Catabolic reactions provide building blocks for **anabolic reactions** and furnish the energy needed to drive anabolic reactions.

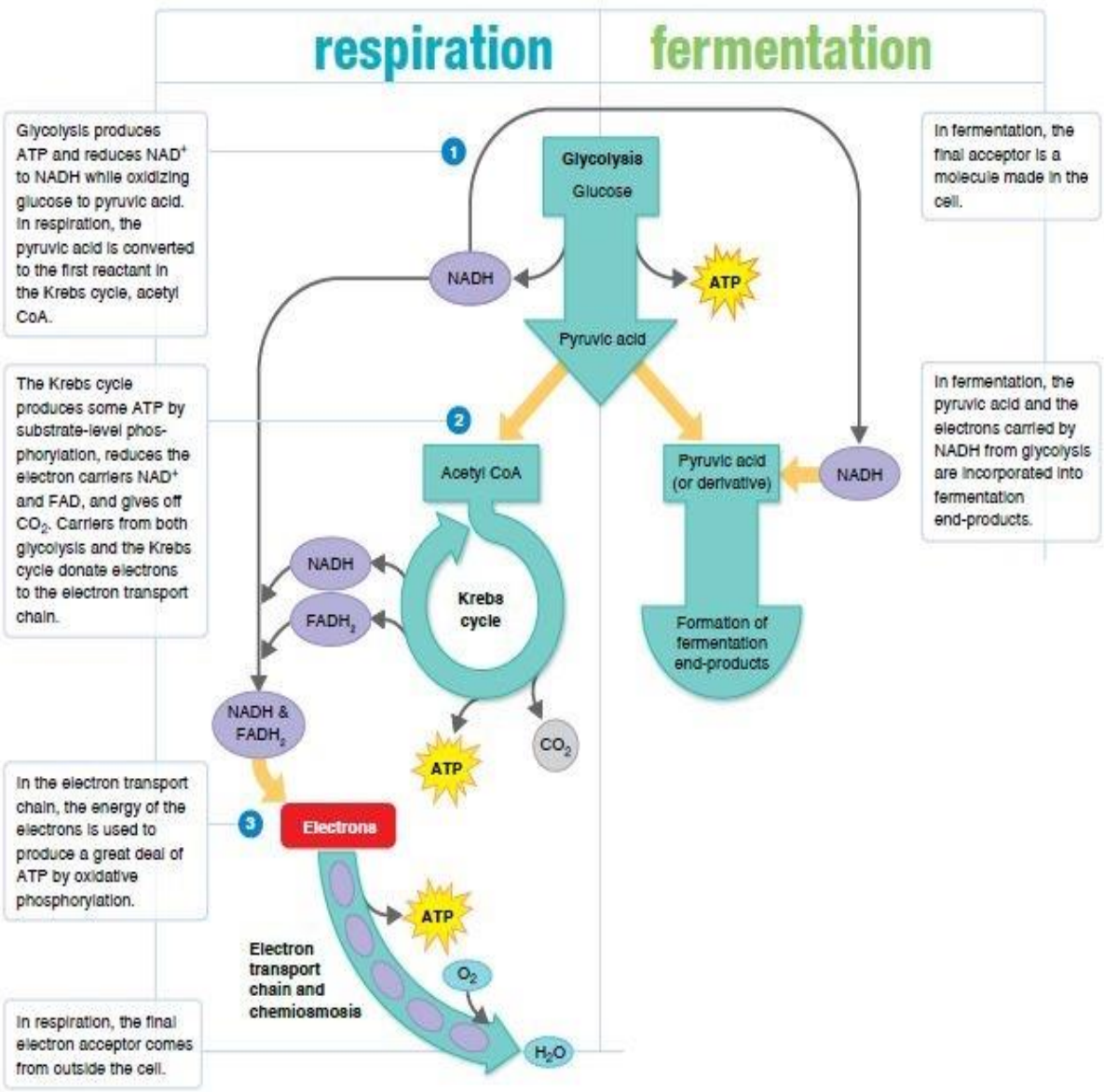
$$ATP \rightarrow ADP + P_i + energy$$

$$ADP + P_i + energy \rightarrow ATP$$

Metabolic Pathways of Energy Production

Organisms release and store energy from organic molecules by a series of controlled reactions rather than in a single burs.

An Overview of Metabolism



Bacterial Genetics

Genetics is the study of inheritance. Bacterial inherited characteristics are encoded in DNA. Bacteria have two types of DNA that contain their genes. These are :

- **Chromosome**
- **Extra chromosome: Plasmid.**

The bacterial chromosome is circular, double stranded DNA attached to bacterial cell membrane. DNA replication in bacteria is semi-conservative i.e. each strand of DNA is conserved intact during replication and becomes one of the two strands of the new daughter molecules.

Plasmids are self-replicating extra chromosomal DNA molecules. It multiplies independent of the host cell. Multiple copies of the same plasmid may be present in each bacterial cell.

Different plasmids are also often present in the same bacterial cell.

Plasmid types

There are many types of plasmid types. The following are examples.

a. R factors: Plasmids which contain genes that code for antibiotic resistance.

b. Col factors: Plasmids which contain genes that code for extracellular toxin (colicines) production that inhibit strains of the same and different species of bacteria.

c. F(fertility) factors: Plasmids that can recombine itself with the bacterial chromosome.

It promotes transfer of the chromosome at a high frequency of recombination into the chromosome of a second (recipient) bacterial cell during mating.

Genetic variation in Bacteria

Mechanisms: Mutation and Gene transfer

1. Mutation: It is due to a chemical alteration in DNA.

It could be spontaneous or induced by chemical and physical meanses. Mutants are variants in which one or more bases in their DNA are altered; which are heritable and irreversible

Types of mutation

1. Substitution: Change of a single base.

2. Deletion: Los of a base.

3. Insertion: Addition of a base.

2. Gene transfer

There are three types of gene transfer that alter the DNA gene content of bacteria.

These are:

- Transformation
- Transduction
- Conjugation

1. Transformation occurs when fragments of exogenous bacterial DNA are taken up and absorbed into recipient bacterial cells.

Transformation of genes from one bacterium to another results in Change in pathogenicity of the bacterium. Change in antibiotic sensitivity pattern of bacterium.

Competence: The recipient bacterium must be competent to absorb the exogenous fragments of bacterial DNA.

