Marfin AA, Hubbs AF, Musgrave KJ, Parker JE. Occupationally Acquired Infections of the Lung. In Stellman JM (ed): Encyclopaedia of Occupational Health and Safety (Fourth Edition). International Labour Office, Geneva, 1998, p. 10.81-10.90.

(IARC 1992). Lung cancer risk was also increased in some, but not all, of these studies. Furthermore, an excess of sinonasal cancer was found in a cohort of workers in isopropanol manufacture using the strong-acid process.

Woodworkers are at increased risk of nasal cancer, in particular adenocarcinoma (IARC 1987). The risk is confirmed for furniture and cabinet-makers; studies on workers in carpentry and joinery suggested a similar excess risk, but some studies produced negative results. Other wood industries, such as sawmills and pulp and paper manufacture, were not classifiable as to their carcinogenic risk. Although carcinogenicity of wood dust was not evaluated by IARC, it is plausible to consider that wood dust is responsible for at least part of the increased risk of nasal adenocarcinoma among woodworkers. Woodworkers do not seem to be at increased risk of cancer in other respiratory organs.

Nasal adenocarcinoma has been caused also by employment in boot and shoe manufacture and repair (IARC 1987). No clear evidence is available, on the other hand, that workers employed in the manufacture of leather products and in leather tanning and processing are at increased risk of respiratory cancer. It is not known at present whether the excess of nasal adenocarcinoma in the boot and shoe industry is due to leather dust or to other exposures. Carcinogenicity of leather dust has not been evaluated by IARC.

Lung cancer has been common among uranium miners, underground hematite miners and several other groups of metal miners (IARC 1988; BEIR IV Committee on the Biological Effects of Ionizing Radiation 1988). A common factor among each of these occupational groups is exposure to α-radiation emitted by inhaled radon particles. The main source of data on cancer following exposure to ionizing radiation is derived from the follow-up of atomic bomb survivors (Preston et al. 1986; Shimizu et al. 1987). The risk of lung cancer is elevated among the atomic bomb survivors as well as among people who have received radiation therapy (Smith and Doll 1982). No convincing evidence, however, is currently available on the existence of an elevated lung cancer risk among workers exposed to low-level ionizing radiation, such as those occurring in the nuclear industry (Beral et al. 1987; BEIR V, Committee on the Biological Effects of Ionizing Radiation 1990). Carcinogenicity of ionizing radiation has not been evaluated by IARC.

An elevated risk of lung cancer among painters was found in three large cohort studies and in eight small cohort and census-based studies, as well as eleven case-control studies from various countries. On the other hand, little evidence of an increase in lung cancer risk was found among workers involved in the manufacture of paint (IARC 1989b).

A number of other chemicals, mixtures, occupations and industries which have been evaluated by IARC to be carcinogenic to humans (IARC Group 1) do not have the lung as the primary target organ. Nonetheless, the possibility of an increased risk of lung cancer has been raised for some of these chemicals, such as vinyl chloride (IARC 1987), and occupations, such as spraying and application of insecticides (IARC 1991a), but the evidence is not consistent.

Furthermore, several agents which have the lung as one of the main targets, have been considered to be possible human carcinogens (IARC Group 2B), on the basis of carcinogenic activity in experimental animals and/or limited epidemiological evidence. They include inorganic lead compounds (IARC 1987), cobalt (IARC 1991b), man-made vitreous fibres (rockwool, slagwool and glasswool) (IARC 1988b), and welding fumes (IARC 1990c).

OCCUPATIONALLY ACQUIRED INFECTIONS OF THE LUNG

Anthony A. Marfin, Ann F. Hubbs, Karl J. Musgrave, and John E. Parker

Although epidemiological studies of occupationally acquired pneumonia (OAP) are limited, work-related lung infections are thought to be declining in frequency worldwide. In contrast, OAPs in developed nations may be increasing in occupations associated with biomedical research or healthcare. OAP in hospital workers largely reflects the prevalent community-acquired pathogens, but the re-emergence of tuberculosis, measles and pertussis in health care settings presents additional risk for health-based occupations. In developing nations, and in specific occupations in developed nations, unique infectious pathogens that do not commonly circulate in the community cause many OAPs.

Attributing infection to occupational rather than community exposure can be difficult, especially for hospital workers. In the past, occupational risk was documented with certainty only in situations where workers were infected with agents that occurred in the workplace but were not present in the community. In the future, the use of molecular techniques to track specific microbial clones through the workplace and communities will make risk determinations more clear.

Like community-acquired pneumonia, OAP results from microaspiration of bacteria that colonize the oropharynx, inhalation of respirable infectious particles, or haematogenous seeding of the lungs. Most community-acquired pneumonia results from microaspiration, but OAP is usually due to inhalation of infectious 0.5 to 10 μm airborne particles in the workplace. Larger particles fail to reach the alveoli because of impaction or sedimentation onto the walls of the large airways and are subsequently cleared. Smaller particles remain suspended during inspiratory and expiratory flow and are rarely deposited in the alveoli. For some diseases, such as the haemorrhagic fever with renal syndrome associated with hantavirus infection, the principal mode of transmission is inhalation but the primary focus of disease may not be the lungs. Occupationally acquired pathogens that are not transmitted by inhalation may secondarily involve the lungs but will not be discussed here.

This review briefly discusses some of the most important occupationally acquired pathogens. A more extensive list of occupationally acquired pulmonary disorders, classified by specific aetiologies, is presented in table 10.26 (see next page).

Occupationally Acquired Infections in Agricultural Workers

In addition to gases and organic dusts that affect the respiratory tract and mimic infectious diseases, several zoonotic (pathogens common to animals and humans) and other infectious diseases associated with rural living uniquely affect agricultural workers. These diseases are acquired by inhalation of infectious aerosols, and are rarely transmitted from one person to another. Such illnesses that occur in agricultural workers include anthrax, brucellosis, Q fever, ornithosis, tuberculosis and plague (table 10.26). Fungal pathogens include histoplasmosis, blastomycosis, coccidioidomycosis, paracoccidioidomycosis and cryptococcosis (table 10.26). Except for the hantaviral diseases, viral diseases are not an important cause of occupational lung disease in agricultural workers.

Some of these infections are thought to be more common but their incidence is difficult to determine because: (1) most infections are subclinical, (2) clinical illness is mild or difficult to

Table 10.26 • Occupationally acquired infectious diseases contracted via microaspiration or inhalation of infectious particles.

