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Incidence and progression of dental erosion among adolescents in Troms Data based on Fit Futures - a health survey among adolescents

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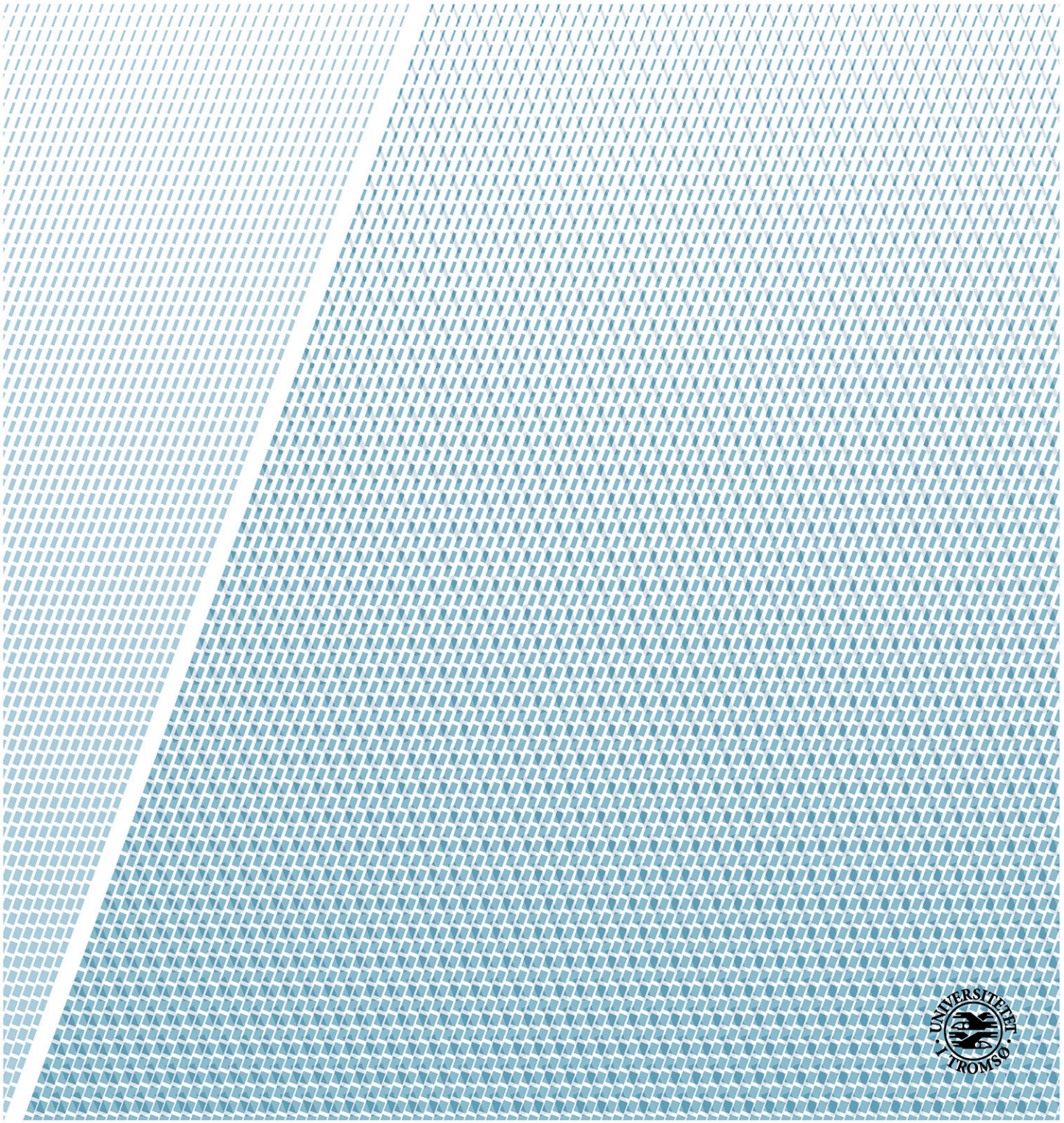

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Incidence and progression of dental erosion among adolescents in Troms

Data based on Fit Futures - a health survey among adolescents:

1.0 Introduction

Caries and periodontitis has for a long time been the main concern among dental practitioners and a lot of research has been directed towards the management of these oral diseases. An improvement of oral health has been observed in most industrialized countries during the last 40 years (2), mostly due to a decline in caries prevalence in the western part of the world. However, during the last 15 years there has been an increased focus from clinicians and researchers on pathological tooth wear caused by acid erosion (3). Dental erosion is a condition that involves the loss of dental hard tissue, caused by exposure to acidic substances without the involvement of microorganisms. Dental erosion has particularly been observed in younger individuals.

The decline in the caries prevalence in western countries may have changed the focus and interest on to dental erosion(4). This condition was however reported as early as in the 19th century (5). There exist only few studies on tooth wear from as far back as 40 years ago and these studies reported a prevalence varying from 18 %-25 % (6-8) which is lower than the prevalence nowadays.

During the last decades, prevalence studies among children and adolescents have been performed in many countries and recent data on dental erosions among adolescents vary from 16-59% (9-15). It has also been observed that the prevalence of dental erosive wear increases linearly with age and also that the lesions progress into more severe lesions (5, 16-18). There are many cross-sectional prevalence studies on dental erosion (19) but longitudinal studies are scarce and the data from these studies vary (3, 20).

What is considered an acceptable amount of wear is dependent on the anticipated life span of the teeth, which is different for primary teeth compared to permanent teeth. Early erosive damage to the permanent teeth occurring in childhood may compromise the growing child`s dentition for their entire lifetime, and may require extensive restorative procedures (21).

Dental practitioners and hygienists play an important role in preventing and treating dental erosion. The diagnosis, treatment and prevention of the condition are often a challenge for the dental healthcare professionals. The focus on diagnosis of the early stage of dental erosion often varies and the initial stages can easily be underestimated. We experienced this during our training at the external dental University clinics (Public Dental Health Clinics) and at the University Clinic at IKO, Tromsø. We believe that awareness, early diagnosis and treatment of dental erosion should be just as important as treating caries and other dental diseases in a dental practice. The purpose of this study was to monitor the progression of dental erosion over a 2-year period.

2.0 Dental erosion

2.1 What is dental erosion?

Dental erosion is defined as the surface dissolution of dental hard tissues by acids without the involvement of microorganisms (22, 23). It is a destructive process that may lead to a pathological loss of dental hard tissue if the teeth are frequently exposed to acids over a long period of time. The eroded tooth surface becomes hypomineralized and is therefore more susceptible to the effects of attrition and abrasion as well (24).
Dental erosion has a multi-factorial etiology and can be categorized in different ways, and it is graded based on quantification of hard tissue loss (5).

The acid sources:
The acids responsible for the erosion are, unlike in the caries process, not products of the intraoral flora, but can be derived from extrinsic and intrinsic sources. The extrinsic sources are mainly in the diet, but also from occupational hazards and sports (swimming pool chemicals) and medication. Intrinsic sources are the stomach acid coming into the oral cavity by reflux, vomiting, regurgitation and rumination. Sometimes the acid sources cannot be identified.

Pathological effect:
Chronic erosive lesions with slow progression are often symptom free; whereas an acute rapid progressive lesion can cause pain. Problems occur when the amount of hard tissue lost leads

to tooth sensitivity, functional problems or aesthetic concerns for the patient. This is often the case if large areas of dentin are exposed.

Localization:

The localization of dental erosions can be described as general, where many teeth are involved, or as localized, where only a few teeth are involved (24).

The initial phase:

The initial phase of erosive lesions begins with erosion of the enamel. The outer surface is demineralized and softened. Remineralization can still occur and the initial phase is therefore to some degree a reversible phase. This phase cannot be detected visually in the clinic or by a scoring system.

The manifested phase:

If the erosive challenge persists it will cause dissolution of the consecutive layers of enamel crystals. The dissolution of the enamel crystals leads to a permanent loss of the dental tissue and remineralization cannot occur. The manifested phase is therefore an irreversible phase (25). The top layer of the remaining tissue will be softened and may be remineralized. Once the dentine becomes exposed, tissue loss accelerates if the erosive challenge persists.

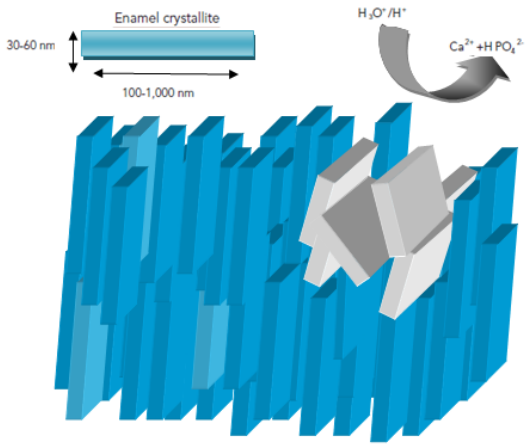
2.2 The chemistry of dental erosion

Enamel and dentine: Minerals and tissue structure

Our teeth are daily exposed to different extrinsic and/or intrinsic erosive challenges, which results in softening of the enamel and dentin. In combination with attrition and abrasion, this may cause an acceleration of the progression of erosive tooth wear (22). Enamel and dentine tissues are very different in their structure, despite consisting of similar components. Understanding the process behind eroded dental hard tissues is essential for applying and developing suitable causal and symptomatic measures. It is therefore important to have an understanding of the chemistry behind dental erosion (26).

Erosion of the teeth occurs when acidic agents interact with the surface of the mineral crystals, but only after they diffuse through the plaque, the pellicle, and the protein coating (27).

