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PART I

Microbiology
CHAPTER I

Microorganisms and disease

CHAPTER OUTCOMES

After reading this chapter, you should be able to:

➤ List the main groups of microorganism causing infection
➤ Explain the terms ‘infection’, ‘colonization’, ‘commensal’, ‘pathogen’, ‘opportunistic’ and ‘virulence’
➤ List the possible signs and symptoms of infection
➤ Give examples of bacteria of the following morphological types: bacilli, cocci, spirochaetes and vibrios
➤ Give an example of each of the following: Gram-negative bacterium, Gram-positive bacterium, acid-fast bacillus, a spore-forming bacterium, an aerobe and an anaerobe
➤ State the ways in which microorganisms gain access to the internal tissues of the host, and give an example for each mechanism suggested
➤ List the ways in which bacteria multiply, and point out their clinical significance
➤ List the main mechanisms by which microorganisms are disseminated, and give an example for each route suggested
➤ Explain how viruses cause disease and give an example
➤ Give one example of a human disease caused by a fungus, a protozoan, a Chlamydia and a helminthic (worm) infestation

Introduction to medical microbiology and microorganisms causing disease

Microbiology is the study of microorganisms – living organisms that are too small to be examined without a microscope. Organisms with a diameter of 0.1 mm are just visible to the naked eye, but magnification is required to study them in detail. Medical microbiology is the study of microorganisms that play a role in human infection.

Infection is caused by bacteria, viruses, fungi, protozoa and a few minor groups (mycoplasmas, rickettsiae and chlamydiae). Parasitic worms are multicellular and often clearly visible to the naked eye, but their eggs and larvae are microscopic, so
the presence of infection is frequently detected in specimens sent to the microbiology department. In recent years, minute virus-like protein particles called ‘prions’ have also been implicated in causing infection. An example is the agent causing Creutzfeldt–Jakob disease (CJD) (Chapter 14).

**Bacteria**

Bacteria live everywhere. Most are saprophytes (organisms that live on dead organic material) present in soil and water. They play a vital role degrading complex organic molecules from dead animals and plants into simple organic ones. These molecules are recycled during metabolism by living organisms.

**Pathogenic activity**

Approximately 50 species of bacteria are ‘pathogenic’ (able to cause disease). Virulence – the ability to generate infection – is a complex phenomenon related to the physiology of both pathogen and host. Some bacteria are always highly virulent. For example, exposure to *Yersinia pestis* (bacterium causing plague) will almost certainly result in infection. However, some bacteria, particularly those causing infections in hospital, are of low pathogenicity. They cause infection only in people whose immune status is compromised by illness, drugs or the invasive procedures they have undergone (for example surgery, intubation or the insertion of an intravenous line). They do not attack healthy tissues. These bacteria are called ‘opportunists’. *Pseudomonas, Klebsiella* and *Proteus* are typical opportunists.

Other bacteria live harmlessly in or on one particular part of the body. These make up the normal flora and are called ‘commensals’ (Table 1.1). They receive shelter and benefit the host by keeping potentially dangerous microorganisms at bay. If they gain access to a different anatomical location, however, they can generate infection. *Escherichia coli* (*E. coli*), normally present in the bowel, can cause urinary tract infection if it gains access to the bladder. This is an example of endogenous (self-) infection, occurring when the organisms responsible originate from the same individual. Exogenous (cross-) infection occurs when microorganisms originate from another source: patients, residents, staff or the environment.

**Table 1.1** The normal human flora

<table>
<thead>
<tr>
<th>Anatomical location</th>
<th>Organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td><em>Staphylococcus epidermidis</em>, micrococi, diphtheroids</td>
</tr>
<tr>
<td>Upper respiratory tract</td>
<td><em>Streptococcus viridans</em>, diphtheroids, <em>Moraxella catarrhalis</em> (Neisseria catarrhalis)</td>
</tr>
<tr>
<td>Large intestine/bowel</td>
<td><em>Bacteroides spp.</em>, <em>Escherichia coli</em>, <em>Streptococcus faecalis</em>, <em>Proteus</em>, clostridia, lactobacilli</td>
</tr>
<tr>
<td>Vagina</td>
<td><em>Lactobacilli, Staphylococcus epidermidis</em></td>
</tr>
</tbody>
</table>
Infection and colonization

Infection occurs when pathogens gain access to host tissues and elicit a response. Infection in a wound is indicated by the appearance of inflammation and pus. The patient may become pyrexial, and a wound swab will indicate the presence of large numbers of the causative organism.

The response to the pathogens may, however, be slight or absent, a situation described as ‘colonization’. A colonized wound is free from inflammation, a swab indicating scanty bacterial growth. When colonization occurs, several species of bacteria may be present, often referred to as ‘mixed bacterial growth’ on laboratory reports. Colonization is of clinical significance because the organism may multiply in large numbers to form a reservoir. Colonization is usually the precursor to infection when outbreaks occur (Muder et al., 1991), and even if the original patient escapes the clinical signs and symptoms of disease, cross-infection may still occur.

There are many situations in which infection may be difficult to diagnose: the very young, older adults, people with communication difficulties and people with some mental health problems or a learning disability (see below).

PRACTICE APPLICATION 1.1

Being Alert to the Possibility of Infection

The expected signs of infection may not be present, for example an elevated temperature may not always be present in older people. Health and social care practitioners need to be alert to other signs, symptoms and changes in behaviour that may indicate an infection.

For example:

➤ Complaints of feeling generally unwell
➤ A rash characteristic of the infection
➤ Chills and shivering
➤ Changes in vital signs other than temperature, such as an increase in respiratory rate
➤ General aches and pains in the muscles and joints
➤ A dry mouth with a furred tongue
➤ Loss of appetite
➤ Nausea and vomiting
➤ Diarrhoea
➤ Headache
➤ Loss of continence in adults
➤ ‘Accidents’ in previously continent toddlers and children
➤ Behavioural changes in children, becoming fretful and miserable
➤ Increasing confusion and disorientation in older adults
➤ Enlarged and tender lymph nodes
NB The knowledge that a particular infection, for example chickenpox or gastroenteritis, is present in the population at the time should alert health and social care practitioners to the possibility of infection.