Disease (pathogen)	Reservoir	At-risk populations
Bacteria, chlamydia, mycoplasma and rickettsia		
Brucellosis (*Brucella spp.*)	Livestock (cattle, goats, pigs)	Veterinary care workers, agricultural workers, laboratory workers, abattoir workers
Inhalation anthrax (*Bacillus anthracis*)	Animal products (wools, hides)	Agricultural workers, tanners, abattoir workers, textile workers, laboratory workers
Pneumonic plague (*Yersinia pestis*)	Wild rodents	Veterinary care workers, hunters/trappers, laboratory workers
Pertussis (*Bordatella pertussis*)	Humans	Employees of nursing homes, health care workers
Legionnaire's disease (*Legionella spp.*)	Contaminated water sources (e.g., cooling towers, evaporator condensers)	Health care workers, laboratory workers, industrial laboratory workers, water well excavators
Melioidosis (*Pseudomonas pseudomallei*)	Soil, stagnant water, rice fields	Military personnel, agricultural workers
Streptococcus pneumoniae	Humans	Health care workers, agricultural workers, subterranean miners
Neisseria meningitidis	Humans	Health care workers, laboratory workers, military personnel
Pasteurellosis (*Pasteurella multocida*)	Variety of domesticated (cats, dogs) and wild animals	Agricultural workers, veterinary care workers
Respiratory tularaemia (*Francisella tularensis*)	Wild rodents and rabbits	Manual labourers, military personnel, laboratory workers, hunters/trappers, agricultural workers
Ornithosis (*Chlamydia psittaci*)	Birds	Pet shop workers, poultry production workers, veterinary care workers, laboratory workers
TWAR pneumonia (*Chlamydia pneumoniae*)	Humans	Health care workers, military personnel
Q Fever (*Coxiella burnetii*)	Domesticated animals (cattle, sheep, goats)	Laboratory workers, textile workers, abattoir workers, dairy cattle workers, veterinary care workers
Atypical pneumonia (*Mycoplasma pneumoniae*)	Humans	Military personnel, health care workers, institutional workers
Fungi/Mycobacteria		
Histoplasmosis (*Histaplasma capsulatum*)	Soil; bird or bat excrement (endemic to eastern North America)	Agricultural workers, laboratory workers, manual labourers
Coccidioidomycosis (*Coccidioides immitis*)	Soil (endemic to western North America)	Military personnel, agricultural workers, manual labourers, textile workers, laboratory workers
Blastomycosis (*Blastomyces dermatitidis*)	Soil (endemic to eastern North America)	Laboratory workers, agricultural workers, manual labourers, forestry workers
Paracoccidioidomycosis (*Paracoccidioides brasiliensis*)	Soil (endemic to Venezuela, Colombia, Brazil)	Agricultural workers
Sporotrichosis (*Sporothrix schenkii*)	Plant debris, tree and garden plant bark	Gardeners, florists, miners
Tuberculosis (*Mycobacterium tuberculosis, M. bovis, M. africanum*)	Human and non-human primates, cattle	Hard rock miners, foundry workers, health care and laboratory workers, abattoir workers, veterinary care workers, military personnel, tavern workers
Mycobacteriosis other than tuberculosis (*Mycobacterium spp.*)	Soil	Silica-exposed workers, including sandblasters
Viruses		
Hantavirus	Rodents	Agricultural workers, herders, rodent control workers
Measles	Humans	Health care and laboratory workers
Rubella	Humans	Health care and laboratory workers
Influenza	Humans	Health care and laboratory workers
Varicella zoster	Humans	Health care and laboratory workers, military personnel
Respiratory syncytial virus	Humans	Health care and laboratory workers
Adenovirus	Humans	Health care and laboratory workers, military personnel
Parainfluenza virus	Humans	Health care and laboratory workers
Lymphocytic choriomeningitis virus (arenavirus)	Rodents	Laboratory workers, veterinary care workers
Lassa Fever (arenavirus)	Rodents	Health care workers
Marburg and Ebola viruses (filovirus)	Human and non-human primates, possibly bats	Laboratory workers, veterinary care workers, health care workers, cotton factory workers

diagnose because of non-specific symptoms, (3) medical and diagnostic services are rarely available for most agricultural workers, (4) there is no organized system for reporting many of these diseases and (5) many of these are rare diseases in the general community and are not recognized by medical personnel. For example, although epidemic nephritis due to Puumala virus, a hantavirus, is rarely reported in western Europe, serosurveys of agricultural workers have shown a 2 to 7% prevalence of antibody to hantaviruses.

Zoonotic infections in developed nations are decreasing due to active disease control programmes directed at the animal populations. Despite these controls, agricultural workers and persons working in agriculturally related fields (such as veterinarians, meat-packers, poultry-processors and hair/hide workers) remain at risk for many diseases.

Hantavirus Infection

Hantavirus infection resulting in haemorrhagic fever with renal syndrome (HFRS) or epidemic nephritis (EN) has been clinically described among agricultural workers, military personnel and laboratory workers in endemic areas of Asia and Europe for more than 50 years. Infection results from inhalation of aerosols of urine, saliva and faeces from infected rodents. Haemorrhagic illness and decreased renal function develop during most hantavirus illnesses rather than pneumonia, but pulmonary oedema due to increased vascular permeability has been reported in HFRS and EN. The profound pulmonary consequences of hantavirus infections were not fully appreciated until a recent outbreak of Hantavirus Pulmonary Syndrome (HPS) associated with infection with a recently isolated hantavirus in the western United States (Muerto Canyon virus, Four Corners virus, or Sin Nombre virus).

Hantaviruses are members of the *Bunyaviridae*, a family of RNA viruses. Five hantaviruses have been associated with human disease. HFRS has been associated with Hantaan virus in eastern Asia, Dobrava virus in the Balkans, and Seoul virus, which has a worldwide distribution. EN has been associated with Puumala virus in western Europe. HPS has been associated with a newly isolated hantavirus in the western United States. From 1951 to 1983, 12,000 cases of HFRS were reported from the Republic of Korea. Disease incidence in China is reported to be increasing with epidemics in rural and urban centres, and in 1980, 30,500 cases with 2,000 deaths were attributed to HFRS.

Clinical presentation

With the viruses causing HFRS or EN, infection usually results in asymptomatic development of anti-hantavirus antibodies. In people who become ill, signs and symptoms of the early phase are non-specific, and hantavirus infection can be diagnosed only with serologic testing. Slow recovery is common, but a few persons progress to HFRS or EN developing proteinuria, microscopic haematuria, azotaemia and oliguria. Persons with HFRS also develop profound haemorrhage due to disseminated intravascular coagulation, increased vascular permeability, and shock. Mortality in persons with the full HFRS syndrome varies from 5 to 20%.

HPS is characterized by diffuse interstitial pulmonary infiltrates and the abrupt onset of acute respiratory distress and shock. Marked leukocytosis may occur as a result of increased cytokines that characterize hantaviral illnesses. In HPS, mortality may be more than 50%. The incidence of asymptomatic infection or unrecognized HPS is incompletely investigated.

Diagnostic tests

Diagnosis is made by demonstrating the presence of immunoglobulin M or rising titre of immunoglobulin G using highly specific and sensitive indirect immunofluorescence and neutralizing antibody assays. Other diagnostic methods include polymerase chain reaction for viral ribonucleic acid and immunohistochemistry for viral antigen.

Epidemiology

Infection results from inhalation of aerosols of urine, saliva and faeces from infected rodents. Infected rodents do not have any apparent illness. Transmission may occur by percutaneous inoculation of urine, saliva or faeces from infected rodents, but there is no evidence of human-to-human transmission.

