

INFECTIONS ASSOCIATED WITH SPORTS

This chapter identifies many of the diseases that the sport physician/coach/athlete is likely to encounter, along with their appropriate therapies. Keep in mind that this is not a comprehensive list of all possible diseases or symptoms. Part 1 presents details of infectious organisms and antimicrobials; in Part 2 the focus is on specific diseases.

Infection in a specific organ can be caused by organisms such as bacteria, viruses, protozoas, fungi, prions, rickettsia, chlamydia and infestation by helminths (worms). There is a wide spectrum of severity including cases that are asymptomatic. Some of these are self limiting and require no treatment

The physician/coach may encounter a wide variety of infectious conditions. Some are quite symptomatic, such as various forms of food poisoning or respiratory illness, but may not require antibiotics. Others are more insidious, for example urethral symptoms may indicate potentially serious sexually transmitted disease requiring detailed diagnosis and complicated therapy.

A. Overview

Infections in athletes may be classified into several categories that vary considerably in their specific relation to sports participation. These include: 1) sports-associated; 2) lifestyle-associated; 3) travel-associated; and 4) general population-related.

1. Sports-Associated Infections or Complications

These infections or complications result directly from participation in sports.

- a. Skin infections are especially common. These include abrasions, cellulitis, and furuncles due to skin damage. Fungal infections, such as tinea and other dermatophytes, affect the inter-triginous areas between the toes (“athlete’s foot”) and the crural areas. Blisters are subject to secondary infections.
- b. Wound infections may occur from puncture wounds (such as from spikes), lacerations, or other deep tissue injuries.
- c. The danger of tetanus must always be considered when there is a deep puncture wound.
- d. Decreased immunity has been associated with intensive training. This may render the athlete more susceptible to a wide variety of infections, especially viral.
- e. Worsening or complications of infections may occur if physical activity persists in the face of an infectious process, particularly viral ones. Examples include myocarditis, pericarditis, and toxic shock with sepsis.

2. Infections Associated with Lifestyle

Infections associated with lifestyle are those to which the athlete becomes predisposed because of participation in sports.

- a. Group living situations such as those associated with team membership, travel and housing, or living in an athletes' village with athletes from many regions, facilitate the spread of infections by droplets, contact, and common-source outbreaks (e.g. food).
- b. Promiscuous behaviour is facilitated by travel opportunities and group living, and peer pressure increases the risk of exposure to many sexually-transmitted diseases.

3. Travel-Associated Infections

Travel markedly increases the possibility of contact with different infectious agents that are more prevalent in the new geographic locale. In addition, contact with other athletes and individuals from throughout the world at major athletic meetings exposes the athlete to many unfamiliar organisms. In either case, the athlete likely lacks adequate immunity to most of these “new” pathogens.

4. General Population Related Infections

The athlete is at least as susceptible, if not more so, to any pathogen that is present in the community at any given time. However, even a “minor” infection can impair the effectiveness of an athlete who is expected to function at peak efficiency.

5. Management

The extent to which an infectious disease can be evaluated and treated will vary considerably depending upon the level of diagnostic technology available, and the range of therapeutic agents that can be obtained.

The circumstances that exist at the time as well as the potential seriousness of the illness will determine the urgency with which diagnosis and treatment will be undertaken. If a critical competition is upcoming, there is usually a tendency to undertake a much more aggressive approach to diagnosis, and to initiate empiric therapy sooner.

B. Sport Hygiene, General Health, and Preventive Measures

1. Healthy Lifestyle

Infectious disease cannot always be prevented. Furthermore, the outcome of an infection depends not only on the specific therapy used but also on the immunologic condition of the individual. Thus, attention to proper diet and nutrition status is important. Athletes should have a sensible attitude about sufficient rest and sleep, and the compatibility of a normal sex life with intense physical activity. Athletes should also be aware of the ill effects of tobacco, alcohol, and drug abuse. (See Appendix 11, *General Health and Hygiene: Recommendations for Athletes.*)

2. Basic Personal Hygiene

High level physical activity necessitates meticulous care of the skin in order to prevent viral (warts), bacterial, and fungal infections. Physical workouts lead to excessive sweating, so regular washing with soap is mandatory. Athletic clothing should be suitable for prevailing weather conditions and fit properly to avoid chafing and abrasions. Soiled clothing should be laundered between workouts. Athletes should be aware, however, of possible allergic reactions and contact dermatitis due to materials or dyes used in apparel, soaps, detergents, and deodorants. Improperly fitted footwear can cause blisters that may become secondarily infected.

The effect of poor dental health on performance should be pointed out and the need for proper dental care, hygiene and examination stressed.

Traveller's diarrhea is a common problem during international travel. Athletes should be aware of the usual causes and basic preventive measures (see Part 2 of this chapter, *Infections at Large*). In general, travellers should avoid tap water and drink only bottled water and beverages. Where necessary, tap water should be boiled or purified with iodine or chlorine. Raw vegetables, undercooked meat or fish, unpasteurised milk and unpackaged foods sold by street vendors should be avoided.

C. Infectious Agents

Infection can be caused by a variety of organisms, including bacteria, viruses, protozoa, fungi, and helminths (worms).

1. Bacteria

Bacteria are in most instances unicellular organisms with well defined nucleus (with the exception of the actinomycetes). They are usually classified according to their mechanism of movement and type of cell walls. A more practical method for sports medicine practitioners is to classify bacteria by their shape (e.g. cocci or bacilli) and staining characteristics (gram-positive or gram-negative). Dependent on the structure of the cell wall, staining characteristics indicate very different biological activity and thus different sensitivity to antibiotics. Refer to Table 12-1 for characteristics and sensitivities of disease-causing bacteria.

Bacteria may affect the human body directly, causing local inflammation and cell destruction, or by means of toxins formed within the body or preformed in ingested food. Virulence may be affected by the size of the inoculum.

Antibiotics are widely used to inhibit biological activities of bacteria (for details, see antibiotics section); however, bacteria often develop resistance to antibiotics by producing enzymes that destroy the drug, changing their permeability to the drug, or altering metabolic pathways. Resistance to antibiotics usually involves genetic changes that can be transferred between diverse bacteria. This means that resistance to new drugs can develop rapidly.

Table 12-1. Characteristics and antibiotic sensitivities of disease-causing bacteria.

Gram-Positive Bacteria**• *Staphylococci***

Found on the skin or noses of most humans. Commonly form a small superficial abscess but have the potential to spread and form large abscesses anywhere in the body. Staphylococci form toxins. 90% of strains are penicillin resistant and 5% methicillin resistant.

• *Streptococci*

A very heterogeneous group of common pathogens. They cause diverse infections such as pharyngitis and pyoderma. Infections may later lead to rheumatic fever and glomerulonephritis. Sensitive to penicillin.

• *Pneumococci*

Cause pneumonia. Sensitive to penicillin.

• *Clostridium perfringens*

Ubiquitous anaerobic bacteria found both in the colon and soil. Frequent cause of food poisoning through the toxin.

• *Clostridium difficile*

Produces both cytotoxin and endotoxin. Cause of antibiotic-associated colitis, most often due to clindamycin but also ampicillin and cephalosporin. Oral vancomycin is the most effective treatment.

• *Clostridium tetani*

Found worldwide in soil. Produces a powerful neurotoxin that causes tetanus. There is a very effective vaccine to protect against it.