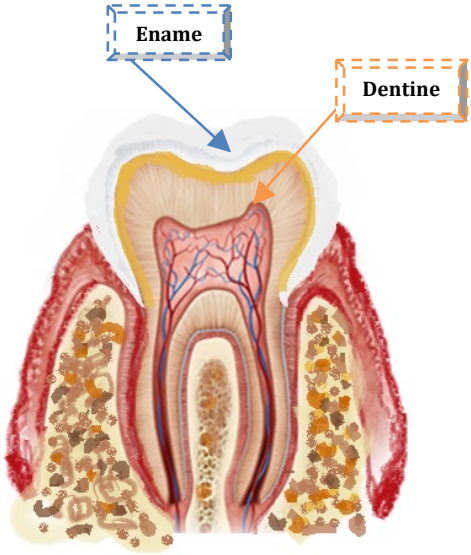
An acid attack will soften the tooth surface due to a demineralization and the tooth will over time lose its natural structural integrity and mechanical strength, as illustrated in Figure 1 (28). If the tooth is exposed to prolonged erosive challenges, it will lead to bulk loss of enamel. The rate of dissolution is crucial to the progression of erosion, and is influenced by the solubility of the dental tissue and also by other factors.(29).



Figur 1 **The development of an erosive lesion(30)**
Illustration where an acidic soft drink destroys the enamel surface by partial and complete dissolution of the enamel crystallites. The result is a release of Ca^{2+} and HPO_4^{2-} ions that loosens the microstructure of the enamel and hydroxyapatite crystallites (grey). These crystallites become demineralised or are lost.

Enamel

Tooth enamel is the most mineralized tissue of the human body and it is designed to last a lifetime. Enamel is a non-vital, densely packed mineralized structure. The mineral is organized in rods of hexagonal structure. The dimension of the rods is difficult to measure, but the studies that have been publishes, have shown results ranging from 50-70 nm in width and a thickness of 20-25 nm (26, 31). Each rods extends from the enamel-dentine junction to the outer enamel surface and are made up of highly organized crystals of calcium phosphate hydroxyapatite (HA_P): $Ca_{10}(PO_4)_6(OH)_2$.



Figur 2: **Illustration of tooth anatomy**

The hydroxyapatite crystals are arranged with their long axes parallel to the long axes of the rods. In the enamel a number of ions can be missing from HA_P, as there is an empty space between each crystal that is filled with organic materials and water.

This allows the passage of small molecules and substitutions within HA_P. For example calcium can be replaced by sodium, magnesium and zinc and carbonate for phosphate, and fluoride for hydroxyl. Since the impurity ions differ from the native HAp ions in size, charge or both, they have the effect of introducing strain in the crystal structure of the Hap, leading to increased solubility at low pH (29, 32).

The composition of enamel in volume is shown in the table below (Table 1). The high minereal content makes the enamel resistant against physical impacts (26).
When enamel is subjected to an acidic solution which is unsaturated with regard to tooth mineral, erosive demineralization occurs. Minerals are dissolved from the surface, resulting in a rough and irregular structure and consequently the microhardness is reduced. Eroded enamel is therefore less resistant against physical wear than sound dental hard tissue. As the acid exposure continues, bulk enamel loss occurs (26).

Dentin:

Dentine is structurally and biologically different than enamel, as it is a vital and permeable tissue. However, similarly to enamel, dentine is an imperfect form of calcium phosphate hydroxyapatite (HA_P): $Ca_{10}(PO_4)_6(OH)_2$. The table below shows that dentine contains more

carbonate and magnesium, compared with enamel. Moreover, the crystals of dentine minerals are much smaller, have a higher concentration of impurities and are less crystallized, compared with enamel (29).

Physiologically and anatomically, dentine is a complex structure and different types of dentins have been identified. If the tissue loss continues beyond enamel, dentin becomes exposed. The dentin crystallites are easily dissolved, the result of which is loss of peri-tubular dentin. If the acid exposures continue for a longer period, the lesion will progress into the inter-tubular dentin followed by loss of mineral in the intertubular areas and lead to an uneven and porous surface. The dentin tubules will become significantly expanded(33).

This explains why the eroded teeth are more sensitive to external stimuli. If the progression of tooth erosion is slow, even severe erosive lesions are without any clinical symptoms. This is due to the formation of reactionary and reparative dentine and obliteration of dentinal tubules in responses to the loss of enamel and dentine (34).

As stated, there are considerable differences in both the structure and the porosity of dentine and enamel. The difference will influence the rate of dissolution in these tissues. Enamel and dentine is composed of an organic matrix, protein and lipid, and water. In enamel 3% of the minerals consist of carbonate while in dentine this percentage is 5.5%. This means that dentine is more susceptible to acid attack than enamel.
The compositions of the two tissues, in volume, are shown in Table 2. (29).

Table 1: **The composition of enamel and dentine, in volume.**

Component	Enamel percent by volume	Dentine percent by volume
Mineral	91,0	49,5
Water	3,4	21,4
Organic material (protein and lipid)	5,3	29,0

Reactions between erosive solutions and dental tissues:

Enamel erosion is a surface phenomenon unlike mineral loss due to dental caries, which forms a subsurface lesion. When enamel is exposed to erosive solutions, the initial dissolution occurs at the surface (34).

When the pH falls to a critical pH, defined as the pH at which a solution is just saturated with respect to a particular mineral, the tooth enamel will be in equilibrium and therefore no mineral dissolution and no mineral precipitation occurs (35).

The chemical process that leads to dental erosion occurs either by the hydrogen ion derived from strong or weak acids or by anions that can bind or complex calcium. For example, citric acid has the possibility of producing three hydrogen ions from each molecule. The H+ ion has the potential to attack the tooth mineral crystals and directly dissolve the tooth minerals by combining with either the carbonate ion or the phosphate ion. The consequence of the hydrogen attacking the tooth surface is that the H^+-ion complex with carbonate and or phosphate and releases all of the ions from the region of the crystal surface, leading to direct surface etching (29).

Below the critical pH, the system becomes undersaturated with respect to tooth mineral and the potential for enamel dissolution can occur. There is no clear-cut critical pH for erosion as there is for caries. However, the value of the critical pH for enamel is often quoted to be 5.5. It is important to have in mind that even when the solution surrounding the tooth has a low pH (<5.5), it is possible that other factors are strong enough to prevent erosion (1, 29, 36).

The extent of tooth erosion depends on the pH of the acidic solution, but also on the type of acid. It can further be influenced by the temperature of the solution. Studies have shown that the erosion rate of enamel exposed to acid solutions increases with increased temperature of the solution (37). The rate of erosion also depends on the velocity of the solution that is covering and flowing over the tooth. This indicates that erosion is controlled by how fast the products are cleared from the tooth. The period of time erosive acids remain in contact with the teeth, and the frequency of contact have some effect on erosion severity and rate of progression, which, clinically, would mean that drinking patterns like swishing will increase the risk of erosive loss. However, also the amount of the acidic fluid, its mineral and protein

composition, ionic strength and chelating properties of the acid itself as well as its flow rate are of influence as further described in the part: "Risk factors"(1, 29, 33, 38)

2.3 Diagnosis

It is often a challenge for clinical professionals to diagnose dental erosions. There is no device available in a routine dental practice for the specific detection of these lesions. The diagnostic procedure therefore relies on a visual rather than an instrumental approach (39). Dental erosions can occur on all surfaces in the dentition. To understand the occurrence and distribution of erosive lesions one has to be aware of the different etiological factors.

Studies report that the distribution of dental erosion is somewhat different in the primary dentition compared with the permanent dentition. It is likely to have a correlation with the difference in anatomical structure and hardness of the teeth (40). In a study of the *primary* dentition of dental erosion in 2- to 4-year-old twins, erosions were observed most frequently on the first molars(33%), followed by the second molars(18%), the canines(18%), the lateral incisors(16%) and the central incisors(15%) (41).

In the permanent dentition, the teeth most commonly affected by erosions are the mandibular and maxillary first molars, and the maxillary incisors. In a study done in the UK, 1,753 12 year-old children were examined. Erosions occurred most frequently on the palatal surface of the maxillary incisors (49%) and the maxillary molars(53%), as well as on the buccal surfaces of the mandibular molars(50%). Occlusal surfaces on the mandibular first molars had the greatest extent of dentine exposure(18(40).

The clinical diagnosis of dental erosion is based on characteristic deviations from the original anatomical tooth morphology (42).
Tooth surface loss or tooth wear can arise as a result of erosion, abrasion and attrition (43). Erosion is the loss of dental hard tissue by a chemical process, which does not involve bacteria. Abrasion is defined as the pathological loss of tooth structure by mechanical forces from a foreign element and attrition is defined as loss of tooth structure as a result of tooth-to-tooth contact.

In the early stages of dental erosion there are only a few visible signs, and even fewer, if any, symptoms. There is no discoloration of the tooth surface or any detectible softening of the surface upon probing (44).

In the late stages, the erosive wear may be easier to diagnose due to the increased loss of tooth substance. Tooth wear is a process that occurs throughout life and in an older dentition it may be hard to distinguish between the influence of erosion compared to abrasion and attrition as these conditions often occur together (6. This is however not as challenging with younger individuals`s dentition, as they have not been exposed to wear as long.
The clinical appearance of the teeth is the most important feature in diagnosing dental erosions.

Clinical appearance in early stages: (39)

- Smooth silky- shining glazed surface.
- Often located coronal from the enamel-cementum junction.
- Absence of pericymata and intact enamel along the gingival margin: The intact enamel band along the gingival margin could be due to some plaque remnants, which can act as a diffuse barrier for acids. It could also be due to an acid neutralization effect of sulcular fluid (45).