Seroepidemiological surveys of humans and rodents have shown that hantaviruses are endemic with a worldwide distribution in rural and urban settings. In endemic rural settings, rodent-human interactions increase when rodents seasonally invade homes or human activity increases in areas with high rodent density. Persons in rural occupations are at greatest risk of infection. In surveys of asymptomatic rural populations in Italy, 4 to 7% of forestry workers, rangers, farmers and hunters had anti-hantavirus antibody, compared to 0.7% of soldiers. In asymptomatic agricultural workers in Ireland and Czechoslovakia, the prevalence of anti-hantavirus antibody was 1 to 2% and 20 to 30%, respectively. Planting, harvesting, threshing, herding and forestry are risk factors for virus infection. Serosurveys in the western United States to determine the occupational risk of hantavirus infection are in progress, but in a study of health care workers (HCWs) caring for HPS patients, no infections were identified. From the first 68 persons with HPS, it appears that agricultural activities in habitats of infected rodents are risk factors for infection. Patients were more likely to hand plow, clean food storage areas, plant, clean animal sheds and be herders. The major reservoir for HPS is the deer mouse, *Peromyscus maniculatus*.

Other affected occupations

In urban settings, the rodent reservoir for Seoul virus is the house rat. Urban workers, such as dockworkers, workers at grain storage facilities, zoo workers and rodent-control workers may be at risk for hantavirus infection. Research laboratories using rodents for research other than hantavirus research have occasionally been unsuspected sources for hantavirus infections of laboratory workers. Other occupations, such as military personnel and field biologists, are at risk for hantavirus infection.

Treatment

Ribavirin has demonstrated *in vitro* activity against several hantaviruses and clinical efficacy against Hantaan virus infection, and has been used to treat persons with HPS.

Public health controls

No vaccine is available for use although there are ongoing efforts to develop live and killed vaccines. Minimizing human contact with rodents and reducing rodent populations in human environments reduces the risk of disease. In hantavirus research laboratories, high-level biosafety facilities limit the risk from virus propagation in cell culture or handling materials with high concentrations of virus. In other research laboratories using rodents, periodic serologic surveillance for hantavirus infection of rodent colonies may be considered.

Lymphocytic Choriomeningitis (LCM)

LCM, like the hantaviral infections, is naturally an infection of wild rodents which occasionally spreads to man. The LCM virus is an arenavirus, but transmission usually occurs by aerosolization. The natural hosts include wild mice, but persistent infection of domestic Syrian hamsters is well documented. Infection is, therefore, possible in most occupations involving the aerosolization of rodent urine. The most recent documented occupational

outbreak of this disease occurred in laboratory personnel exposed to T-cell deficient nude mice persistently infected as a result of inoculation of contaminated tumour cell lines.

Clinical presentation
Most cases of LCM are asymptomatic or associated with non-specific flu-like illness and are, therefore, not recognized. While the respiratory tract is the site of entry, respiratory symptoms tend to be non-specific and self-limited. Meningitis or meningo-encephalitis develops in a small percentage of the patients and may lead to a specific diagnosis.

Diagnostic tests
Diagnosis is usually by serologic demonstration of a rising titre to the virus in the presence of appropriate clinical signs. Virus isolation and tissue immunofluorescence are also occasionally used.

Epidemiology
Approximately 20% of wild mice are infected with this virus. Transplacental transmission of the virus in susceptible rodents leads to T-cell tolerance and congenitally infected mice (or hamsters) who remain persistently infected throughout their lives. Similarly, T-cell deficient mice, such as nude mice, may become persistently infected with the virus. Humans are infected by aerosol transmission. In addition, rodent cell lines can be contaminated with and propagate the virus. Humans usually become infected by aerosols, although transmission may be direct or via insect vectors.

Other affected occupations
Any occupation involving exposure to dusts contaminated with the excreta of wild rodents confers risk of LCM infection. Animal caretakers in laboratory animal facilities, workers in the pet store industry, and laboratory workers working with rodent cell lines may become infected.

Treatment
LCM infection is usually self-limited. Supportive treatment may be necessary in severe cases.

Public health controls
No vaccine is available. Screening of research mice, hamsters and cell lines has limited most laboratory-acquired infections. For T-cell deficient mice, serologic testing requires the use of immunocompetent sentinel mice. The use of routine laboratory safety precautions such as gloves, eye protection and laboratory coats is appropriate. Reducing the number of wild rodents in the human environment is important in the control of LCM, hantavirus and plague.

Respiratory Chlamydiosis

Respiratory chlamydiosis due to *Chlamydia psittaci* is the most frequently reported cause of OAP associated with animal (poultry) slaughter and meat processing. Chlamydiosis and other illnesses are often associated with exposure to ill animals, which may be the only clue to the source and type of infection. Processing infected animals creates aerosols that infect persons who are remote from meat processing, and working near meat-processing plants may be a clue to the type of infection. Respiratory chlamydiosis may be associated with exposure to parrots (psittacosis) or non-psittacine birds (ornithosis). Non-avian sources of *Chlamydia psittaci* are usually not considered potential zoonoses, although spontaneous abortion and conjunctivitis have been reported in humans exposed to sheep and goat strains. Pneumonia due to *C. pneumoniae* is a recently described common cause of community acquired pneumonia distinct from *C. psittaci* infections. Because of its recent discovery, the role of *C. pneumoniae* in OAPs is incompletely investigated and will not be further discussed in this review.

Clinical presentation
Ornithosis varies from mild influenza-like illness to severe pneumonia with encephalitis which, in the preantibiotic era, had a case-fatality rate (CFR) greater than 20%. Prodromal fever, chills, myalgia, headache and non-productive cough may last up to three weeks prior to the diagnosis of pneumonia. Neurologic, hepatic and renal changes are common. Roentgenographic findings include lower lobe consolidation with hilar lymphadenopathy. Clinical suspicion after determining work-related or other exposures to birds is crucial to the diagnosis because there are no pathognomonic findings.

Diagnostic tests
Ornithosis usually results in a high titre of complement fixation (CF) antibody, although early treatment with tetracyclines may suppress antibody formation. A single acute serum titre ≥1:16 dilution of CF antibody with a compatible clinical presentation or four-fold change in CF antibody titre can be used to make the diagnosis. Inappropriately paired serum samples and the high background of Chlamydia antibodies in at-risk groups undermine the utility of antibody assays to diagnose most chlamydial diseases.

Epidemiology
C. psittaci is present in virtually all avian species and is common in mammals. Infection usually results from zoonotic transmission but person-to-person transmission has been reported. Asymptomatic infection is common and up to 11% of agricultural workers without a history of illness have antibodies to *C. psittaci*. Limited outbreaks remain intermittent but pandemics associated with the exotic bird trade most recently occurred in 1930. In the United States, 70 to 100 cases of ornithosis are reported annually, and nearly one-third of these illnesses are occupationally acquired. Most occupationally acquired infections occur in workers in the pet-bird or poultry-processing industries and are related to aerosolization of avian tissue or faeces. In countries where birds are commonly kept as pets and importation quarantines are poorly enforced, outbreaks are more common but occupation is less of a risk factor.

Other affected occupations
Disease most frequently occurs in poultry processing workers, but workers in exotic bird distribution and avian quarantine facilities, breeding aviaries and veterinary clinics are at risk.

Treatment
Tetracycline or erythromycin for 10 to 14 days should be adequate treatment, but clinical relapse is common when treatment is given for an inadequate duration.