Gram-Negative Bacteria**• *Escherichia coli***

A natural inhabitant of the gastrointestinal tract; contributes to normal function and causes no problem in the gut. However, if introduced to other sites in the body it can cause infections. *E. coli* accounts for the majority of urinary tract infection. However, there are specific strains of *E. coli* that can cause diarrhea. They are enterotoxigenic (ETEC), which produces a toxin similar to that of *Vibrio cholerae*, characterised by voluminous watery diarrhea. Enterohemorrhagic (EHEC), enteroinvasive (EIEC), enteroaggregative (EAEC) and enteropathogenic (EPEC) cause bloody diarrhea. No drug is uniformly active against all strains, but ampicillins, cephalosporins, tetracyclines, trimethoprim-sulfamethoxazole and nitrofurantoin may all be useful.

• *Salmonella*

Classification is complex. Over 2000 different serotypes are known. It is important to differentiate between enteric fever caused by *S. typhi* and *S. paratyphi*, and salmonellosis caused by the other serotypes. *S. typhi* is the cause of typhoid fever, usually acquired from contaminated water or food from human carrier. Usually sensitive to chloramphenicol. Other types are called nontyphoidal or *S. enteritidis*. They are commonly found in farm animals and may get into various food products, frequently causing food poisoning. They are usually sensitive to ampicillin, chloramphenicol, third generation cephalosporins, and fluoroquinolones. However, they should not be treated with antibiotics when there are no systemic symptoms

Table 12-1 (cont'd). Characteristics and antibiotic sensitivities of disease-causing bacteria.

- ***Shigella***

A highly communicable human pathogen when orally ingested. It is a major cause of dysentery (blood diarrhea), because of extensive ulceration of the colonic mucosa. They are sensitive to fluoroquinolones and certain cephalosporins, but gaining resistance to trimethoprim/sulfamethoxazole. They are usually resistant to sulfonamides, tetracycline, and chloramphenicol.

- ***Neisseria gonorrhoeae* (gonococci)**

A sexually transmitted disease, causes mainly local infection of mucous membranes but may cause systemic infections. Numerous penicillin resistant strains have appeared.

- ***Neisseria meningitidis* (meningococci)**

Cause meningitis. They are sensitive to penicillin. Outbreaks have occurred among military recruits in camps, but have not been reported among athletes.

- ***Legionella***

Small ubiquitous bacterias found in aquatic environment, first discovered in 1976. Cause pneumonia through inhalation, especially in elderly or debilitated patients. Sensitive to erythromycin.

- ***Vibrio cholerae***

Two biotypes, classic and El Tor, pathogenic only to humans. *V. cholerae* produces enterotoxin that may cause diarrhea up to 20–30L/d. Hydration is the most important aspect of therapy, but antibiotics—particularly tetracycline—may shorten the course.

- ***Vibrio parahaemolyticus***

Present in coastal waters. May cause diarrhea after ingestion of raw or incompletely cooked seafood.

- ***Campylobacter jejuni***

Belong to the intestinal flora of animals. Can contaminate drinking water and cause diarrheal disease in humans.

- ***Yersinia enterocolitica***

Toxin-producing, causing diarrhea. Sensitive to most antibiotics but therapy is rarely needed.

- ***Plesimonas* and *Aeromonas***

Both found in fresh water and have both been associated with diarrhea after ingestion of contaminated fish.

Spirochetes

- ***Trepanoma pallidum***

Causes syphilis. Universally sensitive to penicillin

Rickettsia

Intracellular parasites; numerous types are found throughout the world. They are transmitted through various vectors such as ticks, fleas, or lice. The most fearsome was typhus, now rare. Other types cause Rocky Mountain spotted fever and related diseases. They are sensitive to chloramphenicol and tetracycline.

Mycoplasma

Small, wall-less, fastidious bacteria. *M. pneumoniae* is a common cause of respiratory infections, while *M. hominis* and *Ureaplasma urealyticum* cause non-gonococcal urethritis. They are sensitive to erythromycin and tetracycline, but not penicillin.

Table 12-1 (cont'd). Characteristics and antibiotic sensitivities of disease-causing bacteria.

Chlamydia

Small intracellular parasites, classified as bacteria. *C. trachomatis* was originally known as the cause of trachoma but is now one of the major causes of sexually transmitted diseases. *C. pneumoniae* are recently recognised pathogens causing pneumonia, and are sensitive to erythromycin and tetracycline.

Mycobacteria

Rod-shaped bacteria that have special staining characteristics described as "acid fast". Several species, including *M. tuberculosis*, *M. bovi*, and *M. kansasii* are examples of so-called atypical mycobacteria that are pathogenic for humans. Resistant to chemical agents and can survive for a long time in dried sputum. They infect the host either through the respiratory or GI tract, or the skin. Special groups of antimycobacterial antibiotics in combination therapy are needed for treatment.

2. Viruses

Although classified as microorganisms, viruses differ from all other cellular forms of life. Only 20–300nm in size, they consist of a core of nucleic acid (either DNA or RNA) enclosed in a protein coat, with or without an outer coat.

Inert in the extracellular environment, viruses replicate only in living cells. Host cells usually suffer injury. Many viruses are host specific, pathogenic only to one particular species of animal or plant; other viruses are broadly pathogenic, infecting humans and other animals.

Most infectious viruses enter their hosts through the mucosa of the respiratory or gastrointestinal tract; some enter through the genital tract or directly into the bloodstream during injections.

Viruses vary considerably in size, biologic activity, and virulence. Some viruses can cause tumours or leukemia. Some cause only transient subclinical infections, others chronic persistent infections, and others fulminant infections that quickly lead to death.

Immunologic responses to viruses also differ considerably. Some immune responses result in permanent immunity. Some viruses can be immunised against, others cannot. A few types are sensitive to antiviral agents, but many others are totally resistant.

Viruses are classified in many different ways, including nucleic acid type, size and morphology, immunologic properties, methods of transmission, pathology and symptomatology. Table 12-2 lists some characteristics of the primary disease-causing viruses, the diseases associated with them, drug sensitivities, and immune responses.

3. Protozoa

Protozoa are classified as unicellular animals up to 30 μm in size. Table 12-3 lists the primary disease-causing protozoa.

Table 12-2. Viruses associated with human diseases.

DNA containing viruses**• Parvoviruses**

Cause erythema infection.

• Papovaviruses

Papilloma (wart) viruses, progressive multifocal leucoencephalopathy.

• Adenoviruses

Exist worldwide and are present year-round. May cause diseases in the eye, respiratory, GI and urinary tract. They induce immunity.

• Herpesviruses

Establish lifelong persistent infection in their host and reactivate periodically. They include herpes simplex, varicella-zoster, Epstein Barr (mononucleosis), cytomegalovirus. Herpes virus is sensitive to acyclovir, idoxuridine and related drugs.

• Poxviruses

Cause smallpox, vaccinia, cowpox

• Hepadnaviruses

Cause Hepatitis B.

RNA containing viruses**• Picornaviruses**

Rhinoviruses cause the common cold, enteroviruses (polio, coxsackie), hepatitis A.

• Reoviruses

Rotavirus causes infantile gastroenteritis, orbiviruses cause Colorado tick fever.

• Arboviruses

Cause encephalitis, yellow fever, dengue.

• Togavirus

Causes rubella.

• Arenaviruses

Cause Lassa fever.

• Coronaviruses

Cause acute upper respiratory fever (cold).