Frequency: The frequency of transformation is low.

2. Transduction occurs when fragments of chromosomal DNA is transferred or transduced into a second bacterium by phage.

During phage replication, the bacterial DNA may be accidentally enclosed instead of the normal phage DNA, and when this particle which enclosed the bacterial DNA infects a second bacterial cell, the DNA from the first bacterium is released and incorporated into The chromosome of the second bacterium.

3. Conjugation occurs when plasmid DNA is transferred from donor to recipient bacterium by direct contact via a sex pilus.

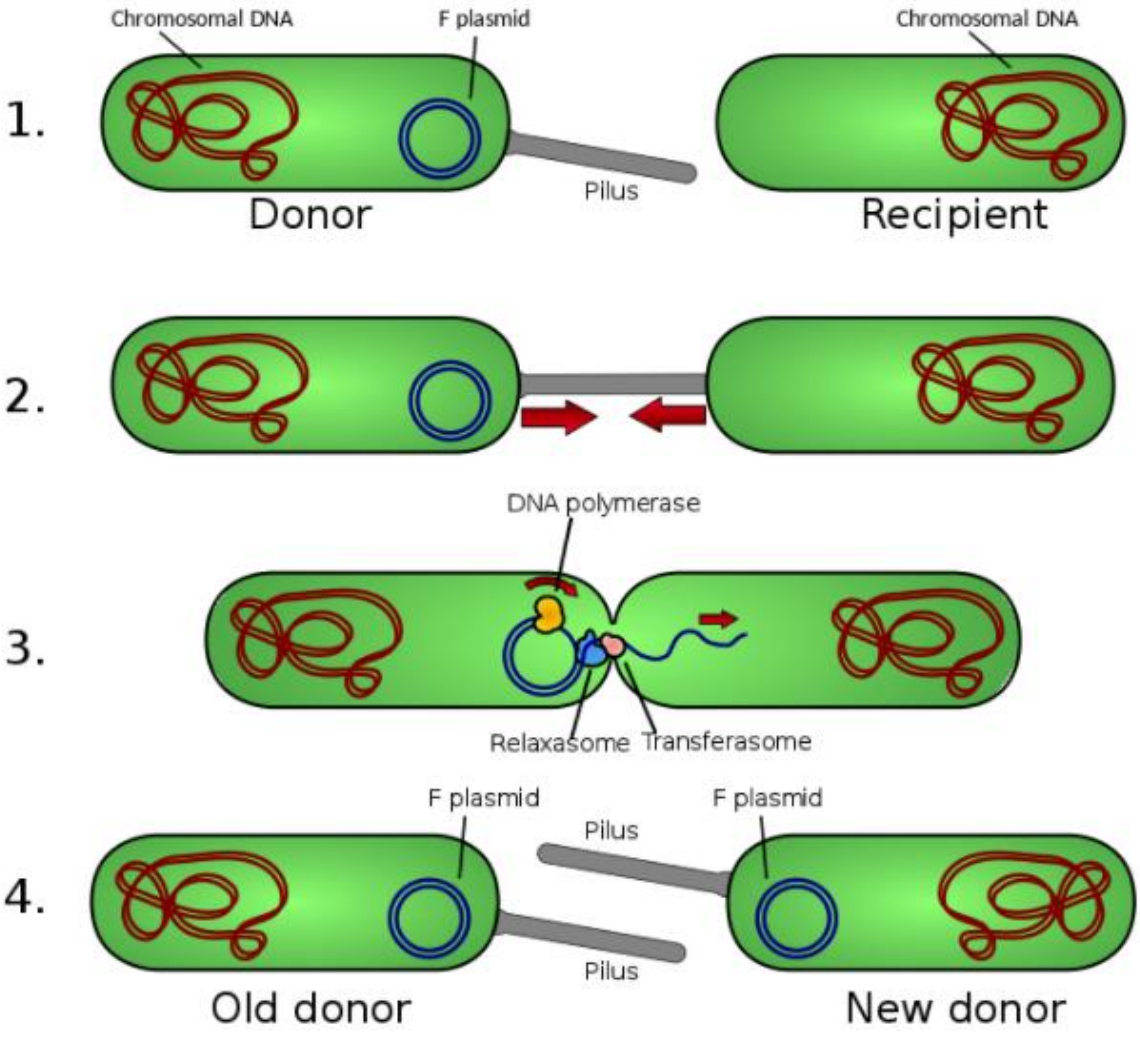
4. Transposition

Mechanism which enhances genetic flexibility among plasmids and bacterial chromosomes.

Transposons(Jumping genes) are segments of DNA that can transpose or move extremely readily, from plasmid to plasmid or from plasmid to chromosome(and viceversa).In this way, plasmid genes become part of the chromosomal component of genes.

When transposons transfer to a new site, it is usually a copy of the transposon that moves, the original transposon remaining in situ.

Transposons code for toxin production, resistance to antibiotics as wellas other fuctions.



