ABSTRACT:
Dental caries, a chronic disease is unique among human and is one of the most common important global oral health problems in the world today. It is the destruction of dental hard acellular tissue by acidic by-products from the bacterial fermentation of dietary carbohydrates especially sucrose. It progresses slowly in most of the people which results from an ecological imbalance in the equilibrium between tooth minerals and oral biofilms which is characterised by microbial activity, resulting in fluctuations in plaque pH due to bacterial acid production, buffering action from saliva and the surrounding tooth structure. The microbial community of caries is diverse and contains many facultatively and obligately-anaerobic bacteria. S. mutans is the most primary associated with it. Dental caries can affect the human in various ways i.e. presence of tooth pain, infection or dysfunction of the stomatognathic system can limit the necessary ingestion of energetic foods, affecting the growth in children and adults as well as their learning, communication skills and recreational activities. Moreover, oral and pharyngeal cancers and oral tissue lesions are also significant health concern. Carious dentin can be life-threatening. Due to this, treatment is needed for dental diseases which cost is normally high and is not feasible for all community due to limited resources such as time, person and money. Therefore, prevention is more affordable. Personal hygiene cares and dietary modification should be recommended.

KEYWORDS: Dental caries, Fluoride, Pathogenesis, Streptococcus mutans, Cavernous sinus thrombosis, Oral Biofilm, Prevention.

INTRODUCTION:
Dental caries is one of the most common preventable diseases which is recognized as the primary cause of oral pain and tooth loss. It is a major public health oral disease which hinders the achievement and maintenance of oral health in all age groups [1]. WHO pointed that the global problem of oral disease still persists despite great improvements in the oral health of population in several countries. WHO claimed that poor oral health may have a profound effect on general health as well as quality of life, and several oral diseases are related to chronic diseases [2]. Dental caries refers to the localised destruction of susceptible dental hard tissues by acidic by-products from the bacterial fermentation of dietary carbohydrates. It is a chronic disease that progresses slowly in most of the people [3] which results from an ecological imbalance in the equilibrium between tooth minerals and oral biofilms (plaque) [4]. The biofilm is characterised by microbial activity, resulting in fluctuations in plaque pH. This is a result of both bacterial acid production and buffering action from saliva and the surrounding tooth structure. The tooth surface is therefore in a dynamic equilibrium with its surrounding environment. As the pH falls below a critical value, the demineralisation of enamel, dentine or cementum occurs, while a gain of mineral (remineralisation) occurs as the pH increases [3]. The process of demineralisation and remineralisation takes place frequently during the day. Over time, this process leads to either caries lesions or the repair and reversal of a lesion [6].

Primary caries can occur on different tooth surfaces. On an approximal surface, the lesion starts and forms beneath the contact area between teeth. Caries on an occlusal surface is also a localised phenomenon in pit and fissure. On both occlusal and approximal surfaces, enamel caries is a three-dimensional subsurface demineralisation that spreads along the enamel prisms. Secondary caries is a lesion located at the margin of a dental restoration. It represents a caries lesion adjacent to the margin and there may be signs of demineralisation (wall lesions) along the cavity wall which could be a consequence of microleakage. However, clinical and microbiological studies indicate that this leakage does not lead to active demineralisation beneath the restoration [7].

Caries may be characterized by the experience of pain, problem with eating, chewing, smiling and communication due to missing, discolored or damaged teeth [2]. The microbial community of caries is diverse and contains many facultatively and obligately-anaerobic bacteria belonging to the genera Actinomyces, Bifidobacterium, Eubacterium, Lactobacillus, Parvimonas and Rothia [8]. It can also be caused by other bacteria, including members of the mites, anginosus and salivarius groups of streptococci, Propionibacterium, Enterococcus faecalis, Scardovi, Prevotella,

doi: 10.15272/ajbps.v6i53.773

Conflict of interest: Authors reported none
Selenomonas, Dialister, Fusobacterium, Pseudoramibacter, Veillonella, Atopobium, Granulicatella, Leptotrichia and Thiomonas species are prevalent on mucosal surfaces and reach very high concentrations in dental plaque, gingival crevices and tonsillar crypts \[9,10,11,12\]. Bacteroides, Prevotella, and Porphyromonas species are prevalent on mucosal surfaces and reach very high concentrations in dental plaque, gingival crevices and tonsillar crypts \[9\]. At present, the distribution and severity of dental caries vary in different parts of the world and within the same country or region. It is affecting 60-90% of school children and the vast majority of adults. It is also a most prevalent oral disease in several Asian and Latin American countries. The prevalence pattern of dental caries varies with age, sex, socio economic status, race, geographical location, food habits and oral hygiene practices. Nowadays, as a consequence of high prevalence of dental caries, the treatment need is increased. However, treatment cost for dental diseases is normally high. In the United States annual treatment costs are estimated to be at least $ 4.5 billion \[13\].