• Retroviruses

Cause of sarcoma and leukemia. Human immunodeficiency virus (HIV) is a nononcogenic retrovirus of the lentivirus subfamily. There are 2 types, HIV-1 and HIV-2. The virus has a selective affinity for the CD4 molecule receptor on T helper-inducer lymphocytes. This leads to infection and destruction of the lymphocytes, but the T4 cells have critical role in the human immune response.

• Bunyaviruses

Hantaviruses cause hemorrhagic fevers and nephropathy.

• Orthomyxoviruses

Influenza viruses. Frequent cause of epidemics of respiratory illnesses throughout the world. Three main immunologic types are known: A, B and C. Type A is highly variable antigenically and is the cause of most cases of epidemic influenza. Type B exhibit antigenic changes and cause epidemics. Type C is antigenically stable and causes only mild illness.

• Paramyxoviruses

Viruses of measles, mumps, parainfluenza and respiratory syncytial virus.

• Prions

Cause Creutzfeldt-Jacob disease.

Table 12-3. Protozoa associated with human diseases.

<p>Amoebas Parasites of the large intestine. May cause severe ulceration of the colon leading to “colitis.”</p> <p>Plasmodium (malaria) Four types, <i>P. vivax</i>, <i>P. ovale</i>, <i>P. malariae</i>, and <i>P. falciparum</i>. Infection through bite of infected female mosquito. Multiply in the liver of the host and later infect red blood cells. (See Part 2 of this chapter, <i>Infections at Large</i>, for more information on malaria.)</p> <p>Giardia lamblia Usually transmitted via contaminated water. Infests the duodenum and jejunum, causes diarrhea.</p> <p>Trichomonas Several types occur; <i>T. vaginalis</i> is the only significant human pathogen. Causes infection of vulva, vagina, and cervix in women and prostate, seminal vesicles and urethra in men.</p> <p>Others <i>Cryptosporidium parvum</i>, <i>Isospora belli</i>, <i>Sarcocystis</i></p>
--

4. Fungi

Fungi are classified as a lower form of plants, and some cause superficial, subcutaneous, or systemic infections. Table 12-4 lists the primary fungi associated with human infections.

Table 12-4. Fungi associated with human diseases.

<p>Dermatophytes <i>Tinea pedis</i> (athlete's foot), <i>T. cruris</i> (jock itch).</p> <p>Candida Normal habitant of the genital and gastrointestinal tract where it may gain dominance and cause infection (vaginitis, oesophagitis).</p> <p>Other <i>Coccidioides immitis</i> <i>Histoplasma capsulatum</i> <i>Aspergillus fumigatus</i> <i>Pneumocystis carinii</i></p>
--

5. Helminthes

A variety of parasitic nematodes, cestodes, and trematodes are associated with human diseases. Table 12-5 lists the helminthes most likely to cause human infections.

Table 12-5. Helminthes associated with human diseases.

Nematodes (roundworm)		
Nematodes have a variety of complex life cycles. A dozen are significant human parasites; another dozen occasionally cause infection. Hosts may become infected via food, water, soil (skin penetration), or mosquito bites. Nematodes primarily associated with diseases in humans include:		
<i>Anisakis</i>	<i>Strongyloides</i>	<i>Ascaris lumbricoides</i>
<i>Trichinella</i>	<i>Enterobius vermicularis</i>	<i>Trichuris trichiura</i> hookworms
Cestodes (tapeworms)		
Tapeworms have ribbon-like segments, each with a complete male and female system. All except <i>Hymenolypis nana</i> use an intermediate host. Tapeworms primarily associated with diseases in humans include:		
<i>Diphyllobothrium latum</i> , copepod-fish-human pathway		
<i>Echinococcus</i> , dog, infected herbivore		
<i>Taenia saginata</i> , beef tapeworm		
<i>Taenia solium</i> , pork tapeworm		
Trematodes (flukes)		
Most flukes are hermaphroditic, use an intermediate host, and cause infection by ingestion. Schistosomes are separate-sexed and cause infection by penetrating the skin. Trematodes primarily associated with diseases in humans include:		
<i>Clonorchis sinensis</i>		
<i>Schistosomes</i>		

D. Antimicrobials

Antibiotics are classified either as bactericidal (killing) or bacteriostatic (inhibiting bacterial multiplication). They act on bacteria by four separate mechanisms:

- Inhibition of cell wall synthesis
- Inhibition of cell membrane function
- Inhibition of protein synthesis
- Inhibition of nucleic acid synthesis

In practice, the physician has to select antibiotics based on a clinical diagnosis of which organ is infected and what is the most likely causative organism. Final selection of appropriate antibiotics may depend on sensitivity tests and even serum assays of bactericidal activity. Table 12-6 lists the major classes of penicillins; Table 12-7 lists additional antimicrobial agents and their applications; Table 12-8 lists the relative costs of oral antibacterials.

Table 12-6. Penicillins and cephalosporins.

 β -Lactam Antibiotics (penicillins and cephalosporins)

This class of antibiotics is named for the β -Lactam ring, which interferes with the synthesis of the bacterial wall by binding to specific proteins called penicillin-binding-proteins (PBP). Bacteria can become resistant to these antibiotics by producing β -Lactamases, enzymes that destroy the antibiotics. Penicillins may cause allergic reactions in many patients.

Penicillins

Penicillin G is mainly used parenterally; penicillin V is used orally. Penicillins are active against *S. pneumoniae* and most types of streptococci, *Treponema pallidum* (syphilis) and meningococci. Gonococci and staphylococci are highly resistant to penicillin.

Aminopenicillins

The best known aminopenicillins are ampicillin and amoxicillins (available in oral and parenteral forms), which are active against many gram-negative bacterias such as *E. coli*, in addition to the Gram-positive types. Both penicillins and aminopenicillins can be destroyed by the β -Lactamase produced by gram-positive and gram-negative bacterias. Clavulanate is a β -Lactamase inhibitor that can be combined, for example, with amoxicillin (e.g. Augmentin®) for use against some lactamase-producing bacterias.

Penicillinase-resistant penicillins

Produced for treatment against staphylococcal infection. Methicillin (injectable) was the first produced, but staphylococci are becoming increasingly resistant. The best known oral versions are cloxacillin and dicloxacillin.

Cephalosporins

Cephalosporins also contain a β -Lactam ring, but with a different structural attachment. They are classified into first-, second- and third-generation cephalosporins, depending on activity. Among them are compounds that are active against both gram-positive and negative bacterias, and are available both in oral and parenteral forms.

D. Antiviral Chemotherapy

As viruses are intracellular parasites, antiviral agents must be capable of selectively inhibiting viral function without damaging the host cell.

Nucleoside analogues are the most common antiviral agents, active mainly against herpes viruses. They inhibit nucleic acid replication by inhibiting enzymes of the metabolic pathways for purines or pyrimidines. They include acyclovir, ganciclovir, zidovudine, AZT (which inhibits replication of HIV), and Iioxuridine.

Other antiviral agents include foscarnet and interferon, and amantadine, which blocks viral penetration of the host cell and is effective prophylactically against influenza.

Table 12-7. Additional antimicrobial agents and their applications.

Vancomycin

A glycopeptide active only against gram-positive bacteria, and used mainly parenterally.

Aminoglycosides

E.g. gentamycin, tobramycin and amikacin are active against various gram-negative bacteria and are used mainly parenterally. Streptomycin is used mainly against mycobacteria.