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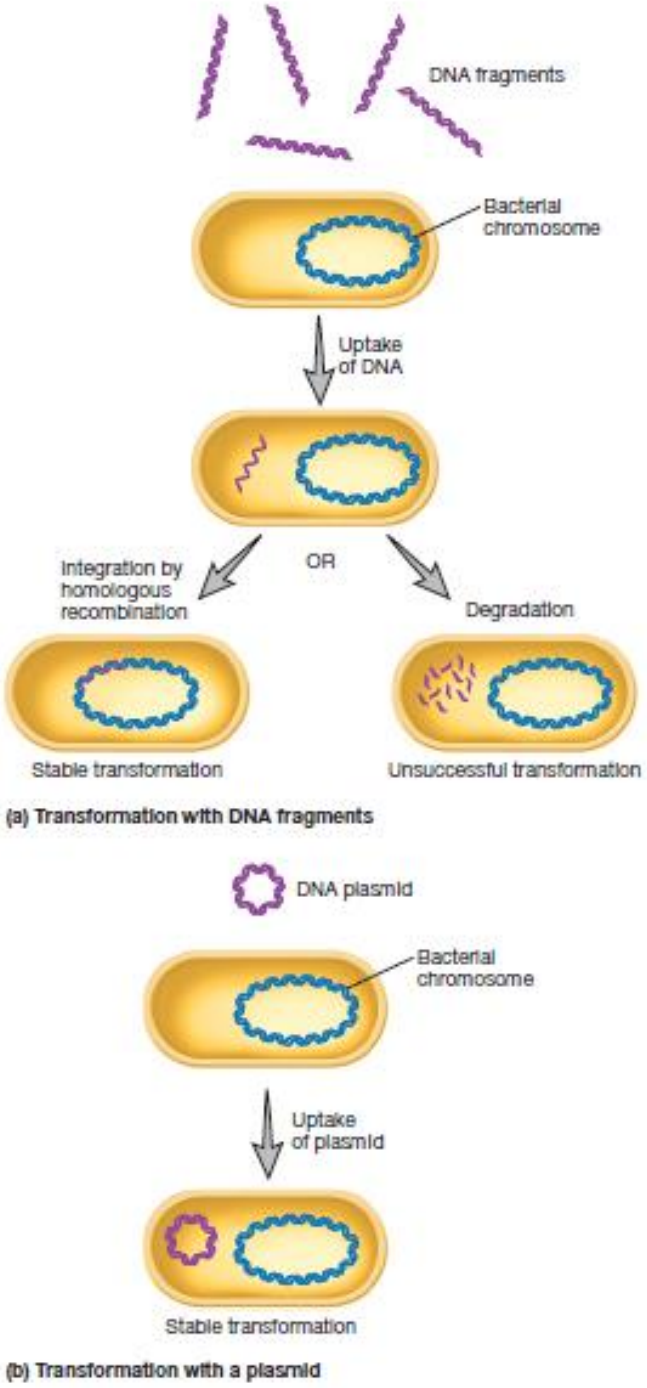
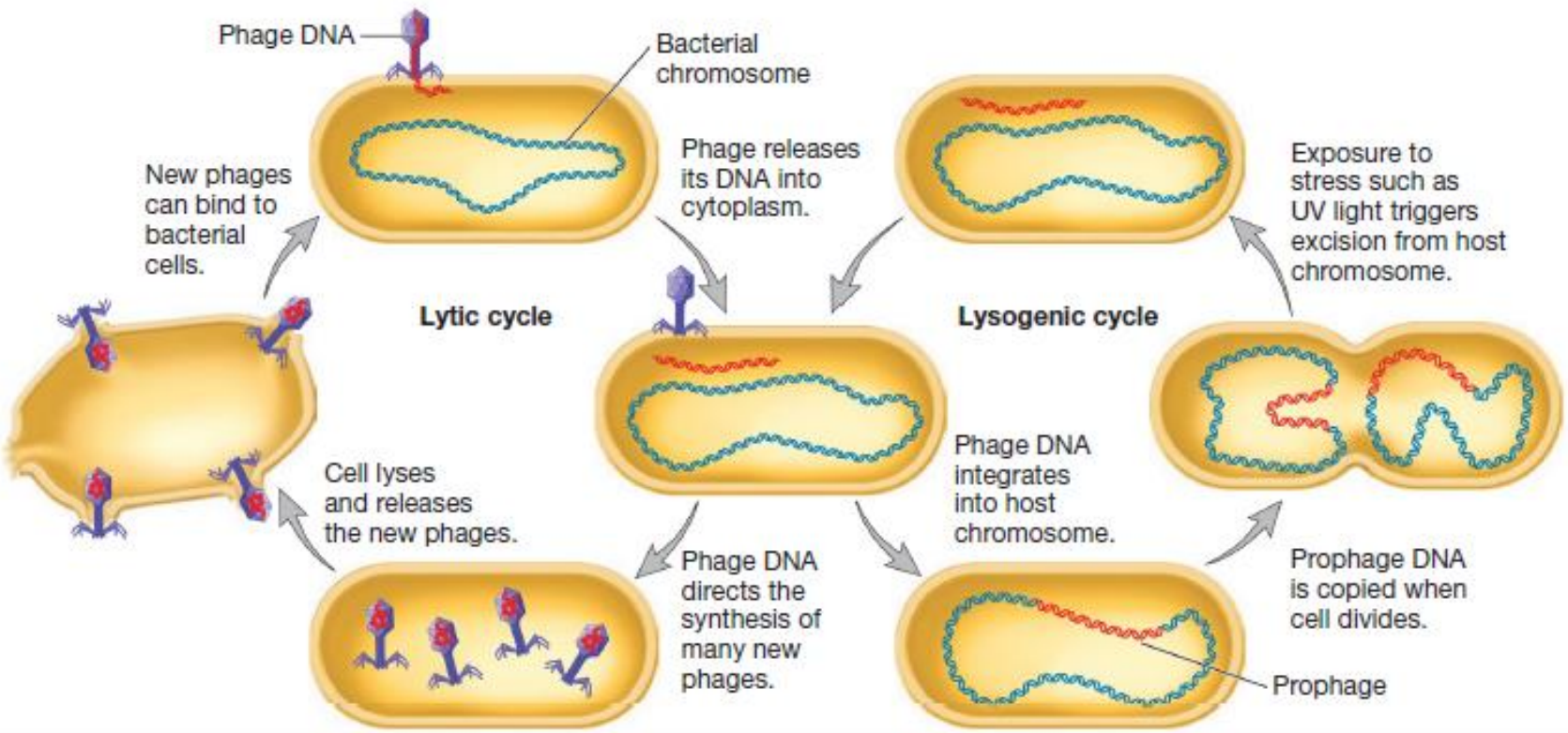


Bacterial Transformation. (Prescotts, p390)



Lytic and Lysogenic Cycles of Temperate Phages (العاثيات المعتدلة (او المتكيفة . Virulent phages undergo only the lytic cycle. Temperate phages have two phases to their life cycles. The lysogenic cycle allows the genome of the virus to be replicated passively as the host cell's genome is replicated. Certain environmental factors such as UV light can cause a switch from the lysogenic cycle to the lytic cycle. In the lytic cycle, new virions are made and released when the host cell lyses.(Prescott's, p.392)

Normal Flora (Microflora or Microbiota)

Introduction

Normal flora (normal microbial flora) is the term used to describe the population of microorganisms that inhabit the skin and mucous membranes of healthy normal persons at certain body sites. The human body is continuously inhabited mostly by bacteria and fungi. Viruses and parasites (protozoa and helminths), which are the other major groups of microorganisms, are usually not considered members of the normal flora but humans can be carriers of some of these organisms). The normal flora organisms are often referred to as commensals. Commensals are organisms that derive benefit from another host but do not damage that host.