Activity
Think about a patient, client or resident in your care who had an infection without the usual signs being present.

➤ What first alerted you to the possibility of an infection?
➤ Which of the features listed above were present?

Describing bacteria
Bacteria can be described in terms of their:

- Morphology (shape)
- Ultrastructure (fine detail)
- Response to dyes used on microscope specimens, for example the Gram stain reaction
- Spore formation
- Oxygen requirement.

Morphology
Four morphological forms exist (Figure 1.1):

- **Cocci** are round. When they are arranged in pairs, they are known as ‘diplococci’. Examples include *Streptococcus pneumoniae* (which causes pneumonia) and *Neisseria gonorrhoeae* (leading to gonorrhoea). Clusters of cocci are termed ‘staphylococci’. Examples include *Staphylococcus aureus*, a constituent of the normal skin flora, which in some members of the population is also able to operate as a wound pathogen, and *Staphylococcus epidermidis*, an opportunist able to cause infection in very sick people, although not in the healthy. ‘Streptococci’ are round bacteria attached to one another in chains. They cause sore throats and a wide range of other infections encountered in hospital and the community.

- **Bacilli** (for example *Pseudomonas, Klebsiella, Proteus and E. coli*) are rod shaped, occurring singly or in chains. They are notorious for their ability to cause serious infection in hospital. An extended-spectrum beta-lactamase (ESBL)-producing *E. coli*, which is resistant to several antibiotics, can cause urinary infection and is responsible for around 2,000 cases of blood poisoning each year in England and Wales (Health Protection Agency, 2007) (see Chapter 4 for beta-lactamase-producing bacteria). Several bacteria which cause food poisoning, including *Shigella* and *Salmonella*, also belong to this group.

- **Vibrios** are curved bacteria. Examples include *Vibrio cholerae* (resulting in cholera) and *Campylobacter* (responsible for food poisoning).
Spirochaetes are very small, flexible, spirally shaped bacteria. Typical members of the group include *Treponema pallidum* (which causes syphilis), *Leptospira interrogans* (serotype *icterohaemorrhagiae*) (Weil’s disease, which is transmitted to human hosts from infected rats) and *Borrelia burgdorferi* (Lyme disease).

**Figure 1.1** Bacterial morphology I

All bacteria are unicellular, but their size and shape vary widely (Figure 1.1). Specimens must be ‘fixed’ (killed) and stained before they can be examined with the light microscope. Advances in electron microscopy have made it possible to study the ultrastructure of cells. The cells are, however, still dead because examination must be performed with the specimens in a vacuum. The image that appears does not represent the dynamic, living state.

**Ultrastructure**

The bacterial cell ultrastructure differs from that of multicellular organisms. The cells of multicellular organisms are ‘eukaryotic’ (that is, they have a true nucleus). Their genetic material is enclosed in a membrane to form this nucleus. Numerous cytoplasmic organelles (minute, subcellular structures in the cytoplasm that perform specific functions in a cell) are also present, a few exceptions being membrane bound. In contrast, bacteria are ‘prokaryotic’ (lacking a true nucleus and nuclear membrane). The chromosome containing the genetic material (nucleic acid) lies directly in the cytoplasm, as do all the organelles, including...
the ribosomes (sites of protein synthesis) and storage granules. The mesosome, an infold of the outer membrane, is the site of respiration, analogous to the eukaryotic mitochondria.

Figure 1.2 depicts a ‘typical’ bacterial cell, although few species display all the possible features shown. Some species (for example *N. gonorrhoeae*) possess hair-like processes called ‘pili’ used to attach the bacterium to a potential host, while other, highly motile forms (for example *Salmonella* and *Proteus*) have one or more flagella (Figure 1.3). However, all bacterial species are surrounded by a rigid cell wall, giving the cell support and protecting its contents. This is absent in eukaryotic cells. Some bacteria have a mucous capsule around the cell wall, reducing the risk of desiccation in dry conditions. Strains of *Klebsiella* equipped with a mucous capsule are particularly likely to contribute to cross-infection and to result in outbreaks of disease because they survive well on dry skin (Casewell and Desai, 1983).

![Figure 1.2 The ‘typical’ bacterial cell](image-url)
The Gram stain reaction

In the natural condition, bacteria are colourless. The Gram staining reaction is used in the first step of laboratory identification (see below).

**Figure 1.3** Bacterial morphology II

**INFORMATION BOX 1.1**

**The Gram stain reaction**

- A thin film of the specimen is smeared onto the surface of a glass microscope slide.
- The slide is passed through the flame of a Bunsen burner 3–4 times to ‘fix’ (kill) the microorganisms.
- The slide is covered with purple dye (methyl or crystal violet) for 15 seconds, the excess fluid then being poured away.
The slide is flooded with Gram’s iodine for up to 1 minute, after which the iodine is drained.

The slide is flooded with acetone for 2–5 seconds before being washed with water or ethanol to rinse away any dye not taken up by the bacteria.

The bacteria are counterstained by pouring a red dye (carbol fuchsin) onto the slide for 20 seconds.

The slide is blotted dry and is then ready for examination. Gram-positive organisms retain the violet dye and appear deep purple. Gram-negative bacteria stain pink because they lose the violet stain, taking up the red counterstain instead.

Examples of typical Gram-positive and Gram-negative bacteria are shown in Table 1.2.

*Mycobacterium* does not respond well to Gram staining because the thick, waxy cell wall is impermeable to the dyes. It can be identified by the acid-fast (Ziehl–Neelsen) staining technique. *Mycobacterium tuberculosis* (tuberculosis) is thus described as being ‘acid fast’ or as the ‘acid-fast bacillus’ (AFB).