**Global scenario of Dental caries**

Dental caries are the most important global oral health problems, although conditions such as oral and pharyngeal cancers and oral tissue lesions are also significant health concern \[14\]. Worldwide, approximately 2.43 billion people (36% of the population) have dental caries in their permanent teeth. In baby teeth it affects about 620 million people or 9% of the population. The disease is most prevalent in Latin American countries, countries in the Middle East, and South Asia, and least prevalent in China \[15\]. In the United States, dental caries is the most common chronic childhood disease, being at least five times more common than asthma \[16\]. It is the primary pathological cause of tooth loss in children \[17\]. Between 29% and 59% of adults over the age of fifty experience caries \[18\].

### Prevalence of dental caries in global scenario.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Age group</th>
<th>Prevalence (%)</th>
<th>Sample size</th>
<th>Country</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>5-9</td>
<td>50</td>
<td>1,598</td>
<td>USA</td>
<td>2004</td>
</tr>
<tr>
<td>2.</td>
<td>17</td>
<td>78</td>
<td>3,249</td>
<td>USA</td>
<td>2004</td>
</tr>
<tr>
<td>3.</td>
<td>6</td>
<td>97.1</td>
<td>4,050</td>
<td>Philippines</td>
<td>2006</td>
</tr>
<tr>
<td>4.</td>
<td>6-12</td>
<td>92.3</td>
<td>1,200</td>
<td>Philippines</td>
<td>2005</td>
</tr>
<tr>
<td>5.</td>
<td>2-6</td>
<td>59-92</td>
<td>993</td>
<td>Philippines</td>
<td>2003</td>
</tr>
<tr>
<td>6.</td>
<td>3-5</td>
<td>55</td>
<td>2,014</td>
<td>China</td>
<td>2007</td>
</tr>
<tr>
<td>7.</td>
<td>5-7-4</td>
<td>100</td>
<td>350,000</td>
<td>China</td>
<td>2008</td>
</tr>
<tr>
<td>8.</td>
<td>5</td>
<td>76</td>
<td>140,712</td>
<td>China</td>
<td>2002</td>
</tr>
<tr>
<td>9.</td>
<td>5-9</td>
<td>84</td>
<td>1,587</td>
<td>China</td>
<td>2001</td>
</tr>
<tr>
<td>10.</td>
<td>6</td>
<td>89.4</td>
<td>178</td>
<td>Taiwan</td>
<td>2006</td>
</tr>
<tr>
<td>11.</td>
<td>1-6</td>
<td>52.9</td>
<td>981</td>
<td>Taiwan</td>
<td>2006</td>
</tr>
<tr>
<td>12.</td>
<td>0-5</td>
<td>40</td>
<td>1,487</td>
<td>Brazil</td>
<td>2007</td>
</tr>
<tr>
<td>13.</td>
<td>1-2.5</td>
<td>20</td>
<td>186</td>
<td>Brazil</td>
<td>2007</td>
</tr>
<tr>
<td>14.</td>
<td>12</td>
<td>53.6</td>
<td>1,151</td>
<td>Brazil</td>
<td>2004</td>
</tr>
<tr>
<td>15.</td>
<td>7-9</td>
<td>78.5</td>
<td>121</td>
<td>Argentina</td>
<td>2006</td>
</tr>
<tr>
<td>16.</td>
<td>6-12</td>
<td>90.2</td>
<td>3,048</td>
<td>Mexico</td>
<td>2006</td>
</tr>
<tr>
<td>17.</td>
<td>6-9</td>
<td>34.7</td>
<td>452</td>
<td>Mexico</td>
<td>2006</td>
</tr>
<tr>
<td>18.</td>
<td>8</td>
<td>50</td>
<td>5,580</td>
<td>UK</td>
<td>2003</td>
</tr>
<tr>
<td>19.</td>
<td>12</td>
<td>59.8</td>
<td>48,168</td>
<td>Norway</td>
<td>2006</td>
</tr>
<tr>
<td>20.</td>
<td>12</td>
<td>86</td>
<td>117</td>
<td>Armenia</td>
<td>2005</td>
</tr>
<tr>
<td>21.</td>
<td>7-9</td>
<td>78.5</td>
<td>121</td>
<td>Peru</td>
<td>2006</td>
</tr>
</tbody>
</table>

(Adams A; et al., 2009) \[19\].