The normal microbiota is new term used recently instead of Normal flora. The genomes of these microbial symbionts are collectively defined as the microbiome. Normal microbiota provides a first line of defense against microbial pathogens, assists in digestion, plays a role in toxin degradation, and contributes to maturation of the immune system. Although the normal flora extensively populates many areas of the body (especially the skin, oropharynx, colon, and vagina), the internal organs usually are sterile. Areas such as the central nervous system, blood, lower bronchi and alveoli, liver, spleen, kidneys, and bladder are generally considered to be sterile.

The human body harbors a variety of microorganisms that can be arranged into two groups: (1) the resident microbiota consists of relatively fixed types of microorganisms regularly found in a given area at a given age; if disturbed, it promptly reestablishes itself; and (2) the transient microbiota consists of nonpathogenic or potentially pathogenic microorganisms that inhabit body sites for hours, days, or weeks. The transient microbiota is derived from the environment, does not produce disease, and does not establish itself permanently. There is a distinction between the presence of these organisms and the carrier state. The term carrier implies that an individual harbors a potential pathogen and therefore can be a source of infection of others.

Distribution of normal flora in the body

The most common sites of the body inhabited by normal flora are those in contact or communication with the outside world, namely, the skin, eye, and mouth as well as the respiratory, gastrointestinal, and urogenital tracts, as summarized in Table 1.

1- **Skin.** The predominant member of the normal flora of the skin is *S. epidermidis*. It is an important cause of infections of prosthetic heart valves and prosthetic joints. *C. albicans*, a yeast also found on the skin, can enter the bloodstream and cause disseminated infections, such as endocarditis in intravenous drug users. *S. aureus* is also present on the skin, but its main site is in the nose. It causes abscesses in the skin and in many other organs.

2- **Eye** :The conjunctiva of the eye is colonized primarily by *S. epidermidis*, followed by *S. aureus*, aerobic corynebacteria (diphtheroids), and *Streptococcus pneumoniae*. Other organisms that normally inhabit the skin are also present but at a lower frequency. Tears, which contain the antimicrobial enzyme lysozyme, help limit the bacterial population of the conjunctiva.

3- **Oropharynx.** The main members of the normal flora of the mouth and throat are the viridans streptococci, such as *S. sanguinis* and *S. mutans*. Viridans streptococci are the most common cause of subacute endocarditis.

4- **Gastrointestinal tract.** The stomach contains very few organisms because of the low pH. The colon contains the largest number of normal flora and the most diverse species, including both anaerobic and facultative bacteria. There are both gram-positive and gram-negative rods and cocci. The members of the colonic normal flora are an important cause of disease outside of the colon. The two most important members of the colonic flora that cause disease are the anaerobe *B. fragilis* and the facultative *E. coli. E. faecalis*, a facultative, is also a very important pathogen.

5- **Vagina.** Lactobacilli are the predominant normal flora organisms in the vagina. They keep the pH of the vagina low, which inhibits the growth of organisms such as *C. albicans*, an important cause of vaginitis.

6- **Urethra.** The outer third of the urethra contains a mixture of bacteria, primarily *S. epidermidis*. The female urethra can become colonized with fecal flora such as *E. coli*, which predisposes to urinary tract infections.

Table 1: Summary of the members of normal flora and their locations

Location	Important Organisms[1]	Less Important Organisms[2]
Skin	*Staphylococcus epidermidis*	*Staphylococcus aureus, Corynebacterium* (diphtheroids), various streptococci, *Pseudomonas aeruginosa,* anaerobes (e.g., *Propionibacterium*), yeasts (e.g., *Candida albicans*)
Nose	*S. aureus*[3]	*S. epidermidis, Corynebacterium* (diphtheroids), various streptococci
Mouth	Viridans streptococci	Various streptococci, *Eikenella corrodens*
Dental plaque	*Streptococcus mutans*	*Prevotella intermedia, Porphyromonas gingivalis*
Gingival crevices	Various anaerobes (e.g., *Bacteroides, Fusobacterium,* streptococci, *Actinomyces*)	
Throat	Viridans streptococci	Various streptococci (including *Streptococcus pyogenes* and *Streptococcus pneumoniae*), *Neisseria* species, *Haemophilus influenzae, S. epidermidis*
Colon	*Bacteroides fragilis, Escherichia coli*	*Bifidobacterium, Eubacterium, Fusobacterium, Lactobacillus,* various aerobic gram-negative rods, *Enterococcus faecalis* and other streptococci, *Clostridium*
Vagina	*Lactobacillus, E. coli,*[3] group B streptococci[3]	Various streptococci, various gram-negative rods. *B. fragilis, Corynebacterium* (diphtheroids), *C. albicans*
Urethra		*S. epidermidis, Corynebacterium* (diphtheroids), various streptococci, various gram-negative rods (e.g., *E. coli*)[3]

[1]Organisms that are medically significant or present in large numbers.

[2]Organisms that are less medically significant or present in smaller numbers.

[3]These organisms are not part of the normal flora in this location but are important colonizers.

BENEFICIAL FUNCTIONS OF NORMAL FLORA Normal flora provides considerable benefits to the host.

➢ The members of the normal flora occupy receptor sites on the skin and mucosal surfaces, thereby preventing pathogens from binding to those receptors. (Also known as Colonization resistance, Figure 1)

➢ some bacteria of the bowel produce antimicrobial substances that is harmful to pathogenic bacteria

➢ Third, bacterial colonization of a newborn infant acts as a powerful stimulus for the development of the immune system. The microbiome plays an important role in "educating" the immune system.

➢ Bacteria of the gut provide important nutrients, such as vitamin K, and aid in digestion and absorption of nutrients.