Public health controls
In the United States, exotic birds are quarantined for chemoprophylaxis with tetracyclines. Similar methods are used in other countries where an exotic bird trade exists. No vaccine has been developed for ornithosis. Programmes to increase ventilation to dilute aerosol concentration, reduce aerosolization or inhalation of infectious particles, or treat ill birds in commercial processing plants have been instituted, but their efficacy has not been demonstrated.

Brucellosis

Annually, approximately 500,000 cases of Brucellosis occur worldwide, caused by several Brucella species. The pathogenicity

of Brucella infections is dependent upon the infecting species, which tend to have different reservoir hosts. The reservoirs for *Brucella abortus, B. suis, B. melitensis, B. ovis, B. canis,* and *B. neotomae* tend to be cattle, swine, goats, sheep, dogs and rats, respectively.

Brucellosis can result from infection by different routes, including aerosolization. However, most illness results from ingestion of non-pasteurized dairy products from goats. The resulting systemic illness is caused by *B. melitensis* but not associated with specific occupations. Pneumonia occurs in 1% of cases, although cough is a frequent finding.

In developed countries, occupational brucellosis is usually caused by *Brucella abortus* and results from ingestion or inhalation of infectious aerosols associated with placentas of swine and cattle. Subclinical infection is common; up to 1% of agricultural workers have antibodies to *B. abortus.* Illness develops in approximately 10% of infected persons. Unlike illness due to *B. melitensis*, illness associated with *B. abortus* is usually occupationally acquired and is less severe. Persons with acute brucellosis develop high daily fevers, arthralgia and hepatosplenomegaly. In primary brucella pneumonia, pneumonic consolidation is actually rare, and pulmonary findings may include hoarseness or wheezing, hilar adenopathy, peribronchial infiltrates, parenchymal nodules or a miliary pattern. Isolation can be made from bone marrow in 90% of acute cases and from blood in 50 to 80% of cases. Diagnosis can be made serologically with a variety of antibody assays. Tetracyclines should be used for four to six weeks, and rifampin may be added for synergy. Cattle, goat, sheep and swine raisers, dairy workers, slaughterhouse workers, veterinarians and butchers are the primary populations at risk. Brucella testing and eradication programmes have greatly reduced the number of infected animals and have identified those herds which pose the greatest risk for disease transmission. When working with Brucella-infected animals, avoidance or personal protection, especially after abortion or parturition, are the only effective disease control methods.

Inhalation Anthrax

Inhalation anthrax occurs worldwide but is less common than cutaneous anthrax. Anthrax is a systemic illness in many animals and is usually transmitted to humans by percutaneous infection from processing or by eating contaminated meat. Inhalation anthrax is caused by inhalation of spores of *Bacillus anthracis* from the bone, hair or hide of sheep, goats or cattle ("woolsorter's disease") or rarely while processing infected meat. Spores undergo phagocytosis by alveolar macrophages and are transported to mediastinal lymph nodes, where they germinate. This results in a haemorrhagic mediastinitis but rarely presents as primary pneumonia. Illness is characterized by a widened mediastinum, pulmonary oedema, pleural effusions, splenomegaly and rapid progression to respiratory failure. The case fatality rate is 50% or greater despite antibiotics and ventilatory support. Positive blood cultures are common but serologic testing using a blotting immunoassay may be used. Ill persons are treated with high-dose penicillin, or intravenous ciprofloxacin as an alternative in persons allergic to penicillin. Animal breeders, veterinarians, veterinary care workers, hair and hide processors, and slaughterhouse workers are at increased risk. Annual vaccination is available for animals in endemic areas and humans at high risk for disease. Specific control measures against inhalation anthrax include formaldehyde decontamination, steam sterilization or irradiation of hair and hides; prohibiting hide importation from endemic areas; and personal respiratory protection for workers.

Pneumonic Plague

Plague, caused by *Yersinia pestis,* is predominantly a flea-borne disease enzootic in wild rodents. Humans usually become infected when bitten by an infected flea and often develop septicaemia. In the United States from 1970 through 1988, secondary pneumonia from haematogenous spread developed in approximately 10% of septicaemic persons. Animals and humans with pneumonic plague produce infectious aerosols. Primary pneumonia in humans can occur from inhalation of an infectious aerosol created around dying animals with secondary pneumonia. Despite the potential for pneumonic spread, person-to-person transmission is rare and has not occurred in the United States in nearly 50 years. Disease controls include the isolation of persons with pneumonic plague and the use of personal respiratory protection by HCWs. Aerosol transmission to hospital workers is possible, and tetracycline prophylaxis should be considered for anyone in contact with humans or animals with pneumonic plague. A number of occupations are at risk for aerosol transmission, including biomedical and hospital laboratory workers and, in endemic areas, a number of rural occupations, including veterinarians, rodent-control workers, hunter/trappers, mammologists, wildlife biologists and agricultural workers. A killed vaccine is recommended for persons in high-risk occupations.

Q Fever

Caused by inhalation of *Coxiella burnetii,* Q fever is a systemic disease that presents as atypical pneumonia in 10 to 60% of infected persons. Many different isolates of *C. burnetii* produce disease, and theories of plasmid-dependent virulence are controversial. *C. burnetii* infects many domestic animals (e.g., sheep, cattle, goats, cats) worldwide; is aerosolized from urine, faeces, milk, placenta or uterine tissues; forms a highly resistant endospore that remains infectious for years; and is extremely infectious.

Clinical presentation

After a 4- to 40-day incubation period, acute Q fever presents as an influenza-like illness that progresses to an atypical pneumonia similar to *Mycoplasma.* Acute illness lasts about two weeks but may persist up to nine weeks. Chronic illness, predominantly an endocarditis and hepatitis, may develop up to 20 years following acute illness.

Diagnostic tests

Primary isolation of *C. burnetii* is rarely performed because it requires a high level of biosafety containment. Diagnosis is made serologically by demonstrating a CF antibody titre of 1:8 or greater in an appropriate clinical setting or a four-fold change in CF titre.

Other affected occupations

Agricultural (especially dairy and wool), hospital laboratory, and biomedical research workers are at risk for infection.

Treatment

No effective vaccine exists for *C. burnetii.* A two-week course of tetracyclines or ciprofloxacin is used to treat acute illness.

Public health controls

Because of its widespread geographic distribution, numerous animal reservoirs, and resistance to inactivation, personal respiratory protection and engineering controls to contain infectious aerosols are the only effective preventive measures. However, these control methods are difficult to implement in many agricultural settings (e.g., sheep and cattle herding). The early diagnosis of Q fever by medical personnel can be facilitated by education of workers at high risk for contracting this rare disease. Transmission to hospital personnel may occur, and isolation may limit the spread of Q fever pneumonia in hospitals.

Miscellaneous Bacterial OAPS of Agricultural Workers

Pseudomonas pseudomallei is a soil- and rodent-associated organism principally of Southeast Asia which causes melioidosis. The disease is associated with soil exposure and a potentially long latency. Military personnel during and after the Vietnam War have been the major victims of melioidosis in the United States. Multifocal, nodular, suppurative or granulomatous pneumonia characterizes the pulmonary form of melioidosis.