### Prevalence of dental caries in National scenario.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Age group</th>
<th>Prevalence (%)</th>
<th>Sample size</th>
<th>District</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>5-6</td>
<td>23.23</td>
<td>638</td>
<td>Bhaktapur</td>
<td>2007-2008</td>
</tr>
<tr>
<td></td>
<td>12-13</td>
<td>50.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>5-6</td>
<td>63.64</td>
<td>638</td>
<td>Lalitpur</td>
<td>2007-2008</td>
</tr>
<tr>
<td></td>
<td>12-13</td>
<td>41.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>5-6</td>
<td>74.46</td>
<td>638</td>
<td>Kathmandu</td>
<td>2007-2008</td>
</tr>
<tr>
<td></td>
<td>12-13</td>
<td>57.06</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>5-7</td>
<td>20.7</td>
<td>376</td>
<td>Pokhara</td>
<td>2012</td>
</tr>
<tr>
<td>8-10</td>
<td></td>
<td>48.2</td>
<td>1174</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-14</td>
<td></td>
<td>52.46</td>
<td>1624</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>5-6</td>
<td>52</td>
<td>361</td>
<td>Chitwan</td>
<td>2013</td>
</tr>
<tr>
<td>6.</td>
<td>1-6</td>
<td>85.2</td>
<td>392</td>
<td>Kathmandu</td>
<td>2013</td>
</tr>
</tbody>
</table>

(Subedi B, et al., 2011; Khanal S, et al., 2013; Adhikari RB, et al., 2012) \[20,21,22\].
Types of dental caries
Different types of caries are found which are as follows.

Table 3. Types of dental caries

<table>
<thead>
<tr>
<th>Types of caries / Primary caries</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incipient caries / Primary caries</td>
<td>Decay at a location that has not experienced previous decay.</td>
</tr>
<tr>
<td>Recurrent caries / Secondary caries</td>
<td>Appears at a location with a previous history of caries and is frequently found on the margins of fillings and other dental restorations.</td>
</tr>
<tr>
<td>Arrested caries</td>
<td>A lesion on a tooth that was previously demineralized but was remineralized before causing a cavitation</td>
</tr>
</tbody>
</table>

(Sonis ST 2003) [23],

Early childhood caries

Early childhood caries (ECC) is a pattern of decay found in young children with their deciduous teeth. The teeth most likely affected are the maxillary anterior teeth, but all teeth can be affected [24]. This type of caries comes as a result of allowing children to fall asleep with sweetened liquids in their bottles or feeding children sweetened liquids multiple times during the day [24]. The risk for ECC also may be determined by pre-existing developmental defects of the enamel called hypoplasia. Hypoplasia predisposes teeth to early colonization by Streptococcus mutans and malnutrition.

ECC exhibits a characteristic pattern related to the emergence sequence of the teeth and the tongue position during feeding. The lower teeth are protected from exposure to ingested liquids by the tongue during feeding and by the pooling of saliva and so usually are not affected. The incisors are the first upper teeth to emerge and are most affected by ECC. Depending on how long the caries process is active, the upper first primary molars are often involved, followed by the upper second molars and canines, and in severe cases, the lower teeth [25].

Rampant caries

Rampant caries are severe decay on multiple surfaces of many teeth [26]. It may be seen in individuals with xerostomia, poor oral hygiene, stimulant use due to drug-induced dry mouth and or large sugar intake [24]. If rampant caries is a result of previous radiation to the head and neck then it is called as radiation-induced caries. Problems can also be caused by the self-destruction of roots and whole tooth resorption when new teeth erupt [26].

Classification of dental caries

Caries can be classified by rate of progression, affected hard tissues and location. These forms of classification can be used to characterize a particular case of tooth decay in order to more accurately represent the condition to others and also indicate the severity of tooth destruction [23].