Tetracyclines

Bacteriostatic agents, active against many gram-positive and gram-negative bacteria and used mainly as oral compounds.

Erythromycin

Active against many gram-positive and gram-negative bacteria and is used mainly in oral form. It is particularly useful for patients with penicillin allergy.

Metronidazole

Originally used mainly orally against trichomonas infections, but is also useful for amoebiasis and giardiasis. It is active against various gram-negative bacteria and is particularly effective in intravenous form against anaerobic bacteria.

Sulfonamides and Trimethoprim

Among the earliest antibiotics, their usage has been renewed in combination with trimethoprim (TMP-SMX). This combination has activity against many gram-positive and negative bacteria. Some patients may show severe form of allergic reaction.

Quinolones

E.g. ciprofloxacin and norfloxacin are chemically synthesised antibiotics and have activity both against gram-positive and negative bacteria.

Ethambutol

Active against *M. tuberculosis*.

INH

Active against *M. tuberculosis*.

Rifampicin

Active against *M. tuberculosis*, some gram-positive and gram-negative cocci, some enteric bacteria, chlamydiae, and poxviruses.

Table 12-8. Relative costs of oral antibacterials.

Name of drug	Dosage	Ratio
Doxycycline	100 mg x 1	1
TMP/SMX	160/800 mg x 2	1.2
Phenoxym. penicillin	1000 mg x 3	1.5
Ampicillin	500 mg x 4	2
Dicloxacillin	250 mg x 4	2
Erythromycin	500 mg x 4	3–5
Cephalexin	500 mg x 4	5–10
Cefuroximum	250 mg x 2	5–25
Augmentin	125/500 mg x 3	6–25
Cefpodoximum	200 mg x 2	10–15
Clarithromycin	500 mg x 2	12
Ciprofloxacinum	500 mg x2	13–15

E. Antifungal and Antiparasitic Agents

Table 12-9 lists the major antifungal and antiparasitic agents.

Table 12-9. Major antifungal and antiparasitic agents.

Antifungal Agents	
Amphotericin	Fluconazole
Flucytocine	Clotrimazole
Ketoconazole	Miconazole
Antiparasitic Drugs	
Iodoquinol	Pentamidine
Mebendazole	Chloroquine phosphate
Thiabendazole	Quinidine gluconate,
Albendazole	Q. dihydrochloride, Q. sulfate
Praziquantel	Pyrimethaminesulfadoxine
Bithionol	Mefloquine
Quinacrine	Halofantrine

F. Immunisation

Prevention is better than a cure, and immunisation has been a major advancement in improving human health.

1. Childhood

In most countries, young children are routinely immunised against diphtheria, tetanus and pertussis (DTP), and polio (OPV). In some countries pediatric immunisation has been expanded to also include measles, mumps and rubella (MMR), and Hemophilus and hepatitis B.

2. Adults

Adults should receive tetanus and diphtheria toxoids every 10 years. Combined measles-mumps vaccine should be given to males who have not been immunised or infected, and seronegative females of reproductive age should receive rubella vaccine.

3. Target Groups

New vaccines are continually being developed, but these are currently targeted at specific groups, few of which include active athletes (but perhaps rather the elderly athletic officials!).

Influenza and pneumococcal vaccines are given mainly to the elderly, although circumstances may require administering them to athletes. Hepatitis B immunisation may be considered for sexually active individuals, and immunoglobulin for non-immunised individuals exposed to infections.

4. Travellers

Prior to travel, the physician should ensure that athletes have received routine vaccinations such as tetanus and polio. Requirements for additional immunisation may vary depending on the travel destination. The team physician should refer to guidelines issued by local health authorities.

Hepatitis A is prevalent in many countries. Routine prevention in the past has utilised passive immunisation with Immune Globulin. A Hepatitis A vaccine (Havrix[®]) is available, although it is expensive. Similarly, new, effective typhoid vaccines are now available, though still relatively costly. These include the oral vaccine Ty21A (Vivitif[®]) and typhoid Vi polysaccharide vaccine (Typhim[®] Vi).

References

1. Eriksson, B. O., T. Mellstrand, L. Peterson, P. Renstrom, and N. Svedmyr. Sports Medicine: Health and Medication. London: Guinness Publishers, 1990.
2. Wilson, M. E. A World Guide to Infections. Oxford: Oxford University Press, 1991.
3. World Health Organization. Travel and Health Vaccination Requirements and Health Advice. World Health Organization Publications Center (USA). 49 Sheridan Avenue, Albany, NY 12210 (revised annually).

INFECTIONS AT LARGE

A. Acute Infectious Diarrheal Diseases

Traveller's diarrhea has been defined as the passage of more than 3 unformed stools in 24 hours with discomfort, occurring in a person visiting another country where food contamination may occur. Food or water contamination by bacterial, viral, or protozoal pathogens is the most common cause of traveller's diarrhea (Figure 12-1). In less than 50% of cases can the infectious organism be found in the stool. Of course where the poisoning is due to preformed toxin such as staphylococci, the organism is hardly likely to be found.

Diarrhea usually starts suddenly; it is often watery and, in approximately 20 percent of cases, bloody. Cramps, nausea, vomiting, and fever may also occur. Symptoms commonly start on the third day after arrival and may last for 3–5 days, with a median of 2 days. Diarrhea may last longer than 7 days in 10% of patients and, rarely, longer than 1 month.

Enterotoxin-producing E. coli, a variant of the most common large bowel bacteria, is the most common cause of this condition. The clinical course may vary considerably, from mild troublesome symptoms to fulminant cholera-type disease.

Staphylococcus aureus is the cause of the classic "food poisoning." Symptoms occur 2 to 6 hours after eating contaminated food, but often last less than 10 hours. Infection is caused by ingesting preformed enterotoxin and may occur in more than 75% of those who have eaten the same food. Vomiting is a very prominent feature of this condition.

Clostridium perfringens is also a significant cause of food poisoning, and infections of this type often appear in a microepidemic pattern following ingestion of contaminated meat or poultry. Typically, two or more individuals who have eaten the same food become sick simultaneously 6–12 hours after eating. Cramps are often more prominent in this condition than in *E. coli* infections.

Campylobacter jejuni causes approximately 10% of food poisoning cases. This bacteria is associated with contaminated water and raw milk from domestic animals. The incubation period may be from 2 to 6 days.

Other organisms that can trigger traveller's diarrhea/food poisoning symptoms include:

- *Shigella*, a frequent cause of bloody diarrhea or dysentery.
- *Salmonella* (nontyphoidal). Salmonella infections occur more frequently in the industrialised world than in developing countries, and cases are increasing among young people. Infection is usually from food, such as poultry and eggs.
- *Viruses* of various types, such as rotavirus and Norwalk agent. These have been associated with epidemics in group situations (e.g. camps) and may account for up to a third of the food-poisoning epidemics that have occurred in the U.S. Incubation period is 18–72 hours.

