CHAPTER III

REVIEW OF LITERATURE
Epidemiological concepts had their beginning many years ago. More than 400 years before Christ, Thucydides wrote of his belief in observing facts and judging by rule of reason and pointed to his observations relating to immunity during the plague of Athens. At least one basic concept has persisted from this early beginning down to the present time—that is, concept that epidemiology is concerned with groups of people. This concept is implied in the roots of the word which mean "cn, upon, or to the people". Epidemiology's concern with groups of people is apparent in the following generally accepted definition of epidemiology: "This is the field of science which is concerned with the various factors and conditions which determine the occurrence and distribution of health, disease, defect, disability and death among aggregations of individuals; it has application also to problem other than those of health and disease".

Epidemiologic principles and methods currently are being applied not only to communicable disease but also to other disorders "on, upon, or to the people" such as: nutritional deficiencies; accidents; congenital defects; mottled enamel; periodontal disease and dental caries.
These disorders are a result of imbalance between a causative agent and man. The extent of imbalance depends on the characteristics of the agent and of man, both of which are in turn controlled by the environment. Thus, within the agent the host and the environment are all of the factors which affect the occurrence and progression of the disease or disorder. The combination of the processes which upset the balance between agent and host and initiate and perpetuate a disease is termed the natural history of the disease.

3.1 AETIOPATHOGENESIS OF DENTAL CARIES:

LEBER and ROTTENSTEIN (1867) reported that presence of micro-organism and the production of acid which dissolved the inorganic constituents of the tooth were the two principle phenomena in the disease process. TOMES (1873) UNDERWOOD & MILLES (1881) also supported this view. MILLER (1890) in a series of studies showed that some oral bacteria were capable of producing acids from certain foods and that the acid formed could decalcify sound dentine. He also observed that other groups of oral micro-organism were capable of liquefying demineralized dentine.

EASLICK (1948) reported that dental caries is a disease of the calcified tissues of the teeth caused by acids resulting from the action of micro-organisms and carbohydrates. It is characterised by a decalcification of the inorganic portion and is accompanied or followed by disintegration of the organic
substance of the tooth. The effect of the carious process on the tooth is made possible by circumstances or structures which retain sufficient acid in contact with the tooth substance including

1. The dental plaque
2. The anatomical characteristics of the tooth
3. The position or arrangement of the teeth in the dental arch, and
4. The presence of dental appliances.

THE ROLE OF BACTERIA IN THE PRODUCTION OF DENTAL CARIES:

Three hundred years ago Anthany Van Leewenhock ground a dual convex lens and scrapped some material from the mouth of an eight year old boy. This was the first view of the bacterial world occupies the oral cavity. Caries results from the interaction of three factors, the tooth, the environmental conditions and Bacteria. (KITE at al., 1950)

ORLAND at al., 1954 maintained caries susceptible rates on a cariogenic diet under germ free environment for 150 days. No carious lesions developed in germ free animals. ORLAND et al., 1955 reported that the oral inoculation of a group of the germ free rates with a culture of an enterococcus resulted in the production of typical carious lesion. FITZGERALD (1963) showed that dental caries in the rate does not occur in the absence of micro-organisms, but can occur in the presence of a single type of streptococcus which need not to be proteolytic.
In hamsters KEYES (1962) has shown that if breeding females of a caries susceptible strain are fed a stock diet containing penicillin, their offspring remain essentially free of caries when fed on experimental diet high in carbohydrate and low in fat.

Proof of the causal role of bacteria was incomplete and depended on circumstantial evidence such as the reduction in the caries incidence when the oral flora was interfered with by mechanical cleaning of teeth, chemicals or antibiotics HANKE, 1940, ROSEN at al., 1956, MCCLURE and HEWITT, 1946).

Two main mechanisms of bacterial activity in the production of caries were postulated
1. Attack on the inorganic constituents of the tooth by acid products of bacterial fermentation and

GOADLY (1903), BURNETT and SCHERP (1951) carried out a comprehensive survey of these two groups of bacteria in carious dentine and found both groups in the superficial layers of the carious tissues. In the deeper parts, however, where the lesion was advancing the bacteria were all of the acid forming type, staphylococci, strepto cocci and Lactobacilli.

Much emphasis was then placed on the Lactobacilli which, because of their highly aciduric and acidogenic nature,
frequently carious than the permanent second molar because of its longer exposure in oral cavity. Stones compared the D.M.F. rates with the post eruptive tooth age values and stated that the 28 permanent teeth (Third molars excluded) fell into five classes of caries susceptibility. These are as follows in order of descending susceptibility.

1. Mandibular first molars and second molars.
2. Maxillary first molars and second molars.
3. Mandibular second premolars, maxillary first and second premolars and maxillary first and second incisors.
4. Maxillary canines and mandibular first premolars.
5. Mandibular first and second incisors and canines.

