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Some examples of bacterial, fungus, and viral infections are listed in Table 6.2.1. All can be aggressive life-threatening infections or nonhostile relatively mild short-term infections. In response to all infectious agents, the body generates antibodies or generates resistance to infection agent. In all cases, bacteria, fungi, and viruses infect one cell or one tissue and then start to spread around the body infecting multiple other cells or tissues, in some cases killing humans. One common symptom of most bacterial, fungal, and virus infections is fever.

Fever or temperature is regulated in the hypothalamus of the brain. A trigger of the fever, called a pyrogen, causes a release of the large folded lipid prostaglandin E2. Prostaglandin E2 acts on the hypothalamus causing heat-creating effects to match a new temperature level. The hypothalamus works like a thermostat. When the set point is raised, the body increases its temperature through both active generation of heat and retaining heat. Vasconstriction reduces heat loss through the skin and causes the person to feel cold. If these measures do not make the blood temperature in the brain match the new setting, then shivering begins in order to use muscle movements to produce heat.

A pyrogen is a substance that induces fever. These can be either internal or external to the body. The bacterial sugary fat substance lipopolysaccharide, present in the cell wall of bacteria, is an example of a pyrogen. In extreme examples bacterial pyrogens can themselves cause rapid and dangerous fevers. All pyrogens, bacteria, fungi, and viruses produce cytokines, molecules that are a part of the immune system. These can cause the increase in the thermoregulation set point in the hypothalamus.

Symptoms of infection depend very much on the type of disease. General symptoms of infection include fatigue, loss of appetite, weight loss, fevers, night sweats, chills, aches, and pains. The classic symptoms of a bacterial infection are local redness, fever, swelling, and pain at the site of infection. In general, viral infections are systemic, involving many different parts of the body at the same time; i.e., a runny nose, cough, and body aches.

Antibacterial, antimycotic, and antiviral drugs have been developed that generally function by
blocking bacteria, fungi, and viruses from replicating and surviving. Penicillin as an antibacterial, for instance, functions by inhibiting synthesis of bacterial cell walls. Examples of penicillin drugs include amoxicillin and ampicillin. Bacteria constantly try to rebuild their peptidoglycan cell wall. Penicillin blocks the formation of peptidoglycan cross-links in the cell wall. The four-membered \( \beta \)-lactam ring of penicillin binds to the enzyme transpeptidase in bacteria. As a consequence, transpeptidase cannot catalyze formation of these cross-links. The imbalance between cell wall production and degradation causes the bacteria cells to rapidly die.

Streptomycin or streptomycin sulfate is an antibacterial; it is a ribosome protein synthesis inhibitor. It binds to the small ribosomal RNA (rRNA), 30S component of the bacterial ribosome causing codon misreading, eventual inhibition of protein synthesis and bacterial replication, and ultimately death of bacterial cells. Humans have structurally very different ribosomes from bacteria, thereby allowing the selectivity of this antibiotic for bacteria. Tetracycline is an antibacterial that also functions by binding the small, 30S, bacterial ribosome, and blocking codon reading and bacterial replication.

A third type of antibacterial is fluoroquinolones. These include drugs such as Cipro and Levaquin. These are synthetic, bactericidal antibacterial agents with broad-spectrum activity. They inhibit the enzyme topoisomerase II, a DNA gyrase that is necessary for the replication of the microorganism. Topoisomerase II enzyme produces a negative supercoil on DNA, permitting transcription or replication and thereby inhibiting enzyme DNA replication and transcription. Blocking transcription blocks any possibility of replication.

Unlike most antibiotics, antiviral drugs do not destroy their target microbe; in its place they inhibit their progress and development of viruses. Antivirals should be distinguished from viricides, which are usually used a disinfectants rather than medications, but deactivate or destroy virus particles, either inside or outside the body. One modern antiviral strategy is to design drugs to interfere with a virus’s ability to infiltrate a target cell. The virus must go through a sequence of steps to do this, starting with binding to a specific “receptor” molecule on the surface of the host cell and ending with the virus “uncoating” inside the infiltrated cell and releasing its contents.
A very early stage of viral infection is viral entry, when the virus attaches to and enters the host cell. A number of “entry-inhibiting” or “entry-blocking” drugs are being developed to fight virus infection. Inhibitors of uncoating have also been investigated. Amantadine and rimantadine have been introduced to combat influenza. These agents act on this penetration and uncoating process. Pleconaril works against rhinoviruses, which cause the common cold, by blocking a pocket on the surface of the virus that controls the uncoating process. This pocket is similar in most strains of rhinoviruses and enteroviruses, which can cause diarrhea, meningitis, conjunctivitis, and encephalitis.

The problem today with antibiotics or antibacterials, antifungicides, and antiviruses is resistance. Antimicrobial resistance is resistance of a microorganism to an antimicrobial drug that was originally effective for treatment of infections caused by it.

Resistant microorganisms (bacteria, fungi, viruses, and parasites) are able to withstand attack by antimicrobial drugs so that standard treatments become ineffective and infections persist, increasing the risk of spread to others. The evolution of resistant strains is a natural phenomenon that occurs when microorganisms replicate themselves erroneously or when resistant traits are exchanged between them.

When resistance occurs it is necessary to form modified drugs to avoid resistance. For instance, ampicillin and amoxicillin are variants of penicillin to work around resistance of common infections.

CHAPTER QUIZ

1. Penicillin kills bacteria and prevent them from replicating by:
   a. Binding their 30S ribosome and blocking protein production
   b. By binding their DNA topoisomerase and blocking it

INTERNET REFERENCES

Use these references to find out more.

en.wikipedia.org/wiki/Antibiotics
en.wikipedia.org/wiki/Antiviral_drug
en.wikipedia.org/wiki/Infection
en.wikipedia.org/wiki/Fever
en.wikipedia.org/wiki/Penicillin
en.wikipedia.org/wiki/Quinolone
en.wikipedia.org/wiki/Streptomycin

CHAPTER QUIZ

Answer these questions. Do you know the answers, holy cow! Check yourself out in this quiz, and prove that you are a humanology wiz.

1. Penicillin kills bacteria and prevent them from replicating by:
   a. Binding their 30S ribosome and blocking protein production
   b. By binding their DNA topoisomerase and blocking it
c. Binding the key proteoglycan blocking replication
d. Binding the cell isomerase receptor and blocking cell replication
e. Binding the membrane transpeptidase and preventing cell wall formation

2. Syphilis is caused by Treponema Pallidum, which is a:
a. Virus
b. Fungus
c. Bacterium
d. Protozoan
e. Prion

3. Fever is generally caused by:
a. The hypothalamus
b. The thyroid
c. The kidney
d. Blood macrophages
e. Blood T-cells

4. Streptomycin kills bacteria and prevent them from replicating by:
a. Binding their 30S ribosome and blocking protein production
b. Binding their DNA topoisomerase and blocking it
c. Binding the key proteoglycan blocking replication
d. Binding the cell isomerase receptor and blocking cell replication
e. Binding the membrane transpeptidase and preventing cell wall formation