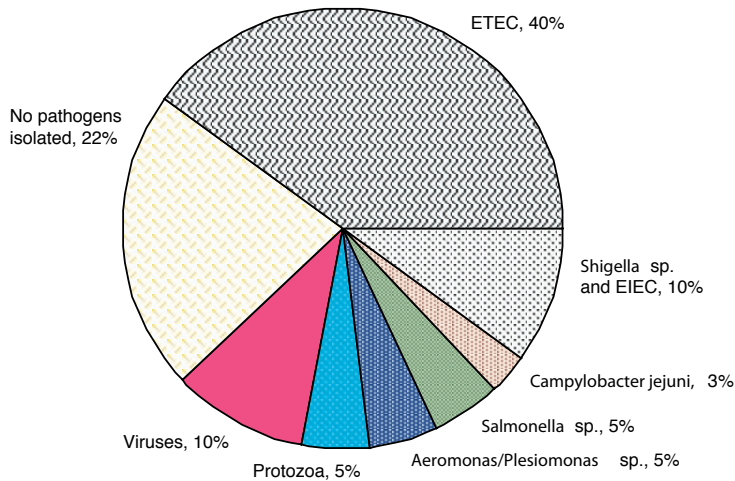


Figure 12-1. Spectrum and approximate prevalence of enteropathogens in traveller's diarrhea. ETEC = enterotoxigenic *E. coli*, EIEC = enteroinvasive *E. coli*.

- *Giardia*, a protozoa that has been associated with waterborne epidemics in the U.S., Russia (former Soviet Union), and developing countries. The incubation period is long (1–3 weeks), and resultant diarrhea may be voluminous, greasy, and floating.
- *Cryptosporidium* and *Entamoeba histolytica* can also cause food poisoning.

1. Prevention

To prevent traveller's diarrhea, athletes should avoid contaminated food, including raw, peeled fruits, vegetables, and other uncooked foods. Water should be bottled or boiled. Bacterial pathogens may survive in food up to 50°C and can be found in ice cubes in drinks.

Many antibiotics as well as bismuth taken prophylactically may decrease the frequency of attacks, but not altogether prevent them. However, antibiotics may at times cause serious adverse effects, and prophylaxis is not recommended except for persons in impaired health or when individuals must undertake a short trip to a high risk area (Figure 12-2).

2. Treatment

Most of these illnesses are clinically mild and self-limiting, and no specific therapy is needed. If the patient has a high fever and frequent bloody diarrhea of any duration, specific diagnosis by stool cultures may be advisable.

Patients should be kept hydrated. In most instances carbonated drinks and salted crackers are sufficient; intravenous fluids are rarely needed. Specific solutions containing electrolytes and glucose should be encouraged. If taken early, bismuth subsalicylate can ease symptoms.

In an athlete with "traveller's diarrhea" and facing an important competition, many antibiotics can shorten the course of illness. These include tetracycline

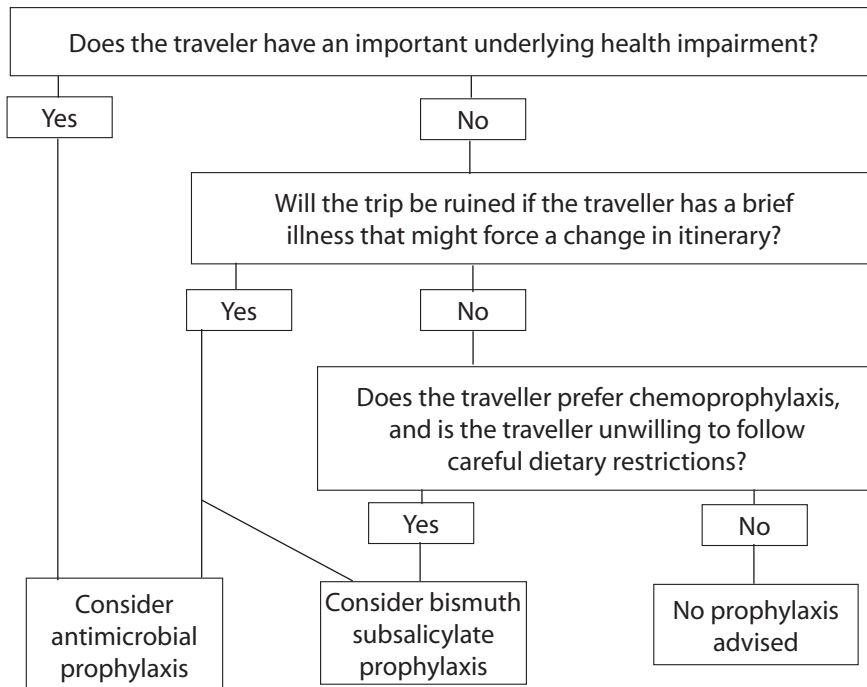


Figure 12-2. Determining the advisability of prophylaxis against traveller's diarrhea during a stay of less than 3 weeks in a high-risk area of Latin America, Africa, or Southern Asia.

products, sulphamethoxazole/trimethoprim, or the quinolone drugs such as ciprofloxacin or norfloxacin. Loperamide or diphenoxylate may relieve abdominal cramps. Antibiotics may be contraindicated in salmonellosis; antiperistaltic agents may be contraindicated in shigellosis.

B. Respiratory Diseases

Acute viral respiratory illness (upper respiratory illness, URI)

These illnesses, which usually involve the upper respiratory tract but can at times also involve the lower respiratory tract, are among the most common afflicting humans. They occur worldwide, with peaks in the fall and spring, and are contagious, spreading by droplets and direct contact. Respiratory illnesses may present with different syndromes, including the common cold, pharyngitis, croup (laryngotracheobronchitis), tracheitis, bronchiolitis, bronchitis, and pneumonia.

Sixty percent to seventy-five percent of respiratory illnesses are caused by one or more of the approximately 200 types of viruses associated with these diseases. The most frequent causative agents are rhinovirus, coronavirus, respiratory syncytial virus, parainfluenza virus, and adenovirus. Clinical symptoms are similar and exact diagnostic methods are rarely necessary.

1. Treatment and Prevention

Respiratory illnesses are usually mild and self-limited. Patients should be kept hydrated, and some may benefit from analgesics and decongestants. Be aware that some formulations contain epinephrine, which is on the list of banned substances. For the athlete facing an important competition, early antibiotic use is a prudent course. Interferon spray applied intranasally may prevent some rhinovirus infections but may also cause irritation. See Appendix 12, *Respiratory Tract Infections*, for additional guidelines on preventing infections, exercise during an episode of respiratory tract infection, and return to exercise following infection.

2. Complications

Physicians should watch for superimposed infections, especially of the sinuses or of the bronchi and lungs, including pneumonia.

Pneumonia

Pneumonia is defined as inflammation of the parenchyma of the lung involving the alveolar units, usually caused by bacteria. Inflammation leads to consolidation of the lung tissue and impairment of gas exchange.

Typically, a pyogenic bacterial pneumonia follows a viral illness and presents with the abrupt onset of chills and fever, a cough with purulent sputum, chest pain, and dyspnea. Symptoms caused by viral or mycoplasma pneumonia may develop more slowly, and pain and respiratory distress may be less prominent.

Pneumococcus is the most common bacteria to be expected in a community-acquired pneumonia in a previously healthy individual. *Mycoplasma pneumoniae* and other types are less frequent and no definite agent is defined in 20–40% of cases.

Examination may reveal a sick individual with high fever, chest pain, dyspnea and tachypnea and abnormal breath sounds on auscultation. Examination of the sputum with Gram stain and cultures and chest radiograph (X-ray) are required for accurate diagnosis.

Treatment

The coach, and even the physician, travelling abroad with an athlete who develops the above-mentioned condition should seek expert local help.

Pneumococcus used to be universally sensitive to penicillin, but in recent years strains have appeared that are resistant to various antibiotics. Sputum culture is therefore very important for specific therapy but high doses of penicillin would still be regarded as the initial drug of choice. If the patient is allergic to penicillin, erythromycin might be considered.