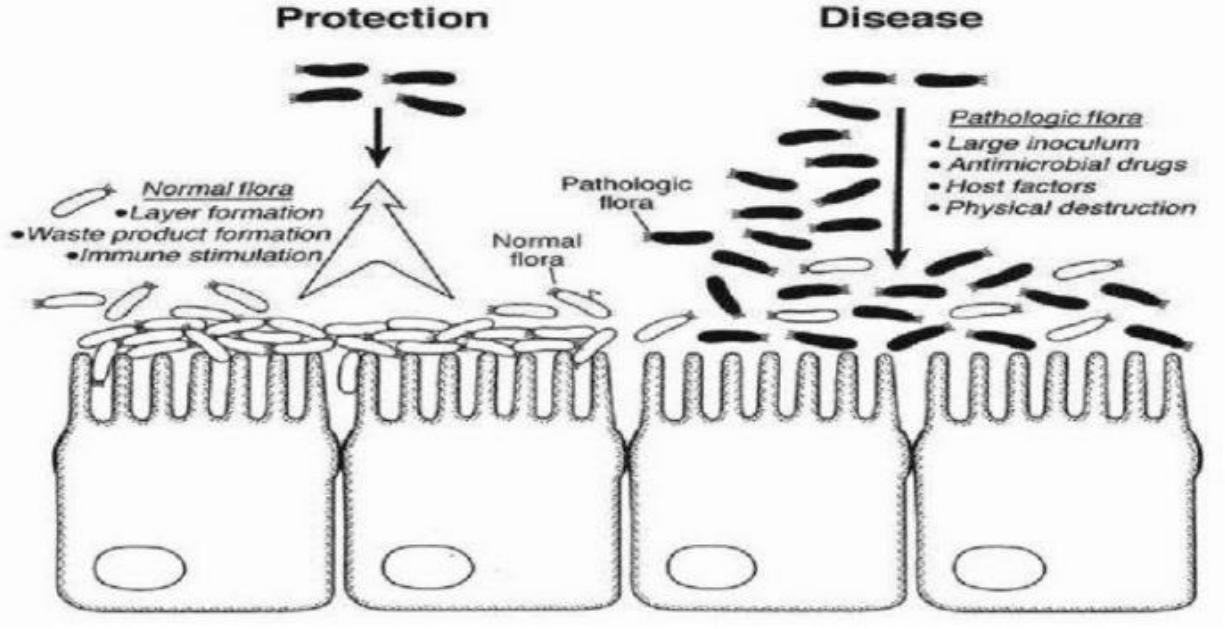


Figure 1: The mechanisms by which normal flora compete with gut pathogens

HARMFUL EFFECTS OF NORMAL FLORA Clinical problems caused by normal flora arise in the following ways:

➢ The organisms are displaced from their normal site in the body to an abnormal site. An example already mentioned is the introduction of the normal skin bacterium, S. epidermidis, into the bloodstream where it can colonize catheters and artificial joints.

➢ Potential pathogens gain a competitive advantage because of diminished populations of the microbiome. For example, normal bowel flora is depleted by antibiotic therapy leading to overgrowth by antibiotic-resistant Clostridium difficile, which can cause severe colitis.

➢ Harmless, commonly ingested food substances are converted into carcinogenic derivatives by bacteria in the colon. A well-known example is the conversion by

bacterial sulfatases of the sweetener cyclamate into the bladder carcinogen cyclohexamine.

➢ When individuals are immunocompromised, normal flora can overgrow and become pathogenic.

10. Bacterial virulence factors

Pathogenicity: is the ability to cause disease by overcoming host defenses, whereas virulence is the degree of pathogenicity**.** To cause disease, most pathogens must gain access to the host, adhere to host tissues, penetrate or evade host defenses, and damage the host tissues. However, some microbes do not cause disease by directly damaging host tissue. Instead, disease is due to the accumulation of microbial waste products.

Some microbes, such as those that cause dental caries and acne, can cause disease without penetrating the body.

Portals of Entry

1. Mucous Membranes

Many bacteria and viruses gain access to the body by penetrating mucous membranes lining the respiratory tract, gastrointestinal tract, genitourinary tract, and conjunctiva, a delicate membrane that covers the eyeballs and lines the eyelids.

Skin

The skin is the largest organ of the body, in terms of surface area and weight, and is an important defense against disease. Unbroken skin is impenetrable by most microorganisms. Some microbes gain access to the body through openings in the skin, such as hair follicles and sweat gland ducts. Larvae يرقة of the hookworm actually bore through intact skin, and some fungi grow on the keratin in skin or infect the skin itself.

Penetration of Host Defenses

Several factors that contribute to the ability of bacteria to invade a host.

1. Capsules: The capsule resists the host's defenses by impairing phagocytosis, the process by which certain cells of the body engulf and destroy microbes.

2. Cell Wall Components:

- **M protein=** mediates attachment of the bacterium to epithelial cells of the host and helps the bacterium resist phagocytosis by white blood cells.

- Some microorganisms use **fimbriae** and an outer membrane protein called **Opa** to attach to host cells. (Bacteria that produce Opa form *opa*que colonies on culture media.)
- The **waxy lipid** (mycolic acid) that makes up the cell wall of *Mycobacterium tuberculosis* also increases virulence by resisting digestion by phagocytes, and the bacteria can even multiply inside phagocytes.

3. **Enzymes**

- **Coagulases** are bacterial enzymes that coagulate (clot) the fibrinogen in blood.
- Bacterial **kinases** are bacterial enzymes that break down fibrin and thus digest clots formed by the body to isolate the infection.
- **Hyaluronidase** is another enzyme secreted by certain bacteria, such as streptococci. It hydrolyzes hyaluronic acid, a type of polysaccharide that holds together certain cells of the body, particularly cells in connective tissue.
- **Collagenase**, produced by several species of Clostridium, facilitates the spread of gas gangrene. Collagenase breaks down the protein collagen, which forms the connective tissue of muscles and other body organs and tissues.

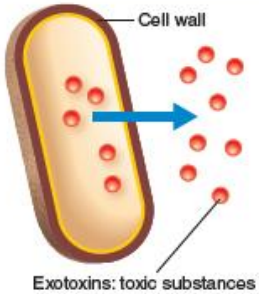
Production of Toxins

Toxins are poisonous substances that are produced by certain microorganisms. They are often the primary factor contributing to the pathogenic properties of those microbes. The capacity of microorganisms to produce toxins is called **toxigenicity**. Toxins transported by the blood or lymph can cause serious, and sometimes fatal, effects. Some toxins produce **fever**, **cardiovascular disturbances** اضطراب الجهاز القلبي الوعائي , **diarrhea**, and **shock** صدمة.