Clinical appearance in more advanced stages:

- Loss of original morphology: Flattening and rounding of surfaces. Restorations often rise above the level of the adjacent tooth surface. In severe cases the whole occlusal morphology disappears and the dentin may be exposed to varying degree (42).

2.4 Scoring systems for dental erosion

A short comparison of two dental erosive wear scoring systems;
The Visual Erosion Dental examination (VEDE) system and the Basic Erosive Wear Examination (BEWE).

During the last decades, dental erosion has attracted considerable attention. To increase the understanding and awareness of dental erosion amongst clinicians and general dental practitioners, it has been mandatory to develop standardized and internationally accepted

indexes for scoring the different stages of dental erosions and follow the progression and incidence of erosive lesions. The scoring systems should enable evaluation and surveillance of the loss of dental hard tissue due to erosive factors on surface level. They provide the clinicians a guide for scoring erosive wear and are helpful both in the dental practice and for research purposes.

A number of indexes for classifying and scoring dental erosions *in vivo* has been described (46). The dental erosive wear scoring systems VEDE and BEWE are commonly used.

In establishing the presence and severity grading of dental erosions, the teeth are examined separately and single surfaces are evaluated (facial, oral and occlusal/incisal surfaces). The VEDE system is a modification of the dental erosion index proposed by Lussi. It measures erosive wear at tooth surface level. VEDE was developed by Espelid and Tveit in 2005 and has been used at the student clinic at the University of Oslo since then. It was implemented at the University of Tromsø in 2013. Helsedirektoratet also recommends the VEDE system for recording dental erosions in The Public Dental Health Service in Norway. In 2007, a new scoring system, BEWE, was proposed. In the BEWE scoring system the most severely affected surface in each sextant is recorded with a four level score. The cumulative score is classified and matched to risk levels which guide the management of the condition (47).

In a study on reliability of the scoring systems VEDE and BEWE (3), the results showed that when comparing the scoring the highest agreement was found for score 0(sound, 86%) and score 3(67%), while the smallest agreement was found for score 1(30%) and score 2(57%). This indicates that the greatest difficulties were found when scoring enamel lesions, especially initial lesions. A weakness of the VEDE system may therefore be in its detailed scale. A weakness of the BEWE system is that it does not distinguish between erosions into enamel and dentin.

There was only a slightly higher inter- and intra- examiner agreement (expressed by mean weighted kappa; κ_w) found in VEDE ($\kappa_w = 0.77$) compared to BEWE ($\kappa_w = 0.69$). This indicates that examiner reliability of both VEDE and BEWE is acceptable when scoring the severity of erosions (48). The authors emphasized that for prognostic purposes an erosion index should distinguish between erosive tooth wear in enamel and dentin. They suggested that the BEWE system seems suitable for clinical screening and for epidemiological purposes

since it has fewer grades and uses index teeth/surfaces. The strength of the VEDE system lies in its ability to diagnose the early stages of the condition and to record progression of the lesions on surface and individual level.

VEDE: Visual erosion dental examination

- Measures erosive wear at tooth surface level.
- Records based on depth of the lesion. Therefore, erosive wear of enamel and dentine are recorded separately.

Table 3: **VEDE system (48).**

Score	Definition
0	No erosion
1	Initial loss of enamel, no dentine exposed
2	Pronounced loss of enamel, no dentine exposed on the surface area
3	Exposure of dentine, <1/3 of the surface involved
4	1/3-2/3 of dentine exposed
5	>2/3 of dentine exposed, or pulp exposed

BEWE: Basic erosive wear examination

- Records the most severely affected surface in a sextant, giving a score summated for all the sextants
- Does not distinguish between enamel and dentine wear. Records lesions as part of the tooth surface in contrast to the depth of the lesion.

Table 4: **BEWE system (48).**

Score	Definition
0	No erosion
1	Initial loss of surface texture
2a	Distinct defect, hard tissue loss less than 50% of the surface area
3a	Hard tissue loss more than 50% of the surface area a) Dentine is often involved

2.5 Risk factors

Dental erosion and the rate of its progression are influenced by multiple factors. It has been suggested by Zero and Lussi (24) that several behavioral and lifestyle factors play important roles in modifying the extent of dental erosion. Similar to caries, dental erosion is an acid-induced loss of mineralized dental hard tissues, and although both conditions involve acids, the pathomechanisms are quite different (33).

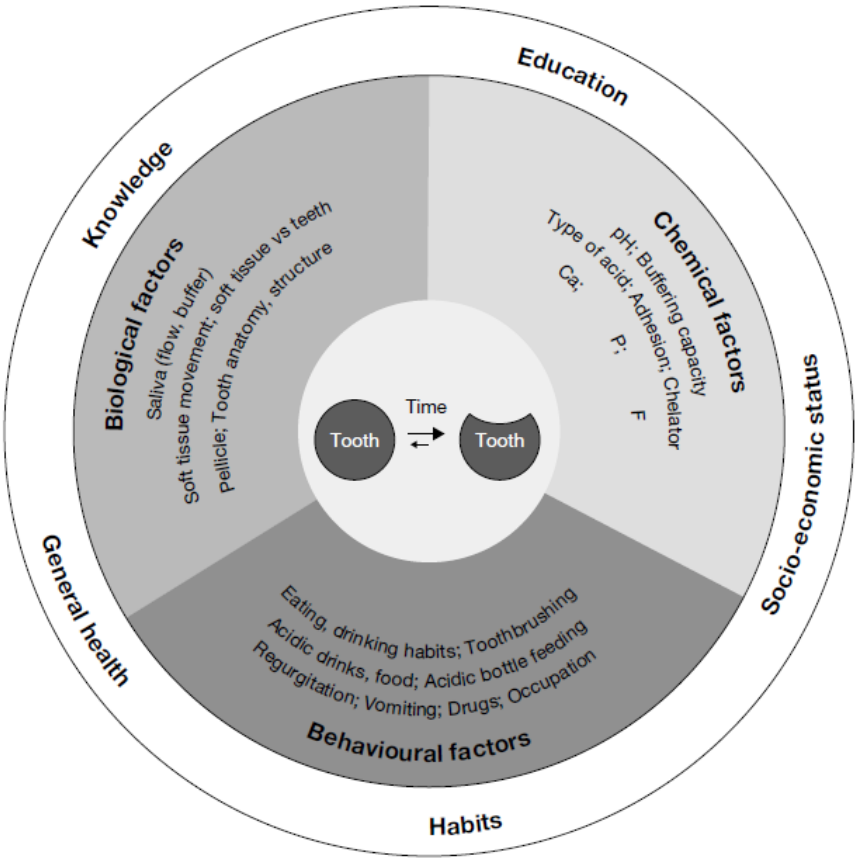


Figure 3: **Interactions of the different factors for the development of erosive tooth wear (1)**

The causes of dental erosion are often divided into external and internal factors, where variation in biological factors such as saliva, acquired pellicle and tooth structure probably influence the development and progression of the condition (45, 49).

Assessment of all the risk factors is helpful for the understanding of the etiology of the disease and allows a more accurate analysis of the risk of developing dental erosion for a particular patient. Knowing the risk factors, the reported symptoms and clinical signs

enables us to initiate adequate preventive and therapeutic measures, giving, the patient the best possible treatment.

The external factors includes

- Behavioral Factors
    - Dietary factors (50)
    - Lifestyle
- Excessive toothbrushing. (51)
- Occupational exposure (52)
- Acidic medications and other drugs

Internal factors:

- Salivary factors(53)
    - Flow
    - Pellicle and
    - Buffer capacity
- Eating disorders (54, 55)
- Gastro-oesophageal reflux diseases (56, 57)
- Tooth quality, anatomy and occlusion

2.5.1 The external factors:

Behavioral Factors

Behavioral factors, such as those listed in Table 5, are likely to increase the risk of dental erosion.

***Table 5*: Behavioral factors influencing erosive tooth wear (58)**
• Unusual eating and drinking habits
• Healthier lifestyle: diets high in acidic fruits and vegetables
• Unhealthy life style: frequent consumption of alcopops and designer drugs
• Alcoholic disease
• Excessive consumption of acidic foods and drinks
• Night-time baby bottle feeding with acidic beverages
• Oral hygiene practices

Dietary factors and lifestyle

An acidic diet is considered to be one of the most important contributors to the presence and progression of dental erosive wear. Studies have shown that the total amount and frequency of consumption of acidic substances have increased because of changes in lifestyle (49, 59)

Table 6: **Chemical factors influencing the erosive potential with respect to food and beverages(1)**
• pH and buffering capacity of the product
• Type of acid (pKa values)
• Adhesion of the product to the dental surface
• Chelating properties of the product
• Calcium concentration
• Phosphate concentration
• Fluoride concentration

Dental erosion can be caused by continued direct contact between the surfaces of the teeth and acidic substances like soft drinks, fruit juices and sport drinks. These products are very acidic, they all have pH values well below the critical pH of 5.5 at which tooth decalcification occurs. The natural intraoral pH is 6.8, but it decreases after drinking an acidic drink to below pH 5 within 2 to 3 minutes. It also requires a large amount of stimulated saliva to be neutralized (35, 60).

Studies have reported a link between a healthy lifestyle and dental erosion. A healthy lifestyle often involves frequent consumption of acidic fruits and vegetables and regular exercise. This will often lead to an increased loss of bodily fluids because of rapid breathing and sweat, and may lead to dehydration and decreased salivary flow. If an athlete in addition consumes sports drinks, fruit juices or other acidic beverages during exercise, the risk of dental erosion is increased (61, 62).