### Table 1.2 Typical Gram-positive and Gram-negative bacteria

<table>
<thead>
<tr>
<th>Gram-positive bacteria</th>
<th>Gram-negative bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Bacillus</em></td>
<td><em>Acinetobacter</em></td>
</tr>
<tr>
<td><em>Clostridium</em></td>
<td><em>Bacteroides</em></td>
</tr>
<tr>
<td><em>Corynebacterium</em></td>
<td><em>Escherichia coli</em></td>
</tr>
<tr>
<td><em>Staphylococcus</em></td>
<td><em>Haemophilus</em></td>
</tr>
<tr>
<td><em>Streptococcus</em></td>
<td><em>Klebsiella</em></td>
</tr>
<tr>
<td></td>
<td><em>Neisseria</em></td>
</tr>
<tr>
<td></td>
<td><em>Proteus</em></td>
</tr>
<tr>
<td></td>
<td><em>Salmonella</em></td>
</tr>
<tr>
<td></td>
<td><em>Vibrio</em></td>
</tr>
<tr>
<td></td>
<td><em>Yersinia</em></td>
</tr>
</tbody>
</table>

The Gram stain reaction is valuable because it distinguishes structural differences between Gram-positive and Gram-negative bacteria and provides an indication of their behaviour. Much of the difference between the two groups is explained by a variation in the chemical composition of the cell wall (see Chapter 4). Gram-positive bacteria tend to be more resistant to desiccation (dehydration) and tolerate dry conditions. Gram-negative species thrive in damp situations and are generally more resistant to antibiotics. Few species of Gram-positive bacteria are flagellated, so they lack motility.

### Spore formation

*Clostridium* and *Bacillus* form spores under adverse conditions. A thick, protective capsule surrounds the cell, and its metabolism slows. In favourable conditions, the spore germinates, releasing the bacterium. Spores are very resistant to heat and desiccation, remaining viable over long periods. The ability to form spores that will survive in adverse environmental circumstances is restricted to the Gram-positive species. The spores of *Bacillus anthracis* (causes anthrax) and *Clostridium tetani* (causes tetanus) survive dormant for years, able to withstand extremes of temperature and exposure to disinfectants that would destroy vegetative cells. Germination occurs when conditions become favourable for growth and reproduction.
Oxygen requirement
Bacteria display a variety of oxygen requirements (Table 1.3):

- Obligate aerobes – their growth demands an environmental oxygen supply
- Obligate anaerobes – those unable to tolerate the presence of oxygen
- Facultative aerobes – can grow whether or not oxygen is available.

Table 1.3 Oxygen requirements of some medically important bacteria

<table>
<thead>
<tr>
<th>Oxygen requirement</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aerobic/facultatively aerobic</td>
<td>Campylobacter</td>
</tr>
<tr>
<td></td>
<td>Escherichia coli</td>
</tr>
<tr>
<td></td>
<td>Klebsiella</td>
</tr>
<tr>
<td></td>
<td>Neisseria gonorrhoeae</td>
</tr>
<tr>
<td></td>
<td>Neisseria meningitidis</td>
</tr>
<tr>
<td></td>
<td>Proteus</td>
</tr>
<tr>
<td></td>
<td>Salmonella</td>
</tr>
<tr>
<td></td>
<td>Shigella</td>
</tr>
<tr>
<td>Anaerobic</td>
<td>Bacteroides</td>
</tr>
<tr>
<td></td>
<td>Clostridium</td>
</tr>
<tr>
<td></td>
<td>Treponema pallidum</td>
</tr>
</tbody>
</table>

Establishing infection
Before infection is possible, a susceptible host must encounter a virulent microorganism. The pathogen must complete the following stages:

- Gain access to the host tissues
- Move to a favourable site
- Multiply successfully in spite of the defence mechanisms mustered by the host
- Reproduce so that new pathogens can escape to be disseminated, thus completing the life cycle.

Invasion: portals of entry
Invasion occurs by inhalation or ingestion, via the urogenital (urinary and genital) tracts, by inoculation and by vertical transmission:

- **Inhalation** occurs via the respiratory tract, the nose or mouth being the route taken by colds and influenza viruses and organisms causing tuberculosis, diphtheria and the infections of childhood (measles and mumps). Infectious airborne particles are released as aerosols. Droplet transmission only occurs when an individual with an infectious respiratory condition exhales forcefully, sneezes or coughs. Only the smallest particles (1–5 µm) can reach the lower airways. The length of contact between the source and the potential new case increases the risk of transmission. This is because the longer the period of exposure, the greater the risk of inhalation.
Ingestion via the mouth into the gastrointestinal tract occurs when food or water is contaminated. *Salmonella*, *Shigella*, *Campylobacter*, *Vibrio* and the virus causing poliomyelitis enter by being ingested.

The urogenital tract is the route taken by pathogens causing sexually transmitted infections (*N. gonorrhoeae*, *T. pallidum* and *Trichomonas vaginalis*). Urinary pathogens, principally Gram-negative bacilli, gain access via the urethra.

The inoculation of pathogens via the skin or mucous membranes can occur during surgical incision, accidental injury or injection with a needle (hepatitis B, hepatitis C and human immunodeficiency virus – HIV), or via the mouthparts of an insect (*Plasmodium* following a mosquito bite).

Vertical transmission occurs via the placenta from the maternal to the fetal circulation (for example rubella virus and *T. pallidum*) or by contamination as the fetus travels down the birth canal at parturition. *N. gonorrhoeae* and other microorganisms can be transferred to the eyes of the infant from an infected mother in this way, resulting in ophthalmia neonatorum (pus discharging from the eyes of an infant commencing within 21 days of birth). *Chlamydia trachomatis* can cause serious respiratory and eye infections in babies exposed to the organism during birth. Women infected with HIV may transmit the infection to the fetus via the placenta, in breast milk or at parturition when the infant is exposed to contaminated blood and cervical secretions. A baby may develop shingles if its mother had varicella (chickenpox) during pregnancy (Enders et al., 1994).

Virulence

The ability to establish an infection depends on virulence. Several factors contribute, including the size of the inoculating dose and the ability to invade host tissues and damage them.

Size of the inoculating dose

Except in the case of very virulent pathogens, a large number of microorganisms is more likely to overwhelm the host defences, and there is a greater chance that at least some will reach a site suitable for growth and multiplication. Most pathogens invade specific sites. *N. gonorrhoeae* invades the delicate cervical and urethral epithelia but not the tough squamous epithelial cells lining the mouth or vagina. Viruses responsible for colds invade the nasal epithelium and conjunctivae but not the oral mucosa.

Ability to invade host tissues

This depends on the bacterium’s morphological characteristics and its production of enzymes and toxins.