Francisella tularensis, the aetiologic agent of tularaemia, is a zoonosis associated with wild rodents and lagomorphs. This is a potential occupational disease of wildlife biologists, mammologists, rodent-control workers, hunters, trappers and veterinarians. Tularaemia may result from inhalation, direct inoculation, cutaneous contact or ingestion, or it may be vector borne. Pulmonary disease results from either direct inhalation exposure or haematogenous spread of septicaemic disease. The pulmonary lesions of tularaemia are acute, multifocal, suppurative and necrotizing.

Histoplasmosis

Histoplasmosis is caused by *Histoplasma capsulatum,* a free-living mould in the soil associated with the faeces of birds or bats. Histoplasmosis is the most important cause of fungal OAPs in agricultural workers. The miscellaneous fungal pneumonias of agricultural workers are described in the next section.

Clinical presentation

Following exposure, attack rates and severity of histoplasmosis vary as a result of the infecting inoculum and the host's antibody levels conferred by prior infection. Following heavy exposure, up to 50% of persons develop self-limited respiratory illness, while others remain asymptomatic. The least severe of the symptomatic syndromes includes "flu-like" symptoms, non-productive cough, and chest pain. Physical examination may be remarkable for erythema nodosum or erythema multiforme. Chest x rays show patchy, segmental infiltrates but no x ray findings can specifically differentiate histoplasmosis from other pulmonary infections. Hilar or mediastinal lymphadenopathy is common in all stages of primary histoplasmosis.

Progressive primary pneumonic histoplasmosis is characterized by profound systemic complaints, cough productive of purulent sputum, and haemoptysis. Progressive x-ray changes include multiple nodules, lobar consolidation and dense, multilobar interstitial infiltrates. Greater exposures increase the severity of the illness and result in severe respiratory disease, the acute respiratory distress syndrome (ARDS) or atelectasis due to obstruction by mediastinal lymphadenopathy.

Approximately 20% of ill persons develop other histoplasmosis syndromes which are idiosyncratic and not the result of greater exposure or progression of primary disease. Syndromes include arthritis-erythema nodosum, pericarditis, and chronic pulmonary histoplasmosis (fibrotic apical lung infiltrates with cavitation). Disseminated histoplasmosis develops in a small percentage of patients, particularly the immunosuppressed.

Diagnostic tests

Definitive diagnosis is made by isolating or histopathologically demonstrating the organism in an appropriate clinical specimen. Unfortunately, the organism is present in low concentrations and the sensitivities of these methods are low. Presumptive diagnoses are often made on the basis of geographic location, exposure history and x-ray findings of the lungs or calcifications in the spleen.

Epidemiology

H. capsulatum is found worldwide associated with specific soil conditions, but illness is reported primarily from the Ohio and Mississippi River valleys of the United States. High concentrations of spores are found in bird roosts, old buildings, poultry houses, caves or schoolyards; they are disrupted by work activity. Microconidia concentration is higher in disrupted, enclosed areas (e.g., building demolition) and results in higher inoculum for workers there than in most outdoor sites. In endemic areas, persons who clean bird roosts, demolish older contaminated buildings or perform excavations for road or building construction are at greater risk than the general population. In the United States, 15,000 to 20,000 persons are hospitalized each year with histoplasmosis, and approximately 3% of them die.

Other affected occupations

Attributing occupational risk for *Histoplasma* infection is difficult because the organism is free-living in soil and the concentration of aerosolized spores is increased by wind and dusty conditions. Infection is predominantly due to geographic location. In endemic areas, rural persons, regardless of occupation, have a 60 to 80% prevalence of positive skin test to *H. capsulatum* antigens. Actual illness results from a large infecting inoculum and is usually restricted to workers involved in the disruption of soil or destruction of contaminated buildings.

Treatment

Antifungal treatment for histoplasmosis and other occupationally acquired fungal infections is not indicated for acute self-limited pulmonary disease. Therapy with amphotericin B (30 to 35 mg/kg total dose) or ketoconazole (400 mg/day for six months) or treatment regimens using both agents is indicated for disseminated histoplasmosis, chronic pulmonary histoplasmosis, acute pulmonary histoplasmosis with ARDS, or mediastinal granuloma with symptomatic obstruction, and may be useful for prolonged, moderately severe primary illness. Treatment results in an 80 to 100% response rate, but relapses are common and may be as high as 20% with amphotericin B and 50% with ketoconazole. Efficacy of newer azole drugs (i.e., itraconazole and fluconazole) for occupational fungal infections has not been defined.

Public health controls

No effective vaccine has been developed. Chemical decontamination with 3% formaldehyde, prewetting the ground or contaminated surfaces to reduce aerosolization, and personal respiratory protection to reduce inhalation of aerosolized spores may reduce infection, but the efficacy of these methods has not been determined.

Miscellaneous Fungal Pneumonias

The miscellaneous fungal pneumonias of agricultural workers include aspergillosis, blastomycosis, cryptococcosis, coccidioidomycosis and paracoccidioidomycosis (table 10.26). These diseases are caused by *Aspergillus spp., Blastomyces dermatitidis, Cryptococcus neoformans, Coccidioides immitis* and *Paracoccidioides brasiliensis,* respectively. Although these fungi have a widespread geographic distribution, disease is usually reported from endemic areas. Relative to viral and bacterial causes of pneumonia, these disorders are rare and are often initially unsuspected. T-cell disorders enhance susceptibility to histoplasmosis, blastomycosis, cryptococcosis, coccidioidomycosis and paracoccidioidomycosis. However, a large initial exposure may result in disease in the immunocompetent worker. Infections with *Aspergillus* and related fungi tend to occur in neutropenic patients. Aspergillosis is most frequently an OAP of the immunosuppressed and will be discussed in the section on infections in the immunosuppressed.

Cr. neoformans, like *H. capsulatum*, is a common inhabitant of soil contaminated by avian faeces, and occupational exposure to such dusts or other dusts contaminated with *Cr. neoformans* may result in

disease. Occupational blastomycosis is associated with outside occupations, especially in the eastern and central United States. Coccidioidomycosis results from exposure to contaminated dusts in endemic areas of the south-western United States (hence the synonym San Joaquin valley fever). Occupational exposure to contaminated soils of South and Central America is often associated with paracoccidioidomycosis. Because of the potentially long latency with paracoccidioidomycosis, this exposure may long precede the appearance of symptoms.

Clinical presentation

The clinical presentation of coccidioidomycosis, blastomycosis, or paracoccidioidomycosis is similar to histoplasmosis. Aerosol exposures to these fungi can produce OAP if the initial inoculum is high enough. However, host factors, such as prior exposure, limit disease in most individuals. In coccidioidomycosis, pulmonary and systemic signs of disease are apparent in a small percentage of those infected; progressive disease with dissemination to multiple organs is rare in the absence of immunosuppression. Although the source of infection is usually the lung, blastomycosis may present as pulmonary disease, cutaneous disease, or systemic disease. The most common clinical presentation of blastomycosis is a chronic cough with pneumonia indistinguishable from tuberculosis. However, the majority of patients with clinically apparent blastomycosis will have extra-pulmonary lesions involving the skin, bones or genitourinary system. Paracoccidioidomycosis is a disease of Mexico, Central and South America which most frequently presents as reactivation of prior infection after a long but variable latency period. The disease may be associated with ageing of infected individuals, and reactivation may be induced by immunosuppression. The pulmonary presentation is similar to other fungal pneumonias, but extrapulmonary disease, particularly of the mucous membranes, is common in paracoccidioidomycosis.

