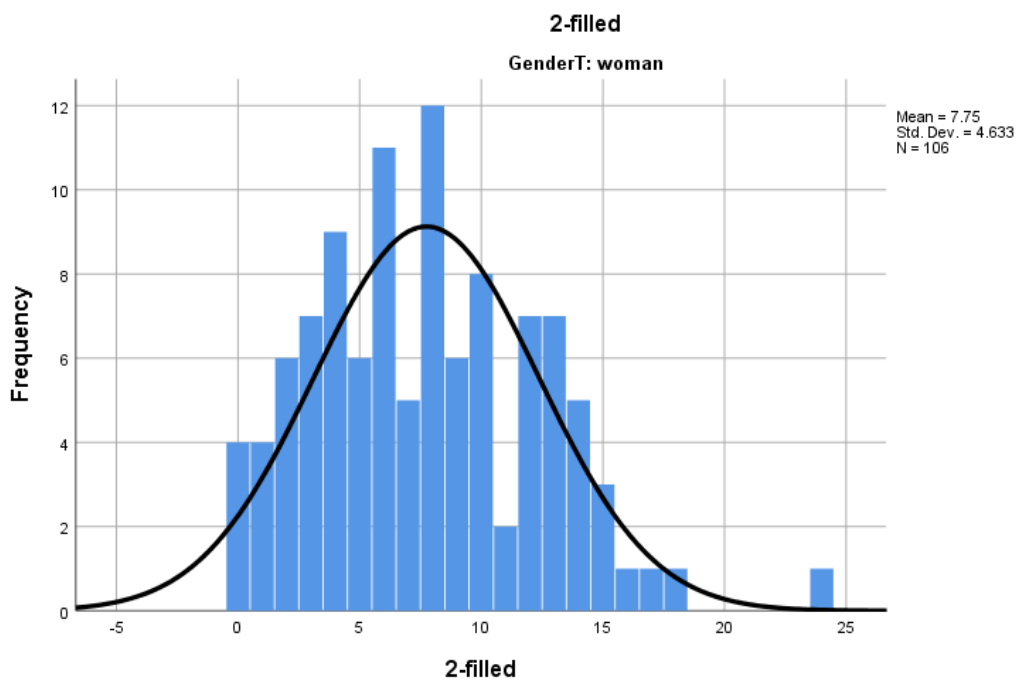


Fig. 4.20 Filled teeth in females

Univariate analysis

t-test was used for variables that are normally distributed – relationships with gender ($t = -2.465$ for sum of healthy teeth with sig. 0.015 with proportions for equal variances assumed $t = -2.465$ with sig. 0.015).

p value < 0.05 is statistically significant.

Multivariate analysis

The dependent variable – the percent of unhealthy teeth. The independent variables: age and gender. Both were statistically significant in the univariate analysis.

We built a linear regression model to explain the association between the percent of unhealthy teeth and the independent variable (Fig. 4.21). The model explains 36.5% of changes in the percent of unhealthy teeth (adjusted $R^2 = 0.365$).

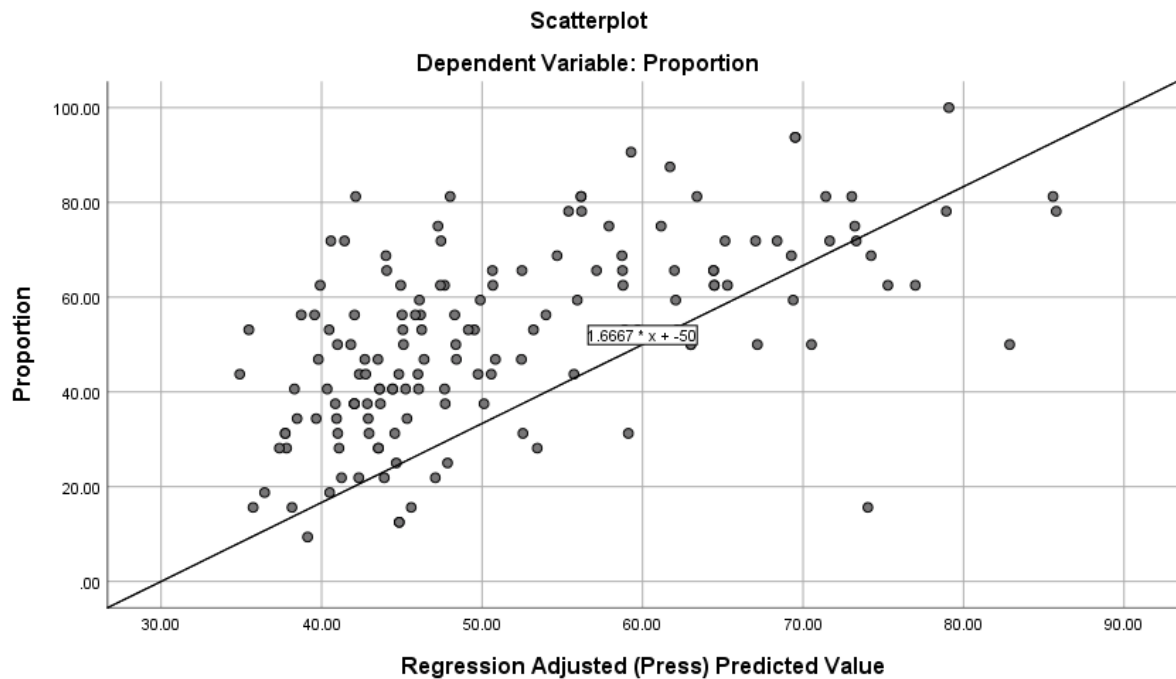


Fig. 4.21 Linear regression model of the association between the percent of unhealthy teeth and the independent variable

Mann-Whitney test was used for variables that distributed non-normally (Tab. 4.2).