PARFITT (1955) has confirmed and findings and adds that in children below the age of 8 years the occlusal surfaces of the deciduous second molars are the most common sites for caries, while above this age the occlusal surfaces of the permanent first molars become the most commonly affected.

Thus the molar teeth are the most susceptible and this is probably due to their deep fissures and posterior position which favours food stagnation. The relative immunity of the mandibular front teeth is probably due to the continual flushing with the saliva.
could produce a high level of acidity (KLIGLER, 1915) Capable of
demineralizing enamel in vitro (MC INTOSH et al. 1922) and
which are found in practically every lesion of active dental
caries (BUNTING, 1930; HADLEY, 1933; JAY, 1947). HEMMENS et al.,
1946 showed that lactobacilli were isolated with greater frequency
from plaques on human teeth during the period when caries is
active.

HADLEY (1933), DIAMOND (1950) made attempts to
estimate caries activity by plate counts of lactobacilli in saliva.
The usefulness of the lactobacilli count as a measure of caries
activity remains controversial having been both supported
(WINKLER and DIRTES, 1946; KITCHIN and ROBINSON, 1948) and
denied (WHYTE, 1943a & b; SPEIDEL et al., 1939, BOYED et al.,
1949 GLASS, 1952) in varying degree.

In these earlier studies oral lactobacilli were usually
Lactobacillus acidophilus or Lactobacillus oranslyticus but more
recent studies of the various species of Lactobicilli found in
oral cavity (MORRIS, 1953; ORLAND, 1953; ROGOSA et al., 1953;
DAVIS, 1955; 1956; HAYWARD, 1957; and BAIRD-PARKER et al.,1958).

STEFHAN and HEMMENS (1947) pointed out that
streptococci, staphylococci, neisserias, yeasts, actinomyces and
diphtheroids are all capable of lowering the pH of a carbohydrate
medium to 5.0 or less and HOWELL et al., 1962 have reported
Actinomyces israeli to be present in early carious lesions and
in deep cavities of children with rampant caries. Streptococci are much more numerous in saliva and in both pre-carious and carious plaques than are Lactobacilli. The total number of streptococci in plaques has not been shown to increase during caries, as was the case with lactobacilli, but the more aciduric streptococci are more numerous in carious than in non carious plaques.

Snyder (1941) devised and other bacteriological test for caries activity based on the total acid producing potentiality of the oral flora. This is carried out by adding 6.1-0.2 ml of saliva to melted glucose infusion agar where the acid potential of the saliva is shown by the speed with which the color of brom-cresol green indicator changes to yellow in the medium.

3.1.1 Acidity Required to Decalcify Teeth:

The pH necessary to effect fairly rapid demineralization of the teeth has been reported variously as 4.0 (Mc Intosh, et al., 1922); 4.5 (Hoff and Keszey, 1937) and 5.2 (Klinger, 1938). Demineralization of tooth substance by fluids containing citrates has been demonstrated by Mc Clure and Ruzicka (1946) at pH between 5.5 and 7.2, at which nearly neutral pH a soluble calcium citrate complex is formed.

The acids concerned in caries are produced by the action of bacteria on food debris lodging around the teeth.
Protein and fat are little concerned in the process, the most important bacterial substrate being the carbohydrate portion of the food. Cellulose and uncooked starch do not undergo degradation in the mouth though cooked starch may be acted upon by the salivary amylase, ptyalin with the production of maltose. Disaccharides (e.g. sucrose, maltose), either formed from starch or eaten as such in the diet are first hydrolysed by bacterial enzymes to monosaccharides (e.g. glucose, fructose) and then further degraded to yield a mixture of simple organic acids of which lactic acid has been the one most frequently named, though FOSDICK and WESSINGER (1937) have drawn attention to the importance of Pyruvic acid. NEUWIRTH and SUMMERSAN (1951) reported that these two acids probably do not account for more than one half to two thirds of the total acid production and that other acids including acetic, lutyric and propionic acids are also produced as end products of the action of bacteria on glucose.

The acid when formed are buffered by the saliva which usually has a pH of 6.5 to 6.9, and this is not acid enough to dissolve the enamel.

The pH of the plaque has been examined, using a micro-colorimetric technique, by ELKERINGTAN and TRIMBLE (1934) and STEPHEN (1940).

STEPHEN (1940) found that in 211 plaques the pH
varied from 4.6 to 7.0 with an average of 5.9; also the average pH of debris in 38 carious lesion was 5.2. STEPHAN (1944) found that in every plaque there was an increase in acidity, the extent and duration of which was most marked in cases with extensive caries activity. A pH lower than 5.0 occurred only if there was caries activity and the fall in the pH was more marked on the surfaces of the maxillary anterior teeth than on the mandibular anterior teeth. This finding has been confirmed by ENGLANDER et al., 1954, who observed no pH of 5.5 or below in "Caries immune" individuals.