Morphological characteristics

Pili on the surface of *N. gonorrhoeae* allow it to attach to epithelial cells on the cervix uteri and urethra. Mutant strains without pili lack virulence. The presence of a
protective mucous capsule surrounding the cell wall reduces the risk of desiccation in particular strains of Gram-negative bacteria, so they survive longer on the hands and are more likely to cause cross-infection (Cooke et al., 1981).

Enzyme production

Enzyme production is a property of many bacteria. Staphylococci, streptococci and Clostridium perfringens release haemolytic enzymes, which destroy erythrocytes (red blood cells). Staphylococcus aureus releases an enzyme called ‘coagulase’, which clots plasma, thus protecting the bacteria from phagocytosis (Chapter 2).

Toxins

Toxins are of two types, depending on the mechanism of synthesis and secretion:

1. **Exotoxins** are secreted by Gram-positive bacteria and released outside the cell into the surrounding extracellular fluid, dissolving and being carried throughout the tissues. Exotoxins destroy host cells or inhibit specific metabolic functions. They include some of the most lethal chemicals known. The exotoxin secreted by Clostridium botulinum (which causes botulism) interrupts the transmission of nervous impulses, paralysing the victim. Clostridium tetani releases an exotoxin that excites neurones in the central nervous system. The muscular spasms of ‘lockjaw’ result. Exotoxins released by Staphylococcus aureus and Bacillus cereus result in food poisoning.

2. **Endotoxins** develop as part of the cell wall of Gram-negative bacteria. They include Salmonella enterica serovar Typhi (which causes typhoid), Neisseria meningitidis (meningococcal meningitis) and Shigella sonnei (dysentery). The release of endotoxins corresponds with the symptoms of fever and malaise experienced by the host.

Ability to damage host tissues

The ability to damage host tissues is closely related to the ability to invade. Damage may be structural (the tissues being physically destroyed) or physiological (normal function becoming disturbed). In most cases, both types occur. Staphylococcus aureus destroys tissue because the infection causes abscess formation. Pyrexia occurs simultaneously with this.

**Bacterial growth requirements**

Knowledge of bacterial growth requirements is essential when attempts are made to grow and identify organisms in the laboratory. Bacteria are unicellular and therefore more susceptible to environmental fluctuations than larger, more complex multicellular organisms. As with higher forms of life, their growth requirements include:
■ Water

■ An energy source

■ A suitable pH

■ A suitable temperature

■ Protection from ultraviolet rays.

Water

Water accounts for more than 80 per cent of the bacterial cell volume and is essential for the growth and survival of vegetative bacterial cells. Some Gram-positive species (for example *Bacillus* and *Clostridium*) avoid desiccation by forming resistant spores under adverse conditions.

Energy source

Nourishment is derived from substances available within the environment. Bacteria vary enormously in their ability to utilize different sources of nourishment:

■ Phototrophs use carbon dioxide as their sole source of carbon to synthesize all the complex organic molecules they need. Like plants, they obtain their energy from sunlight.

■ Chemotrophs obtain energy by oxidizing inorganic material.

■ Heterotrophs require a supply of nutrients such as carbohydrates or amino acids produced by other organisms. Most pathogens are heterotrophs. Generally speaking, the more adapted the organism is to a strictly pathogenic existence, the more demanding its growth requirements (for example *Pseudomonas*, *E. coli* and *Klebsiella*). In contrast, *N. gonorrhoeae* has complex growth requirements and cannot survive long outside the human host. *T. pallidum*, which is even more fastidious, has never been cultured outside living tissues.

Bacteria also vary in their ability to use sources of energy during respiration (see Oxygen requirement, above):

■ Obligate aerobes (for example *M. tuberculosis*) are unable to grow in the absence of oxygen.

■ Facultative aerobes are tolerant of the presence of free atmospheric oxygen in their environment and will grow whether or not it is available. Most human pathogens belong to this group.

■ Obligate anaerobes cannot grow unless all traces of oxygen are removed from their environment. They tend to cause infections deep within the tissues. *Clostridium* spp. cause gangrene and tetanus, infections originating when the bacteria gain access to the deep tissues.

■ Microaerophilic bacteria grow more rapidly in the presence of only traces of free oxygen.
A suitable pH

Bacteria vary widely in their tolerance of acidic or alkaline conditions, ranging from pH 4 to 9. Human pathogens generally prefer a pH within the range 7.2–7.6, but there are exceptions. Cholera vibrios, for example, thrive best at pH 8. They affect the small intestine, which receives pancreatic fluid at the same pH. Lactobacilli (part of the normal flora) inhabiting the vagina grow best at a pH of about 4.

A suitable temperature

All species have a preferred temperature range, but within this there is an optimum temperature at which they grow best:

- **Mesophilic** bacteria thrive within the 25–40 °C range. Human pathogens fall into this group, thriving optimally at 37 °C.
- **Psychrophilic** bacteria grow best at approximately 20 °C and slowly at 4 °C. They influence health not by causing infection, but by their ability to spoil food that has not been properly refrigerated.
- **Thermophilic** bacteria, growing at temperatures of 55–90 °C do not operate as human pathogens.

Protection from ultraviolet rays

Most pathogenic bacteria grow best in darkness and are rapidly destroyed by ultraviolet light, whether it is natural, in sunlight or arising from an artificial source. This is the rationale behind ‘airing’ clothing in the sun as it dries.

Bacterial reproduction and genetics

Bacteria reproduce asexually by simple binary fission, or by sexual reproduction in which there is transfer of genetic material.

Binary fission

Binary fission is a simple, asexual process involving the division of a bacterial cell into two genetically identical daughters. The rate of binary fission depends on the particular species and the environmental circumstances. In ideal conditions (for example a warm, damp hospital ward), a typical Gram-negative bacillus such as *E. coli* will divide about once every 20 minutes. Others, for example *M. tuberculosis*, divide very slowly. The results of laboratory tests for *E. coli* are available within 24 hours, but a diagnosis of tuberculosis may not become available for weeks. Treatment for tuberculosis is, however, started on the basis of clinical findings and other tests, for example skin tests, radiography and the presence of AFBs in a sputum specimen.

