- Normal flora, Micro-organisms that are permanently resident but not produce disease.

-- At birth, microbial populations begin to establish themselves.

-(**before a woman gives birth, lactobacilli multiply rapidly in her vagina)**

-- The newborn's first contact with lactobacilli which become predominant organisms in his intestine.

-- Breathing and feeding also introduce different kinds of other micro-organisms.

-- These micro-organisms may increased or decreased according to the change in the environmental conditions. The total No. of the harmless orgs. Exceeds the total No of the body cells.

Types of The Normal Flora

- - Different regions of the body fluid have different kinds of micro-organisms.(see the enclosed Table 1):
- - Residence Flora: are permanently exists (colonize) without harm animal.
- - Transient Flora: They stay for several days, weeks or months and then disappear.
- -

Normal Flora of different Regions

Pathology, Infection, and Disease

Pathology is the scientific study of disease (*pathos* means suffering; *logos* means science). Pathology is first concerned with the cause, or **etiology**, of disease. Second, it deals with **pathogenesis**, the manner in which a disease develops. Third, pathology is concerned with the structural and functional changes brought about by disease and with its final effects on the body.

Although the terms *infection* and *disease* are sometimes used interchangeably, they differ somewhat in meaning. **Infection** is the invasion or colonization of the body by pathogenic microorganisms. **Disease** occurs when an infection results in any change from a state of health. Disease is an abnormal state in which part or all of the body is not properly adjusted or is not capable of carrying on its normal functions. An infection may exist in the absence of detectable disease. For example, the body may be infected with the virus that causes AIDS, but there may be no symptoms of the disease.

The presence of a particular type of microorganism in a part of the body where it is not normally found is also called an infection—and may lead to disease. For example, although large numbers of *E. coli* are normally present in the healthy intestine, their infection of the urinary tract usually results in disease.

Few microorganisms are pathogenic. In fact, the presence of some microorganisms can even benefit the host. Therefore, before we discuss the role of microorganisms in causing disease, let us examine the relationship of the normal flora to the human body.

Normal Flora

Animals, including humans, are generally germ-free in utero. (Viruses and some bacteria may infect a fetus in the uterus.) At birth, however, normal and characteristic microbial populations begin to establish themselves. Just before a woman gives birth, lactobacilli in her vagina multiply rapidly. The newborn's first contact with microorganisms is usually with these lactobacilli, and they become the predominant organisms in the newborn's intestine. More microorganisms are introduced to the newborn's body from the environment when breathing begins and feeding starts. After birth, *E. coli* and other bacteria acquired from foods begin to inhabit the large intestine. These microorganisms remain there throughout life and, in response to altered environmental conditions, may increase or decrease in number and contribute to disease.

Many other normally harmless microorganisms establish themselves inside other parts of the normal adult body and on its surface. It is estimated that a typical human body contains 1×10^{13} body cells yet harbors 1×10^{14} bacterial cells. This gives you an idea of the abundance of normal organisms residing in the human body. The microorganisms that establish more or less permanent residence (colonize) but that do not produce disease under normal conditions are known as **normal flora** or **normal microbiota** (Figure 14.1).

FIGURE 14.1 Representative normal flora for different regions of the body. **(a)** Bacteria on the surface of the skin. **(b)** Plaque on enamel near the gums. The bacteria that cause dental plaque, while part of the normal flora, will cause gum disease and caries unless removed frequently. **(c)** Bacteria of the large intestine.

Table 1

Table 1

The relationships Between The Normal Flora & Host

- - Microbial Antagonism: The normal flora can benefit the host by preventing the overgrowth of harmful microorganisms. When the balance the NF & pathogenic
- Microbes changed, disease will result. e.g. the NF of adult
- Human vagina maintains a local pH at 3.5- 4.5. At this pH,
- The *Candida albicans* can not grow. However, if the bacterial population is eliminated by antibiotics, or excessive deodorants, or antiseptics and the pH reverts to

Symbiosis

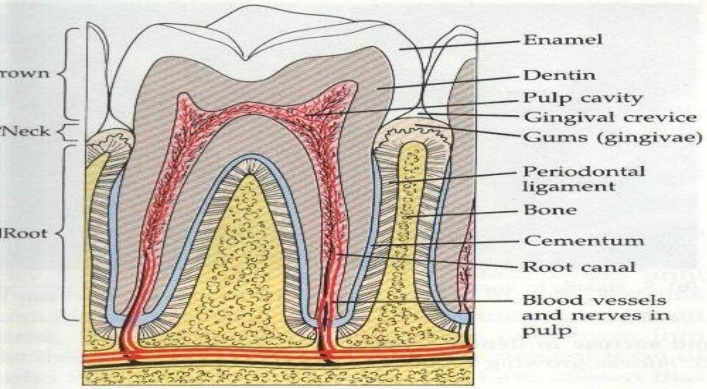
- - Symbiosis is the relationship between the normal flora &
- the host. It means living together and can be classified to:
- A) commensalism : one of the organisms gets benefit and the other does not affected. e.g. corynebacteria that inhibit
- The surface of the eye and certain saprophytic mycobacteria that inhibit the ear and external genitals.
- B) Mutualism: Both organisms gain benefits. E.g. the bacteria of the large intestine (*E.coli).*
- C) Parasitism: One organism benefited at the expense of the other as in case of many disease-causing bacteria.