The lung is the usual site for primary infection with *Cryptococcus neoformans*. As with the previously discussed fungi, pulmonary infections may be asymptomatic, self-limited or progressive. However, dissemination of the organism, particularly to the meninges and brain, may occur without symptomatic respiratory disease. Cryptococcal meningoencephalitis without evidence of pulmonary cryptococcosis, while rare, is the most common clinical manifestation of *Cr. neoformans* infection.

Diagnostic tests

Direct demonstration of the tissue form of the organism permits a definitive diagnosis in biopsies and cytologic preparations. Immunofluorescence can be a useful confirmatory procedure if morphologic details are insufficient for establishing the aetiologic agent. These organisms can also be cultured from suspicious lesions. A positive latex cryptococcal agglutinin test in cerebrospinal fluid is consistent with cryptococcal meningoencephalitis. However, demonstration of organisms may not be sufficient for the diagnosis of disease. For example, saprophytic growth of *Cr. neoformans* is possible in airways.

Other affected occupations

Laboratory workers isolating these fungi are at risk for infection.

Treatment

Antifungal therapy is similar to that for histoplasmosis.

Public health controls

Engineering controls are indicated to reduce the risk to laboratory workers. Respiratory protection when working with soils heavily contaminated with avian faeces will reduce exposure to *Cr. neoformans*.

Occupationally Acquired Infections in Health Care and Laboratory Workers

Inhalation of infectious aerosols is the most common source of infection in hospital workers, and many types of viral and bacterial pneumonias have been attributed to work-related transmission (table 10.26). The majority of infections are viral and self-limited. However, potentially serious outbreaks of tuberculosis, measles, pertussis and pneumococcal pneumonia have been reported in hospital workers. Infections in immunocompromised workers are discussed at the end of this section.

Diagnostic laboratory workers are at risk for occupationally acquired infections resulting from airborne transmission. Transmission occurs when pathogens are aerosolized during the initial processing of clinical specimens from patients with uncertain infectious diseases, and is rarely recognized. For example, in a recent community outbreak of brucellosis, one-third of the laboratory technicians developed brucellosis. Employment in the laboratory was the only identified risk factor. Person-to-person transmission between laboratory employees, food or waterborne transmission, or contact with a particular clinical specimen could not be shown to be risk factors. Rubella, tuberculosis, varicella-zoster and respiratory syncytial virus are occupational illnesses similarly acquired in the laboratory by technicians.

Despite rigorous veterinary care, biosafety containment procedures and the use of commercially reared, pathogen-free laboratory animals, inhalation remains the principal mode of infectious disease transmission associated with biomedical research workers. In addition, newly discovered micro-organisms or previously unrecognized zoonotic reservoirs may be encountered and undermine these disease control strategies.

Measles

Measles, as an occupationally acquired illness, has become an increasing problem among hospital workers in developed nations. Since 1989, there has been a resurgence of measles in the United States due to poor compliance with vaccine recommendations and the failure of primary immunization in vaccine recipients. Because of the high morbidity and potential mortality associated with measles in susceptible workers, special consideration should be given to measles in any occupational health programme. From 1985 to 1989, more than 350 cases of occupationally acquired measles were reported in the United States, representing 1% of all reported cases. Nearly 30% of hospital workers with occupationally acquired measles were hospitalized. The largest groups of hospital workers with measles were nurses and physicians, and 90% of them acquired measles from patients. Although 50% of these ill persons were eligible for vaccination, none had been vaccinated. The increased measles morbidity and mortality in adults has increased the concern that infected workers may infect patients and co-workers.

In 1989, the Immunization Practices Advisory Committee recommended two doses of measles vaccine or evidence of measles immunity at the time of employment in a health care setting. Serologic and vaccination status of workers should be documented. In addition, when patients with measles present, re-evaluation of the immune status of HCWs is appropriate. Implementing these recommendations and appropriate isolation of patients with known and suspected measles curtails the transmission of measles in medical settings.

Clinical presentation

In addition to the common presentation of measles seen in non-immune adults, atypical and modified presentations of measles

must be considered because many hospital workers had previously received killed vaccines or have partial immunity. In classic measles, a two-week incubation period with mild upper respiratory symptoms follows infection. During this period, the worker is viremic and infectious. This is followed by a seven- to ten-day course of cough, coryza and conjunctivitis and the development of a morbilliform rash and Koplik spots (raised white lesions on the buccal mucosa), which are pathognomonic for measles. Diffuse reticulonodular infiltrates with bilateral hilar lymphadenopathy, often with a superimposed bacterial bronchopneumonia, are noted on x ray. These signs occur well after the person has had the opportunity to infect other susceptible persons. Pulmonary complications account for 90% of the measles deaths in adults. No specific antiviral treatment is effective for any form of measles, although high-titre anti-measles immunoglobulin may ameliorate some symptoms in adults.

In atypical measles, which occurs in persons vaccinated with a killed vaccine developed in the 1960s, severe pulmonary involvement is common. The rash is atypical and Koplik spots are rare. In modified measles, which occurs in persons previously receiving a live vaccine but developing partial immunity, signs and symptoms are similar to classic measles but milder, and often go unnoticed. Persons with atypical and modified measles are viremic and can spread measles virus.

Diagnosis

Measles in hospital workers is often modified or atypical, and is rarely suspected. Measles should be considered in a person with an erythematous maculopapular rash preceded by a three- to four-day febrile prodrome. In persons with a first time infection and without previous immunization, viral isolation or antigen detection is difficult, but enzyme-linked immunosorbent or fluorescent antibody assays may be used for rapid diagnosis. In persons with previous immunizations, interpreting these assays is difficult, but immunofluorescent antibody stains of exfoliated cells may be helpful.

Epidemiology

Susceptible nurses and physicians are nearly nine times more likely to acquire measles than persons of the same age who are not HCWs. As with all measles infections, person-to-person transmission occurs via inhalation of an infectious aerosol. Hospital workers acquire measles from patients and co-workers and, in turn, transmit measles to susceptible patients, co-workers and family members.

Other affected occupations

Epidemic measles has occurred in academic institutions in developed nations and among agricultural workers restricted to collective lodgings on plantations.

Public health controls

Public health intervention strategies include immunization programmes as well as infection control programmes to monitor measles illness and antibody status of workers. If natural infection or an appropriate two-dose vaccination cannot be documented, antibody assays should be performed. Vaccination of pregnant workers is contraindicated. Vaccination of other at-risk workers is an important aid in disease prevention. After exposure to measles, removal of susceptible workers from patient contact for 21 days may reduce the spread of disease. Restricted activity of workers with measles for 7 days after the appearance of the rash may also curtail disease transmission. Unfortunately, appropriately vaccinated workers have developed measles despite protective antibody levels that were documented prior to illness. As a result, many recommend personal respiratory protection when caring for patients with measles.