Table 4.2. Mann – Whitney test for non-normally distributed variables

Test Statistics^a

	E-extracted	3- decayed	2-filled
Mann-Whitney U	2023.500	1999.000	1461.500
Wilcoxon W	3013.500	7670.000	2451.500
Z	-1.300	-1.380	-3.604
Asymp. Sig. (2-tailed)	.194	.168	.000

a. Grouping Variable: GenderT

In the multivariate linear regression model age was statistically significantly associated with the percent of unhealthy teeth: each year of age increases the possibility to have the percent of unhealthy teeth by 0.8 (effect estimate, $\beta = 0.80$, 95% confidence interval 0.62; 0.98, p value < 0.01). Gender did not display statistical significance in this model (p value = 0.07). However, the sample size in this study is very small, and it is possible that in the case of an increase of the sample size gender would be significantly associated with the percent of unhealthy teeth. In this case, women's gender became a risk factor for the increasing percent of unhealthy teeth. Null hypothesis - increase in the DMFT index may be observed with increasing of patient age was proven with $p < 0.01$.

5 DISCUSSION

Oral health is an indispensable component of general health and is considered a determinant of good quality of life (Petersen et al., 2007). Dental caries is the most prevalent oral problem, affecting more than 2.4 billion people globally in 2010. (Kassebaum et al., 2007) In addition, according to the WHO report, dental caries is the fourth most expensive chronic disease to treat. (Petersen et al., 2008)

DMFT-index is used in order to statistically determine the number of carious, extracted and filled teeth per person and the average value of this index in the tested population. DMFT is chosen because it is commonly used index for assessment of dental status in population. Primary, DMFT was developed for use in childhood and later is adapted for use in the adults. Some authors believe that DMFT index has low validity for assessment of caries in adults, primarily because it is not able to reveal the true impact of caries on the oral health. This index refers only to the presence of the disease, its consequences and the need for treatment, and it can present high values especially in adult populations, where the presence of caries and extracted teeth is high.

This index does not take into consideration the reasons for tooth loss - whether the tooth is lost due to caries or periodontal disease, risk for caries occurrence and dental assessment of the need for treatment. (Petrovski et al., 2015)

This study determined the mean DMFT index values different age and sex groups of patients treated by dentistry students. The study was conducted on patients who were visiting the Students Dental Clinic of University of Latvia. Dental students treat patients while supervised by the academic staff, taking responsibility for the students actions, according to the rules of the department and University of Latvia. The Students Dental Clinic provides students with well- equipped clinic rooms, so that they can provide patients with the best treatment available. Students perform dental examination and take care of the main complaints that patients have. In case the patient needs some complicated treatment, what student can't provide, patient is referred to another more competent institution.

In our study the analysis of the result revealed that unhealthy teeth among examined groups of the patients was relatively high. This is largely due to factors for example like socioeconomic status, low dental knowledge and poor oral hygiene habits. Most patients treated by students cannot afford to cover the private treatment expenses so that they prefer to be treated by students at the Students Dental Clinic at the University of Latvia, where all treatment (except prosthetic restoration) is for free. Various studies show that ones socioeconomic status is one of the risk factors for dental caries. (Petersen et al., 2005)

The mean of DMFT index in woman and men was the next factors what was emphasized in this work. Woman demonstrated higher values mostly in all study groups. This might be due to the fact that there were more female participants in the study. Other than that, some authors also noticed that caries are more prevalent in woman than men. These studies have shown that woman are more exposed to caries because of their different rate of saliva and hormonal fluctuations. (Lukacks et al., 2006)

The age of the patients was the next factor was analyzed in this study. The females who were participating in this research was slightly more amount than males. The group of 18-25 years old was reported the highest result in female group what was 14,5 % and male 10,5%. The group of 26-35 years old females have the highest percent the males (F-14,32%; M-11,27%). The group of 36-45 years old females have the higher results than the male group (M-17,09%; F-19%). The group of 46-60 years old have higher result in male population then in female. And in group of more than 60 years old, this age in literature described like old adult age the male result was higher than in the female group. This result shows us that the prevalence of unhealthy teeth increases with age. The prevention of dental caries should be a priority in any age, with special care given to the predisposing factors that lead to dental caries. (Szymanska et al., 2011)

Our pilot study what was are performed during our research work showed very good significance, emphasizing that age and DMFT is associated. In Latvia we have only research studies what are performed to study caries prevalence in child populations, during work writing process we didn't find investigations about adult population groups. Our study is unique in this field and give the opportunity to continue this research project in future, with more participants and more opportunities for this research.

6 CONCLUSION

It is possible to conclude that caries prevalence (DMFT index) depends from the age and gender. The aim of the study was approved, and the null hypothesis (the patient age increase then the prevalence of caries) was proved with good statistical significance. As well some other fact was highlighted - the mean of DMFT in females was higher in this study comparing with males.