Table 4. Classification of dental caries

<table>
<thead>
<tr>
<th>S.No.</th>
<th>On the basis of</th>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate of progression</td>
<td>Acute</td>
<td>Signifies a quickly developing condition</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic</td>
<td>Signifies an extended time to developing condition</td>
</tr>
<tr>
<td></td>
<td>Affected hard tissue</td>
<td>Enamel</td>
<td>Early in its development and may affect only enamel.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dentinal</td>
<td>The extent of decay reaches the deeper layer of dentin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cementum</td>
<td>The decay on roots of teeth</td>
</tr>
<tr>
<td></td>
<td>Location (G.V. Black)</td>
<td>Class I</td>
<td>Pit and fissure caries (anterior or posterior teeth)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Class II</td>
<td>Approximal surfaces of posterior teeth</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Class III</td>
<td>Approximal surfaces of anterior teeth without incisal edge involvement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Class IV</td>
<td>Approximal surfaces of anterior teeth with incisal edge involvement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Class V</td>
<td>Gingival/cervical surfaces on the lingual or facial aspect (anterior or posterior)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Class VI</td>
<td>Incisal edge of anterior teeth or cusp heights of posterior teeth</td>
</tr>
</tbody>
</table>

Figure 1. Caries model by Fisher-Owens and co-workers
Caries aetiology
Historically, researchers have focused on biological and dietary effects on children’s oral health to explain caries development. In recent years, children’s oral health outcomes using a broader framework, which incorporates psychosocial and environmental predictors as well as the biological and dietary effects [27]. These frameworks generally classify conditions associated with disease into five broad domains: genetics and biology, social environment, physical environment, health influencing behaviors and medical care [27]. These relevant variables explain why some children, despite use of fluoride and abundant information about caries prevention, develop carious lesions. The caries model by Fisher-Owens and co-workers includes different levels of the environment that can affect caries development: child-level; family-level; and community-level.

Child-level
Visible plaque, early colonisation by caries-related bacteria, the presence of mutans streptococci, frequent intake of sweetened drinks, infrequent tooth brushing, illness and use of antibiotics have all been associated with caries developments in preschool children [28].

Family-level
Family level characteristics associated with caries risk in children included are demographic factors of the family, parental oral health behaviors and attitudes, dental anxiety and dental attendance, maternal health and lifestyle in pregnancy and early childhood [29].

Community-level
Children’s oral health is likely to be better in a community that values good oral health [30]. Cultural aspects and the neighbourhood may have implications for caries development [28]. The dental care system and amount of dental care available may affect oral health and the development of caries in preschool children [29].

Caries-promoting factors

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Primary risk factors</th>
<th>Reasons</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Saliva</td>
<td>(1) Ability of minor salivary glands to produce saliva (2) Consistency of unstimulated (resting) saliva (3) pH of unstimulated saliva (4) Stimulated salivary flow rate (5) Buffering capacity of stimulated saliva</td>
</tr>
<tr>
<td>2.</td>
<td>Diet</td>
<td>(6) Number of sugar exposures per day (7) Number of acid exposures per day</td>
</tr>
<tr>
<td>3.</td>
<td>Fluoride</td>
<td>(8) Past and current exposure</td>
</tr>
<tr>
<td>4.</td>
<td>Oral biofilm</td>
<td>(9) Differential staining (10) Composition (11) Activity</td>
</tr>
<tr>
<td></td>
<td>Modifying factors</td>
<td>(12) Past and current dental status (13) Past and current medical status (14) Compliance with oral hygiene and dietary advice (15) Lifestyle (16) Socioeconomic status (17) Tooth location</td>
</tr>
</tbody>
</table>

(Islam B et al 2007) [32]

Pathogenesis of dental caries
The classic description of the cause of dental caries includes three factors: host, bacteria and diet. Dental caries occurs when a susceptible tooth surface is colonized with cariogenic bacteria and dietary source of sucrose or refined sugar is present. Bacterial pathogen produced lactic acid from fermentation of carbohydrates and this acid dissolves the hydroxyapatite crystal structure of the tooth which causes caries [32].