- **Exotoxins:** Exotoxins are produced inside some bacteria as part of their growth and metabolism and are secreted by the bacterium into the surrounding medium or released following lysis. (*Exo*- refers to "outside,").
- **Endotoxins:** Endotoxins differ from exotoxins in several ways. Endomeans "within," in this context referring to the fact that the endotoxins are located within the bacterial cells. Endotoxins are part of the outer portion of the cell wall of gramnegative bacteria**.** The lipid portion of LPS, called **lipid A**, is the endotoxin. Thus, endotoxins are lipopolysaccharides, whereas exotoxins are proteins.

exotoxins

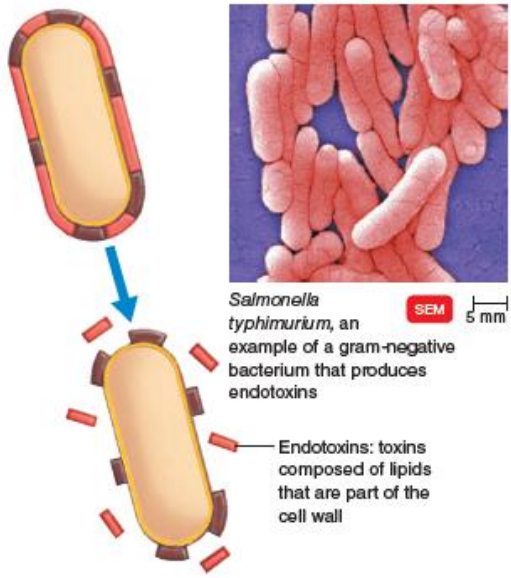
Proteins produced inside pathogenic bacteria, most commonly gram-positive bacteria, as part of their growth and metabolism. The exotoxins are then secreted into the surrounding medium during log phase.



Clostridium botulinum, an example of a gram-positive bacterium that produces exotoxins

endotoxins

Lipid portions of lipopolysaccharides (LPS) that are part of the outer membrane of the cell wall of gram-negative bacteria (lipid A). The endotoxins are liberated when the bacteria die and the cell wall lyses, or breaks apart.

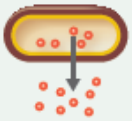


Salmonella typhimurium, an example of a gram-negative bacterium that produces endotoxins

KEY CONCEPTS

- Toxins are of two general types: exotoxins and endotoxins.
- Bacterial toxins can cause damage to host cells.
- Toxins can elicit an inflammatory response in the host, as well as activate the complement system.
- Some gram-negative bacteria may release minute amounts of endotoxins, which may stimulate natural immunity.

Mechanisms of Exotoxins and Endotoxins (Tortora, p. 425)

Property	Exotoxins	Endotoxins
		
Bacterial Source	Mostly from gram-positive bacteria	Gram-negative bacteria
Relation to Microorganism	Metabolic product of growing cell	Present in LPS of outer membrane of cell wall and released with destruction of cell or during cell division
Chemistry	Proteins, usually with two parts (A-B)	Lipid portion (lipid A) of LPS of outer membrane (lipopolysaccharide).
Pharmacology (Effect on Body)	Specific for a particular cell structure or function in the host (mainly affects cell functions, nerves, and gastrointestinal tract)	General, such as fever, weaknesses, aches, and shock; all produce the same effects
Heat Stability	Unstable; can usually be destroyed at 60–80°C (except staphylococcal enterotoxin)	Stable; can withstand autoclaving (121°C for 1 hour)
Toxicity (Ability to Cause Disease)	High	Low
Fever-Producing	No	Yes
Immunology (Relation to Antibodies)	Can be converted to toxoids to immunize against toxin; neutralized by antitoxin	Not easily neutralized by antitoxin; therefore, effective toxoids cannot be made to immunize against toxin
Lethal Dose	Small	Considerably larger
Representative Diseases	Gas gangrene, tetanus, botulism, diphtheria, scarlet fever	Typhoid fever, urinary tract infections, and meningococcal meningitis

11. Chemotherapy

- ✓ **Chemotherapy** is the chemical treatment of a disease.
- ✓ Two types of chemotherapeutic agents are **synthetic** drugs (chemically prepared in the laboratory) and **antibiotics** (substances produced naturally by bacteria and fungi that inhibit the growth of bacteria).
- ✓ Paul Ehrlich introduced an arsenic-containing chemical called salvarsan to treat syphilis (1910).
- ✓ Alexander Fleming observed that the Penicillium fungus inhibited the growth of a bacterial culture. He named the active ingredient penicillin (1928).
- ✓ Researchers are tackling the problem of drug-resistant microbes.

A sterilizing agent is called a **sterilant**. Liquids or gases can be sterilized by filtration.

Control directed at destroying harmful microorganisms is called **disinfection**. It usually refers to the destruction of vegetative (non–endospore-forming) pathogens, which is not the same thing as complete sterility.

In practice, the term is most commonly applied to the use of a chemical (a **disinfectant**) to treat an inert surface or substance. When this treatment is directed at living tissue, it is called **antisepsis**, and the chemical is then called an **antiseptic**.

Names of treatments that cause the outright death of microbes have the suffix **-cide**, meaning **kill**. A **biocide**, or **germicide**, kills microorganisms.

Other treatments only **inhibit** the growth and multiplication of bacteria; their names have the suffix **-stat** or **-stasis**, meaning to **stop** or to **steady**, as in bacteriostasis.

Sepsis, from the Greek for decay or putrid, indicates bacterial contamination.

Aseptic means that an object or area is free of pathogens.

Antimicrobial agents are often classified as **narrow-spectrum drugs**—that is, they are effective only against a **limited** variety of pathogens—or **broad spectrum** drugs that attack **many** different kinds of bacteria.

Some idea of the effectiveness of a chemotherapeutic agent against a pathogen can be obtained from **the minimal inhibitory concentration (MIC).** The MIC is the lowest concentration of a drug that prevents growth of a particular pathogen. On the other hand, **the minimal lethal concentration**

(MLC) is the lowest drug concentration that kills the pathogen. A cidal drug generally kills pathogens at levels only **two to four** times more than the MIC, whereas a static agent kills at much higher concentrations, if at all.