We can emphasize that community programs should be considered and developed in order to improve knowledge and behaviors related with caries prevention programs for all populations not differencing them to the age groups. Good oral health is a major resource for social, economic and personal development. Government can allocate money from the state budget that dentistry field availability can be for all citizens useful and country can show good levels on GDP in yearly statistic around the world. This study can be improved, for example to include more participants into this study and improvement of the epidemiological situation in the country. For the future task it is interesting to compare this study with different study years what was performed previously or can be performed in future.

ACKNOWLEDGEMENT

First of all and most important I wish to thank my supervisor lecturer dr. Baiba Krauze for the most continuous guidance, invaluable advice and mentorship she has provided throughout my time as a student. This human with great and unstoppable energy, good academic, life and work experience, without her this thesis wouldn't happened. Dr. Baiba Krauze was always helping and was in communication due to all process of writing the thesis. Her advice has motivating me to study and write this thesis. I am very thankful for all of you feedback and advices. Dr. Baiba Krauze did very perfect and priceless work.

Also I want to thank the medical faculty at the University of Latvia and the director of dentistry program Dr Romualds Razuks for supporting during the all period of my study. Also, all our clinical stuff who work with us throughout the whole our study years.

Last but not least I would like to thank my family, especially to my brother Vladimir and my best cat Kuzjanka, they supporting me for all of this 5 study years.

BIBLIOGRAPHY

1. Aas J, Paster B, Stokes L, Olsen I, Dewhirst F. Defining the normal bacterial flora of the oral cavity. *J Clin Microbiol.* 2005; 43(11):5721-32
2. Agustsdottir H, Gudmundsdottir H, Eggertsson H, *et al.* Caries prevalence of permanent teeth: a national survey of children in Iceland using ICDAS. *Community Dent Oral Epidemiol*, 38 (2010), pp. 299-309
3. Anil, S.; Anand, P.S. Early childhood caries: Prevalence, risk factors, and prevention. *Front. Pediatr.* 2017; 5, 157.
4. Antunes J, Peres M. *Epidemiologia da Saúde Bucal.* 1st ed. Rio de Janeiro, Brazil: Guanabara Koogan; 2006.
5. Aoba T. Solubility properties of human tooth mineral and pathogenesis of dental caries. *Oral Dis.* 2004; 10:249-57
6. ARCHIVES OF THE AMERICAN DENTAL ASSOCIATION. Internet. https://www.mouthhealthy.org/~media/ADA/Education%20and%20Careers/Files/dental_history.pdf
7. Axelsson P. *Diagnosis and Risk Prediction of Dental Caries.* 1st ed. Slovakia: Quintessence Publishing; 2004
8. Bastos J, Gigante D, Peres K, Nedel F. Social determinants of odontalgia in epidemiological studies: theoretical review and proposed conceptual model. *Cien Saude Colet.* 2007; 12(6):1611-21.
9. Benjamin RM. Oral health: the silent epidemic. *Public Health Rep.* 2010;125(2):158-159. doi:10.1177/003335491012500202
10. Bowen WH. Wither or whiter caries research? *Caries Res* 1999;33:1-3.
11. Braga MM, Oliveira LB, Bonini GA, *et al.* Feasibility of the International Caries Detection and Assessment System (ICDAS-II) in epidemiological surveys and comparability with standard World Health Organization criteria. *Caries Res*, 43 (2009), pp. 245-249
12. Braga, M.M.; Mendes, F.M.; Martignon, S.; Ricketts, D.N. & Ekstrand, K.R. In vitro comparison of Nyvad's system and ICDAS-II with Lesion Activity Assessment for evaluation of severity and activity of occlusal caries lesions in primary teeth. *Caries Research*, Vol.43, No.5, (September), (2009), pp. 405-412

13. Brambilla E, Twetman S, Felloni A, Cagetti M, Canegallo L, Garcia-Godoy F, et al. Salivary mutans streptococci and lactobacilli in 9- and 13-year-old Italian schoolchildren and the relation to oral health. *Clin Oral Investig*. 1999; 3(1):7-10
14. Branden S, Broucke S, Leroy R, Declerck D, Hoppenbrouwers K. Oral health and oral health-related behaviour in preschool children: evidence for a social gradient. *Eur J Pediatr Dent*. 2013; 172:231-7
15. Bratthall D, Hänsel-Petersson G, Sundberg H. Reasons for the caries decline: what do the experts believe? *Eur J Oral Sci* 1996;104:416-422.
16. Broadbent JM, Thomson WM, Poulton R. Trajectory patterns of dental caries experience in the permanent dentition to the fourth decade of life. *J Dent Res*, 2008; 87(1):69–72.
17. Broadbent JM, Thomson WM. For debate: Problems with the dmf index pertinent to dental caries data analysis. *Community Dent Oral Epidemiol*, 2005; 33(6): 400–9.
18. Brook I. Anaerobic Infections in Children. *Adv Exp Med Biol*. 2011; 697:117-52.
19. Buldur, B. Pathways between parental and individual determinants of dental caries and dental visit behaviours among children: Validation of a new conceptual model. *Community Dent. Oral Epidemiol*. 2020, 48, 280–287.
20. Burt BA. Definitions of risk. *J Dent Educ*. 2001 Oct;65(10):1007–8
21. Cadavid AS, Lince CM, Jaramillo MC. Dental caries in the primary dentition of a Colombian population according to the ICDAS criteria. *Braz Oral Res*, 24 (2010), pp. 211-216
22. Cappelli DP, Mobley CC. *Prevention in Clinical Oral Health Care*, 2007. Elsevier; Philadelphia.
23. Caries diagnosis and risk assessment. A review of preventive strategies and management. *J Am Dent Assoc.*, 1995. 126 p. 1S-24S6. Kutsch, V., C. Kutsch, and B. Nelson, A clinical look at CAMBRA. *DPR* 2007. 41(8): p. 62-67.
24. Chapple ILC, Bouchard P, Cagetti MG, Campus G, Carra M-C, Cocco F, Nibali L, Hujoel P, Laine M L, Lingström P, Manton DJ, Montero E, Pitts N, Rangé H, Schlueter N, Teughels W, Twetman S, Van Loveren C, Van der Weijden F, Vieira AR, Schulte AG. Interaction of lifestyle, behaviour or systemic diseases with dental caries and periodontal diseases: consensus report of group 2 of the joint EFP/ORCA workshop on the boundaries between caries and periodontal diseases. *J Clin Periodontol* 2017; 44: S39–S51.
25. Chen M. Oral health of disadvantaged populations. In: Cohen LK, Gift H, editors. *Disease prevention and oral health promotion. Socio-dental sciences in action*. Copenhagen: Munksgaard; 1995.