C. Sexually Transmitted Diseases

Athletes must be made aware that any sexual contact without adequate protection might lead to an incapacitating illness. Condom use must be stressed, and condoms should be included in the medical supplies. The most common sexually transmitted pathogens include the following:

- **Bacteria:** *Neisseria gonorrhoea* (gonococcus), *Chlamydia trachomatis*, *Treponema pallidum* (syphilis), *Calymmatobacterium granulomatis* (granuloma inguinale), *Hemophilus ducreyi* (chancroid).
- **Viruses:** Herpes simplex viruses (HSV), Human immunodeficiency viruses (HIV), Hepatitis viruses (HBV).
- **Others:** *Trichomonas vaginalis*, Pubic lice, Scabies, Candida.

The most frequently encountered symptoms of sexually transmitted disease include the following:

- *Urethritis in men.* Classified as gonococcal or nongonococcal (NGU), urethritis presents with burning on urination. Discharge is more prominent with gonococcal infection. NGU is most often caused by chlamydia, but also by HSV. This disease may progress to epididymitis.
- *Lower genitourinary tract infection in women.* Symptoms are mainly burning on urination, vaginal discharge, vulvar irritation, and dyspareunia. Infection may involve the urethra and bladder, vulva, vagina, and cervix and may lead to infections of the uterus and fallopian tubes.
- *Genital ulcers.* Herpes simplex virus is the most frequent cause of genital ulcers, but syphilis and chancroid should also be considered.

1. Treatment

The physician must obtain a careful sexual history and perform a detailed physical examination to assess the possible extent of the infection. STDs are reportable. Whenever a patient presents with symptoms that are thought to be those of STD, multiple coinfections must be suspected. Gram stain and cultures should be done, as well as immunologic studies for chlamydia, syphilis, hepatitis, and HIV.

Assuming that *N. gonorrhoea* and/or *C. trachomatis* are the cause of the STD, the patient should be given a single dose of ceftriaxone 250 mg intramuscularly and doxycycline 100 mg orally twice daily for 10 days.

AIDS

AIDS is caused by the human immunodeficiency virus (HIV). Transmission occurs mainly through sexual contact, infected blood transfusions, and needle sharing among IV drug users (including anabolic steroid users). Infection with the virus affects lymphocytes, causing immunosuppression.

Development of the clinical disease may take 8–10 years from the initial infection. The most common manifestation is infections with unusual opportunistic organisms such as *Pneumocystis carinii* and *Toxoplasma gondii* (protozoa), *Candida albicans* and *Cryptococcus neoformans* (fungi), *Mycobacterium avium* and *Mycobacterium tuberculosis* (bacteria), as well as the better-known bacteria such as Salmonella, Hemophilus, Streptococcus, and Staphylococcus. Kaposi sarcoma is a frequent complication of AIDS, as are various lymphoid neoplasms. Numerous immunologic laboratory tests are now available to diagnose the disease.

Education and behaviour modification remain the cornerstones of prevention, and must be continually stressed to sexually active men and women. Travelling athletes are particularly at risk, especially in areas of high HIV-prevalence. Athletes who may have been exposed to HIV should be tested. Significant advances have been made in the treatment of patients with HIV infection. The cornerstone is a combination of antiretroviral therapy and appropriate therapy of opportunistic infections.

D. Hepatitis

Viral hepatitis is a systemic infection that mainly affects the liver. Five categories of viral agents are presently known; all cause similar clinical illness. The viruses can be distinguished from one another by their antigenic properties. They include A (HAV); B (HBV); Delta agent associated with HBV (HDV); and 2 types of non-A, non-B, one bloodborne (called C); and the other enterically transmitted (called E):

- **Hepatitis A.** HAV is spread almost exclusively by the fecal-oral route, enhanced by poor personal hygiene and overcrowding. Outbreaks have been traced to food, water and shellfish. Incubation lasts from 15–45 days and the virus can then be found in liver, blood, and stool samples. The infection causes the formation of antibodies to the virus (anti-HAV), initially of the IgM class but later IgG. These remain indefinitely and cause lasting immunity to HAV.
- **Hepatitis B.** The major route of HBV infection is through inoculation (via skin or mucous membranes) of infected serum or blood products, but as most body fluids (particularly semen and saliva) contain virus, any intimate contact—especially sexual—may cause infection. Incubation period is from 30–180 days. With HBV, the concentration of antigens and viral particles in blood may reach a very high concentration. Three types of antigens have been identified: hepatitis B surface antigen HBsAg, hepatitis B core antigen HBcAg, and hepatitis Be antigen HBeAg. Of these the HBsAg is the most important. All of these lead to formation of antibodies: anti-HBs, anti-HBc and anti-HBe. By measuring the different antigens and antibodies it is possible to assess the stage of infection and infectivity of the body fluids.
- **Hepatitis D.** The Delta agent or virus is a defective RNA virus that requires the helper function of the HBV virus. In some areas of the world it is endemic among those with hepatitis B and spread by nonpercutaneous means. In other areas it is mainly associated with blood transfusions.
- **Non-A, non-B hepatitis.** Two different types of non-A, non-B have been identified, called C and E:
 - HCV is associated with blood donations. Infection is spread via pooled donor products such as concentrates of blood factors. Products such as albumin and immune globulin constitute no risk because of prior treatment.
 - HEV causes waterborne non-A, non-B hepatitis.

1. Clinical Features

Symptoms of hepatitis are systemic and variable. Patients may experience anorexia, nausea, vomiting, fever, fatigue, arthralgias, myalgias, headache, and cough for 1–2 weeks prior to the onset of jaundice and passing of dark urine for several days. These symptoms may subside with the onset of jaundice, when the patients might have hepatosplenomegaly and adenopathy.

Increase in serum aminotransferases AST and ALT (formerly SGPT and SGOT) may precede rise in bilirubin. The enzymes may reach 400–4000 IU but do not necessarily correlate with the liver injury. Jaundice is visible when the bilirubin rises to 43 $\mu\text{mol/L}$ (2.5 mg/dL). Typical range is 85–340 $\mu\text{mol/L}$ (5–20 mg/dL), usually equally divided between conjugated and unconjugated fractions.

Serologic tests can be used to diagnose the various types of hepatitis. HAV-hepatitis is based on detection of IgM anti-HAV and HBV infection on HBsAg. Diagnosis of non-A, non-B can be made if IgM anti-HAV, HBsAg, and IgM anti-HBc are not present.

2. Course

Almost all previously healthy patients with hepatitis A recover completely, as well as approximately 90% of hepatitis B patients; 10% of patients with hepatitis B experience a more severe course leading to early death, or chronic hepatitis and cirrhosis. Superinfection of delta agent in Hepatitis B may increase the severity. Long-term carriers of HBsAg have an increased risk of hepatocellular carcinoma.

After transfusion-associated hepatitis C a significant number of patients continue to have biochemical abnormalities and histology consistent with chronic hepatitis. Approximately 10% of those who contract hepatitis C may develop cirrhosis after 10 years.

HEV infection often leads to chronic hepatitis.

3. Treatment

No specific therapy is needed for typical acute viral hepatitis. In fulminant hepatitis, complicated supportive measures are necessary, leading even to liver transplant.