FOSDICK (1949) suggested that the plaque could act as a semipermeable membrane so that solutions of sugar which have a high osmotic pressure would pass through the plaque and be retained for considerable periods in contact with the tooth surface. Here they could become converted in acid by the micro-organisms directly in contact with the tooth surface before becoming diluted by saliva or neutralized by the strong buffering capacity of the dental plaque.

These findings suggest that at localised areas such as in plaques, a sufficient acidity can be reached to cause demineralization of the tooth substance.

3.2 DENTAL PLAQUE:

One of the prime culprits of dental caries is plaque—a soft, adherent collection of salivary products and bacterial
colonies on the teeth (WILLIAMS, 1897) It accumulates on the surface of the teeth continuously throughout the life span of most of the people in varying degree. The only hope a patient has in eliminating this disease producing material is to continually remove it by toothbrushing and dental flossing.

Plaque growth begins approximately 6 hrs. after a thorough cleaning of teeth. The first phase of plaque development is the deposition of the adherent products from the saliva. These products are primarily composed of mucin, which forms a thin adherent layer on the teeth called the pellicles. Once the pellicle has been deposited on the clean tooth surface, bacteria that inhibit the oral cavity attaches themselves to the pellicle. After attachment, the bacteria multiply to form large masses of bacterial colonies. This begins to occur approximately 18 hrs. after a thorough cleaning of teeth and continues until the plaque is fully matured by the end of 3 weeks.

The mature plaque is an accumulation of material consisting of mucin, desquamated epithelial cells, food debris and many varieties of bacteria. STARLFARS (1950) has estimated that there are more than 400 millions micro-organisms per mg. of plaque material, bacteria this accounting for at least 70% of the plaque by volume (WINKLER et al., 1958). Some bacteria produces harmful chemical substances and other produces substances that are needed by neighbouring bacteria to survive.
Still other organisms produces adherent substances that are interspread with the bacteria and hold the plaque intact on the tooth surface. Additional minor component of plaque includes salivary mucin, dead epithelial cells and food debris.

Saliva contains 0.05 to 0.6% of mucoid (KNOX 1953), which has the property of forming a thin film over all the surfaces of the mouth. On those areas of the teeth liable to stagnation of food, bacteria and food particles will adhere to this film. Any local production of acid will then cause a further precipitation of mucoid in this area (KEISER-NIELSON 1946). The layer will thus increase in thickness and more food debris and bacteria will be incorporated. Plaque will therefore be thicker in areas where acid production is favoured by food retention over smooth surfaces which are cleaned naturally (Mo DONGAL, 1963) Plaque is very tenacious and can not easily be removed with a toothbrush, and is formed anew very quickly after instrumental cleaning (HAUKE, 1940).

Streptococcus mutans is one of the organism which attaches to the pellicle and multiply. The streptococcus are capable of producing both polysaccharides and acids from carbohydrates, that are consumed by the patient. This is important because polysaccharides help to attach streptococcus to the pellicle. The acid they produce is capable of demineralizing the enamel layer of the tooth. The demineralization is the first stage of Dental caries.
The plaque, because of its thickness and density, protects acid produced within it from being either diluted by saliva or neutralised by chemicals present in saliva. So acid remains concentrated adjacent to the tooth surface and is able to break down the enamel more quickly.

Another view is that plaque is concerned with proteolysis of the organic fraction of the enamel (BODECKER 1948).

3.3 EFFECT OF CARBOHYDRATE:

Basic relationship can be explained by use of following formulas.

1. Carbohydrate + Bacteria = Acids
   (Dental Plaque)

2. Acids + Susceptible tooth structure = Decay

Much of the evidence for the importance of carbohydrates in the etiology of human caries is circumstantial. Studies using caries susceptible rats have demonstrated conclusively that in this animal caries does not occur in the absence of fermentable carbohydrates from the mouth. (KITE et al. 1950, HALDI, et al. 1953) WINN et al 1953 also observed that sucrose fed in solution was much less cariogenic than when fed in solid form.
KAMRIN (1954), SHAH (1954) found that when caries susceptible rats were fed for long periods on diets free from carbohydrate, carious lesions did not develop even after surgical removal of the salivary glands. The results of surveys and clinical investigations on human caries are, in general, are in support of these findings.

BUNTING (1934), and BUNTING et al. (1936) examined children in institutions where the diet could be controlled and modified. Some of the diets were found to be deficient in the essentials required by modern standards. It was reported that a restriction of sugar reduced the lactobacillus acidophilus count in the mouth considerably, and that dental caries that was previously present became inactive over a period of several years even though some of the cavities were large. Few new cavities developed when candy was given in unrestricted amounts, the lactobacillus counts increased and active dental caries developed in a few months.

