



BY: PROF. FRANCIS G. MACIGO

2. OVERVIEW OF CARIES: EPIDEMIOLOGY, CLINICAL PRESENTATION & PREVENTION



OBJECTIVES



- * Epidemiology
- * Clinical presentation
- * Prevention



Definition



- *Dental caries is a disease of the hard tissues of the teeth characterized by the demineralization of the inorganic component of the tooth followed by the breakdown/dissolution of the organic component.



It is a dynamic process



- * Demineralization (removal/ leaching of CaPO_4) → remineralization (precipitation of Ca & PO_4 ions)
- * Remineralization → demineralization
- * Dental caries occurs if there is more demineralization than there is remineralization.
- * Demineralization leads to softening and weakening of the affected tooth surface



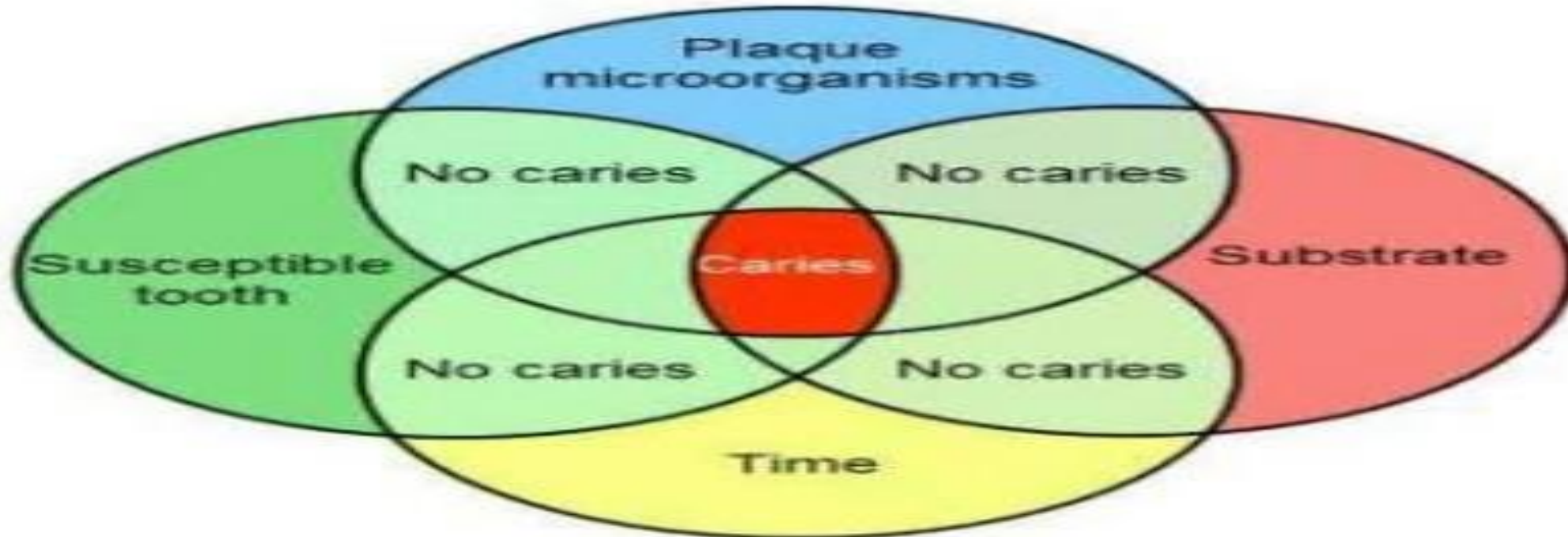
Etiology



- * Dental Caries (DC) is a product of a complex interaction among 4 major factors:
 1. Bacterial agents concentrated in plaque
 2. Suitable substrate: mainly sucrose
 3. Susceptible host/ teeth (enamel etc.)
 4. Time factor: there must be continuous demineralization with no time for remineralization
- * 4 factors form an **ecosystem**. All factors must be present for DC to occur.



Image of dental caries ecosystem



Preventive measures are aimed at upsetting the ecosystem



Role of the 4 factors



Plaque bacteria.