26. Chen, L.; Hong, J.; Xiong, D.; Zhang, L.; Li, Y.; Huang, S.; Hua, F. Are parents' education levels associated with either their oral health knowledge or their children's oral health behaviors? A survey of 8446 families in Wuhan. *BMC Oral Health* 2020 Children. *The Medical Journal of Cairo University*, 79(2).
27. Cohen LK, Bryant PS, editors. *Social sciences and dentistry: a critical bibliography*. Vol. II. London: Quintessence Publ Co.; 1984.
28. Coppa A, Bondioli L, Cucina A, Frayer DW, Jarrige C, Jarrige JF, et al. Palaeontology: Early neolithic tradition of dentistry. *Nature* 2006;440:755-6.
29. Cortelli S, Cortelli J, Prado J, Aquino D, Jorge A. DMFT in school children relate to caries risk factors. *Cienc Odontol Bras* 2004; 7(2):75-82.
30. D'Cruz, A.M.; Aradhya, S. Impact of oral health education on oral hygiene knowledge, practices, plaque control and gingival health of 13- to 15-year-old school children in Bangalore city. *Int. J. Dent. Hyg.* 2013, 11, 126–133
31. Daniel S, Harfst S, Wilder R, Francis B, Mitchell S. *Mosby's Dental Hygiene: Concepts, Cases and Competencies*. 2nd ed. St Louis, USA: Mosby Elsevier; 2008
32. de Amorim RG, Figueiredo MJ, Leal SC, *et al.* Caries experience in a child population in a deprived area of Brazil, using ICDAS II. *Clin Oral Invest*, 16 (2012), pp. 513-520
33. Department of Global Oral Health. Oral health promotion and oral health education. 2014 [Internet]. (access in 3 April 2014). Available from: http://www.globaloralhealth-nijmegen.nl/ohp_and_oh.html.
34. Diniz M., Rodrigues J., Lussi A., Traditional and novel caries detection methods. *ResearchGate*. 2012
35. Dr. R. Divya Dr. R. Jayasrikrupaa,. Dr. N. Aravindha Babu,. Dr. Kmk Masthan. Dental caries: recent update. *European journal of molecular and clinical medicine*. Volume 07, issue 5, 2020.
36. FDI World Dental Federation. FDI policy statement on Classification of caries lesions of tooth surfaces and caries management systems. *Int Dent J*, 2013; 63(1):4–5
37. Fejerskov O, Kidd E (Eds.), *Dental Caries – The Disease and its Clinical Management* (2nd ed), Blackwell Munksgaard, Frederiksberg 2008; pp. 123-146
38. Fejerskov O, Kidd E. *Dental Caries: The Disease and its Clinical Management*. 2nd ed. Oxford: Blackwell Munksgaard; 2003
39. Fejerskov O, Kidd EAM, Nyvad B, Baelum V. Defining the disease: an introduction. In: *Dental caries: The disease and its clinical management*, 3d edn. Copenhagen: Blackwell Munksgaard 2013.

40. Fejerskov O. Changing Paradigms in Concepts on Dental Caries: Consequences for Oral HealthCare. *Caries Res.* 2004;38:182–191
41. Fejerskov O. Concepts of dental caries and their consequences for understanding the disease. *Community Dent Oral Epidemiol.* 1997 Feb;25(1):5–12.
42. Fernández-Alvira, J.M.; Börnhorst, C.; Bammann, K.; Gwozdz, W.; Krogh, V.; Hebestreit, A.; Barba, G.; Reisch, L.; Eiben, G.; Iglesia, I.; et al. Prospective associations between socio-economic status and dietary patterns in European children: The Identification and Prevention of Dietary- And Lifestyle-induced Health Effects in Children and Infants (IDEFICS) study. *Br. J. Nutr.* 2015, 113, 517–525
43. Figueiredo MJ, de Amorim RG, Leal SC, *et al.* Prevalence and severity of clinical consequences of untreated dentine carious lesions in children from a deprived area of Brazil. *Caries Res*, 45 2011, pp. 435-442
44. Fisher-Owens SA, Gansky SA, Platt LJ, Weintraub JA, Soobader M-J, Bramlett MD, Newacheck P W. Influences on children’s oral health: a conceptual model. *Pediatrics* 2007; 120 (3): e510–e520.
45. Fleming E, Afful J. Prevalence of total and untreated dental caries among youth: United States 2015-2016. NCHS Data Brief, no 307. Hyattsville (MD): National Center for Health Statistics; 2018.
46. Frencken JE, de Amorim RG, Faber J, *et al.* The Caries Assessment Spectrum and Treatment (CAST) index: rationale and development. *Int Dent J*, 61; 2011, pp. 117-123
47. Giugliano D, d’Apuzzo F, Majorana A, Campus G, Nucci F, Flores-Mir C, Perillo L. Influence of occlusal characteristics, food intake and oral hygiene habits on dental caries in adolescents: a cross-sectional study. *Eur J Paediatry Dent* 2018; 19 (2): 95–100
48. Glick M et al., FDI Vision 2020: shaping the future of oral health. *Int Dent J*, 62(6), 2012; pp. 278–291.
49. Halonen H, Pesonen P, Seppa L, Peltonen E, Tjaderhane L, Anttonen V. Outcome of a community- based oral health promotion project on primary schoolchildren’s oral hygiene habits. *Int J Dent.* 2013
50. Hals, E. Histology of natural secondary caries associated with silicate cement restorations in human teeth. *Archives of Oral Biology*, Vol. 20(4), 1975a; pp. 291-296.
51. Harris N, Garcia-Godoy F. *Primary Preventive Dentistry.* 6th ed. New Jersey: Pearson Prentice Hall; 2004
52. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. *Community Dent Health.* 2004; 21(Suppl 1):71–85.