BECKS et al. (1944) and BECKS (1950) by lessening the intake of refined carbohydrates have obtained similar results, reducing the lactobacillus counts and the caries incidence. As in Jay's studies the caloric value of the diet was maintained by an increased intake of meat, eggs, vegetables, milk and milk products.

During world war II the amount of sugar consumed
in many European countries was considerably curtailed and there have been various reports of a subsequent reduction in the incidence of dental caries. (SOGNNAES 1948; TOVERUP 1949). This reduction was least marked in the older children whose teeth had begun to develop before the war and was most marked in the younger children whose teeth developed during the war. In the immediate post-war period the individual consumption of sugar doubles between 1944 and 1948 yet the incidence of dental caries for the children in the same age group was only slightly higher in 1948. SHAW (1950 d) in an excellent review suggests that these findings indicate that there are important developmental factors as yet unknown that considerably influence the susceptibility of the teeth to caries.

Other studies have shown that not only the total carbohydrate intake, but also other factors including the frequency of administration and the retention of the carbohydrate around the teeth are important. GUSTAFSON et al (1954) added three times the normal amount of sugar to the diet of 436 inmates of a mental hospital and found no corresponding increase in caries incidence when the sugar was administered only during meals. However, when half the sugar was fed as sticky candy between the meals, caries activity rose sharply and then immediately subsided when the candy was withdrawn from the diet. One group of 60 inmates received a carbohydrate-poor & high fat diet practically free from refined sugar. A slight
keto-urea was produced in some patients and caries activity was almost entirely suppressed. In an investigation in Great Britain, twice and three times the normal amount of sugar was incorporated into the diet of institutional children of various age. After one to two years there was no significant difference between the increase in caries in control and experimental groups (KING, et al. 1955).

It has been suggested that naturally occurring sugars are less likely to produce caries than the refined sugars. Thus STANNUS (1917) reports that among certain of the Bantu races in South Africa who regularly suck sugar cane there is little caries. WAAGH (1940) has studied the effects of natural and refined sugars on oral lactobacillus and caries among primitive Eskimos and finds that natural sugars in the diet do not initiate or cause an increase in dental caries, but refined sugars initiate caries in over 70% of cases previously caries free. On the other hand DREIZEN and SPIES (1952) find a high caries incidence in cubans who habitually chew sugar canes.

Some research has indicated that certain chemicals contained in the saliva actually help in repair or remineralize softened enamel. This phenomenon occurs in between meals. Snacking between meals not only adds to the frequency of acid production by bacteria but also interferes with the process
of remineralization by the saliva. Thus between the meals snacking with foods containing sugars has a two fold negative effect. If a patient insists on snacks between the meals, it should be of sugar free foods.

One simple method of reducing the frequency of acid production is to limit eating three meals a day. If sweets are eaten, they should be consumed at meal-time.

3.4 SALIVA AND DENTAL CARIES:

The saliva has an acid neutralizing power and a base neutralizing power. Therefore it acts as a buffer. This buffering capacity is mainly due to the phosphates, bicarbonates and proteins in the saliva. ERICSSON (1962) studied the influence of dietary proteins, carbohydrates and vegetables on salivary buffering in normal subjects. Proteins and vegetables were found to increase, carbohydrates to decrease the buffering action. DREIZEN et al. (1946) reported that individuals relatively free from caries have the highest buffering capacity. LILIENHAL (1955) has confirmed these results and in addition has shown a direct relationship between the volume of saliva secreted in unit time and its buffering effect. KRASSE and GUSTAFSON (1958) found significantly higher buffer values in stimulated saliva collected at mealtime than in saliva between meals.

RAE and CLEGGE (1949) find no evidence of positive
correlation between the buffering capacity and salivary lactobacillus count. Clements and Rae (1953) report a high buffering capacity in a sample of Bakalogadi children and bushman who have little caries. Hartles and McLean (1953) confirm previous observations that lactic and other acids are produced rapidly in the mouth after a glucose rinse and that there is a rapid removal mechanism. They consider that if a high acid production concides in the same subject with a low buffering capacity, the acid formed will not be neutralized, but that with a low acid production and high buffering capacity the acid will be neutralized.

In severe diminution of salivary flow, however, as may occur unilaterally following irradiation of the parotid gland area there is a greatly increased caries activity on the affected side. Similarly the caries incidence in susceptible rats maintained on a cariogenic diet is greatly increased following surgical removal of the major salivary glands.