Recent research has reported that a high intake of fruit is a causal factor for dental erosive lesions. Järvinen *et al*. (63) reported that individuals with an intake of citrus fruits more than twice a day shows a greater risk of developing erosive tooth wear. The risk was actually found to be 37 times higher in people with a twice-daily intake of fruit, than in persons eating fruit less often.

In contrast, an unhealthy lifestyle, for example involving alcoholism, use of drugs and extreme use of tooth whitening products can increase the risk of dental erosion.

Some alcoholic drinks, such as dry wine, cider and alcohols are also acidic. A study done by Robb and Smith (64) reported a higher prevalence of tooth wear in 37 alcoholic patients compared to healthy controls of the same age and sex.

Environmental factors and occupation

Environmental factors mainly involve exposure to acid fumes without proper safeguards.

Several studies have investigated professionals in certain industries, for example employers in the battery, chemical, tin and dyestuff manufacturing industries. Workers in these industries are more likely to be exposed to airborne acids such as sulfuric, hydrochloric, nitric, tartaric, chromic, phosphoric, and acetic acids. The results showed that these workers are at a greater risk of developing dental erosion (65, 66).

Workers in the wine industry exhibit a higher prevalence and severity of dental erosive wear, compared with non-wine consumers. A study done on Norwegian wine tasters by Mulic showed that there were significant differences in the prevalence of dental erosive wear between the wine tasters and the control group. The wine tasters had a higher prevalence and more severely affected surfaces (52).

There has also been suggested that swimming pools with low pH, due to inadequate maintenance, could increase the occurrence and progression of erosion (61). However, these considerations are often ambiguous.

2.5.2 The internal factors

The internal factors include different types of diseases and bad habits that may lead to acidic stomach contents rising up into the oral cavity, thereby affecting the teeth and increasing the risk of erosive wear. Examples of such cases are patients suffering from acid reflux or from an eating disorder where self-induced-vomiting is a symptom. Pregnancy and obesity can also cause reflux problems and represent an increased risk of erosions. In addition to these factors, reduced saliva flow rate is one contributory and modifying cause, because it results in reduced clearance and reduced buffering of acidic substances.

Saliva

Saliva is one of the most important protective factors against dental erosion and contributes to neutralization, thinning, remineralization and dilution of the acid. It also plays a role in the formation of a protective membrane, the pellicle, which covers the tooth surface.

Saliva flow rate

The average unstimulated salivary flow rate is reported to be >0.3 ml/min with normal daily production between 0.5 and 1.5 liters. Hyposalivation can be defined as the decrease of unstimulated and stimulated salivary flow rates to less than 0.1 and 0.7 ml/min (67). It has been shown that people who are suffering from hyposalivation are at higher risk for erosive damage, compared with those who have a normal saliva flow rate.

Different conditions that can lead to hyposalivation(68, 69):

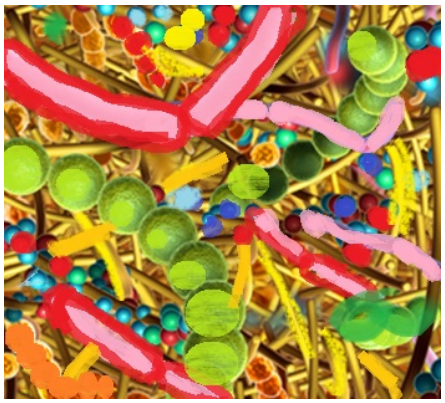
- Radiation therapy in the head and neck region(70).
- Medication, a combination of 3 medicines or more or some specific medications(1)
- Diseases
    - Sjögren`s syndrome(71)
      pathology in salivary glands
    - diabetes 1 and 2.
- Disturbance in the water balance(61).

The elimination (clearance) of an acidic substance varies individually with saliva flow rate, but also on ability to swallow. A lower flow rate decreases the capacity of saliva in

neutralizing and buffering acids, and as a result increasing the chances for erosion development.

Pellicle

When an acidic solution comes in contact with enamel, it first has to diffuse through the pellicle. The pellicle acts as a diffusion barrier or a perm-selective membrane preventing direct contact between the acids and the tooth surface. The acid can only interact with the enamel if it passes through the pellicle. This barrier is an organic film, devoid of bacteria, formed by the adsorption of proteins, peptides, lipids and other macromolecules present in saliva. The formation of the pellicle is a dynamic process, which initiates as soon as peptides and proteins come into contact with the cleansed tooth surface (72).



Figur 4: **The pellicle:** an organic film not containing bacteria, formed by the adsorption of proteins, peptides, lipids and other macromolecules present in saliva.

The pellicle covers the oral hard and soft tissues, and it varies in thickness between individuals, but it also varies in thickness between different locations in the mouth. A thinner pellicle leads to increased risk of erosion compared to a thicker layer.

A study done by Amaechi *et al*.showed that the thickness of the acquired salivary pellicle varies within the dental arches, which may be responsible for the site specificity of dental erosion. The thickest pellicle occurred at the lower posterior lingual surface, while the thinnest occurred at the upper anterior palatal surface (73).

GERD

Gastro-esophageal reflux disease (GERD) is a common condition affecting up to 65% of the western population at one point during their lifetime.

GERD and other intrinsic factors can cause dental erosion since they may reduce the saliva pH to the levels below the critical pH at which hydroxyapatite crystals in the dental enamel dissolves. At a pH of less than 2.0, gastric reflux is potentially capable of causing dental erosion. A typical clinical sign of acidic gastric juice entering the mouth is palatal dental erosion. As the condition becomes more chronic, the erosive wear becomes more widespread (54).

Eating disorders

It is known that eating disorders (ED) can be a problem among children and adolescents and the population of Norway is no exception (74).

Eating disorders such as anorexia and bulimia nervosa have long been recognized as a factor in the development of dental erosion, especially among those with an eating disorder in combination with vomiting. The oral status of these patients has been assessed in several studies. Johansson examined the oral health of patients with eating disorders and compared them to gender- and age-matched controls. In the group they studied, with a mean age of 21 years, patients with eating disorders were 8.5 times more likely to exhibit dental erosion. The patients with a longer history of eating disorders more commonly possessed teeth with dental erosive lesions. Patients with bulimia nervosa are also shown to have more acidic oral mucosa than age-matched controls as well as a decreased salivary flow rate (44, 75)

Results from a study performed in Oslo on a group of Norwegian bulimic patients experiencing self-induced vomiting (SIV), showed that dental erosion is more commonly affecting individuals with ED experiencing SIV. The distribution of the lesions are more often found on palatal/lingual surfaces than on buccal surfaces. The study showed that out of 72 referred patients, 66 were or had been experiencing SIV. Dental erosions were found in 46 individuals, 19 had enamel lesions only, while 27 had both enamel and dentine lesions. Ten or more teeth were affected in 26.1% of those with erosions, and 9% had >/=10 teeth with dentine lesions. Of the erosions, 41.6% were found on palatal/lingual surfaces, 36.6% on occlusal surfaces and 21.8% on buccal surfaces. Dentine lesions were most often found on lower first molars, while upper central incisors showed enamel lesions most frequently. The majority of the erosive lesions were found in those with the longest illness period, and 71.7% of the lesions extending into dentine were also found in this group (55).

Tooth quality, anatomy and occlusion

With regard to biological factors like the quality of dental tissues, tooth position and anatomy of the soft tissues might affect the development of dental erosion (42).

The anatomy of the teeth and the positioning in the mouth in relation to method of drinking and swallowing may also affect the development of erosion. Eroded enamel is more susceptible to attrition, and thus, occlusion plays a major role in the formation of the wear pattern (49).

Medicine

Acidic medicines with a low pH value have the potential to soften dental hard tissues, especially if used frequently and/or over a long period of time. Some medicines such as tranquilizers, antihistamines, antiemetic and antiparkinson can contribute to increased erosion, as they affect the salivary flow rate and buffering capacity. The salivary flow rate is decreased leading to a reduction in the modifying and protective effects of saliva (68).

The intake of asthma medication in the form of inhalation has often been associated with dental erosion. It is assumed that the content of the inhalers might have an acidic pH and thereby lead to a greater risk for erosive wear. However, only one controlled study has supported this (76).

2.6 Prevalence and progression

During the last few decades there has been extensive research into the prevalence and etiology of dental erosion. A number of epidemiological studies amongst children and adolescents have been published (11, 15, 16, 20, 43, 59).

The studies show a high variation in the prevalence of dental erosion and it is therefore difficult to compare and judge the outcome. The reported variations can be explained by difference in the study sample, the methods and the examiners (20). Factors influencing the result are age, population, ethnicity and socio-economic background. The clinicians also used different scoring systems and the dental examination was not always standardized. Another reason why dentists now report that dental erosion is more prevalent compared to 10-15 years ago (3), could be due to the change in focus of the public on dental erosive wear.

I Norway, the prevalence of dental erosion in Oslo have been reported to be 38% in 18 year-olds. Out of the individuals diagnosed with erosions 32,2% had at least one lesion extending into the dentine (15). In Sweden the prevalence of dentine erosive wear has been reported to be 11,9% among 13-14 year-olds and 22,3% in 18-19 year-olds (11).
In addition, many studies report on tooth wear prevalence in general. Despite these variations, it is safe to say that the prevalence of dental erosions is growing steadily.

More studies regarding the prevalence of dental erosion are necessary to predict the future needs for prevention, treatment and assessment of progression, but also to stimulate the resources required to handle the problem (4).