4. Prevention

- a. **Hepatitis A.** Active immunisation with vaccine and passive immunisation with IG are available. All preparations of IG contain anti-HAV. Athletes travelling to areas of high risk should strongly consider use of immunisation. When given before exposure or early in the incubation period, immunisation may prevent or attenuate clinically apparent hepatitis A and cause long-lasting passive immunity.
- b. **Hepatitis B.** Vaccine for active immunisation has now been prepared by recombinant DNA technology or from healthy HBsAg carriers. Prior to potential exposure, immunoprophylaxis individuals in high risk groups are given injection at 0, 1, and 6 months. Post-exposure immunisation is given by both HBIG and vaccine.

- c. **Delta hepatitis** can be prevented by giving hepatitis B vaccine.
- d. **Hepatitis C and E.** The efficacy of IG prophylaxis has not been proven.

E. Malaria

Malaria is caused by the protozoon *Plasmodium*, which is transmitted by the bite of the *Anopheles* mosquito, and occasionally by blood transfusion or needle sticks. It is the most serious human parasitic disease, affecting approximately 200 million people and causing over 1 million deaths each year.

1. Epidemiology

Malaria occurs throughout most of the tropics. Four species of the genus *Plasmodium* infect humans: *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*. *Plasmodium falciparum* is predominant in Africa, New Guinea, and Haiti. *Plasmodium vivax* is more common in Central Africa and the Indian subcontinent. The prevalence of both species is approximately equal in South America, east Asia, and Oceania. *Plasmodium malariae* is found in most areas, but is much less common. *Plasmodium ovale* is relatively unusual outside Africa.

Some areas have experienced a resurgence of the disease due to increasing drug resistance. Most cases experienced by U.S. and European travellers are acquired in sub-Saharan Africa. Malaria symptoms may begin as early as 8 days after initial exposure and as late as several months after departure from a malarious area, after chemotherapy has been discontinued. Almost all deaths are caused by *P. falciparum*.

2. Etiology

Human infection begins when a female anopheline mosquito inoculates plasmodium sporozoites from its salivary glands during a blood meal. They are carried via the bloodstream to the liver where they infect hepatocytes, reproduce asexually, and thus create thousands of merozoites. The liver cell eventually bursts and after entering the blood stream the merozoites invade the erythrocytes, beginning the symptomatic infection.

The disease is caused by the direct effects of red cell invasion and destruction and the host reaction to this process. The merozoite gradually grows to occupy most of the red cell, divides, and when the cell ruptures, numerous merozoites are released, which again can infect red cells and repeat the cycle. These cycles occur at 48 or 72 hour (*P. malariae*) intervals.

After invading the red cell, the parasite progressively consumes and degrades intracellular proteins. This leads to sequestration of mature forms of the parasite in vital organs, such as the heart and brain, where they interfere with microcirculatory flow and metabolism and continue to develop away from the principal host defense, i.e., splenic processing and filtration.

The specific immune response to malaria limits the rising parasitemia and, with exposure to sufficient strains, eventually confers protection from disease, but not from infection. The complexity of the immune response in malaria, and the parasite's evasive mechanism, have caused slow progress towards a vaccine.

3. Symptoms

The first symptoms of malaria are nonspecific, and may resemble those of a viral illness. They can include headache, fatigue, and muscle pains followed by fever. Diagnosis rests on identifying the parasite in peripheral blood smears.

The most severe form is seen in *P. falciparum* infection. The patient may have multifactorial anemia, with accelerated red cell destruction, dysfunction of the spleen, and bone marrow suppression. Renal failure may occur, as well as lactic acidosis, hypoglycemia, and coma with convulsions.

Chronic complications (hyperreactive malarial splenomegaly) lead to hypergammaglobulinemia, normochromic anemia and splenomegaly with increased vulnerability to infections.

4. Prevention and Prophylaxis

The Anopheles mosquito feeds primarily between dusk and dawn. Exposure can be minimised by wearing protective clothing, and using repellent (“DEET”) on exposed skin and in impregnated bed nets.

Chemoprophylaxis depends on which area the traveller intends to visit, and the risk of encountering chloroquine-resistant *P. falciparum* (Table 12-10). Up-to-date information should be sought from the appropriate health authority and/or the World Health Organisation.

- a. Chloroquine is recommended for travel to areas of risk where chloroquine-resistant *P. falciparum* has NOT been reported. The drug should be taken for 1–2 weeks before and continued for 4 weeks after travel. Daily doxycycline is also effective against *P. falciparum* but should be used only for areas of mefloquine resistance. It should be taken 1–2 weeks before travel and then for 4 weeks after leaving the area.
- b. Mefloquine is recommended for areas of risk where chloroquine-resistant *P. falciparum* has been reported. The drug should be taken for 1–2 weeks before and continued for 4 weeks after travel. Resistance has been reported in both Asia and South America. A combination of proguanil and dapsone has also been used in areas with chloroquine-resistant strains of *P. falciparum*.
- c. Pyrimethamine-sulfadoxine Fansidar® and proguanil have been used, but resistance has limited their use.

5. Self-treatment

Travellers taking chloroquine alone have been advised to take 3 tablets of Fansidar® if they suspect that they have developed malaria.

6. Treatment

If the team physician suspects the diagnosis of malaria, he or she should at once consult with an infectious disease expert who is familiar with effective drug therapy for the region.

Table 12-10. Malaria chemoprophylaxis according to geographic area.

Regimen	Drug of choice	Alternative Drug
<i>Chloroquine sensitive</i> Central America Caribbean	Chloroquine	proguanil mefloquine
<i>Chloroquine resistant</i> 1. South America Amazon (Brazil) 2. Asia 3. Africa (sub Saharan) 4. SE Asia and Oceania	mefloquine mefloquine mefloquine or mefloquine mefloquine	chloroquine + pyrimethamine sulfadoxine ^a doxycycline ^b chloroquine + pyr/sulfadoxine chloroquine + proguanil + pyr/sulfadoxine ^a doxycycline

^a(Fansidar) Single-dose presumptive therapy when prompt medical attention is not available.

^bDoxycycline is recommended for those staying overnight along the Thai-Cambodian or Thai-Myanmar (Burma) borders.

F. Helminthic Infections

Diverse helminthic pathogens can inhabit the small and large bowel of humans. Helminths are multicellular, multisystem organisms, with complex life cycles. They consist of roundworms (nematodes) and flatworms (platyhelminths), which include flukes (trematodes) and tapeworms (cestodes). Most of them develop in intermediate hosts or soil before they infect humans; some are capable of autoinfections.

Warm temperatures help ova survive for a long time and facilitate the maturation of ova and larvae into infectious forms. Poor sanitation favours the spread of helminths. Infection may be caused by ingestion of contaminated food or water, or invasion of larvae through exposed skin.

The life cycle and sensitivity to therapeutic agents are often similar. In some instances ova are ingested; they develop in the small intestine, where adults reside for the remainder of their lives. Other helminths follow a complex path through the body before coming to reside in the small intestine. Eosinophilia occurs when larvae migrate through tissue, but may not be seen when adult worms are present only in the gastrointestinal tract.

The clinical signs of infection depend on the number of parasites, pathogenicity, and the immune response of the host. Tissue damage can be caused by direct toxic effects or immune responses. The signs and symptoms of infections may differ, and include abdominal pain, diarrhea, and weight loss. The individual can be infected with multiple species of parasites as well as other organisms, which may complicate the picture. In general the diagnosis is made by identifying larvae, ova, or cysts in the feces. In some instances assays have been developed to detect antigens in the stool. Chemotherapy is available for the most of the helminthic pathogens. Successful vaccines have not yet been developed.