Another point to be considered is the steadily increasing number of individuals (trying to keep up with the fast pace of life) who are being compelled to take medication producing a sharp drop in saliva secretion eg. sleeping tablets, oral nasal drops, and the betablockers used in hypertensive patients. A decrease in salivary secretions primarily entails the risk of more wide spread caries. Fibre rich, well prepared
food which smells good and looks appetizing should be the first line of action in attempting to stumulate saliva.

3.5 PREVALENCE OF DENTAL CARIES:

3.5.1 Prehistoric races:

From the few fragmentary remains of prehistoric man that have been found, the teeth would appear to have been remarkably free from caries; attrition was usually well marked. An exception is the Rhodesian Man who is said to belong to the pliocene period, and estimated to be about one quarter of a million years old. Most of the teeth are present in the maxilla and many shows caries. There is some resorption of the alveolar bone (WOODWARD, 1922).

VON LENHOSSEK (1919) noted that human skulls before the neolithic period (about 12000 B.B.) were dolichocephalic and free from caries. Skulls of Neolithic period (12000 to 3000 B.C.) shows the presence of dental caries.

3.5.2 Ancient Races:

In Egypt, before 3000 B.C. dental caries was comparatively rare. In the pyramid age the disease became more prevalent in lower Egypt (ELLIOT SMITH, 1929). TURNER and BENNETT (1913) who examined 104 skulls of the twenty fifth dynasty found 40.38% affected with caries.
MUMMERY (1870) found dental caries in ancient British skulls in the following percentage:

- Neolithic - 2.94%
- Roman Conquest - 28.67%
- Anglo Saxon - 15.78%

COLYER and SPRAWSON, (1953) estimated 14.6% caries in the skulls of 17th and 18th century Londoners.

MUMMERY (1870) made investigation about the teeth of Indians in skulls and found 14% caries in South Indians and 9.5% caries in North Indians.

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Race</th>
<th>Diet</th>
<th>Skulls examined</th>
<th>Attrition (% of skull)</th>
<th>Caries % of skull</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>South Indian</td>
<td>Rice, Sweet meals</td>
<td>71</td>
<td>10</td>
<td>14.0</td>
</tr>
<tr>
<td>2.</td>
<td>North Indian</td>
<td>Vegetables, wheat</td>
<td>152</td>
<td>8</td>
<td>9.5</td>
</tr>
</tbody>
</table>

Table 3.1 reveals that **D.M.F.Trend** in India is increase from 1.17 to 4.97 in last 15 years. It signifies the very high rate of increase of Dental Caries incidence rate in India.
Table 3.1 Trends in Dental Caries prevalence in Children from selected developing countries

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Country</th>
<th>D.M.F.Trend From</th>
<th>To</th>
<th>No.of year taken</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Ethiopia</td>
<td>0.2</td>
<td>1.6</td>
<td>17</td>
</tr>
<tr>
<td>2.</td>
<td>Kenya</td>
<td>0.1</td>
<td>1.7</td>
<td>21</td>
</tr>
<tr>
<td>3.</td>
<td>Iraq</td>
<td>0.7</td>
<td>3.5</td>
<td>9</td>
</tr>
<tr>
<td>4.</td>
<td>Thiland</td>
<td>0.7</td>
<td>4.5</td>
<td>15</td>
</tr>
<tr>
<td>5.</td>
<td>Vietnam</td>
<td>2.0</td>
<td>6.3</td>
<td>15</td>
</tr>
<tr>
<td>6.</td>
<td>India</td>
<td>1.17</td>
<td>4.96</td>
<td>15</td>
</tr>
</tbody>
</table>

Source: Moller 1978

3.6 Heredity in Dental Caries:

The question of whether the caries experience of the parents affects that of the children has been a devatable point for many years. KLEIN (1946) found a close relationship between caries experience of the offspring with that of the father or mother considered separately. The results suggest that dental caries involves familial vectors, but further surveys are necessary before reaching definite conclusions.

Animal experiments have shown that rats born to mothers maintained throughout pregnancy and lactation
on a cariogenic diet show a higher susceptibility to caries that do control animals (SOGNNAES, 1948). This effect of parental environment may simulate a hereditary influence.

3.7 Age and Dental Caries:

It is considered that the susceptibility to caries is very much higher during childhood than in the adult. In the later years, following recession of the gums and exposure of the cementum, decay may commence at the cervical margin.

The incidence and degree of caries increases with the age of the child, that is with the number of years that the teeth have been erupted and exposed in the mouth.

3.8 Susceptibility Pattern of Individual Teeth to Dental Caries:

Although the pattern of permanent teeth emergence is well known, no such pattern has so far been established for the loss of permanent teeth (GANGULY 1974). The loss of teeth is not a direct consequence of ageing, most of the teeth are lost due to disease. Dental caries is the most widespread dental illness in children and young people, whereas periodontal disease (GINGIVITIS, PYORRHOEA) are common in older individuals. The loss of teeth from caries decreases with age, while the loss from periodontal diseases increases with age (ROBINSON & BOLING 1952). The loss may also be caused by an injury.
The susceptibility to caries is very much higher during childhood than in the adult. In later years, following recession of the gums and exposure of the cementum, decay may commence at the cervical margin.