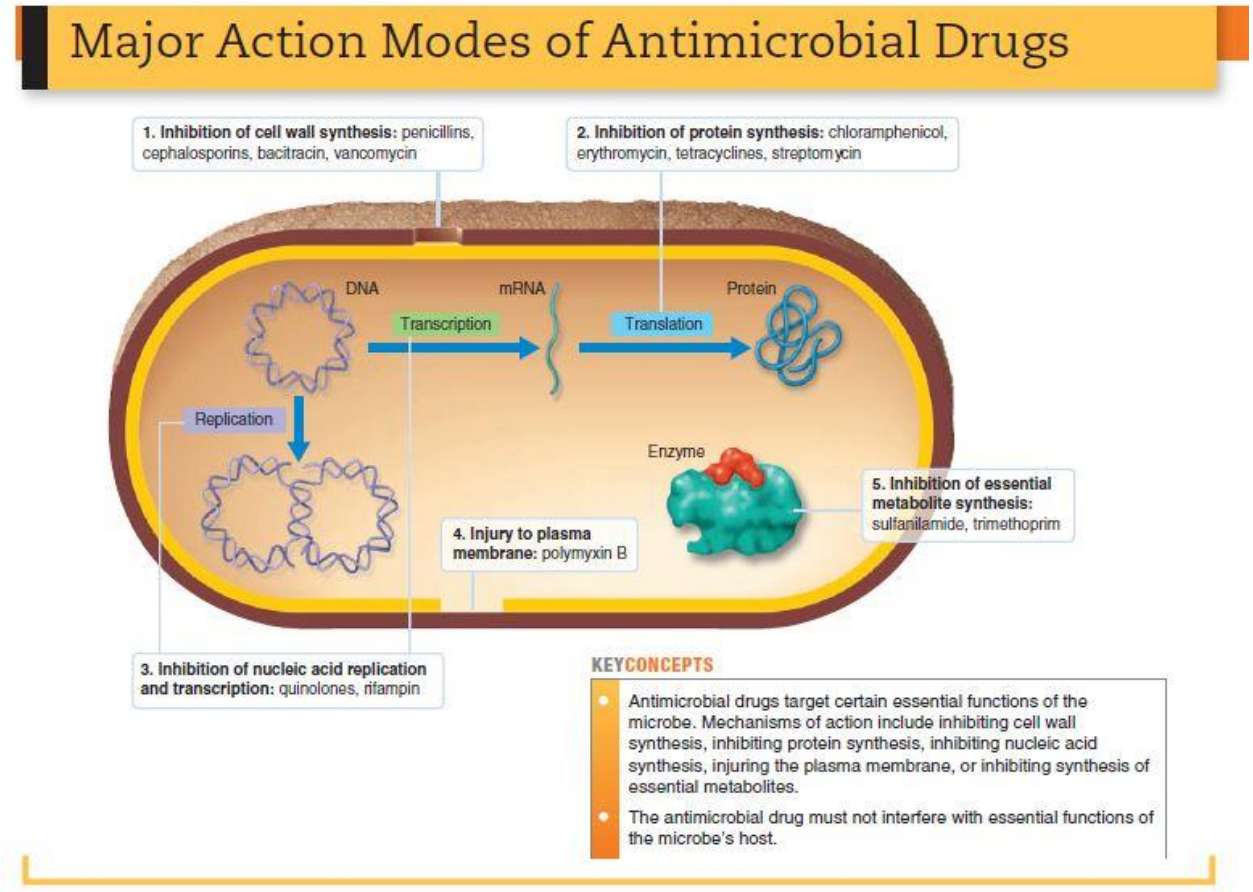
Properties of Some Common Antibacterial Drugs

Antibiotic Group	Primary Effect	Mechanism of Action	Example Members	Spectrum	Common Side Effects
Cell Wall Synthesis Inhibition					
Penicillins	Cidal	Inhibit transpeptidation enzymes involved in cross-linking the polysaccharide chains of peptidoglycan Activate cell wall lytic enzymes	Penicillin G, penicillin V, methicillin Ampicillin, carbenicillin	Narrow (Gram-positive) Broad (Gram-positive, some Gram-negative)	Allergic reactions (diarrhea, anemia, hives, nausea, renal toxicity)
Cephalosporins	Cidal	Same as above	Cephalothin, cefoxitin, cefaperazone, ceftriaxone	Broad (Gram-positive, some Gram-negative)	Allergic reactions, thrombophlebitis, renal injury
Protein Synthesis Inhibition					
Aminoglycosides	Cidal	Bind to small ribosomal subunit (30S) and interfere with protein synthesis by directly causing misreading of mRNA	Neomycin, kanamycin, gentamicin Streptomycin	Broad (Gram-negative, mycobacteria) Narrow (aerobic Gram-negative)	Ototoxic, renal damage, loss of balance, nausea, allergic reactions
Tetracyclines	Static	Same as aminoglycosides	Oxytetracycline, chlortetracycline	Broad (including rickettsia and chlamydia)	Gastrointestinal upset, teeth discoloration, renal and hepatic injury
Macrolides	Static	Bind 23S rRNA of large ribosomal subunit (50S) to inhibit peptide chain elongation during protein synthesis	Erythromycin	Broad (aerobic and anaerobic Gram-positive, some Gram-negative)	Gastrointestinal upset, hepatic injury, anemia, allergic reactions
Nucleic Acid Synthesis Inhibition					
Quinolones and Fluoroquinolones	Cidal	Inhibit DNA gyrase and topoisomerase II, thereby blocking DNA replication	Norfloxacin, ciprofloxacin, Levofloxacin	Narrow (Gram-negatives better than Gram-positives) Broad spectrum	Tendonitis, headache, light-headedness, convulsions, allergic reactions
Rifampin	Cidal	Inhibits bacterial DNA-dependent RNA polymerase	R-Cin, rifacilin, rifamycin, rimactane, rimpin, siticox	*Mycobacterium* infections and some Gram-negatives (e.g., *Neisseria meningitidis* and *Haemophilus influenzae* b)	Nausea, vomiting, diarrhea, fatigue, anemia, drowsiness, headache, mouth ulceration, liver damage
Cell Membrane Disruption					
Polymyxin B	Cidal	Binds to plasma membrane and disrupts its structure and permeability properties	Polymyxin B, polymyxin topical ointment	Narrow—mycobacterial infections, principally leprosy	Can cause severe kidney damage, drowsiness, dizziness
Antimetabolites					
Sulfonamides	Static	Inhibit folic acid synthesis by competing with *p*-aminobenzoic acid (PABA)	Silver sulfadiazine, sulfamethoxazole, sulfanilamide, sulfasalazine	Broad spectrum	Nausea, vomiting, and diarrhea; hypersensitivity reactions such as rashes, photosensitivity
Trimethoprim	Static	Blocks folic acid synthesis by inhibiting the enzyme tetrahydrofolate reductase	Trimethoprim (in combination with a sulfamethoxazole)	Broad spectrum	Same as sulfonamides but less frequent

Antimicrobial Activity Can Be Measured by Specific Tests….. Practical

There are different mode of action of antimicrobial drugs:

- Inhibition of cell wall synthesis.
- Inhibition of protein synthesis.
- Inhibition of nucliec acid replication and transcription.
- Injury of plasma membrane.
- Inhibition of essential metabolite synthesis.