53. Hart T, Corby P, Hauskrecht M, Ryu O, Pelikan R, Valko M, et al. Identification of Microbial and Proteomic Biomarkers in Early Childhood Caries. *Int J Dent*. 2011; Article ID:196721:1-13
54. Hattne K, Folke S, Twetman S. Attitudes to oral health among adolescents with high caries risk. *Acta Odontol Scand*. 2007; 65:206-13
55. Hobdell M. Poverty, oral health and human development. *J Am Dent Assoc*. 2009; 9(2):27-30
56. Holst, D. Schuller, A.A. & Gimmestad, A. Changing dental health. From risk of disease to oral health potential. *Nor Tannlegeforen Tid*. 2004; 114: 866-871. In Norwegian, with English summary
57. Hua Xi Kou Qiang Yi Xue Za Zhi. Cause of secondary caries and prevention. *Apr*;32(2), 2014; pp. 107-10.
58. Hugoson, A., Koch, G., Göthberg, C., Nydell Helkimo, A., Lundin, S-Å., Norderyd, O., Sjödin, B. & Sondell, Katarina. Oral health of individuals aged 3-80 years in Jönköping, Sweden during 30 years (1973-2003). II. Review of clinical and radiographic findings. *Swedish Dental Journal*. 2005; 29: 139-155
59. Ismail AI et al., Caries management pathways preserve dental tissues and promote oral health. *Community Dent Oral Epidemiol*, 41(1), 2013; e12–40.
60. Ismail AI, Hasson H, Sohn W. Dental Caries in the Second Millennium. *J Dent Edu*. 2001;65:953– 9, PubMed.
61. Ismail, A.I.; Sohn, W.; Tellez, M.; Amaya, A.; Sen, A.; Hasson, H. & Pitts, N.B. The International Caries Detection and Assessment System (ICDAS): an integrated system for measuring dental caries. *Community Dentistry and Oral Epidemiology*, Vol.35, No.3, (June), 2007; pp. 170-178
62. Ito, S.; Misaki, T.; Naka, S.; Wato, K.; Nagasawa, Y.; Nomura, R.; Otsugu, M.; Matsumoto-Nakano, M.; Nakano, K.; Kumagai, H.; et al. Specific strains of *Streptococcus mutans*, a pathogen of dental caries, in the tonsils, are associated with IgA nephropathy. *Sci. Rep*. 2019, 9, 20130.
63. Kassebaum NJ, Bernabé E, Dahiya M, Bhandari B, Murray CJ, Marcenes W. Global burden of untreated caries: a systematic review and metaregression. *J Dent Res*. 2015 May;94(5):650-8. doi: 10.1177/0022034515573272. Epub 2015 Mar 4. PMID: 25740856.
64. Kelly, S.E.; Binkley, C.J.; Neace, W.P.; Gale, B.S. Barriers to care-seeking for children's oral health among low-income caregivers. *Am. J. Public Health* 2005, 95, 1345–1351.