![Figure 2. Flowchart of pathogenesis of dental caries](image)

The sucrose connection related to caries
Sucrose is a major environmental contributor to dental caries because of the many oral streptococci possess extracellular enzymes capable of cleaving the α-1 and α-2 glycosidic bond of sucrose and harnessing the energy to yield glucose polymer (glucans and mutans) and fructose. This group of enzymes called the glucosyltransferases (GTFs), accounts for this special relationship between sucrose and caries. Formation of glucan-mutan polymer allows the cariogenic bacteria to accumulate into biofilm to form a critical mass. Without formation of critical mass cariogenic bacteria would colonize the oral cavity but not be massed so as to cause the destruction of the enamel surface. Accordingly the formation of critical mass that is uniquely associated with glucosyltransferases and sucrose is a biological reason for man’s recent affliction with caries [33].

Stages of dental caries

**White spot stage**
The acid produced by bacteria and yeast in dental plaque dissolve the mineral matrix of teeth. In the earliest stage, dental caries appears as a chalky white spot on the tooth. At this stage, the surface is intact, and the subsurface lesion is reversible. White spot resulting from incipient caries can be difficult to distinguish from developmental hypocalcification. Further, white spot changes to black staining stage [32].

![Figure 3. Black staining stage](image)
If mineral continues to be lost because of acid challenge, the surface is eventually broken or “cavitated” and the lesion cannot be reversed. If the lesion progresses, larger areas of tooth can be lost. Active cavitated lesions are usually golden brown. Long standing lesions are darker, sometimes nearly black. Depth of the color is not a good indicator of the severity of the lesions because arrested decay is often the darkest [32].

Pathophysiology

Enamel

Demineralization of enamel by caries follows the direction of the enamel rods, the different triangular patterns between pit and fissure and smooth-surface caries develop in the enamel [34]. As the enamel loses minerals, the enamel develops several distinct zones: translucent zone, dark zones, body of the lesion, and surface zone [35]. The translucent zone coincides with 1/2% loss of minerals [36]. Dark zone is slight remineralization of enamel. The greatest demineralization and destruction is in the body of the lesion. The surface zone remains relatively mineralized until the loss of tooth structure results in a cavitation [37].

Dentine

In dentine from the deepest layer to the enamel, the distinct areas affected by caries are the advancing front, the zone of bacterial penetration, and the zone of destruction [34]. The advancing front represents a zone of demineralised dentine due to acid and has no bacteria present. The zones of bacterial penetration and destruction are the locations of invading bacteria and ultimately the decomposition of dentin. The zone of destruction has a more mixed bacterial population where proteolytic enzymes have destroyed the organic matrix [36].

Cementum

The incidence of cemental caries increases in older adults as gingival slump occurs from either trauma or periodontal disease. It is a chronic condition that forms a large, shallow lesion and slowly invades first the root’s cementum and then dentin to cause a chronic infection of the pulp [38].

Signs and symptoms of dental caries

The signs and symptoms of cavities vary, depending on their extent and location. When a cavity is just beginning and may not have any symptoms at all. As the decay gets larger, it may cause signs and symptoms such as:

- Toothache and mild to sharp pain when eating or drinking something sweet, hot or cold called tooth sensitivity [39].
- Visible holes or pits in teeth [40]
- Brown, black or white staining on any surface of a tooth [40]
- Bad breath and foul tastes [41]
- Fever, chills, abscess, and trismus (www.rightdiagnosis.com).

Complications

- Cavernous sinus thrombosis and Ludwig angina can be life-threatening [42]
- Toothache, pulpitis, tooth loss and dental discoloration (www.rightdiagnosis.com)

Diagnosis of dental caries

Primary diagnosis

Initially it may appear as a small chalky area (smooth surface caries) which may eventually develop into a large cavitation. Inspection of all visible tooth surfaces using a good light source, dental mirror and explorer. Dental radiographs (X-rays) are used for less visible areas of teeth in particular caries between the teeth. Lasers without ionizing radiation also now used for detection of interproximal decay (between the teeth). Visual and tactile inspection along with radiographs are employed frequently among dentists, in particular to diagnose pit and fissure caries [43]. Early, uncavitated caries is often diagnosed by blowing air across the suspect surface, which removes moisture and changes the optical properties of the unmineralized enamel [44].

Differential diagnosis

Dental fluorosis and developmental defects of the tooth including hypomineralization of the tooth and hypoplasia of the tooth are used for dental caries [44].