Over the last decades there has been a change in lifestyle. Today's lifestyle promotes an increase in acidic challenges to the dentition as the consumption and frequency of acidic food and beverages have changed (39, 44)

The increased consumption of acidic food and beverages is a contributing factor to the progression of erosion, but it is important to remember that dental erosion has a multifactorial etiology (24). Studies have shown an increase in the prevalence of diseases resulting in stomach acid reaching the mouth and as a result promoting dental erosion (44). It is therefore essential to diagnose and identify the causative factors early in, order to prevent further progression of lesions (22).

To assess the progression of dental erosions it is necessary to have incidence data from studies. The need for a population based incidence study is apparent in assessing the progression. Incidence data in any form is however sparse. This is partly due to difficulties in obtaining access to the same subjects at two or three different occasions.

In a study by Dugmore and Rock a total of 1753 children were examined at age 12 years and 1308 were successfully traced and re-examined 2 years later at age of 14 years. Erosions were present in 56.3% of the12 year-olds increasing to 64.1% by the age of 14. The proportion of subjects with deep enamel lesions increased from 4.9% to 13.1% and those with exposed dentine from 2.4% to 8.7%. 161 children (12.3%) who had no erosions at age of 12, developed tooth erosion at the age of 14. Most of the erosions were graded score 1; loss of enamel surface characteristics. Approximately 1% had erosions grade 2, deep enamel loss or grade 3, exposure of dentine (77).

2.7 Management and prevention

To prevent erosive tooth wear, early diagnosis and identification of causative factors are essential (22).

Ideally, the etiology of dental erosion should be identified prior to the patient management. This is however not always possible due to several reasons. It may be difficult to gain an accurate and up to date patient history or the patient may withhold important information regarding lifestyle or behavior (78).

Identification and reduction of risk factors: Patient medical history and dietary intake.

Patient medical history: It is important to do a thorough anamnesis.

The anamnesis should contain questions regarding gastro-oesophageal reflux and vomiting. These conditions are known to increase the risk of dental erosion. Side effects of medications such as reduced saliva flow also leads to increased susceptibility to acidic substances (54).

Dietary intake: Careful questioning regarding dietary intake, with particular reference to specific items of food and drinks known to have a high acidic content.

A dietary journal is a useful aid where the patient records a minimum three-day diet history (including a weekend). Times of food and drink consumption must also be recorded. From the dietary journal and an intraoral examination, the dentist can determine the erosive potential of the diet and give dietary counseling to the patient (1, 39).

Another aid can be patient information leaflets containing information regarding dental erosion. By handing out these leaflets we allow patients to gain information about risk factors, prevention and treatment on their own time.

When diagnosed, the erosions must be recorded and monitored using tools like study casts, tooth wear indexes and photographs. If tooth wear indexes are used it is important that the dentist is aware of the diagnostic criteria and the need to maintain good intra-examiner reproducibility.

Patient cooperation and management:

Early diagnosis of dental erosion may stop the progression, provided the patient complies with the dentist's advice. The most important factor in prevention of further progression is for the patient to change their lifestyle (22, 39).

Using the dietary journal and an intraoral examination the dentist should suggest an individually tailored treatment program to the patient. It should comprise of dietary advice, oral hygiene instructions and motivation (use of buffering agents, optimization of fluoride regime, stimulation of salivary flow rate and instructions for a non-destructive tooth brushing technique (1).

As long as the patient has no complaints regarding symptoms of pain/sensitivity, reduced function or esthetical aspects, a "wait and see" approach regarding restorative treatment is recommended. If the patient however has complaints, interventional treatment may be necessary.

3.0 Selection of literature

Before initiating the study we searched for relevant literature. We used the following databases:

- Pubmed

We used the following *keywords* in our search:

- Progression + erosion
- Grading + dental erosion
- Dental erosion
- Erosion + lifestyle
- Dental erosion + prevalence
- Dental erosion + risk factors
- Dental erosion + adolescents
- Dental erosion + scoring system
- Dental erosion + chemistry

4.0 The study: Progression of dental erosion among adolescents in Troms

Aim

The aim of the present study was to determine whether there was any progression of dental erosion over a two-year period among adolescents in Northern Norway.

5.0 Material and methods

5.1 Selection of study population

The raw data used in our master project was collected from the previously conducted studies FitFutures 1 and 2- a part of the Tromsø study. The Tromsø study was first established in 1974 as a response to the high mortality rate related to heart and cardiac diseases in Northern Norway. The FitFutures study aims to explore health and lifestyle, including oral health, among adolescents in Northern Norway. It was divided into two parts, FF1 and FF2, carried out two years apart 2010-2012.

In 2010-2011 school students, in the two neighbouring municipalities Tromsø and Balsfjord, were invited to join the cross-sectional health survey FitFutures. A total of 1038, 15-18-years of age participated. The patients were examined clinically by an experienced dentist, assisted by a dental hygienist. The examination included x-rays, impression of the upper and lower jaw and intraoral clinical photos.

The Norwegian Data Protection Authority and The Regional Committee of Medical and Health Research Ethics (reference number 2009/1282 and 2011/1702/REK nord) approved the study in July 2010 and October 2011, respectively. All participants gave written informed consent.

Photographs (Canon EOS 60D; Canon 105mm; Sigma EM-140 DG) were taken in the following order: the buccal surfaces of the teeth in the first and fourth quadrant (#1), corresponding surfaces in second and third quadrant (#2), the buccal surfaces of the upper and lower front teeth (#3), the occlusal surfaces of the upper teeth (#4 & 5) and lower teeth (#6 & 7), and palatinal surfaces of the upper front teeth (#8).

In order to assess the progression of the dental erosive lesions, the clinical photos and records of dental erosions collected in FF1 and FF2 were studied. The number and the severity of all dental erosions in each patient were recorded and scored using the Visual Erosion Dental Examination system (VEDE) (48).

5.2 Calibration

We received training in calibration from one of the three calibrated examiners (ABT) who had previously used the VEDE system to grade the erosions in FF1. The calibration manual consisted of photos that have been used for this purpose several times at the University of Oslo.

The calibration meetings were done in two sessions prior to examinations of the actual clinical photos in this project. In the first calibration session, 76 intra-oral photos, with both erosive and non-erosive lesions were used. Three weeks after the first session, a reproducibility test was carried out using the same 76 photos.

The meetings were carried out under identical conditions:

- The same room
- Same light condition (time of day)

5.3 Examination of photos, grading and standardization:

Intraoral close-up clinical photos of 50 individuals with erosive wear in FF1 and the same individuals in FF2 were selected by one of the calibrated clinicians from FF1. Patients were selected randomly, not based on gender, health condition or ethnicity. The photos were mixed so the examiners TAD and JG were blind to whether the photos were from FF1 or FF2.

We were not given any information about the baseline scoring. We graded the selected samples individually twice, three weeks apart. After scoring the photos separately, the two sets of results were compared and a final score was given based on joined decision. The scoring was done in the same room, under the same light conditions and using the same LCD screen.

5.4 Scoring system

We scored t sion (on

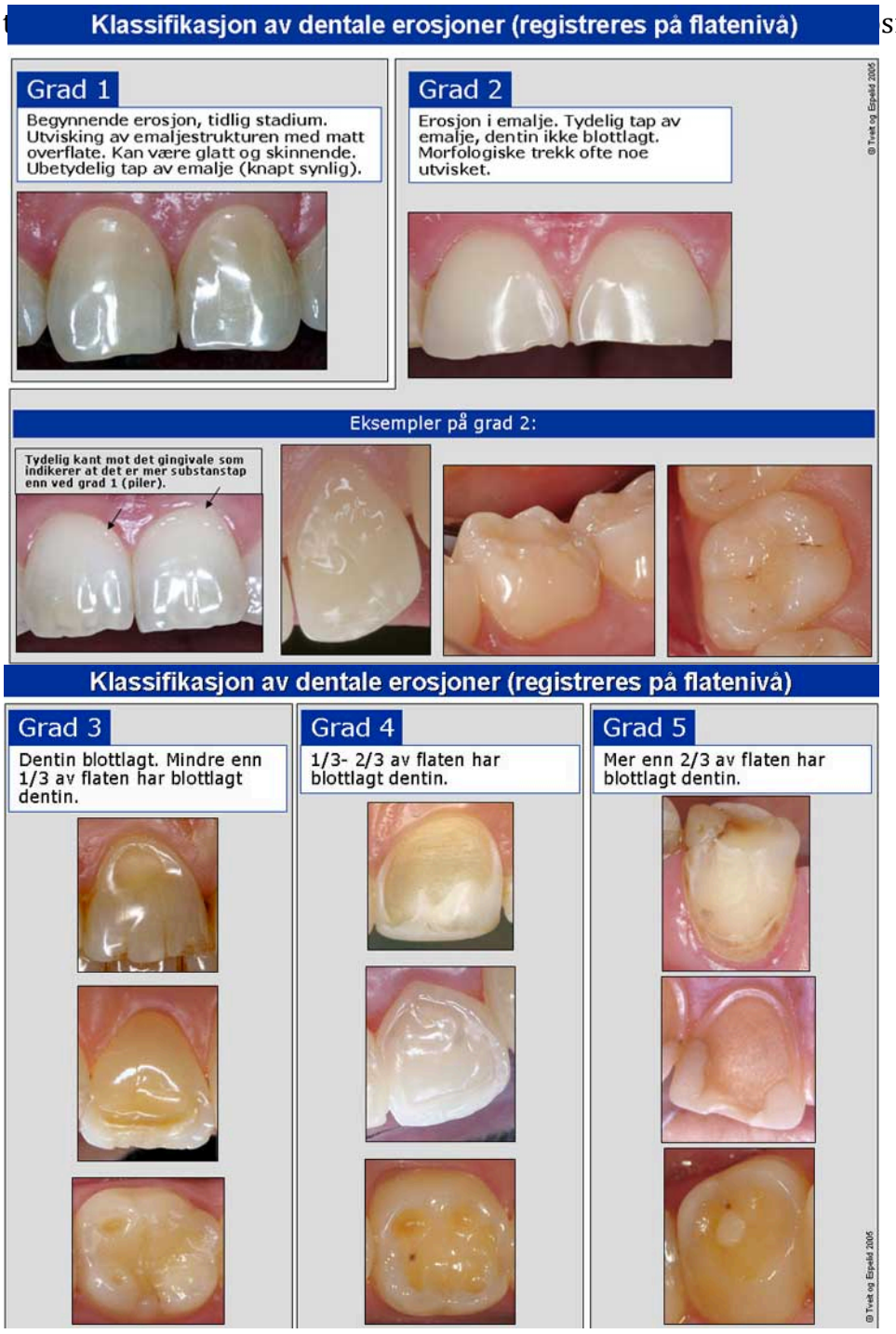


Figure 5: **The Visual Erosion Dental Examination (VEDE).**

5.5 Teeth examined

Index teeth:

Due to early eruption, first permanent molars (16, 26, 36, 46) and maxillary incisors (12, 11, 21, 22) were chosen since these teeth have been exposed to erosive challenges for several years. As a result, they are likely to show a high propensity for erosion (15). The buccal and palatal surfaces were examined on the four maxillary incisors, while the occlusal surfaces were studied on all first molars (59).

5.6 Statistic analysis

The statistical analyses were performed using IBM Statistical Package for the Social Sciences (SPSS) version 20.The absolute frequencies and proportions were obtained for data analysis (descriptive).

The inter- and intra-observer agreement was expressed by Cohen's linear weighted kappa(κ_w) and calculated using a spreadsheet programme (Microsoft Excel).

6.0 Results

6.1 Calibration

The calibration data are presented in Table 7. The mean inter-examiner agreement value expressed by linear weighted Cohen's Kappa (κ_w) at the surface level in the photo calibration session at time I was 0.62 (range 0.39-0.85) and at time II 0.66 (range 0.45-0.87). This indicates that the κ_w agreement was moderate at time I and substantial at time II, according to the scale suggested by Landis and Koch (79).

6.2 Severity and progression of dental erosions

In Figure *6, 7*, 8 and 9 an overview of the distribution and severity grade of the first permanent molars and the upper front teeth at both time I and II are presented. Enamel lesions dominated compared to dentine lesions. The first molars had more often dentine lesions than the anterior teeth. The results show that the maxillary molars were mostly affected by erosions into enamel, in contrast to the mandibular molars where dentin lesions were more frequent. The majority of the dentin lesions was grade 3 (92.5 %) and grade 4 (7.5 %). Only one lesion was graded to 5 (Figure 6-7). Overall, 18 participants developed one or more new lesions, varying from one to four in the two year period.

When looking at the progression rate of the lower first molars, tooth 36 had 22 dentin lesions (grade 3) that remained unchanged after 2 years, five had a progression from enamel to dentine and five changed status from dentine to enamel according to the registrations (Table10.). Regarding 46 and 27, dentine lesions remained unchanged, only one progressed. A total of five enamel lesions progressed to dentine and four changed status from dentin to enamel (Table 11).

The progression rate of lesions in the maxillary molars showed that tooth 16 had seven dentin lesions, which remained unchanged, seven, progressed from enamel to dentine and five changed status from dentin to enamel lesion (Table 8). Concerning 26, three dentine lesions remained unchanged, two progressed into dentine and six changed status from dentine to enamel lesion (Table 9).

A total of 15 patients had progression of erosive lesions on their lower first molars. The majority of these lesions (ten in number) progressed from grade 2 to grade 3 that means enamel to dentine. Regarding the maxillary first molars ten patients showed progression of

lesions. The majority of these lesions (eight) progressed from enamel to dentin (grade 2 to grade 3).

A slightly higher frequency of erosive lesions was observed on the upper central incisors, compared with the first molars (Figure 1 and 3). Erosive lesions were found on the central incisors in all of the 50 participants. The palatal surfaces of 12, 11, 21 and 22 had more lesions than the buccal surfaces (Fig. 8) and calculating at the surface level, 11 and 21 were most often affected (Figure 3).

Regarding 11 and 21, almost all lesions were registered as enamel lesions at both times while 45 % (17 of 38) of the surfaces registered as healthy (time I) were recorded as enamel lesions two years later (Tables 14-17). When looking at lesions in 12 and 22, 47.2% of the healthy surfaces were recorded as enamel lesions two years later (Tables 12, 13, 18 and 19).Tooth surface 22 P (time I) and 22 B (time II) were the only ones with dentin erosion grade 3 (one in each group) (Figure 8). Only one patient had a progression from enamel to dentine.

7.0 Discussion:

The erosion data were recorded using maxillary incisors and molars as index teeth. They were chosen as they are known to have a high propensity for dental erosion (15, 40, 59). The sites scored, were the buccal and palatal surfaces. At the age of 15-18 years these teeth have been present and exposed to erosive challenges for the longest.

It was considered important to distinguish erosive wear from attrition and abrasion. In adolescences there is higher probability of finding surfaces exposed to erosive challenge only (80) since attrition and abrasion are more common with age. A lesion involving dentin may be interpreted as serious in an adolescent, while the same lesion at older ages may be taken as a reflection of normal wear over the lifespan (81).

As hypothesized and in agreement with results from other studies, dentine lesions were mostly found in lower first molars, whereas lesions restricted to enamel dominated in upper anterior teeth (15, 59). Results from a Swedish study (59) showed that in a group of adolescences with registered erosive lesions, 74% of the lesions were on molars. In a similar conducted study in Norway, 44% of the registered lesions were on the molars and 46% on the

incisors (15). A study by Peres *et al.* reported that among a group of 12-year-old school children in Brazil, enamel loss was the most prevalent type of dental erosion, which was the finding in the present study as well.(90).

We also registered cuppings and the majority was seen on the lower first molars. This is in agreement with a study done on prevalence and severity of dental erosive wear among a group of Norwegian 18-year-olds. Of the participants registered with erosive lesions, 62% were registered with cuppings, out of which 85% where on the first molars. The cuppings were usually in addition to erosive lesions on other surfaces (15).

Concerning the incisors, the prevalence may be explained by the abrasive effect of the tongue in combination with erosive components. The consumption and distribution pattern of acidic drinks may also contribute to the greater loss of tooth substance (82). Looking at the progression rate, the majority of these lesions remained in enamel during the whole 2-year period.

Regarding the first molars, lower molars had a higher prevalence of dentine erosive lesions than upper. This may be explained by the significantly thinner enamel in lower molars than in upper molars (83). In addition, it has also been suggested that occlusal surfaces in lower molars are more likely to have contact with acidic fluid due to the gravity (84).

Different diagnostic tools in the assessment of progression can be used, like clinical examination, intraoral photos and study models. These can be assessed alone or in combination (46).

The advantage of using casts compared to a clinical approach is that the evaluation can be performed repeatedly without the patients present and under optimal illumination. The casts can be viewed from all sides and in addition, an excellent assessment of occlusal surfaces and the occlusal relation can be preformed. However, one cannot address difference in color and reflection of enamel and the diagnosis of very early stages may therefore be difficult. This can result in underestimation of initial lesions. In a clinical examination one of the criteria for estimating the severity is the amount of dentin exposed. This is not possible in the diagnosis of lesions on study models. (46, 81)

Clinical photos are a method to assess progression of tooth wear over long periods of time. It offers an advantage in research studies, as they are easy to select, arrange and rearrange, making randomizing and making blinding easier. They also give the examiners an opportunity to repeat assessment without the patient's presence as they can be archived. However, technical difficulties may affect the validity. Lack of sharpness, loss of contrast and focus may affect the distinguishing of difference in severity. The fact that the photos from FF1 and FF2 were not taken in the same room and under the same conditions may have influence the credibility of comparison.

In a clinical examination, one has the benefit of archiving optimal drying conditions. This makes the extent of dentine exposure and initial enamel loss easier to detect. We experienced difficulties in distinguishing between healthy surfaces and initial enamel loss due to presence of saliva in many of the clinical photos. In the present study, these aspects may have resulted in a shift from the diagnose healthy surface towards a grade 1 lesion and vice versa. This may be an explanation to the numbers of "reversals". Considering the difficulties in distinguishing between healthy surfaces and grade 1, one could consider if these grades should have been merged. This holds true if grade 1 lesions are interpreted as normal features of functioning teeth even at an early age(81).

Scoring dental erosion based on clinical photos without the benefit of direct clinical comparison is therefore not ideal, but in this case it was the only way to assess the longitudinal progression.

The variation in the results between our study and other studies may be partially explained by differences in the diagnostic criteria. Prevalence studies have shown varied findings and may reflect the difficulty in finding an universal index among researchers for measuring and scoring dental erosions (17).
As discussed earlier, the VEDE, has a very detailed scale. It distinguish between no dental erosive wear, initial loss of enamel and markedly loss of enamel, which sometimes makes it difficult for the examiners to differentiating between intact enamel and early enamel lesion (46).