Asexual reproduction does not involve the exchange of genetic material so there can be no provision for genetic variation, a disadvantage as the organisms are thus limited in their ability to respond and adapt to environmental pressures.
Sexual reproduction

Sexual reproduction is, however, possible in particular bacteria containing a small amount of extrachromosomal deoxyribonucleic acid (DNA) lying within the cytoplasm. This is called a ‘plasmid’. It accounts for approximately 1 per cent of the total amount of genetic material present in those cells which contain it. A transfer of genetic material between bacteria is possible according to three mechanisms: conjugation, transduction and transformation (Figure 1.4).

(a) Conjugation

Conjugation (Figure 1.4a) is an important means of genetic exchange, particularly among Gram-negative bacilli. Sex pili coded by the DNA of a donor or ‘male’ cell attach to the recipient or ‘female’ cell. Plasmid replication follows, one copy passing to the recipient, the other remaining within the cytoplasm of the donor.

(b) Transduction

Extrachromosomal genetic material from another bacterium carried by a bacteriophage

(c) Transformation

Extrachromosomal genetic material absorbed into the bacterial cell cytoplasm

Figure 1.4 Sexual reproduction in bacteria (diagrammatic)
Transduction

Transduction (Figure 1.4b) occurs when a bacteriophage (a viral parasite of bacteria) invades a bacterial cell. Bacteriophages (or phages) operate in a manner similar to that of conventional viruses, entering the bacterium and replicating to release a large number of new infective agents, which in turn attack more bacteria. Transduction results when new phages carry extrachromosomal genetic material from the old host to a new one that previously lacked a plasmid.

Transformation

Transformation (Figure 1.4c) takes place when a strand of extrachromosomal DNA is absorbed via the cell wall into the cytoplasm of a bacterium.

Sexual reproduction in bacteria is of great clinical significance as genes conferring antibiotic resistance can be exchanged, resulting in the emergence of antibiotic-resistant strains (see below). The widespread, indiscriminate use of antibiotics encourages the survival of bacteria carrying plasmids conferring antibiotic resistance on their hosts (Chapter 4).

PRACTICE APPLICATION 1.2

Plasmid-mediated Antibiotic Resistance

Plasmid-mediated antibiotic resistance occurs between enterococci. Plasmids carrying the genes for vancomycin resistance can also spread between enterococci and other more virulent bacteria, including *Staphylococcus aureus* (Tenover et al., 2004).

Commonly, resistance to glycopeptide antibiotics (vancomycin and teicoplanin) occurs in the bowel commensals *Enterococcus faecium* and *Enterococcus faecalis*. These enterococci are known as glycopeptide-resistant enterococci (GRE) or vancomycin-resistant enterococci (VRE).

**Activity**

> Consider the implications of a vancomycin-resistant gene transferring from enterococci to meticillin*-resistant *Staphylococcus aureus* (MRSA). For example, how it will influence the choice of antibiotic therapy for infections caused by *Staphylococcus aureus*.

* The new British Approved Name (BAN) for methicillin.

Escape and dissemination

In many cases, bacteria leave the body via the entry route, but there are exceptions. Those causing gastroenteritis gain access via the mouth and leave in the faeces, thus being said to be disseminated by the faecal-oral route.

Microorganisms are spread from one individual to the next by direct and indirect contact. Dissemination is also possible via the airborne route, in contaminated food and water, and by insects.
Contact

Contact is the major route of spread in hospital and probably in the community too (Gould, 1991).

In hospital, bacteria are spread chiefly on the hands of staff because patients and equipment are handled so frequently, increasing the number of opportunities for cross-infection. Ignaz Semmelweiss first demonstrated the relationship between handwashing and a reduction in infection rate in a series of epidemiological studies in the 1840s. Since this time, controlled trials in hospital have been notable by their absence because withholding hand decontamination would be ethically and aesthetically undesirable (Larson, 1988). There is, however, a wealth of indirect evidence to implicate hands as vectors of cross-infection.

Persuasive evidence is provided by Casewell and Phillips (1977), who demonstrated that the hands of staff in an intensive care unit were contaminated with Klebsiella of the same strain as those colonizing and infecting the patients. Laboratory studies indicated that the bacteria could remain viable for up to 150 minutes following artificial inoculation onto the hands of volunteers – ample time for cross-infection to occur during normal nursing activities. Clothing, air and ward dust were seldom contaminated with the same strains, confirming earlier views that Gram-negative bacteria are not readily disseminated by the airborne route (Noble et al., 1976). In later studies within the same unit, the rate of cross-infection declined following the introduction of a strict regimen of hand decontamination (Casewell and Phillips, 1977).

In the community, there is evidence that many pathogens traditionally thought to rely on droplet spread are in fact disseminated by contact (Worsley et al., 1994). Laboratory simulations demonstrate that individuals are more likely to develop upper respiratory tract infection after contact with hands and objects (fomites) contaminated with the virus than after exposure to virus-laden aerosols (Gwaltney et al., 1978). It has been suggested that coughing and sneezing release infected droplets that settle onto surfaces, including clothes, in the immediate environment. Hands then transfer them to other objects (crockery, door handles and so on), reaching new victims after their hands have in turn become contaminated. The virus reaches the nose and conjunctivae when the face is touched. Hand hygiene can reduce the incidence of upper respiratory tract infection.

Similarly, rotavirus, responsible for vomiting and diarrhoea, although released in droplets, appears to be spread by hand contact. In an experimental incidence study conducted in a day nursery, a reduction in the rate of infection was demonstrated when handwashing was promoted among children and the staff attending them (Black et al., 1981). It is worth remembering that handwashing is a simple and cost-effective way to reduce infection (Gould, 1997) (see below).

PRACTICE APPLICATION 1.3

Handwashing

Handwashing is the most effective infection control measure, but it is performed too seldom by hospital staff, often because they are too busy. Hands should be washed even when gloves are worn because virus particles can leak through and contamination can occur as the gloves are removed (Gould, 1994).
Hand hygiene is equally important in the community, where it can be more difficult to achieve. For example, difficulties arise when a large number of people are seen quickly in clinics and health centres (Gould, 1997).

**Activity**

- Reflect on the frequency of your own handwashing when dealing with patients, clients or residents.
- Have there been occasions when it was difficult to wash your hands?