Opportunistic Organisms

- Opportunists are potentially pathogenic organisms usually do not cause disease in their normal habitat in a healthy person.
- Examples: *S. auruas* of the skin may cause infection through broken skin or mucous.
- *E. coli* will cause infection if gains access to other
- body sites such as urinary bladder, lungs, spinal cord or wounds.

Opportunistic Organisms(cont.)

- Tooth decay & gum disease are caused by normal flora of the mouth where the accumulation of bacteria and their products make the **plaques** which initially involved in the formation of **dental caries** or tooth decay by converting sucrose to lactic acid which in turn attacks the tooth enamel.
- - more than 300 species have been identified on and around the teeth & the most important cariogenic bacterium is *Streptococcus mutans*.



IGURE 25.2 Normal tooth.

Oral bacteria convert sucrose and other carbohy-rates into lactic acid, which in turn attacks the tooth namel. The microbial population on and around the eeth is very complex; more than 300 species have been lentified. Probably the most important *cariogenic* ausing caries) bacterium is *Streptococcus mutans*, a ram-positive coccus (Figure 25.3a). Some other spe-es of streptococci are also cariogenic but play a lesser ole in initiating caries.

The initiation of caries depends on the attachment f *S. mutans*, or other streptococci, to the tooth. These acteria do not adhere to a clean tooth, but within min-tes a freshly brushed tooth will become coated with a ellicle (thin film) of proteins from saliva. Within a ouple of hours, cariogenic bacteria become estab-shed on this pellicle and begin to produce a gummy olysaccharide of glucose molecules called *dextran* igure 25.4). In the production of dextran, the bacteria rst hydrolyze sucrose into its component monosac-narides, fructose and glucose. The enzyme glucosyl-ansferase then assembles the glucose molecules into extran. The residual fructose is the primary sugar fer-ented into lactic acid. Accumulations of bacteria and extran adhering to the teeth make up dental plaque. he bacterial population of plaque is predominantly reptococci and filamentous members of the genus ctinomyces (Figure 25.3b). (Older, calcified deposits of laque are called *dental calculus* or *tartar*.) *S. mutans* es-ecially favors crevices or other sites on the teeth pro-cted from the shearing action of chewing, or the ushing action of the liter or so of saliva produced in

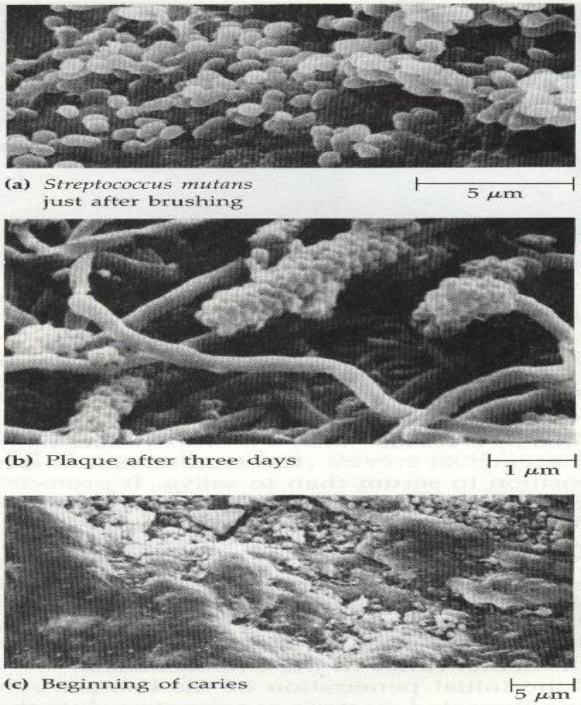
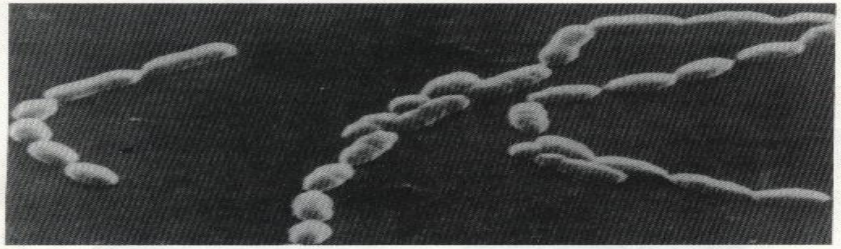


FIGURE 25.3 Initiation of dental caries. **(a)** Spherical cells of *Streptococcus mutans* adhere to tooth enamel shortly after brushing. **(b)** Dental plaque formed on tooth after three days without brushing. Both cocci and filamentous bacteria are present. **(c)** The beginning of a dental caries lesion. Numerous cells of *S. mutans* can be seen in the eroded area.