Table 3.2 No. of Specified Teeth Affected by Caries Experience

(D.M.F.)per 100 Children at age of 16 years.

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Corresponding Teeth of either side(Rt or Lt)</th>
<th>Upper Jaw</th>
<th>Lower Jaw</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Ist Incisors</td>
<td>21.58</td>
<td>2.70</td>
</tr>
<tr>
<td>2.</td>
<td>IIInd Incisors</td>
<td>22.70</td>
<td>2.70</td>
</tr>
<tr>
<td>3.</td>
<td>Canines</td>
<td>2.48</td>
<td>0.34</td>
</tr>
<tr>
<td>4.</td>
<td>Ist Premolars</td>
<td>15.96</td>
<td>3.49</td>
</tr>
<tr>
<td>5.</td>
<td>IIInd Premolars</td>
<td>16.07</td>
<td>11.69</td>
</tr>
<tr>
<td>6.</td>
<td>Ist Molars</td>
<td>74.27</td>
<td>84.16</td>
</tr>
<tr>
<td>7.</td>
<td>IIInd Molars</td>
<td>40.23</td>
<td>60.45</td>
</tr>
</tbody>
</table>

Source-Kelvin, Kelvin & Palmer 1941

The incidence and degree of caries increases with the age of child, that is with the number of years that the teeth have been erupted and exposed in the mouth. Hence, this must be taken into account when comparing the susceptibility of the different teeth.

In adolescence the permanent first molar is more
The maxillary teeth are more susceptible to loss than mandibular. But there is no difference observed between the right and left sides in either of the job. The canine is the sturdiest tooth in the sense that it tends to outlive the other permanent teeth.

3.9 Nutrition and Dental Caries:

MELLANBY and PATTISON (1928) is of the opinion that a supplement of vitamin D to the diet, especially when given to young children, renders the teeth less susceptible to caries and that cereals have an adverse effect on mineralization and tend to antagonize the beneficial effect of vitamin D.

Lady Mellanby conducted a comprehensive study regarding the effect of various diets on children in institutions. Three groups of children were fed on a similar basal diet with the following additions:

1. Ordinary diet plus treacle.
2. Ordinary diet plus olive oil.
3. Ordinary diet plus cod liver oil (Vitamin A & D.)

An examination of teeth was made before modifying the diets and at subsequent six monthly intervals for three years. It was found that the group taking treacle developed
the highest incidence of dental caries, next came the olive oil group, finally the least incidence occurred in the group taking cod liver oil.

It must be recognized that the incidence of caries is lower in institutional children that in those living under normal family conditions and that the decrease in caries activity becomes greater with increased length of institutional residence. This has been demonstrated for deciduous teeth by KNOWLES (1948) and MELLANBY and MELLANBY (1950) and for both dentitions by STONES, et al. (1950 A).

BOYD (1943) has noted the markedly low incidence of dental caries in children with diabetes mellitus who have a strict dietary regime, BOYD (1944) finds no significant difference in the average rates of progress of caries whether on the high or low fat diets. On the other hand ZISKIN et al. 1944, COHEN (1947) have reported that no significant reduction is found in the incidence of caries in diabetic children.

BRANSBY, et al. 1946 have reported that no discernible effect of the vitamin supplements on the incidence of caries.

TAYLOR and DAY (1940) have carried out observations in the Punjab. In certain districts the diet consists chiefly of rice and chapatti. It is deficient in Vitamin D. Calcium
and phosphorus. There is a high incidence of rickets and osteomalacia but the teeth are very well formed with not much hypoplasia and little caries. The children of mothers with severe osteomalacia also have only a slight amount of caries. MANN et al. (1947) from an examination of 124 malnourished patients showing single or multiple deficiencies of vitamins A, B and C, report that the incidence of dental caries is only 30.5% as great as in 99 control patients free from deficiency disease.

In the new industrialised set-up in India, sweets are increasingly becoming an integral part of the life experience of a child. Added to this is the role of Indian made sweets as a symbol of auspiciousness. Indian sweetmeats are exchanged at weddings, engagements, on getting a new job, religious festivals, or even on passing an examination. Further more the drinking of a certain sweet non-alcohol drink known as shikanji is very wide spread during the hottest part of the day. Shikanji is a beverage made up of fresh lemon, sugar and water. It is also considered as a health drink. Research has shown a high cariogenic potential of the shikanji drink with a slow recovery of plaque pH after taking the drink.