One study recorded wear only on certain surfaces (palatal and buccal surfaces)(85), while a prevalence study in Sweden used two modified grading systems bases on Hasselkvist *et al.* and Eccles and Johansson *et al.* The erosions where categorized into mild, moderate and severe (11, 59, 86, 87).

Aine *et al*. graded dental erosion according to their clinical appearance, being graded from 0 (no erosion) to 3 (dentine exposure on occlusal surfaces or dentine affected on other surfaces). The study was done on both primary and permanent teeth affected by gastro-oesophageal reflux disease (GORD). This index is limited as it does not specify the site of the lesion or the extent of the involved surface(88).

Regarding the severity and progression rate of the lesions, one could argue that these adolescences do not suffer severely from erosion and that the rate of progression is slower than first assumed. When taking in account the age of the participants, it is likely that many of the lesions will develop into more severe lesions. It is, however, impossible to predict if the worst cases would progress more rapidly as erosions are a measure of past conditions and not a predictor for the future. One can on the other hand assume that without any intervention these lesions will progress (77).

There are only a few studies that have investigated the incidence and progression of dental erosion. A study done by Ganss *et al.* on study models showed that over a five year period the proportion of permanent teeth with moderate lesions increased from 5.3% to 23%, while teeth with more severe lesions increased from 0.4% to 1.5% (81).
Another study done by Dugmore *et al*. examining the incidence of erosions over a two year period in children aged 12 and 14 years showed that erosion were present in 56.3% of the 12-year-olds, increasing to 64.1% by the age of 14 .The proportion of subjects with exposure of deep enamel increased from 4.9% to 13.1%, and those with exposed dentine from 2.4% to 8.7% (77). In contrast to the low progression rate found in the present study, a relatively high incidence of enamel lesions was seen on the upper front teeth (45% for the incisors and 47% for the laterals). This indicates erosive activity. Longer observations times may be required to follow lesions progression and to evaluate risk indicator for the individuals.

8.0 Conclusion:

There are concerns that the assessment of dental erosion, particularly in the early stages, is difficult. This is not unlike the problem in recording early caries lesions (91). In this study, dental erosion did not progress markedly over a two year period when assessed by comparing clinical photos, less than reported in other studies (9, 59, 77). However, the incidence was relatively high. Increasing prevalence of dental erosion during the last two decades (4, 9, 15, 59) require further cross-sectional and longitudinal prevalence studies. We also believe that there is a great need for further research into scoring method that can be used worldwide, which makes comparison easier between studies. Awareness of dental erosion should be enhanced to allow early diagnosis and preventive measures.

9.0 Appendix
9.1: Calibration

Table 7: Calibration

	κw	Agreement (%)	CI
A1-B1	0.62	53	0.39-0.85
A2-B2	0.66	53	0.45-0.87
A1-true value	0.54	41	0.30-0.79
A2- true value	0.64	53	0.43-0.86
B1- true value	0.58	47	0.35-0.81
B2- true value	0.68	57	0.47-0.88
A1-A2	0.61	50	0.38-0.84
B1-B2	0.70	59	0.50-0.90

Table 7: A:student 1. B:student 2. 1: first calibration, 2: second calibration, 14 days later."true value" is based on the registration of a calibrated examiner ABT.

9.2: Tables and figures: Distribution and severity

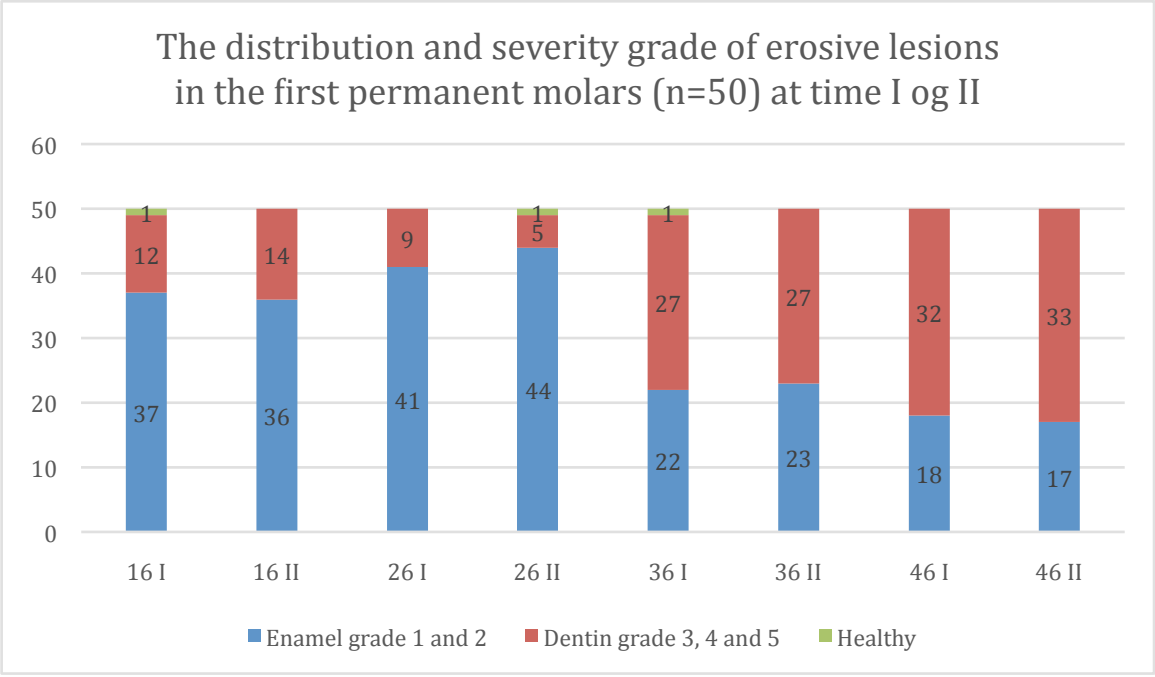


Figure 6. The number of erosions expressed in severity (enamel=grade 1 and 2, dentin= grade 3, 4 and 5) on the occlusal surfaces of 16, 26, 36 and 46 (n=50) at time I and II (2 years later).

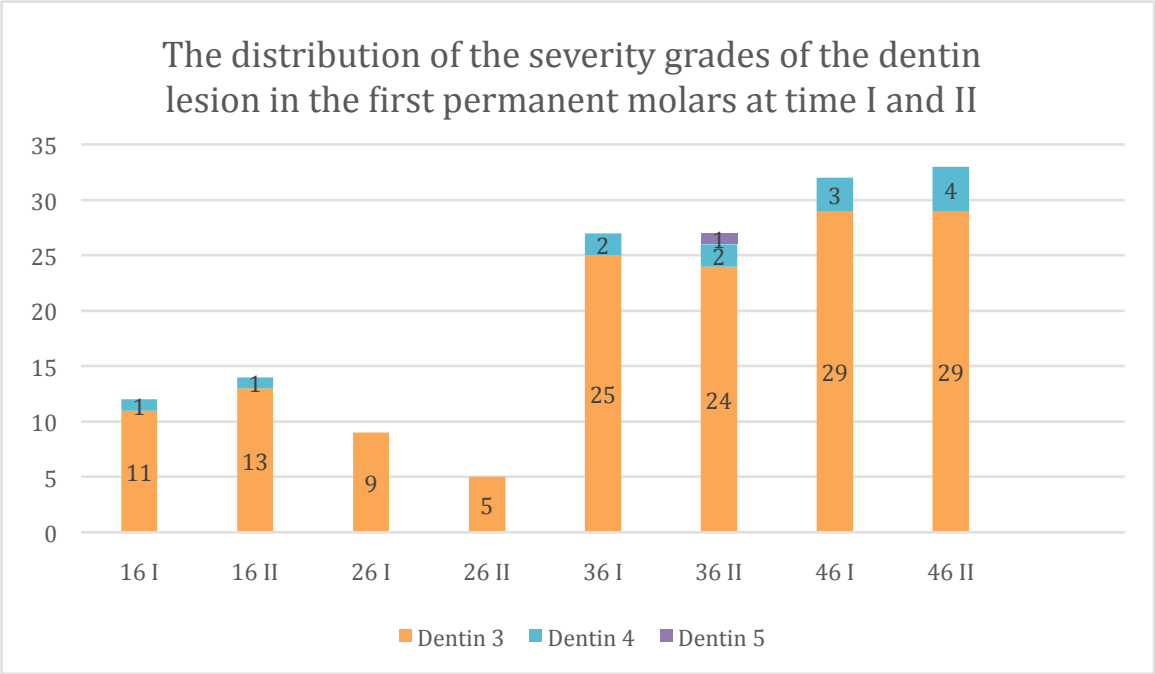


Figure 7. Teeth 36 and 46 had a higher frequency of erosions into dentin (grade 3, 4 and 5) compared to 16 and 26 at both time I and II (59% versus 21% respectively).

Tooth 16 (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy
Enamel erosions	37	30 Unchanged		30		
		Enamel-Healthy				
		7 Enamel-Dentin			7	
Dentin erosions	12	7 Unchanged			7	
		5 Dentin-Enamel		5		
Healthy	1	1 Healthy-Enamel		1		
Total Surfaces	50	50		36	14	0

Table 8. The distribution of healthy surfaces and erosive lesions on **tooth 16** at time I and II, expressed by unchanged-, progressed- and "reversals".

Tooth 26 (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy
Enamel erosions	41	38 Unchanged		38		
		1 Enamel-Healthy				1
		2 Enamel -Dentin			2	
Dentin erosions	9	3 Unchanged			3	
		6 Dentin-Enamel		6		
Healthy	0					
Total surfaces	50	50		44	5	1

Table 9. The distribution of healthy surfaces and erosive lesions on **tooth 26** at time I and II, expressed by unchanged-, progressed- and "reversals".