Miscellaneous viral respiratory tract infections

A variety of viruses which are not unique to the health care environment are the most common cause of OAPs in health care workers. The aetiological agents are those which cause community-acquired OAPs, including adenovirus, cytomegalovirus, influenza virus, parainfluenza virus and respiratory syncytial virus. Because these organisms are also present in the community, establishing these as the cause of an individual OAP is difficult. However, serologic studies suggest that health care and day care workers are at increased risk for exposure to these respiratory pathogens. These viruses are also responsible for disease outbreaks in many situations where workers are brought together in a confined space. For example, outbreaks of adenoviral infection are common in military recruits.

Pertussis

Pertussis, like measles, has been increasingly reported in hospital workers in developed nations. In 1993, nearly 6,000 cases of pertussis were reported in the United States, an 80% increase over 1992. Unlike previous years, 25% of the reported cases occurred in persons over ten years old. The number of occupationally acquired illnesses in hospital workers is unknown but is felt to be underreported in developed nations. Because of waning immunity in adults and the potential for hospital workers to infect susceptible infants, there is greater emphasis on diagnosis and surveillance of pertussis.

Clinical presentation

Pertussis may persist for six to ten weeks without intervention. In the first week, when the ill person is most contagious, dry cough, coryza, conjunctivitis and fever develop. In previously vaccinated adults, the persistent, productive cough may last several weeks and pertussis is rarely considered. Clinical diagnosis is difficult, and clinical suspicion should be aroused when one encounters any worker with a cough that lasts for more than seven days. A white count greater than 20,000 with a predominance of lymphocytes may be the only laboratory abnormality, but this is rarely noted in adults. Chest radiographs show confluent bronchopneumonia in the lower lobes that radiate from the heart to give the characteristic "shaggy heart" sign, and atelectasis is present in 50% of cases. Because of the extreme infectiousness of this agent, strict respiratory isolation is necessary until treatment with erythromycin or trimethoprim/sulphamethoxazole has continued for five days. Close contacts of infected person and hospital workers who were not using respiratory precautions should receive 14 days of antibiotic prophylaxis regardless of immunization status.

Diagnosis

Isolation of *Bordetella pertussis*, direct immunofluorescent staining of nasal secretions, or development of a *B. pertussis* antibody response is used to make a definitive diagnosis.

Epidemiology

B. pertussis is highly contagious, transmitted person-to-person via inhalation of infectious aerosols, and has an attack rate of 70 to 100%. In the past, it has not been a disease of adults and has not been appreciated as an OAP. During a community outbreak of pertussis in the western United States, many hospital workers were exposed at work and developed pertussis despite antibiotic prophylaxis. Because of waning protective antibody levels in adults who have never had clinical disease but received cellular vaccine after 1940, there is a growing population of pertussis-susceptible hospital workers in developed nations.

Public health controls

Identification, isolation and treatment are the main disease control strategies in hospitals. The role of acellular pertussis vaccine for hospital workers without adequate levels of protective antibody is unclear. During the recent outbreak in the western United States, one-third of vaccinated hospital workers reported mild to moderate side effects to the vaccine but 1% had "severe" systemic symptoms. Although these more severely affected workers missed days of work, no neurologic symptoms were reported.

Tuberculosis

During the 1950s, it was generally recognized that health care workers in developed nations were at greater risk for tuberculosis (TB—granulomatous disease due to *Mycobacterium tuberculosis* or closely related organisms *M. bovis*) than the general population. From the 1970s through the early 1980s, surveys suggested that this had become only a slightly increased risk. In the late 1980s, a marked increase in the number of cases of TB admitted to US hospitals resulted in the unsuspected transmission of *M. tuberculosis* to hospital workers. The high background prevalence of positive tuberculin skin test (TST) in certain socio-economic or immigrant groups from which many hospital workers came, and the poor association of TST conversion with work-related exposures to TB, made it difficult to quantify the risk of TB occupational transmission to workers. In 1993 in the United States, an estimated 3.2% of reported persons with TB were health care workers. Despite problems in defining risk, work-related infection should be considered when hospital workers develop TB or convert their TST.

M. tuberculosis is spread almost exclusively person to person on infectious particles with a diameter of 1 to 5 µm that result from coughing, talking or sneezing. The risk of infection is directly related to the intensity of exposure to infectious aerosols—small shared spaces, increased density of infectious particles, poor clearance of infectious particles, recirculation of air containing infectious particles, and prolonged contact time. In health care settings, procedures such as bronchoscopy, endotracheal intubation and nebulized aerosol treatment increase the density of infectious aerosols. Approximately 30% of close contacts—persons who share a common space with an infectious person—become infected and undergo skin-test conversion. After infection, 3 to 10% of persons will develop TB within 12 months (i.e., primary disease) and an additional 5 to 10% will develop TB in their lifetime (i.e., reactivation disease). These higher rates occur in developing nations and situations when malnutrition is more prevalent. HIV-infected persons reactivate TB at higher rates, approximately 3 to 8% per year. CFR varies; in developed nations, it is between 5 and 10%, but in developing nations, these rates range from 15 to 40%.

Clinical presentation

Prior to the HIV epidemic, 85 to 90% of persons with TB had pulmonary involvement. Chronic cough, sputum production, fever and weight loss remain the most frequently reported symptoms of pulmonary TB. Except for rare amphoric breathing or post-tussive crackles over the upper lobes, physical examination is not helpful. An abnormal chest x ray is found in nearly all cases and is usually the first finding to suggest TB. In primary TB, a lower- or middle-lobe infiltrate with ipsilateral hilar lymphadenopathy with atelectasis is common. Reactivation TB usually results in an infiltrate and cavitation in the upper lobes of the lungs. Although sensitive, chest x rays lack specificity and will not give a definitive diagnosis of TB.

Diagnosis

Definitive diagnosis of pulmonary TB can be made only by isolating *M. tuberculosis* from sputum or lung tissue, although a presumptive diagnosis is possible if acid-fast bacilli (AFB) are found in sputum from persons with compatible clinical presentations. The diagnosis of TB should be considered on the basis of clinical signs and symptoms; isolation and treatment of persons with compatible illnesses should not be delayed for the result of a TST. In developing nations where TST reagents and chest x rays are not available, WHO suggests evaluating persons with any respiratory symptom of three weeks' duration, haemoptysis of any duration or significant weight loss for TB. These persons should have a microscopic examination of their sputum for AFB.

Other affected occupations

Worker-to-worker and client-to-worker airborne transmission of *M. tuberculosis* has been documented among hospital workers, airline flight crews, miners, correctional facility workers, animal caretakers, shipyard workers, school employees and plywood factory workers. Special consideration must be given to certain occupations such as farmworkers, animal caretakers, manual labourers, housekeepers, janitors and food preparation workers, although most of the risk may be due to the socio-economic or immigration status of the workers.

Special consideration should be given to pulmonary TB among miners and other groups with silica exposure. In addition to an increased risk of primary infection from fellow miners, persons with silicosis are more likely to progress to TB and have a greater TB-specific mortality compared to non-silicotic workers. As in most persons, TB reactivates among silicotic persons from long-standing *M. tuberculosis* infections that predate silica exposure. In experimental systems, silica exposure has been shown to worsen the course of infection in a dose-dependent fashion, but it is unclear if silica-exposed, non-silicotic workers are at greater risk for developing TB. Silica-exposed foundry workers without radiographic silicosis are at a three-times greater risk of TB-specific mortality compared to similar workers without silica exposure. No other occupational dust exposures have been associated with enhanced progression of TB.