3.10 Physical Effect of Diet on Dental Caries:

The physical character of the diet is one of the most important factors in the incidence of dental caries. It
has been pointed out that the teeth of primitive races have been far less affected that is now observed in civilized communities and the latter undoubtedly partake of foods that are more refined and less fibrous. WALLACE (1912) has stressed the importance of food containing some roughage. Should this be absent, the soft food is just bolted without any serious attempt at mastication. The more fibrous diet requires thorough trituration and, when chewing, the fragments of food that have not been comminuted are continually being returned between the teeth by the action of the tongue and cheeks until they are thoroughly disintegrated. A bolus is then formed at the back of the tongue and swallowed. The friction has a cleansing effect on the teeth and tends to prevent the lodgment of food at any rate on the exposed areas.

Under primitive conditions the food also was not entirely free from sand or gritty particles and hence these caused attrition of the teeth. This wear tended to eliminate the occlusal fissures which lessened the amount of caries. Further interproximal attrition also occurred and this reduced the liability to caries at that site.

The modern method of refining flour by roller milling has been said to be the cause of the prevalent extensive dental caries of today. Previously the grain was stone milled and hence not ground as finely, so that the germ and small
fragments of the husk remained. With roller milling these are all removed and the granules are so minutely crushed that the resulting flour is very refined. As the fibrous element has been removed, bread made from such flour requires little mastication and rapidly goes into a sticky mess. This clings to the teeth so that local enzymatic action is more apt to occur than with bread made from stone milled flour.

SULLIVAN and HARRIS (1958) reported a near caries free children population whose diet is free from meat and refined carbohydrate and consists mainly of raw fruits, vegetables and diary products.

SLACK and MARTIN (1958) have supplied slices of apple to a group of children at the end of each meal and after any snack eaten between meals. At the end of two years they have observed a significant reduction in the number of new carious lesions compared with those observed in a control group living in the same institution who did not receive apple slices.

The caries producing potentialities of 96 carbohydrates have been studied by BIBBY, et al. (1951). They have revised methods of measuring the retention of carbohydrate in the mouth and the amount of acid formed from them in the saliva, the combination of these two measurements being termed the food demineralization potential.
3.11 Structural Defects and Dental Caries:

There are certain areas on all the teeth, which become carious more rapidly and more frequently than others. The areas which are not self cleaning normally are more prone to caries. The general areas can be localised as the pits and fissures of the molars and anterior teeth, the cervical and proximal areas located in otherwise resistant part of teeth.

The enamel layer of the tooth does not provide a perfect covering for all teeth. Particularly in posterior teeth, the occlusal surface contain several voids in the enamel called enamel pit and fissure. The pit and fissure vary in extent from one tooth to another. Some teeth do not contain any.

Teeth containing rather deep pits and fissures have been shown to be more vulnerable to dental caries in these voids. Oral bacteria and their nutrient can easily enter into the fissure and initiate decay. It is nearly impossible to adequately clean these voids by tooth brushing. Hence the bacteria are harboured in pits and fissures.

Studies have shown that 44% of all carious lesion in young patient occurs at occlusal surface with deep pits and fissures.

Periodic examination of teeth is indicated for all persons regardless of age. The interval of examination depends
upon many factors like rate of development of caries, lactobacillus count, sugar intake, age, susceptibility to decay.

Occlusal pits and fissure caries resembles a notorious iceberg in the sea. Clinically only one third of the lesion is seen with the remaining two third hidden beneath. Due to the orientation of enamel rods in groves, the surface shows a small point of decay with a large carious lesion lying beneath. Regarding the correct diagnosis of a deep carious lesion, it must be confirmed whether caries extends only upto dentin or whether the carious process has affected the underlying pulp.

Dental caries is a multifactorial disease and when it involves the deeper structure, it leads to irritation of the pulp and usually becomes one of the commonest cause of pulpal injury. The degree of pulpal inflation is directly proportional to the depth of the carious lesion.

3.12 Fluoride and Dental Caries:

MAC KAY, (1929) had found an inverse relationship between caries incidence and the occurrence of mottled enamel which developed due to ingestion of water containing fluoride in excess of 1ppm Throughout the period of dental development. At a level of 1.0 ppm of fluoride the incidence of caries is strikingly low. (BULL, 1943; KLEIN 1048; BOX and HODGINS
1944; MURROY and WILSON (1942).

The cariostatic mechanism of systemic fluoride can be explained under the following heads-

1. Rendering enamel more resistant to acid dissolution.

2. Inhibition of bacterial enzyme systems - enzymatic action.

3. By reducing tendency of the enamel surface to absorb proteins.


3.12.1 Effect of Fluoride in Reducing enamel Solubility:

The systemic action of fluoride on caries being due to stabilization of the apatitic lattice. Such a stabilization is a result of hydroxy apatitic having inherent voids due to missing hydroxyl groups. Fluoride ions fill these voids and add their hydrogen bonding tendency to the forces which hold the crystals together.