Tooth 36 (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy
Enamel erosions	22	17 Unchanged		17		
		Enamel-Healthy				
		5 Enamel -Dentin			5	
Dentin erosions	27	22 Unchanged			22	
		5 Dentin-Enamel		5		
Healthy	1	1 Healthy -Enamel		1		
Total surfaces	50	50		23	27	0

Table 10. The distribution of healthy surfaces and erosive lesions on **tooth 36** at time I and II, expressed by unchanged-, progressed- and reversed surfaces.

Tooth 46 (n=50)	Time I	Progression	/»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy
Enamel erosions	18	13 Unchanged		13		
		0 Enamel-Healthy				
		5 Enamel -Dentin			5	
Dentin erosions	32	28 Unchanged			28	
		4 Dentin-Enamel		4		
Healthy	0					
Total surfaces	50	50		17	33	

Table 11. The distribution of healthy surfaces and erosive lesions on **tooth 46** at time I and II, expressed by unchanged-, progressed- and reversed surfaces.

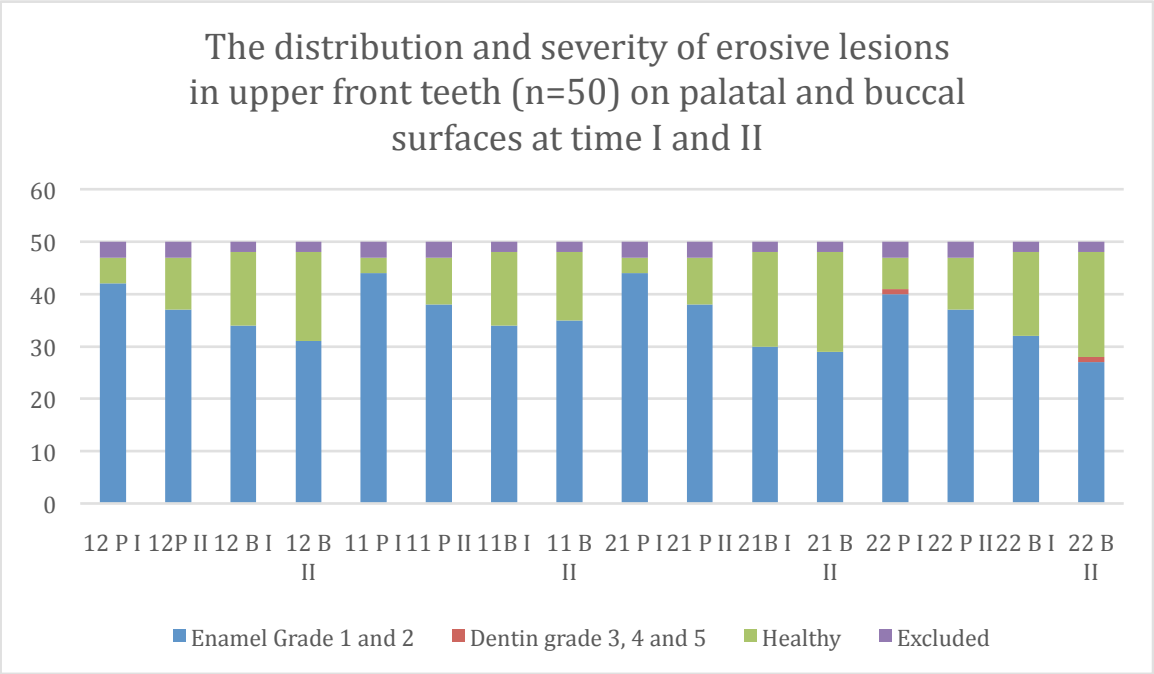


Figure 8. Number of erosions expressed in severity (enamel=grade 1 and 2, Dentin= grade 3, 4 and 5) of the erosions on the palatal (n=50) and buccal (n=50) surfaces of 12, 11, 21 and 22 at time I and II (2 years later).

Tooth 12 P (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	42	Unchanged		35			
		Enamel-Healthy				7	
		Enamel -Dentin					
Dentin erosions	1	Unchanged					
		Dentin-Enamel					
Healthy		Unchanged				3	
	5	Healthy-Enamel		2			
Excluded	3						3
Total surfaces	50	50		37		10	3

Table 12. The distribution of healthy surfaces and erosive lesions on **tooth 12P** at time I and II, expressed by unchanged-, progressed- and "reversals"

Tooth 12 B (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	34	Unchanged		23			
		Enamel-Healthy				11	
		Enamel -Dentin					
Dentin erosions	0	Unchanged					
		Dentin-Enamel					
Healthy	14	Unchanged				6	
		Healthy-enamel		8			
Excluded	2						2
Total surfaces	50	50		31	0	17	2

Table 13. The distribution of healthy surfaces and erosive lesions on **tooth 12B** at time I and II, expressed by unchanged-, progressed- and "reversals".

Tooth 11 P (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	44	Unchanged		38			
		Enamel-Healthy				6	
		Enamel -Dentin					
Dentin erosions	0	Unchanged					
		Dentin-Enamel					
Healthy	3	Unchanged				3	
		Healthy-Enamel					
Excluded	3						3
Total surfaces	50	50		38		9	3

Table 14. The distribution of healthy surfaces and erosive lesions on **tooth 11P** at time I and II, expressed by unchanged-, progressed- and "reversals".

Tooth 11 B (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	34	Unchanged		26			
		Enamel-Healthy				8	
		Enamel -Dentin					
Dentin erosions		Unchanged					
		Dentin-Enamel					
Healthy	14	Unchanged				5	
		Healthy to Enamel		9			
Excluded	2						2
Total surfaces	50	50		35		13	2

Table 15. The distribution of healthy surfaces and erosive lesions on **tooth 11P** at time I and II, expressed by unchanged-, progressed- and "reversals" surfaces.

Tooth 21 P (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	44	Unchanged		38			
		Enamel-Healthy				6	
		Enamel -Dentin					
Dentin erosions		Unchanged					
		Dentin-Enamel					
Healthy	3	Unchanged				3	
		Healthy -Enamel					
Excluded	3						3
Total surfaces	50	50		38		9	3

Table 16. The distribution of healthy surfaces and erosive lesions on **tooth 21P** at time I and II, expressed by unchanged-, progressed- and "reversals".

Tooth 21 B (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	30	Unchanged		21			
		Enamel-Healthy				9	
		Enamel -Dentin					
Dentin erosions		Unchanged					
		Dentin-Enamel					
Healthy	18	Unchanged				10	
		Healthy -Enamel		8			
Excluded	2						2
Total surfaces	50	50		29		19	2

Table 17. The distribution of healthy surfaces and erosive lesions on **tooth 21B** at time I and II, expressed by unchanged-, progressed- and "reversals".

Tooth 22 P (n=50)	Time I	Progression	»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	40	Unchanged		34			
		Enamel-Healthy				6	
		Enamel -Dentin					
Dentin erosions	1	Unchanged					
		Dentin-Enamel		1			
Healthy	6	Unchanged				4	
		Healthy-Enamel		2			
Excluded	3						3
Total surfaces	50	50		37		10	3

Table 18. The distribution of healthy surfaces and erosive lesions on **tooth 22P** at time I and II, expressed by unchanged-, progressed- and "reversals" surfaces.

Tooth 22 B (n=50)	Time I	Progression	/»Reversals»	Time II Enamel	Time II Dentin	Time II Healthy	Time II Excluded
Enamel erosions	32	Unchanged		22			
		Enamel-Healthy				9	
		Enamel -Dentin			1		
Dentin erosions		Unchanged					
		Dentin-Enamel					
Healthy	16	Unchanged				11	
		Healthy-Enamel		5			
Excluded	2						2
Total surfaces	50	50		27	1	20	2

Table 19. The distribution of healthy surfaces and erosive lesions on **tooth 22B** at time I and II, expressed by unchanged-, progressed- and "reversals".

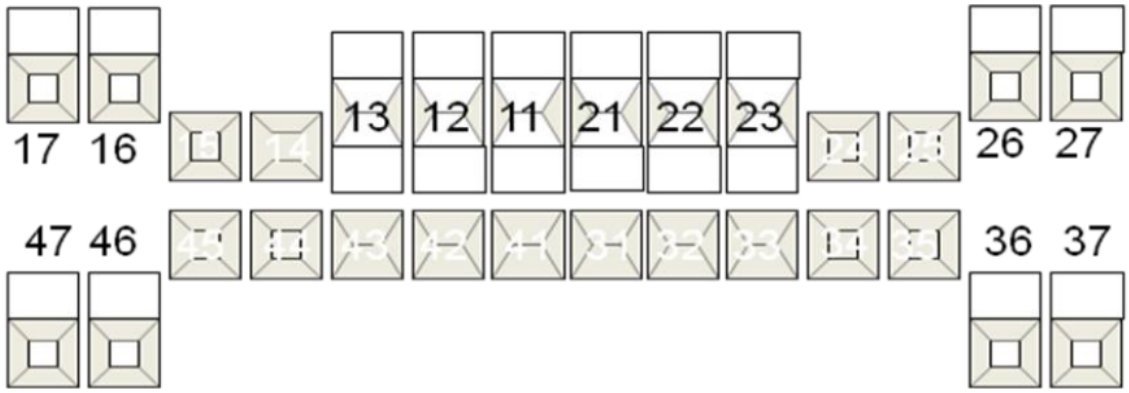
9.3: Registration form used in the examination

Registration nr:

Examiner:

Clinic:

Date:



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