- * The most important are:
 - * **Acidogenic bacteria: produce acid**
 - * **Aciduric bacteria: can survive at low pH. Most important**
 - * *Streptococcus mutans*
 - * *Lactobacilli acidophilus*
 - * *Actinomyces*



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Suitable substrate.

- * Mainly refined/ fermentable carbohydrates
- * Sucrose is the most important substrate for bacterial metabolism resulting in acid production e.g. lactic acid and pyruvic acids
- * Sucrose: the arc criminal



Dietary practices that increase the risk of DC



- *Diet rich in fermentable CHO
- *Frequency of consumption
- *Timing of intake – between meals: favor growth of Lactobacilli
- *Bed – time snacks



Cont.



Susceptible host.

- * Tooth enamel is susceptible to dissolution by acids
- * Characteristics of teeth that increase susceptible to dental caries:
 - * Tooth morphology – pits, fissures, grooves – not cleansable
 - * Tooth composition – quantity of minerals. Inorganic matter in relation to organic matter
 - * Increased minerals → increased resistance



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Time factor

- * There must be sufficient time and repeated attack from bacterial acids with insufficient time for recovery
- * Brushing teeth helps



Interaction and consequences



- * Bacteria on tooth surface + sucrose → fermentation into acids → demineralization (interplaying with remineralization) → time factor → demineralization supersedes progressive destruction of tooth substance → time → tooth mortality



Epidemiology



- * Indices of measuring DC:

- * **DMF (T) index:** measures caries experience in **permanent teeth** i.e. the number of teeth decayed (D), teeth missing due to caries (M) & teeth filled due to caries (F)

- * $D = 3; M = 1; F = 0$: DMF (T) score = 4

- * **dmf (t) index:** measures caries experience in **deciduous teeth**



DC prevalence and experience in Kenya



- * The Kenya National Oral Health Report of 2015 showed the following:
 - * The DC prevalence in the ages 5, 12 & 15 yrs. \rightarrow 23.9%
{caries experience DMF(T)/ dmf(t) \rightarrow 0.8}
 - * DC prevalence in the 5 yrs. Age group only is 46.3%
{1.87}
 - * DC prevalence in adults: 34.3%



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- * By international standards, DC experience in Kenya is lower than that in many other countries
- * However evidence from various studies shows DC experience in Kenya is increasing.



Sugar consumption and DC in Kenya



- * International Dental Journal 2016 (Macigo F. G., James R. M et. Al.)
- * Findings showed DC in Kenya is increasing due to increasing sugar consumption
- * Per capita sugar consumption increased from 35.5g/d in 1969 to 60.8g/d in 2009. critical sugar consumption is 50g/d according to studies.



Cont.



- * DC experience in deciduous teeth in 3 – 5 yrs. Increased from dmf (t) index of 1.5 in 1980s to 2.95 in the early 2000s
- * Caries prevalence increased from 43.2% to 59.5%
- * DC experience for permanent teeth at 12 yrs. of age increased from DMFT of 0.2 to DMFT of 0.92 over the same period.
- * Caries prevalence increased from 11.7% to 44.5% over the same period



Other risk factors predisposing to DC



- * Age: DC may occur at any age post – eruption of deciduous and permanent teeth
- * Advice mothers to start oral hygiene measures early before the tooth erupts.
- * Prevalence increases with age due to cumulative effects of the disease



Cont.



- * Sex: many studies have demonstrated that females have a higher DC prevalence than males. Also demonstrated by the 2015 Kenya National Oral Health Survey.
- * Why? Early eruption of teeth in girls & dietary habits.



Other



- * Familial/ hereditary factors – role of genetic factors.
- * Emotional disturbances – affecting mental health – high caries experience.
- * Ethnicity and race.
- * Culture & religion.
- * Socioeconomic factors.
- * Low level of parental education esp. the mother.



Cont.



- * Unemployment
- * Low family income
- * Single parent hood
- * Geographic factors
- * Deficient quantities of dietary microelements e.g. fluorides, calcium
- * Systemic illness
- * ISS
- * Diseases, drugs with manifestations of xerostomia



Clinical presentation



- * First clinical sign of the process of DC is a white spot/ white opaque area of the tooth surface (**incipient carious lesion → indicative of demineralization**)
- * With time, the lesion may become brown, grey or dark in color (uptake of proteins from drinks and feeds eaten)
- * If the lesion progresses, there is breakdown of the tooth surface forming a physical defect leading to formation of a cavity.