65. Khamis A. Re-Visiting the Decay, Missing, Filled Teeth (DMFT) Index with a Mathematical Modeling Concept Article in *Open Journal of Epidemiology* · February 2016
66. Khan, S. Y. Impact of Sociodemographic Factors on Dental Caries Among. 2011
67. Kleinberg I. A Mixed-bacteria Ecological Approach to Understanding the Role of the Oral Bacteria in Dental Caries Causation: an Alternative to *Streptococcus mutans* and the Specific-plaque Hypothesis. *Crit Rev Oral Biol Med.* 2002; 13(2):108-25
68. Kolenbrander, P., Palmer,R., Saravanan,P., Jakubovics, N. Oral multispecies biofilm development and the key role of cell-cell distance. 2010. PubMedi.
69. Krstrup, U. & Petersen, P.E. Periodontal conditions in 35-44 and 65-74-year-old adults in Denmark. *Acta Odontologica Scandinavica.* 2006; 64: 65-73
70. Krstrup, U. *Clinical-epidemiological study of oral health among adults in Denmark 2000/2001.* Ph.D. Thesis. University of Copenhagen. 2004.
71. Kumar, S.; Tadakamadla, J.; Zimmer-Gembeck, M.J.; Kroon, J.; Lalloo, R.; Johnson, N.W. Parenting practices and children's dental caries experience: A structural equation modelling approach. *Community Dent. Oral Epidemiol.* 2017, 45, 552–558.
72. Levin K, Nicholls N, Macdonald S, Dundas R, Douglas G. Geographic and socioeconomic variations in adolescent toothbrushing: a multilevel cross-sectional study of 15 year olds in Scotland. *J Public Health.* 2015; 37(1):107-15
73. Lima J. Cárie Dentária: um novo conceito. *Rev Dental Press Ortodon Ortop Facial.* 2007; 12(6):119- 30
74. Lingström P, Holm AK, Mejåre I, Twetman S, Söder B, Norlund A, Axelsson S, Lagerlöf F, Nordenram G, Petersson LG, Dahlgren H, Källestål C. Dietary factors in the prevention of dental caries: a systematic review. *Acta Odontol Scand* 2003;61:331-340.
75. Loesch WJ. Clinical and Microbiological aspects of Chemotherapeutic Agents Used According to the Specific PlaqueHypothesis. *J Dent Res.* 1979;58:2404–12.
76. Lukacks JR, Laigaespada L. Explaining sex differences in dental caries prevalence: saliva, hormones, and life-history etiologies. *Am J Hum Biol.* 2006; 18(4): 540-55
77. Machiulskiene,V., Campus, G., Carvalho, J., Dige, I. Terminology of dental caries and dental caries management: Concensus report of a workshop organized by ORCA and cariology research group of IADR. *Caries research.* 54, 2020; 7–14.
78. Macri D et al: Caries classification. *Dimens Dent Hyg.* 15(7):17-8, 21, 2017
79. Marsh PD. Microbial Ecology of Dental Plaque and its Significance in Health and Disease. *AdvDent Res.* 1994;8:263–71.
80. Marthaler TM. Changes in dental caries 1953–2003. *Caries Res,* 2004, 38(3): 173–181.

81. Monse B, Heirich-Weltzien R, Benzian H, *et al.* PUFA – an index of clinical consequences of untreated dental caries. *Community Dent Oral Epidemiol*, 38 (2010), pp. 77-82
82. Nadanovsky P, Sheiham A. Relative contribution of dental services to the changes in caries levels of 12-year-old children in 18 industrialized countries in the 1970s and early 1980s. *Community Dent Oral Epidemiol*, 1995, 23(6): 331–9.
83. Nibali L, Di Iorio A, Tu, Y-K, Vieira AR. Host genetics role in the pathogenesis of periodontal disease and caries. *J Clin Periodontol* 2017; 44 Suppl 18: S52–S78.
84. Nicolau B, Marcenes W, Bartley M, Sheiham A. Association between socio-economic circumstances at two stages of life and adolescents' oral health status. *J Public Health Dent*. 2005; 65(1):14-20
85. Nordblad, A., Vehkalahti, M. & the working group for oral health. (2004b). Oral health. In Arpo Aromaa & Seppo Koskinen (Eds). *Health and Functional Capacity in Finland. Baseline Results of the Health 2000 Health Examination Survey*. Helsinki: Publications of the National Public Health Institute (KTL), B12/2004
86. NOU 2005:11. *Det offentlige engasjement på tannhelsefeltet. Et godt tilbud til de som trenger det mest.* Norges offentlige utredninger. Statens forvaltningstjeneste Informasjonsforvaltning. Oslo. In Norwegian. 2005
87. Okeyo, A.P.; Seekoe, E.; de Villiers, A.; Faber, M.; Nel, J.H.; Steyn, N.P. Dietary practices and adolescent obesity in secondary school learners at disadvantaged schools in south africa: Urban–rural and gender differences. *Int. J. Environ. Res. Public Health* 2020, 17, 5864
88. Oliveira L, Sheibam A, Bonecker M. Exploring the association of dental caries with social factors and nutritional status in Brazilian preschool children. *Eur J Oral Sci* 2008; 116:37-43.
89. Pereira A. *Odontologia em Saúde Colectiva - Planejando acções e promovendo saúde*. 1st ed. Porto Alegre: Artmed Editora; 2003
90. Peres K, Latorre M. Impact of dental caries and dental fluorosis on 12-year-old schoolchildren's self-perception of appearance and chewing. *Cad Saúde Pública*. 2003; 19(1):323-30
91. Peres M, Latorre M, Sheiham A, Peres K, Barros F, Hernandez P, *et al.* Social and biological early life influences on severity of dental caries in children aged 6 years. *Community Dent Oral Epidemiol*. 2005; 33:53-63