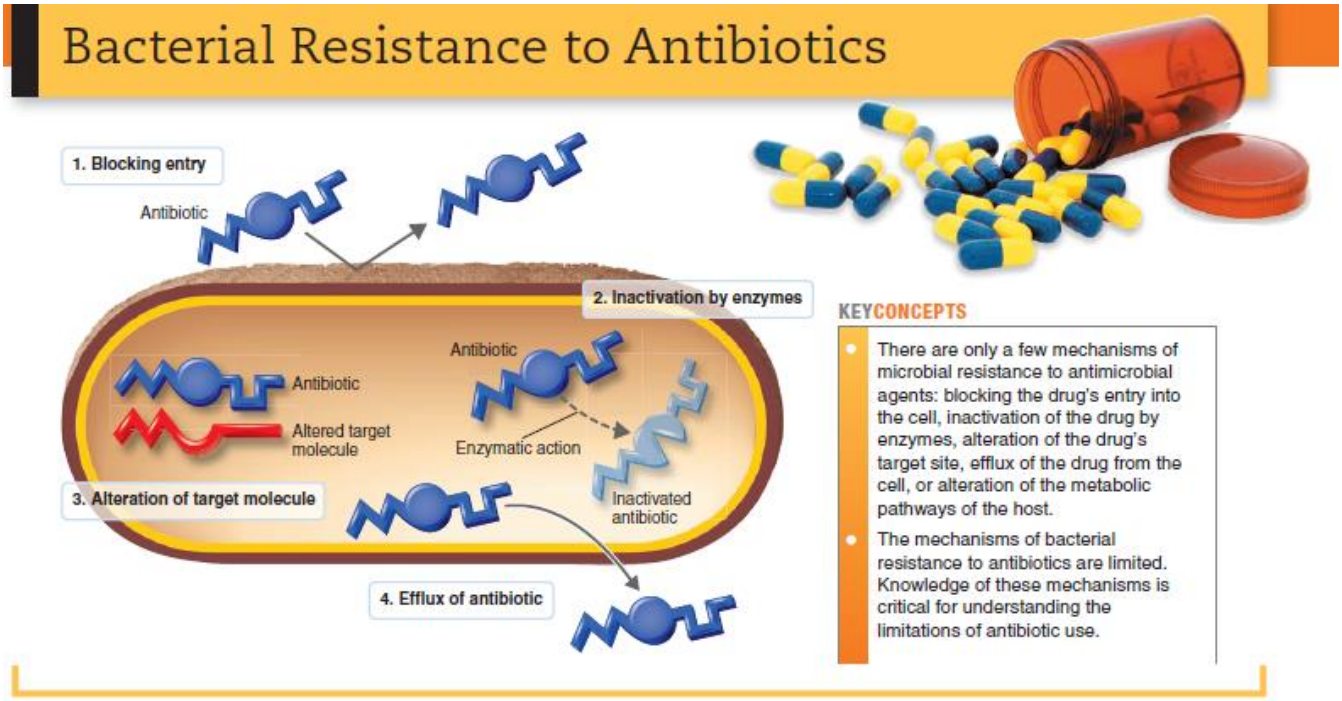


Tortora, 561

Bacterial cell (according to the species) developes different types of resistance,and this issue concidered the problem of the world.

The resistant mechanisms are:

- Blocking the antibiotic entry.
- Inactivation by enzymes.
- Alteration of the target molecule.
- Efflux of antibiotic.


Bacterial Resistance to Antibiotics
1. Blocking entry
Antibiotic
2. Inactivation by enzymes
Antibiotic
Antibiotic
Altered target molecule
Enzymatic action
Inactivated antibiotic
3. Alteration of target molecule
4. Efflux of antibiotic
KEYCONCEPTS
There are only a few mechanisms of microbial resistance to antimicrobial agents: blocking the drug's entry into the cell, inactivation of the drug by enzymes, alteration of the drug's target site, efflux of the drug from the cell, or alteration of the metabolic pathways of the host.
The mechanisms of bacterial resistance to antibiotics are limited. Knowledge of these mechanisms is critical for understanding the limitations of antibiotic use.

Tortora, 580

Staphylococci

The most clinically important species of Staphylococci include *Staphylococcus aureus*, *S. epidermidis* and *S. saprophyticus*. They are Gram-positive cocci; usually arranged in clusters; non-motile; catalase positive; non-sporing; grow over a wide temperature range (10–42 ˚C), with an optimum of 37 ˚C; aerobic and facultatively anaerobic; grow on simple media.

Classification

1 Colonial morphology: ***S. aureus*** colonies are **grey** to **golden yellow**; ***S. epidermidis*** and ***S. saprophyticus*** colonies are white. Staphylococci may produce haemolysins, resulting in haemolysis on blood agar.

2 Coagulase test: ***S. aureus*** possesses the enzyme **coagulase**, which acts on plasma to form a clot. Other staphylococci (e.g. ***S. epidermidis*** and ***S. saprophyticus***) do not possess this enzyme and are often termed, collectively, 'coagulase-negative staphylococci' (CoNS). There are three methods to demonstrate the presence of coagulase:

(a) tube coagulase test: diluted plasma is mixed with a suspension of the bacteria; after incubation, clot formation indicates S. aureus

(b) slide coagulase test: a more rapid and simple methodin which a drop of plasma is added to a suspension of staphylococci on a glass slide; visible clumping indicates the presence of coagulase.

(c) latex agglutination test: cells are mixed with coated latex particles; visible agglutination provides simultaneous detection of staphylococci containing coagulase and/or protein A.

3 Deoxyribonuclease (DNAase) production: ***S. aureus*** possesses an enzyme, DNAase, which depolymerises and hydrolyses DNA; other staphylococci rarely possess this enzyme.