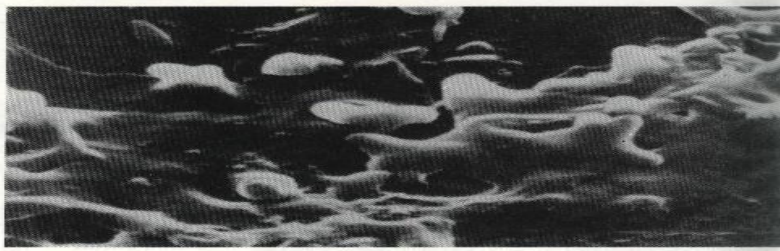
the mouth each day. On protected areas of the teeth, plaque accumulations can be several hundred cells thick. Because plaque is not very permeable to saliva, the lactic acid produced by bacteria is not diluted or neutralized, and it breaks down the enamel of the teeth to which the plaque adheres.

While saliva contains nutrients that encourage the growth of bacteria, it also contains antimicrobial substances, such as lysozyme (see Chapter 16), that help protect exposed tooth surfaces. Some protection is also

DENTAL CARIES



(a) *S. mutans* in glucose — 5 μm



(b) *S. mutans* in sucrose — 5 μm

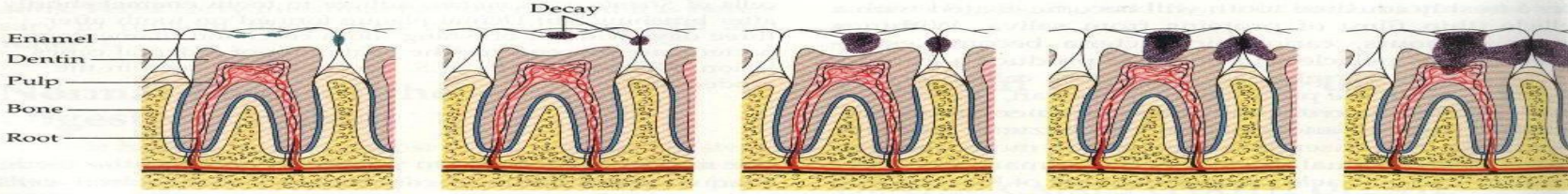
FIGURE 25.4 Involvement of *S. mutans* and sucrose in dental caries. **(a)** *S. mutans* growing in glucose broth. **(b)** *S. mutans* growing in sucrose broth. Note the large accumulation of dextran.

provided by *crevicular fluid*, a tissue exudate that flows into the gingival crevice (Figure 25.2) and is closer in composition to serum than to saliva. It protects teeth by virtue of both its flushing action and its phagocytic cell and immunoglobulin content.

Localized acid production within deposits of dental plaque results in a gradual softening of the external *enamel* (Figures 25.3c and 25.5a, b). Enamel low in fluoride is more susceptible to the effects of the acid. This is the reason for fluoridation of water and toothpastes, which has been an important factor in the recent decline in dental caries in the United States.

If the initial penetration of the enamel by caries remains untreated, bacteria can penetrate into the interior of the tooth. The composition of the bacterial po
ulation involved in advancement of the decayed ar
from the enamel into the *dentin* is entirely differe
from that of the population initiating the decay. Th
dominant microorganisms are gram-positive rods ar
filamentous bacteria; *S. mutans* is present in sma
numbers only. Although once considered the cause
dental caries, *Lactobacillus* species actually play no ro
in initiating the process. However, these very proli
lactic acid producers are important in advancing th
front of the decay once it is established.

The decayed area eventually advances to the *pul*
which connects with the tissues of the jaw and co
tains the blood supply and the nerve cells (Figu



(a) Normal tooth with plaque (b) Decay in enamel (c) Advanced decay (d) Decay in dentin (e) Decay in pulp

FIGURE 25.5 Stages of dental caries. **(a)** Tooth with plaque accumulation in hard-to-clean areas. **(b)** Decay begins as enamel is attacked by acids formed by bacteria. **(c)** Decay advances through the enamel. **(d)** Decay advances into the dentin. **(e)** Decay enters the pulp and may form abscesses in the tissues surrounding the root.