92. Petersen P, Kjoller M, Christensen L, Krusturup U. Changing dentate status of adults, use of dental health services, and achievement of national dental health goals in Denmark by the year 2000. *J Public Health Dent.* 2004; 64(3):127-35.
93. Petersen PE. Sociobehavioural risk factors in dental caries – international perspectives. *Community Dent Oral Epidemiol* 2005; 33: 274–9. Blackwell Munksgaard, 2005
94. Petersen PE. World Health Organization global policy for improvement of oral health– World Health Assembly 2007. *Int Dent J*, 115–124. 14. Pitts NB (Guidance Development Group Chair) and the SDCEP Guidance Development Group. Oral Health Assessment and Review - Full Guidance Scottish Dental Clinical Effectiveness Programme, 2011 (<http://www.sdcep.org.uk/published-guidance/oral-health-assessment>). 2008.
95. Petersen, P., World Health Organization global policy for improvement of oral health - World Health Assembly 2007. *Int Dent J*, 2008. 58: p. 115-121.
96. Petrovski M, Ivanovski K, Minovska A. DMFT index among institutionalized elderly. *Balkan journal of dental medicine.* 2015. ISSN: 2335-0245.
97. Pitts N et al., Caries risk assessment, diagnosis and synthesis in the context of a European Core Curriculum in Cariology. *Eur J Dent Educ*, 2011; 15 (Suppl. 1):23–31.
98. Pitts N. *Detection, Assessment, Diagnosis and Monitoring of Caries.* Karger, Basel 2009, pp. 1-41
99. Pitts N. ICDAS – an international system for caries detection and assessment being developed to facilitate caries epidemiology, research and appropriate clinical management. *Community Dent Health*, 21 2004; pp. 193-198
100. Pitts NB, Ekstrand KR, ICDAS Foundation. International Caries Detection and Assessment System (ICDAS) and its International Caries Classification and Management System (ICCMS) - methods for staging of the caries process and enabling dentists to manage caries. *Community Dent Oral Epidemiol*, 2013; 41(1):e41–52.
101. Pitts NB, Zero DT, Marsh PD, Ekstrand K, Weintraub JA, Ramos-Gomez F, et al. Dental caries. *Nat Rev Dis Primers.* 2017 May;3(1):17030
102. Pitts NB. Are we ready to move from operative to non-operative/ preventive treatment of dental caries in clinical practice? *Caries Res* 2004; 38(3):294–304.
103. Pitts, N. B. & Zero, D. T. White paper on dental caries prevention and management. FDI World Dental Federation http://www.fdiworlddental.org/sites/default/files/media/documents/2016-fdi_cpp-white_paper.pdf (2016).
104. Pitts, N.B. & Richards, D. Personalized treatment planning. *Monographs in Oral Science*, Vol.21, (June), 2009; pp. 128-143

105. Pitts, N.B. Modern concepts of caries measurement. *Journal of Dental Research*, Vol.83, No. Spec No C, 2004; p. C43-C47
106. Qualtrough A, Satterthwaite J, Morrow L, Brunton P. *Principles of operative dentistry*. Blackwell. 2005. Pp.15-16.
107. Rashid EG: Operative dentistry. In: Scheid RC et al, eds: *Woelfel's Dental Anatomy*. 7th ed. Lippincott Williams & Wilkins; 2007:432-465
108. Relch E, Lussi A, Newbrun E-. Caries-risk assesement. *Int Dent J*. 1999; 49:15-26
109. Reyes C, Dalmacio L. Bacterial Diversity in the Saliva and Plaque of Caries-free and Caries-active Filipino Adults. *Philippine J Sci*. 2012; 141(2):217-27.
110. Richards ND, Cohen LK, editors. *Social sciences and dentistry. A critical bibliography*. The Hague: A Sijthoff, 1971.
111. Roberson TM, Roberson TM, Heymann HO, Swift EJ. *Sturdevant's Art and Science of Operative Dentistry*. 5th ed. St. Louis, Missouri: Mosby: Cariology: The lesion, etiology, prevention and control; 2006; pp. 71–80.
112. Roberts K, Condon L. How do parents look after children's teeth? A qualitative study of attitudes to oral health in the early years. *Community Pract*. 2014; 87(4):32-5
113. Roland E, Gueguen G, Longis MJ, Boisselle J. Validation of the reproducibility of the dmf index used in bucco-dental epidemiology and evaluation of its 2 clinical forms. *World Health Stat Q*, 1994; 47(2): 44–61.
114. Schulte AG et al., European Core Curriculum in Cariology for undergraduate dental students. *Eur J Dent Educ*, 15 Suppl 1, 2011; pp. 9–17.
115. Selwitz RH, Ismail AI, Pitts NB. Dental caries. *Lancet*, 2007; 6;369(9555):51–59.
116. Sgan-Cohen H. Health, oral health and poverty. *J Am Dent Assoc*. 2009; 9(2):33-8
117. Skudutyte-Rysstad, R. & Eriksen, H.M. Changes in caries experience among 35-year-old Oslo citizens, 1973-2003. *Acta Odontologica Scandinavica*. 2007; 65: 72-77
118. Slayton RL et al: Evidence-based clinical practice guideline on nonrestorative treatments for carious lesions: a report from the American Dental Association. *J Am Dent Assoc*. 149(10):837-49.e19, 2018
119. Sosial- og helsedirektoratet. *Utviklingstrekk i helse- og sosialsektoren 2007*. Oslo 2007. In Norwegian. 2007
120. SOU 2007:19. *Friskare tänder - till rimliga kostnader*. Slutbetänkande av Utredningen om ett nytt tandvårdsstöd för vuxna. Statens offentliga utredningar. Stockholm. In Swedish. 2007.
121. Štefanová, E.; Baška, T.; Holubčíková, J.; Timková, S.; Tatarková, M.; Sovičová, M.; Hudečková, H. Selected behavioural factors affecting oral health in schoolchildren: Results

from the health behaviour in school-aged children (HBSC) Slovak study. *Int. J. Environ. Res. Public Health* 2020, 17, 7516.