Migrant farmworkers are more likely to develop reactivation TB than the general population. Estimates of positive TSTs in migrant farmworkers range from approximately 45% in 15 to 34 year-old persons to nearly 70% in workers more than 34 years old.

Clinical laboratory workers are at increased risk for occupationally acquired TB through airborne transmission. In a recent ten-year survey of selected hospitals in Japan, 0.8% of laboratory workers developed TB. No community sources were identified, and work-related exposures were identified in only 20% of the cases. Most cases occurred among the workers in the pathology and bacteriology laboratories and autopsy theatres.

Treatment

Several treatment regimens have been shown to be effective in different outpatient settings. Among compliant patients in developed nations, daily doses of four drugs (including isoniazid and rifampin) for two months followed by daily doses of isoniazid and rifampin for the next four months has become a standard treatment regimen. Directly observed, twice weekly administration of the same drugs is an effective alternative in less compliant patients. In developing nations and in situations where anti-tuberculous medications are not readily available, 9 to 12 months of daily dosing with isoniazid and rifampin has been used. A treatment regimen should be consistent with the national policy and take into consideration the organism's susceptibility to standard, available anti-tuberculous medications and duration of therapy. Because of limited resources to control TB in developing nations, efforts may focus on the primary sources of infection—patients with sputum smears that demonstrate AFB.

In health care settings, work restrictions are indicated for infectious workers with pulmonary TB. In other settings, infectious workers may simply be isolated from other workers. In general, persons are considered non-infective after two weeks of appropriate antituberculous medications if there is symptomatic improvement and decreasing density of AFB in the sputum smear.

Public health controls

The main public health control of occupationally or community-acquired TB transmission remains identification, isolation and treatment of persons with pulmonary TB. Ventilation to dilute infectious aerosols; filtration and ultraviolet lights to decontaminate the air containing the aerosol; or personal respiratory protection may be used where the risk of transmission is known to be exceptionally high, but the efficacy of these methods is still unknown. The utility of BCG in worker protection remains controversial.

Miscellaneous bacterial infections in the health care environment

Common bacterial infections of the lung may be acquired from patients or within the community. Work-related airborne transmission of bacterial pathogens such as *Streptococcus pneumoniae, Haemophilus influenza, Neisseria meningitidis, Mycoplasma pneumoniae* and *Legionella spp* (table 10.26) occurs and the resulting illnesses are included in many hospital surveillance programmes. Occupational bacterial respiratory tract infections are also not restricted to health care workers. Infections with *Streptococcus spp* are, for example, a well-established cause of disease outbreaks among military recruits. However, for a specific worker, the prevalence of these disorders outside the workplace complicates the distinction between occupational and community-acquired infections. The clinical presentation, diagnostic tests, epidemiology and treatment of these disorders are described in standard medical textbooks.

Infections in the immunosuppressed worker

Immunosuppressed workers are at increased risk from many OAPs. In addition, a number of organisms which do not cause disease in normal individuals will produce disease in the immunosuppressed. The type of immunosuppression will also affect disease susceptibility. For example, invasive pulmonary aspergillosis is a more frequent complication of chemotherapy than of acquired immunodeficiency syndrome (AIDS).

Invasive pulmonary aspergillosis is usually seen in the immunosuppressed, particularly individuals with neutropenia. However, invasive pulmonary aspergillosis is occasionally reported in individuals without an apparent predisposition to disease. Invasive pulmonary aspergillosis normally presents as a severe, necrotizing pneumonia with or without systemic involvement in a neutropenic patient. While invasive aspergillosis is most frequently seen as a nosocomial infection in chemotherapy patients, this is a highly fatal disease in any neutropenic worker. Techniques which reduce nosocomial aspergillosis—for example, the control of dusts from construction projects—may also protect susceptible workers.

A variety of animal pathogens become potential zoonoses only in the immunosuppressed patient. The zoonoses transmitted by aerosol exposure seen only in the immunosuppressed include encephalitozoonosis (due to *Encephalitozoon cuniculi*), avian tuberculosis (due to *Mycobacterium avium*) and *Rhodococcus equi* infections. Such diseases are of particular concern in agriculture. Methods for the protection of immunosuppressed workers are incompletely investigated.

In the immunosuppressed worker, many potential pathogens cause invasive and severe disease not seen in normal patients. For example, severe infections with *Candida albicans* and *Pneumocystis carinii* are classical manifestations of AIDS. The spectrum of occupational pathogens in the immunosuppressed worker, therefore, potentially involves disorders not present in immunologically normal workers. The diseases of immunosuppressed individuals have been thoroughly reviewed elsewhere and will not be further discussed in this review.

Public Health Controls: Overview

OAPs predominantly occur in five groups of workers: hospital workers, agricultural workers, meat production workers, military personnel and biomedical laboratory workers (table 10.26). Avoidance of infectious aerosols is the most effective way of reducing infection in most situations but often is difficult. For example, *Coxiella burnetii*, the aetiological agent for Q fever, may be present in any environment previously contaminated with the biological fluids of infected animals, but avoidance of all potentially infected aerosols would be impractical in many low-risk situations such as sheep herding or rodeos. Control of concomitant diseases may also reduce the risk of OAPs. Silicosis, for example, increases the risk for reactivation of TB, and reducing silica exposure may reduce the risk of TB in miners. For OAPs that have significant mortality and morbidity in the general population, immunization may be the most important public health intervention. Education of workers about their risk of OAPs assists in worker compliance with occupational disease control programmes and also aids in the early diagnosis of these disorders.

Among hospital workers and military personnel, human-to-human transmission is usually the main route of infection. Worker immunization may prevent disease and may be useful in the control of pathogens of high morbidity and/or mortality. Because there is a risk for persons who may not have been adequately immunized; identifying, isolating and treating ill persons remains a part of disease control. When immunization and respiratory isolation fail or the associated morbidity and mortality is intolerable, personal protection or engineering controls to reduce the density or infectiousness of aerosols may be considered.

For agricultural, meat production and biomedical laboratory workers, animal-to-human transmission is a common transmission pattern. In addition to immunization of susceptible persons when possible, other disease control strategies may include immunization of the animals, veterinarian-controlled antibiotic prophylaxis of well-appearing animals, quarantine of newly arrived animals, isolation and treatment of ill animals, and purchase of pathogen-free animals. When these strategies have failed or there is high morbidity and mortality, strategies such as personal protection or engineering controls may be considered.

Environment-to-human transmission of infectious agents is common among agricultural workers, including many labourers. Worker immunization is possible when a vaccine is available, but for many of these pathogens, disease incidence in the general population is low and vaccines are rarely feasible. In agricultural settings, the sources of infection are widespread. As a result, engineering controls to reduce the density or infectiousness of aerosols are rarely feasible. In these settings, wetting agents or other methods to reduce dust, decontaminating agents and personal respiratory protection may be considered. Because control of OAP in agricultural workers is often difficult and these diseases are rarely seen by medical personnel, education of workers and communication between workers and medical personnel is essential.

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