Symptoms



- * Mild to severe sensitivity due to thermal, chemical changes or tactile touch
- * Mild to severe pain esp. at night (may be due to changes in temp. or pressure)
- * Discoloration on the tooth surfaces
- * Cavities on tooth surfaces
- * Food sticking between teeth – proximal cavities
- * Resulting complications such as bleeding or swelling of gums, mandible, maxilla



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* In children

- * Anxiety, fear
- * Refusal to feed
- * Loss of sleep
- * Uncooperative behavior
- * Loss of attention



Diagnosis



- * Visual inspection (tooth must be clean & dry) look for:
 - * White opaque spots, brown, grey discolored areas
 - * Physical defects
 - * Discontinuity of tooth surface (breaching of enamel)
 - * Frank cavities with or without discoloration
 - * Food impaction between tooth



Cont.



- * Use of diagnostic tools
 - * Dental mirror & sickle shaped probe: The probe usually catches on a softened floor of a cavity (resists withdrawal)
- * Bitewing radiographs: DC appears as radiolucent lesions on one or several surfaces of a tooth. This is good for detecting cavities on proximal surfaces (between teeth) that are difficult to see
- * Exploration/ trial cavity: where clinical and radiographic examination fail to detect a cavity but symptoms persist.



Is dental caries life threatening?



- * May lead to complications that are life threatening:
 - * Cellulitis e.g. Ludwig's angina
 - * Death through e.g. airway obstruction, septicemia



Management



- * Incipient carious lesion with no cavitation: fluoride application to reverse the lesion
- * Removal of carious lesion and filling (radio – opaque on imaging) to:
 - * Repair the damage
 - * Restore physical appearance/ aesthetic
 - * Restore function
- * Root canal therapy and filling where there is pulpal involvement.



Cont.



- * Surgical intervention where there is associated pathological lesion e.g. periapical abscess with bone loss
- * Crowning of decayed tooth: artificial crown fabrication and fixation where there is marked destruction of the natural crown
- * Tooth extraction: when other methods of treatment are not possible, not accessible, not affordable, not available or on patient demand (last resort); it is becoming less common due to increased patient awareness on restorative tooth treatment modalities.



Prevention of DC



- *Basis of prevention: etiological model well established



Methods of prevention: communities based methods



1. Use of fluorides; the most effective means of DC prevention in community based programmes.

* Mechanism of fluoride action:

- * Reduction in susceptibility of tooth enamel to dissolution by acids
- * Interference with plaque bacterial metabolism and growth
- * Enhancement of remineralization: repair of early carious lesions



Cont.



* Methods of fluoride use

- * Fluoridation of public, school water supplies

- * Fluoridation of salt

- * Fluoridation of milk U.K, Chile, China, Russia → school milk programs for ages up to 6 yrs.



Cont.



* Limitations in Kenya

- * Variation in distribution of fluorides
- * Requires piped water supplies. Majority have no piped water in rural areas
- * Lack of adequate data on dietary sources of fluorides
- * Lack of official policy
- * Logistics and variation in consumption patterns e.g. in milk



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2. Promotion of health diet/ control of cariogenic diet

- * Food modification: substituting sucrose with non – cariogenic sugar sweeteners e/g/ xylitol, sorbitol
- * Legislation & regulation: aim. Control of production, labelling, advertising, marketing. Influence consumption patterns.



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3. Public health education: aim → inculcate better dietary habits

4. Bans/ restrictions: sale of cariogenic foods to vulnerable groups – school children



Limitations of control of cariogenic diet



- * National economic considerations
- * Monetary interests of powerful groups of manufacturers
- * Biological needs



Individual based methods



- * Use of fluorides
 - * Supervised fluoride use in children
 - * Fluoride tablets
- * Individual self care
 - * Fluoride tooth pastes in oral hygiene
 - * Fluoride mouth rinses 7 years or more
 - * Dietary control



TYPED BY NAILA KAMADI



Every time you smile at someone, it is an action of love, a gift to that person, a beautiful thing. 😊.

- Mother Teresa