Treatment

The goal of treatment is to preserve tooth structures and prevent further destruction of the tooth. Most importantly, whether the carious lesion is cavitated or noncavitated dictates the management.

Noncavitated lesions can be arrested and remineralization can occur with extensive changes to the diet i.e, reduction in frequency of refined sugars [45]. It can be treated with non-operative method by tooth remineralization.

Tooth remineralization

Tooth remineralization is a process in which minerals are returned to the molecular structure of the tooth itself. Destroyed tooth structure does not fully regenerate, although remineralization of very small carious lesions may occur if dental hygiene is kept at optimal level such as toothbrushing twice per day with fluoride toothpaste and flossing, and regular application of topical fluoride. Such management of a carious lesion is termed “non-operative treatment” [44].

Cavitated lesion, especially if dentin is involved, remineralization is much more difficult and a dental restoration is usually indicated. Such management of a carious lesion is termed “operative treatment”.

Dental restoration

A dental restoration or dental filling is a process in which dental restorative material (including dental amalgam, composite resin, porcelain, and gold) is used to restore the function, integrity and morphology of missing tooth structure. Composite resin and porcelain can be made to match the color of a patient’s natural teeth and are more frequently used [41]. Local anesthetics, nitrous oxide ("laughing gas"), or other prescription medications may be required in some cases to relieve pain during or following treatment or to relieve anxiety during treatment [41].

Tooth extraction

The removal of the decayed tooth is performed if the tooth is too far destroyed from the decay process to effectively restore the tooth [46].

Others measures

Dental sealants

A sealant is a thin plastic-like coating applied to the chewing surfaces of the molars to prevent food from being trapped inside pits and fissures [47].

Prevention and control

Oral hygiene

Personal hygiene care consists of proper brushing and flossing daily [41]. Proper brushing and flossing is to remove and prevent the formation of plaque or dental biofilm. Professional hygiene care consists of regular dental examinations and professional prophylaxis (cleaning) [46].

Dietary modification

Minimizing snacking is recommended, since snacking creates a continuous supply of nutrition for acid-creating bacteria in the mouth. Chewy and sticky foods (such as dried fruit or candy) tend to adhere to teeth longer, brushing the teeth after meals is recommended. For children, the ADA and the EAPD recommend limiting the frequency of consumption of drinks with sugar, and not giving baby bottles to infants during sleep. Chewing gum containing xylitol (a sugar alcohol) helps in reducing dental biofilm [48].

Calcium and fluoride

Calcium is found in food such as milk and green vegetables, is often recommended to protect against dental caries. Fluoride helps prevent decay of a tooth by binding to the hydroxyapatite crystals in enamel [37]. The incorporated calcium makes enamel more resistant to demineralization and, thus, resistant to decay [40]. Topical fluoride include a fluoride toothpaste or mouthwash or varnish is now more highly recommended than systemic intake such as by tablets or drops to protect the surface of the teeth. After brushing with fluoride toothpaste, rinsing should be avoided. Fluoride have pre-eruptive and post-eruptive effects on caries prevention [50].

CONCLUSION:
The present review documents several risk factors of dental caries which had numerous interventions to prevent caries. Since extensive damage from caries can lead to major problems for the individual, affecting quality of life both functionally and esthetically. Increasing the awareness and knowledge about dental caries in general can increase their knowledge and skills in oral health care. Having the ability to identify potential health risk factors such as lifestyle, ethnicity, health status, and social determinants associated with oral health status risk, health care providers can take an active role in health screening to discover any need for clinical preventive services, including dental preventive services, and can detect health problems. With proper knowledge and oral health behavior, health care professionals can play an important role in the oral health education of individuals and groups and act as role models for patients, friends, families and the community at large. Good general health also includes good oral health. Hence, preventing caries is an important element in public health efforts. Personal hygiene cares (proper brushing with fluoride tooth paste and flossing daily) and dietary modification (minimizing snacking, chewing gum, milk and green vegetables) should be recommended. Raising public awareness about dental check-up may assist in early diagnosis.

REFERENCES

22. Adhikari RB, Mall N and Bhandari PS. Prevalence and treat-
40. Richie SK. An incipient carious lesion is the initial stage of structural damage to the enamel, usually caused by a bacterial infection that produces tooth-dissolving acid. The New York Times. 2011; pp 23.