Nematodes (roundworm)

Among the most prevalent parasites of the humans are *Ascaris lumbricoides*, *Necator americanus*, and *Ancylostoma duodenale*, which reside in the small bowel and *Trichuris trichiura*, which resides mainly in the colon. In many areas of the tropics residents can be infected with more than one of these parasites. *Ascaris* and *Trichuris* infections occur when ova are ingested in fecally contaminated food and water. Larvae of the hookworm invade the skin. An eruption may be seen at the site of entry. After a series of developmental stages, they pass through the lung, resulting occasionally in symptoms, infiltrates, and eosinophilia. These nematodes then settle down in the intestinal tract. They produce prodigious number of ova, which are excreted.

1. Signs, Symptoms, and Treatments

Infected individuals may complain of abdominal discomfort and diarrhea. As they may also be infected with other enteropathogens, it can be difficult to decide which of the parasites is responsible for their symptoms.

Clumps of *Ascaris* can cause obstruction of the small bowel. Individual ascaris, which can reach more than 30 cm in length, may migrate into the common bile duct or pancreatic duct and cause inflammation. Hookworms can produce abdominal pain, bloody diarrhea, and anemia.

Trichuris trichiura is associated with colonic lesions and chronic bloody diarrhea. These three parasites can produce malnutrition in children. The diagnosis of roundworm infection is made by finding ova in the stool. These parasites are susceptible to a number of agents such as mebendazole and albendazole.

Strongyloides stercoralis is endemic in many tropical areas. Like hookworm, larvae invade through the skin and elicit pulmonary infiltrates with eosinophilia as they migrate through the lungs. *Strongyloides* in the intestine can produce abdominal discomfort, diarrhea, and eosinophilia. Autoinfection can occur. Disseminated hyperinfection may occur in immunocompromised individuals. The diagnosis is made by finding larvae in the stool. Thiabendazole and Ivermectin are effective as treatment.

Trichinella species are prevalent worldwide. The intestinal phase occurs in the first week, after ingestion of meat infected with cysts. The larvae penetrate the small bowel epithelium and develop into the adult stage. This phase is associated with nausea, abdominal pain, and diarrhea, followed by periorbital oedema and muscle aches when larvae penetrate into muscle cells.

A number of additional roundworms should be kept in mind, such as: *Trichostrongylus* species in cattle raising areas; *Capillaria philippinensis* in areas of South East Asia and the Philippines; *Angiostrongylus costaricensis* in scattered areas of Latin America; and *Anisakiasis* in coastal areas.

Trematodes (flukes)

Schistosomiasis is the most prevalent and important of the helminthic infections. Their life cycle is complex, involving snails. They infect their human hosts by

entering the skin, or when they are consumed in uncooked food. *Schistosomiasis mansoni* is endemic throughout Africa and is found in many areas of Latin America and the Middle East. *Schistosomiasis japonicum* is found in Asia.

1. Signs, Symptoms, and Treatments

Adults reside in venules in the mesenteric plexus where they release their ova, causing mucosal inflammation in the intestines with hypertrophy and ulceration. Patients complain of abdominal pain and bloody diarrhea. Eggs reaching the liver produce granulomas, leading to fibrosis, portal hypertension, and hepatosplenomegaly.

The diagnosis is made by finding ova in the stool or in biopsy specimens, or suggested by anti-schistosomal antibodies. Mucosal friability may be seen at colonoscopy. Praziquantel is effective against all forms of schistosomiasis and Oxamniquine against *S. mansoni*.

Several flukes live within the gastrointestinal tract. *Fasciolopsis buski* is acquired when people ingest contaminated raw water plants, resulting in inflammation of the small intestine and epigastric pain, nausea, and diarrhea of varying severity. *Heterophyes heterophyes* and *Metagonimus yokogawai* cause similar symptoms.

Tapeworms

Tapeworms have complex life cycles. Adults live in the intestinal tract of their final host while larvae are found encysted in the tissue of intermediate hosts such as cattle or pigs.

1. Signs, Symptoms, and Treatments

Taenia saginata and *Taenia solium* tapeworms reach great lengths in humans but cause minimal disease. The ova of *Taenia solium* can produce cysts in the brain and other tissues, a condition known as neurocysticercosis, which is an important cause of morbidity in Latin America and in some areas of Asia and Africa.

Humans serve as both the intermediate and definitive host of the dwarf tapeworm *Hymenolepis nana*, and autoinfection is common. Light infections are often asymptomatic, but heavy infections can produce loss of appetite, abdominal discomfort, diarrhea, and anorexia. Niclosamide and praziquantel are active against adult tapeworms in the intestinal tract; praziquantel or albendazole can be used in the treatment of neurocysticercosis.

References

1. Dupont, H. L. Prevention and treatment of traveller's diarrhea. *New Eng. J. Med.* 328(25):1821-7, 1992.
2. Dupont H. L., Z. D. Jaing, P. C. Okhuysen, C. D. Ericsson, et.al. A randomized, double-blind, placebo-controlled trial of Rifamaximin to prevent travelers' diarrhea. *Ann. Int. Med.* 142:805-812, 2005.
3. Dybul M., et al. Guidelines for using antiretroviral agents among HIV-infected adults and adolescents. Recommendation of the Panel on Clinical Practice for

- Treatment of HIV. MMWR Recomm. Rep. 51(RR-7):1, 2002. Updates available at www.aidsinfo.nih.gov.
4. Eriksson, B. O., T. Mellstrand, L. Peterson, P. Renstrom, and N. Svedmyr. *Sports Medicine: Health and Medication*. London: Guinness Publishers, 1990.
 5. Gleeson M., B. Pyne, and R. Callister. The missing links in exercise effects on mucosal immunity. *Exercise Immunology Review* 10:107-128, 2004.
 6. Gulick, R. M. New antiretroviral drugs. *Clin Microbiol Infect* 9:186, 2003.
 7. Hoffman, S. L. Diagnosis, prevention, and treatment of malaria. *In Travel Medicine*, Wolfe, M.S. (ed.). *Med. Clin. N. Am.* 176:1327-1356, 1992.
 8. Kaplan J. E., et al. Guidelines for preventing opportunistic infections among HIV-infected persons—2002. Recommendation of the U.S. Public Health Service and the Infectious Disease Society of America. MMWR Recomm. Rep. 51(RR-8):1, 2003. Updates available at www.aidsinfo.nih.gov.
 9. Margolis H. S., et al. (eds.). *Viral Hepatitis and Liver Disease*. Atlanta/London: International Medical Press, 2002.
 10. Nieman D. C. Exercise and resistance to infection. *Canadian J. of Physiology and Pharmacology* 76:583-580, 1998.
 11. Peters, E. M. Exercise immunology and upper respiratory infections. *Int. Journal of Sports Medicine* 1:S69-S 77, 1997.
 12. Ronsen, O. Prevention and management of respiratory tract infections in athletes. *New Studies in Athletics* 20(3):49-56, 2005.
 13. U.S. Public Health Service. Updated U.S. Public Health Service guidelines for the management of occupational exposure to HBV, HCV, and HIV and recommendations for postexposure prophylaxis. MMWR Recomm. Rep. 50 (RR-11):1. Updates available at www.aidsinfo.nih.gov.
 14. Wilson, M. E. *A World Guide to Infections*. Oxford: Oxford University Press, 1991.