### Airborne spread

Airborne spread occurs only over short distances for Gram-positive pathogens and for viral infections such as chickenpox. An extensive review of the literature confirms that cross-infection by this route is unusual outside high-risk environments such as operating theatres and burns units (Ayliffe and Lowbury, 1982). In theatre, skin scales laden with staphylococci gain access to open tissues, often by landing on the drapes from the air. They may originate from either the patient or the attendants. The airborne route is also important in burns units. The skin is the body's chief defence against bacteria, and when it is no longer intact, patients become extremely susceptible to infection.

### Contaminated food and water

Contaminated food readily operates as a vehicle for bacteria. Such infection is the result of poor hygiene in homes, restaurants, fast-food outlets, shops and factories (North, 1989). In most cases, contamination occurs via the hands. *Salmonella* contaminating the fingers from infected food sources can survive handwashing. Spread is therefore by the faecal-oral route.

Waterborne spread occurs in areas where sanitation is poor. Cholera is endemic throughout much of the developing world, including Asia, but outbreaks rarely occur in the UK. Typhoid is also transmitted via contaminated water. Legionnaires’ disease (caused by the bacterium *Legionella pneumophila*) is disseminated in contaminated aerosols (Woo et al., 1986); outbreaks of this have occurred in the UK.

### Insect vectors

Insect vectors disseminate infection by mechanical and biological transmission. Mechanical transmission occurs when pathogens are transferred from one locality to another via the surface of the insect, often on its feet. Houseflies operate as mechanical vectors for *Shigella* (Cohen et al., 1991). In hospital, flies, Pharaoh’s ants and other arthropods may carry pathogenic bacteria present within the clinical environment (Fotedar et al., 1992).

Biological transmission involves a complex interaction between pathogen and vector. *Plasmodium*, the agent responsible for malaria, multiplies within the gut of the mosquito, increasing the number of protozoa available to contribute to an infective dose. Transmission occurs when the insect bites a human host.
Reservoirs of infection

Reservoirs of infection develop when favourable conditions promote the growth and reproduction of a large number of bacteria. Reservoirs may develop on the skin of staff, patients or residents, leading to cross-infection. The contribution of environmental reservoirs to cross-infection depends on their situation. A large reservoir of bacteria in a drain is unlikely to contribute to healthcare-associated infection (HCAI) because there are few opportunities for transfer to susceptible individuals, but if the reservoir involves objects that have the potential for contact with patients, residents or staff, the risks are considerable.

Viruses

Viruses are the smallest microorganisms known to be infective agents. They vary in size between 10 and 300 nm, being visible only under the electron microscope. Each virus particle consists of a core of nucleic acid – either DNA or ribonucleic acid (RNA) but never both (Table 1.4). The nucleic acid is surrounded by a protein coat (or capsid) to protect it from adverse environmental conditions (Figure 1.5). Prions (see above) are less complex structures that consist of proteins but no nucleic acids. ‘Enveloped’ viruses are surrounded by a lipid and protein capsule with structures permitting them to attach to their hosts. Attachment is always at specific sites on the cell surface for which the virus has particular affinity. For example, the influenza virus attaches itself to mucoprotein receptors. Viruses lacking a capsule are described as ‘naked’. Viruses are classified by their shape and by the type of nucleic acid they contain – DNA or RNA.

![Figure 1.5 The structure of a typical virus](image)
## Table 1.4 Examples of some medically significant viruses

<table>
<thead>
<tr>
<th>Viruses</th>
<th>Diseases/conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DNA viruses</strong></td>
<td></td>
</tr>
<tr>
<td>Adenoviruses</td>
<td>Sore throat, conjunctivitis</td>
</tr>
<tr>
<td><strong>Herpes viruses</strong></td>
<td></td>
</tr>
<tr>
<td>Herpes simplex types 1 and 2 (HSV-1, HSV-2)</td>
<td>Cold sores, genital infections</td>
</tr>
<tr>
<td>Varicella zoster virus (VZV)</td>
<td>Chickenpox (varicella), shingles (herpes zoster)</td>
</tr>
<tr>
<td>Epstein–Barr virus (EBV)</td>
<td>Glandular fever (infectious mononucleosis),</td>
</tr>
<tr>
<td>Cytomegalovirus (CMV)</td>
<td>Burkitt’s lymphoma</td>
</tr>
<tr>
<td></td>
<td>Cytomegalovirus infection</td>
</tr>
<tr>
<td><strong>Hepadnavirus</strong></td>
<td></td>
</tr>
<tr>
<td>Hepatitis B virus (HBV)</td>
<td>Hepatitis B</td>
</tr>
<tr>
<td><strong>Papovaviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Human papilloma virus (HPV)</td>
<td>Warts, tumours (for example cervix)</td>
</tr>
<tr>
<td><strong>Poxvirus</strong></td>
<td></td>
</tr>
<tr>
<td>Smallpox virus</td>
<td>Smallpox (variola)</td>
</tr>
<tr>
<td><strong>RNA viruses</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Picornaviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Enteroviruses, poliovirus, echoviruses, coxsackie viruses</td>
<td>Poliomyelitis, respiratory infection</td>
</tr>
<tr>
<td>Rhinovirus</td>
<td></td>
</tr>
<tr>
<td>Hepatitis A virus (HAV)</td>
<td>Common cold (coryza)</td>
</tr>
<tr>
<td></td>
<td>Hepatitis A</td>
</tr>
<tr>
<td><strong>Togaviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Flaviviruses</td>
<td></td>
</tr>
<tr>
<td>Rubella virus</td>
<td>Yellow fever, dengue, West Nile fever, hepatitis C</td>
</tr>
<tr>
<td></td>
<td>German measles (rubella)</td>
</tr>
<tr>
<td><strong>Reoviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Reovirus</td>
<td></td>
</tr>
<tr>
<td>Rotavirus</td>
<td>Respiratory tract infection, gastroenteritis</td>
</tr>
<tr>
<td><strong>Caliciviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Norovirus</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td><strong>Rhabdovirus</strong></td>
<td></td>
</tr>
<tr>
<td>Rabies virus</td>
<td></td>
</tr>
<tr>
<td><strong>Arenavirus</strong></td>
<td></td>
</tr>
<tr>
<td>Lassa virus</td>
<td>Lassa fever</td>
</tr>
<tr>
<td><strong>Orthomyxovirus</strong></td>
<td></td>
</tr>
<tr>
<td>Influenza viruses</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Influenza</td>
</tr>
<tr>
<td><strong>Paramyxoviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Parainfluenza virus</td>
<td></td>
</tr>
<tr>
<td>Respiratory syncytial virus (RSV)</td>
<td>Parainfluenza</td>
</tr>
<tr>
<td>Mumps virus</td>
<td>Respiratory infection</td>
</tr>
<tr>
<td>Measles virus</td>
<td>Mumps (infectious parotitis)</td>
</tr>
<tr>
<td></td>
<td>Measles (morbilli)</td>
</tr>
<tr>
<td><strong>Retrovirus</strong></td>
<td></td>
</tr>
<tr>
<td>Human immunodeficiency virus (HIV-1, HIV-2)</td>
<td>HIV disease</td>
</tr>
<tr>
<td>Human T cell lymphotropic viruses (HTLV-I, HTLV-II)</td>
<td>Leukaemia</td>
</tr>
<tr>
<td><strong>Filoviruses</strong></td>
<td></td>
</tr>
<tr>
<td>Ebola virus</td>
<td>Ebola fever</td>
</tr>
<tr>
<td>Marburg virus</td>
<td>Marburg fever</td>
</tr>
</tbody>
</table>
Viruses are responsible for a wide range of human, animal and plant infections. Some, called ‘bacteriophages’ (phages), attack bacteria. Viruses depend on living organisms to provide a host; they are not capable of growth or reproduction outside living cells. Lacking cellular structure and the characteristics of living organisms, they may occupy the ‘grey’ zone between animate and inanimate organisms, perhaps resembling life as it first appeared on earth. It is, however, more likely that they represent degeneration into highly successful and sophisticated parasites. Their existence as the earliest form of ‘life’ in the absence of potential victims is hard to explain.