The other mechanism which have been postulated in rendering decrease enamel solubility in addition to void replacement are -

1. Under the influence of fluoride, large crystals are formed with fewer imperfections thus
stabilizing the lattice and presenting a smaller surface area/unit volume for dissolution.

2. Enamel which mineralizes under the influence of fluoride has a lower carbonate content, thus giving a reduced solubility.

3. Fluoride brings about remineralization of the enamel at 1 ppm in early carious lesions

3.12.2 Enzymatic Action:

A further important property of fluoride ion is its ability to inhibit enzyme action and so exert a direct effect on plaque bacteria. BIBBY et al. 1940, WRIGHT & JENKINS 1954 have shown that small amounts of fluoride ion (1-10 ppm) will decrease acid production by saliva - glucose mixture. Higher levels of ionic fluoride are required to reduce acid production by plaque (JENKINS 1959).

Fluoride is concentrated within plaque (DAWES et al. 1965) which originates from the oral fluids rather than enamel. Most of the fluoride is bound at neutral pH with only 2-5 percent being in ionic form (JENKINS et al. 1977). The bound fluoride represents a fluoride reservoir in that it can dissociate when acid is produced by plaque organisms to make available much more ionic fluoride has several different modes of action on bacterial metabolism.
1. The concentration of fluoride above 2ppm in solution progressively decreases transport or uptake of glucose into cells of oral streptococci (WEISS et al. 1965; SCHACHTEL et al. 1973).

2. When plaque has been depleted of it's exogenous sugar supply, fluoride inhibits metabolism of iodophilic polysaccharides by the microorganisms present in plaque and also by salivary bacteria thus indirectly interfering with acid production (WEISS et al. 1965).

3.12.3 Effects Mediated by Surface Absorption:

Fluoride incorporated in enamel by substitution of hydroxyl ions altered the surface charge or free energy and thus indirectly alters the deposition of pellicle and subsequent plaque formation (ERRICSSON et al. 1987).

3.12.4 Effects Mediated by Tooth Morphology:

Fluoride is believed to alter the tooth morphology i.e. reduction in the cusp height, fissure depth and increase in the fissure width (LEVIUS et al. 1969 and ASSENDEN et al. 1974). Thus making teeth less susceptible to caries.

Fluoride has been described as an essential nutrient by WHO expert committee for normal development and
growth of human beings.

It is widespread in fresh and sea water, Vegetables and milk. The main source of fluoride to human body is through drinking water. Foods like dried fish and salmon (84.5 and 19.3 ppm F), tea leaves and turmeric are good source of fluoride. Fluoride is readily absorbed into the body mainly from stomach.

Systemic research in the various physiological, metabolic and toxic aspects of fluoride has revealed that 1 ppm of fluoride in drinking water has no biological side effects on the vital organs of human body.

In the human system thus fluoride has a dual personality a derogatory effect (greater than 4ppm) and caries preventive and health promoting effect at 1ppm).

The optimum recomended fluoride levels varies with climate because the average consumption of water increases in warmer climates. In cold climates the recommended fluoride levels may be as high as 1.2 ppm while in extremely hot climates a level of about 0.7 ppm is recommended. In moderate climates this optimum fluoride levels has been shown to be 1 ppm.

Communal water fluoridation appears to be the most effective, practical and economical public health measure
for prevention of dental caries. But the only short coming is that it can be implemented only in areas which have central pipe water supply system. Currently most of the cities and towns in India covering 30 percent of the population have piped water supply. School water fluoridation is one of the potential areas to be explored.

WESPI et al (1961) found 20-25% reduction in dental caries after the use of salt fluoridation. Tooth from HUNGARY (1976) found 41% decrease in d.m.f.t. in 2-6 years of age after salt fluoridation.

INAMURA (1959) from Japan reported a 36.3% caries reduction when 2.5 Mg NaF was added to milk and served daily in school mels. RUSOFF et al. (1962) from U.S.A. reported a 35% caries reduction with 3.5 ppm of fluoridation in milk. WIRZ (1964), ZIEGLER (1964), BENCGZY (1983) also found similar results.

Available facts about water fluoride concentration in drinking water reveals that only 5% of population live in high fluoride areas like Biharsharif in Bihar, Bhatinda belt in Punjab, Etawah area in U.P. (RAMA SUBRAMANIAN et al. 1979).

Only 3% population lives in optimal fluoride areas and the rest 90-92% of population consumes water, deficient
in fluoride.

About 66% are using water with fluoride contents less than 0.5 ppm which is well below the optimum fluoride level required for protection against dental caries.