122. Steinberg D, Eskander L, Zini A, Sgan-Cohen H, Bajali M. Salivary levels of mutans streptococci and Lactobacilli among Palestinian school children in East Jerusalem. *Clin Oral Investig.* 2014; 18(3):979-83

123. Szymanska J, Szalewski L. Deciduous teeth caries in the population of the Polish children aged 0.5-6 years. *Pol J Public Health.* 2011; 121(1): 86-9.

124. The World Oral Health Report, in Continuous improvement of oral health in the 21st century - the approach of the WHO Global Oral Health Programme, World Health Organization. 2003

125. Thylstrup A, Bruun C, Holmen L. In vivo caries models—mechanisms for caries initiation and arrestment. *Adv Dent Res.* 1994 Jul;8(2): 144–57.

126. Timis T, Danila I. Socioeconomic status and oral health. *J Prev Med.* 2005

127. Touger-Decker R, Loveren C. Sugars and dental caries. *Am J Clin Nutr.* 2003; 78(suppl)

128. Uhrbom, E. & Bjerner, B. *EpiWux03. Vuxnas mun- och tandhälsa i Dalarna, utveckling och framtid. En undersökning avseende 35-, 50-, 65- och 75-åringar i Dalarna 2003.* Skriftserie nr 75. Falun: Centrum för Oral Rehabilitering. In Swedish. 2003

129. Usha C, R S. Dental caries - A complete changeover (Part I). *J Conserv Dent.* Apr; 2009; 12(2):46-54.

130. Vanobbergen J, Martens L, Lesaffre E, Bogaerts K, Declerck D. Assessing risk indicators for dental caries in the primary dentition. *Community Dent Oral Epidemiol.* 2001; 29:424-34

131. Vehkalahti, M., Varsio, S. & Hausen, H. Hampaiden kunto. In Liisa Suominen- Taipale, Anne Nordblad, Miira Vehkalahti & Arpo Aromaa (Eds.). *Suomalaisten Aikuisten Suunterveys. Terveys 2000 – tutkimus.* [Health 2000]. Helsinki: Publications of the National Public Health Institute B16/2004. In Finnish, with English summary. 2004

132. Wänman, A., Forsberg, H., Sjödin, L., Lundgren, P. & Höglund Åberg, C. *Tillståndet i mun och käkar bland Västerbottens vuxna befolkning år 2002. En rapport baserad på en epidemiologisk undersökning bland 35-, 50-, 65- och 75-åringar.* Umeå: Umeå Universitet och Västerbottens läns landsting. In Swedish. 2004

133. World Health Organization **Oral Health Surveys:Basic Methods**(4th ed), WHO, Geneva (1997).

134. World Health Organization. Health promotion and oral health. 2014 [Internet]. (access in 28 September 2014). Available from: http://www.who.int/oral_health/strategies/hp/en/.

135. World Health Organization. Oral Health - Fact sheet No 318. Geneva: WHO; 2012.

136. World Health Organization. Oral Health: important target groups. 2014 [Internet]. (access in 22 April 2014). Available from: http://www.who.int/oral_health/action/groups/en/.
137. World Health Organization. WHO information series on School-health. Oral health promotion: An essential element of health-promoting schools. Document 11. Geneva: WHO; 2003.
138. Wu, L.; Li, J.; Zhang, Y.; Zhou, Y.; Liang, Y.; Huang, S. Oral Health Status and Risk Factors for Caries in Permanent Teeth among 12-year-old Students in Guangdong, Southern China: A Population-based Epidemiological Survey. *Oral Health Prev. Dent.* 2020, 18, 731–740.
139. Yamaga R, Nishino M, Yoshida S, Yokomizo I. "Diammine Silver Fluoride and Its Clinical Application". *J Osaka Univ Dent Sch.* 1972; 12: 1–20
140. Yip, K., Smales, R. Oral diagnosis and treatment planning: part 2. Dental caries and assessment of risk. *Medicine. BDJ.* 2012
141. Young DA et al: The American Dental Association Caries Classification System for clinical practice: a report of the American Dental Association Council on Scientific Affairs. *J Am Dent Assoc.* 146(2):79-86, 2015
142. Zandoná, A.F. & Zero, D.T. Diagnostic tools for early caries detection. *Journal of the American Dental Association*, Vol.137, No.12, (December), 2006; pp. 1675-1684

DOCUMENTATION PAGE

DOCUMENTATION PAGE

This Diploma Thesis „ **DMFT INDEX AND CARIES PREVALENCE OF THE PATIENTS ATTENDING STUDENTS DENTAL CLINIC AT UNIVERSITY OF LATVIA**” was developed at the Faculty of Medicine of the University of Latvia.

With my signature, I attest, that this research has been carried out without aid or assistance. Used information was obtained only from indicated sources and the electronically submitted copy of this diploma work complies with printout.

Author: KRISTINA KOVALENKO Kristina Kovalenko
(name, surname) (signature)

I recommend the work for presentation.

Supervisor: LECT. BAIBA KRAUZE, DDS [Signature] 20.05.2022
(position, name, surname, degree) (signature) (date)

Reviewer: ASSOC. PROF. SIGNE MEŽINSKA _____
(position, name, surname, degree) (signature) (date)

The diploma thesis was submitted to the Faculty of Medicine on: _____
(date)

International students' coordinator, _____
(signature)

The diploma thesis is presented at the meeting of the State Examination Commission of Second Level Higher Professional Study Program „Dentistry” _____ 2022. Protocol No.

Secretary of Commission: _____
(position, name, surname, degree) (signature)