**Life cycle**

The virus gains entry by ‘endocytosis’ (a bulk transport process that transfers material into cells) and is carried into the cytoplasm in a vacuole via the cell membrane (plasma membrane), leaving its protein capsule redundant on the cell’s surface (Figure 1.6). Viral nucleic acid is then released to take over the genetic

![Figure 1.6 The life cycle of a typical virus](image_url)
machinery of the host cell. Viral DNA becomes incorporated into the DNA of the host, assuming command of genetic control. The host synthesizes viral proteins rather than its proteins, so that new virus particles are generated and eventually released, completing the life cycle. RNA viruses use the enzyme ‘reverse transcriptase’ to manufacture DNA templates of their own RNA for incorporation into the genome of the host. Some viruses lie dormant within the host cell for long periods of time but can become activated to produce active infections, a good example being herpes zoster (shingles).

**Viruses and malignancy**

The earliest relationship between viruses and malignancy was demonstrated in 1908 when it was established that, in poultry, a certain type of leukaemia could be transmitted to previously healthy birds from those with the disease. It is now known that viruses are responsible for malignancies in many animals, and they appear to play a role in the development of some human cancers. There is an established association between the human papilloma virus (HPV) and cervical cancer, the Epstein–Barr virus and Burkitt’s lymphoma, and the hepatitis viruses and hepatocellular cancer (Campbell, 2006). A vaccine against HPV types 6, 11, 16 and 18 has been licensed for use for females aged 9–26 years. In the UK, the vaccine against the HPV is to be added to the routine NHS immunization programme (Department of Health, 2007). It will be routinely offered to girls aged 12–13 years from the autumn of 2008, with a later catch-up programme for girls aged up to 18 years.

**Fungi**

Fungi are classified independently of plants and animals. Over 300,000 species are known but like bacteria, most are harmless saprophytes. Approximately 200 species cause human disease. In common with other microorganisms, some fungi (for example *Candida albicans*) can cause opportunistic infections in people who are immunocompromised (Arkell, 2003), especially those with a malignant disease (Krcmery and Barnes, 2002). *Aspergillus* species can cause severe, frequently fatal infections in people who are already immunocompromised (Kibbler, 2003). All fungi are eukaryotic, and because of the similarities between fungal and mammalian cells, it has never been easy to develop antifungal agents. The drugs used to treat fungal infections are often highly toxic, and few are available without a prescription. Some fungi, for example yeasts, assume a simple structure and exist as single cells, but complex forms exist with filamentous hyphae branching to form an extensive interwoven mesh called a ‘mycelium’ (Figure 1.7). These forms are visible to the naked eye, but as microscopic examination is necessary for identification, the diagnosis of fungal infection (mycosis) is made in the microbiology laboratory.

There are three types of mycosis:

1. **Superficial mycoses** occur when infection is superficial or restricted to the skin and its appendages (hair and nails), for example athlete’s foot (*Trichophyton interdigitale*), or mucous membranes, as in the case of vaginal thrush (*Candida albicans*).
2. **Subcutaneous mycoses** (for example mycetoma) affect the skin, subcutaneous tissues and bone. Slow, localized spread occurs.

3. **Systemic mycoses** (caused by, for example, *Cryptococcus*) develop, and then the hyphae penetrate the deeper tissues. In temperate climates, systemic mycoses are uncommon except in the immunocompromised patient.

**Figure 1.7** Fungal morphology

Table 1.5 gives examples of fungi that may cause human disease.

<table>
<thead>
<tr>
<th>Fungus</th>
<th>Mycosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Candida albicans</em></td>
<td>Thrush (candidiasis/candidosis)</td>
</tr>
<tr>
<td><em>Trichophyton interdigitale</em></td>
<td>Athlete's foot (tinea pedis)</td>
</tr>
<tr>
<td><em>Cryptococcus neoformans</em></td>
<td>Meningitis (immunocompromised patients)</td>
</tr>
<tr>
<td><em>Microsporum audouini</em></td>
<td>Ringworm (commonly affecting the scalp)</td>
</tr>
<tr>
<td><em>Aspergillus fumigatus</em></td>
<td>Respiratory infection (immunocompromised patients)</td>
</tr>
</tbody>
</table>

**Protozoa**

Protozoa are unicellular, microscopic animals (Figure 1.8). Most species are harmless, but some operate as human pathogens, especially in hot climates. Others are a threat to the immunocompromised host (Table 1.6). *Plasmodium*, the protozoan responsible for malaria, is discussed in Chapter 14.
Figure 1.8 Pathogenic protozoa

<table>
<thead>
<tr>
<th>Protozoan</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Trichomonas vaginalis</em></td>
<td>Vaginal infection</td>
</tr>
<tr>
<td><em>Plasmodium</em> spp.</td>
<td>Malaria</td>
</tr>
<tr>
<td><em>Trypanosoma rhodesiense</em>, <em>Trypanosoma brucei gambiense</em></td>
<td>Trypanosomiasis (some types known as ‘sleeping sickness’)</td>
</tr>
<tr>
<td><em>Leishmania donovani</em></td>
<td>Leishmaniasis – kala-azar (generalized visceral form)</td>
</tr>
<tr>
<td><em>Entamoeba histolytica</em></td>
<td>Amoebic dysentery</td>
</tr>
<tr>
<td><em>Toxoplasma gondii</em></td>
<td>Latent infection, damage to fetus in utero</td>
</tr>
</tbody>
</table>

Rickettsiae and chlamydiae

These microorganisms bridge the gap between viruses and bacteria. Like viruses, they are small and rely on their hosts to grow and reproduce, but they are susceptible to antibiotics. Typhus, caused by *Rickettsia prowazeki*, is spread by human head and body lice. *Chlamydia trachomatis*, responsible for nonspecific urethritis (inflammation of the urethra), is discussed in Chapter 13. The microorganism also causes
an eye condition known as trachoma or trachoma inclusion conjunctivitis (TRIC), which can lead to blindness.

**Mycoplasmas**

Mycoplasmas are similar to bacteria but lack cell walls. Without a rigidly supporting outer structure, they change shape readily during growth, often becoming filamentous. The most significant mycoplasma operating as a human pathogen is *Mycoplasma pneumoniae*, which infects the lungs.

**Helminths**

Numerous species of helminths (worms) give rise to human infestation. Some are large and multicellular, others microscopic (Figure 1.9). There are two main groups: round and flat (Table 1.7). It is impossible to cover all types and with that in mind only the threadworm will be discussed.

<table>
<thead>
<tr>
<th>Helminth</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Enterobius vermicularis</em></td>
<td>Round (threadworm/pinworm)</td>
</tr>
<tr>
<td><em>Ascaris lumbricoides</em></td>
<td>Roundworm</td>
</tr>
<tr>
<td><em>Toxocara canis</em></td>
<td>Dog roundworm</td>
</tr>
<tr>
<td><em>Trichinella spiralis</em></td>
<td>Pork roundworm</td>
</tr>
<tr>
<td><em>Necator spp.</em></td>
<td>Roundworm (hookworm)</td>
</tr>
<tr>
<td><em>Strongyloides stercoralis</em></td>
<td>Roundworm</td>
</tr>
<tr>
<td><em>Taenia saginata</em></td>
<td>Beef tapeworm</td>
</tr>
<tr>
<td><em>Taenia solium</em></td>
<td>Pork tapeworm</td>
</tr>
<tr>
<td><em>Schistosoma haematobium</em></td>
<td>Fluke</td>
</tr>
</tbody>
</table>

**Threadworms**

*Enterobius vermicularis*, the threadworm, is probably the most common helminthic parasite in the Western world. Cats, dogs or any other domestic animals do not carry it; humans are the only hosts. The eggs are swallowed, hatch in the small intestine and migrate to the large intestine, where they live. Within two weeks, the worms reach maturity, mate and migrate to the rectum, emerging at night to lay their eggs on the perianal (around the anus) skin. The eggs adhere to the skin by a sticky fluid, which causes intense itching. When the victim scratches, large numbers of eggs are transferred to the hands and fingernails. These are thence transferred back to the mouth, recommencing the cycle of infection. People of any age can become infested with threadworms, but children are the most commonly affected (Blake, 2003). The entire family should be treated, however, as the eggs are easily transferred onto towels, soap and upholstery, and may be ingested with food if it is touched with inadequately washed hands (see Practice Application 1.4). The eggs can survive in the environment for several weeks. Threadworms are not dangerous but they can be a nuisance, causing discomfort, irritability and sleeplessness.
Figure 1.9 Helminthic infestation

(a) **A roundworm** (*Ascaris lumbricoides*)

- mouth
- intestine
- testis
- ovary
- anus

Female Male

(b) **A tapeworm**

- scolex (head)
- sucker
- neck
- hooks
- tapes
- Egg-filled mature tapes exit the body in the faeces

The hooks and sucker anchor the tapeworm to the gut wall of host.
PRACTICE APPLICATION 1.4

Controlling Threadworm Infestation

Control is achieved by:

➤ All household members taking one of the proprietary antihelminthic agents such as piperazine, which paralyses the worms, or mebendazole, which starves them by preventing sugar absorption. These preparations are available over the counter but instructions must be followed carefully.

➤ Good handwashing before eating and after using the lavatory and scrubbing of the nails, which should be kept short.

➤ Vacuuming the house (carpets and upholstery) to remove eggs and avoid reinfection.

➤ Avoiding sharing towels and flannels. Towels, flannels and bed linen should be frequently laundered.

SELF-ASSESSMENT

1. Most bacteria and fungi are pathogenic. True? or False?
2. Which of the following are typical opportunists?
   (a) Staphylococcus aureus
   (b) Candida albicans
   (c) Legionella pneumophila
   (d) None of these
3. Infection always causes an elevation in body temperature. True? or False?
4. Which of the following are bacilli?
   (a) Pseudomonas
   (b) Staphylococcus epidermidis
   (c) Mycobacterium pneumoniae
   (d) Treponema pallidum
5. What is virulence?
6. Staphylococcus aureus never forms spores, even under dry conditions. True? or False?
7. Most microorganisms are disseminated by the airborne route. True? or False?
8. Diseases caused by viruses include influenza, rubella and hepatitis A. True? or False?
9. Plasmodium is a protozoan. True? or False?
10. List the measures taken to control threadworm infestation.
REFERENCES


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**FURTHER READING AND INFORMATION SOURCES**


Health Protection Agency (UK agency) – www.hpa.org.uk/.

INFECTION PREVENTION AND CONTROL

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