CLINICAL REVIEW

Soil-Related Bacterial and Fungal Infections

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A variety of classic and emerging soil-related bacterial and fungal pathogens cause serious human disease that frequently presents in primary care settings. Typically, the growth of these microorganisms is favored by particular soil characteristics and may involve complex life cycles including amoebae or animal hosts. Specific evolved virulence factors or the ability to grow in diverse, sometimes harsh, microenvironments may promote pathogenesis. Infection may occur by direct inoculation or ingestion, ingestion of contaminated food, or inhalation. This narrative review describes the usual presentations and environmental sources of soil-related infections. In addition to tetanus, anthrax, and botulism, soil bacteria may cause gastrointestinal, wound, skin, and respiratory tract diseases. The systemic fungi are largely acquired via inhalation from contaminated soil and near-soil environments. These fungal infections are particularly life-threatening in those with compromised immune systems. Questions regarding soil exposure should be included in the history of any patient with syndromes consistent with tetanus, botulism or anthrax, traumatic wounds, recalcitrant skin lesions, gastroenteritis, and nonresponsive, overwhelming, or chronic pneumonia. (J Am Board Fam Med 2012;25:734–744.)

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Significant attention is given to food- and water-related infections. Questions regarding such exposures are routinely included in patient histories when infectious diseases are included in the differential diagnosis of disease presentations. There are, however, a wide variety of soil-related infections[1,2] that also should be considered, particularly in the case of wound, respiratory tract, or gastrointestinal infections. The purpose of this article is to review bacterial and fungal infections for which the source of contact is primarily the soil (eg, *Clostridium tetani*) or for which soil is an important or emerging secondary site of contact (eg, *Legionella*). The emphasis of this article will be on the epidemiology of significant soil-related human pathogens and their common disease presentations such that these entities will be considered promptly during relevant patient evaluations. (The reader is referred to recent texts and manuscripts for details regarding unusual presentations and the diagnosis and treatment of these infections and for discussion of superficial soil-related infections, such as dermatophyte infections, which will not be covered here.)

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Soil Microbiology

Soil[1,3] is a multilayered surface complex of mineral and organic (humus) constituents present in solid, liquid, and gaseous states. The mineral portion of soil results from the actions of weathering and erosion on rock. Broad soil type—sand, silt, or clay—is defined, largest to smallest, by particle size. These particles pack loosely, and pore spaces of varying sizes are formed. Particle surfaces, pore spaces, and plant roots are particular habitats for microorganisms, often in biofilms. Soil also contains plants, animals, carcasses, and man-made materials.

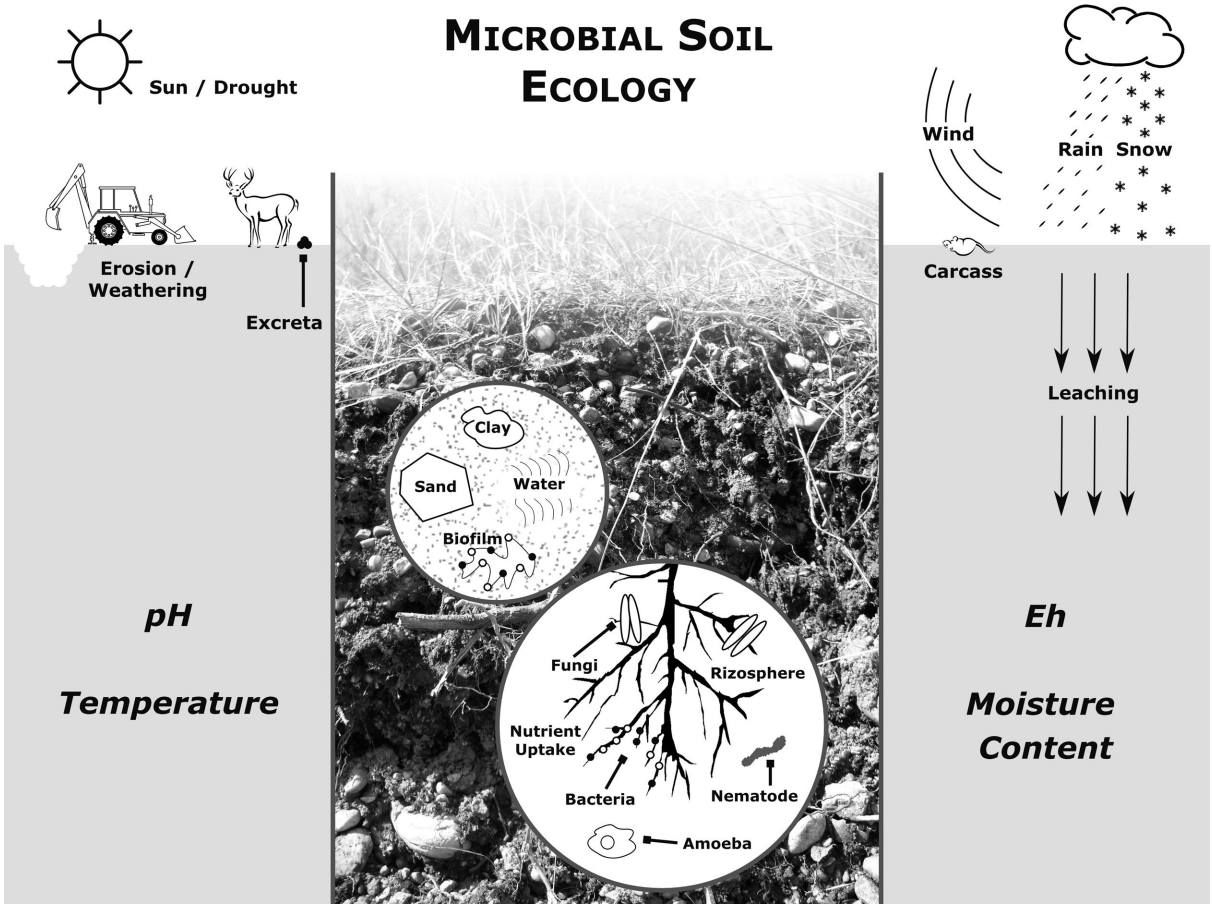
The quantity and type of microorganisms in a particular portion of soil are determined by a complex interaction of varying amounts of sunlight, temperature, moisture, soil pH, nutrients, and re-

dox potential. Pathogens may be indigenous or enter the soil through deliberate introduction (for purposes of biological control) or indirectly by animal deposits, manure application, or from flooding with sewage or contaminated water. Competition, infection, predation, or all three by other bacteria, archaea, viruses, fungi, and larger organisms such as protozoa further select the predominant species, particularly in diverse, nutrient-rich zones such as the rhizosphere (soil surrounding a plant root)[1–5] (Figure 1). Strategies that bacteria acquire to survive this in-soil competition may, in turn, provide the ability to infect animals. Horizontal gene transfers and the occurrence of pathogenicity islands (blocks of acquired DNA encoding for multiple virulence factors) may further allow the emergence of opportunistic pathogens from these microenvironments.[6]

Other adaptations to the soil environment may promote pathogenicity. The ability of *Legionella* to survive within human macrophages likely has evolved from the ability to survive within grazing amoeba in the environment.[7] Fungal adaptation to extreme environmental biotic and abiotic stress likely has resulted in the ability to infect human hosts,[8] even in the absence of known virulence factors.[9]

Pathogenic fungi or bacteria may enter humans via direct inoculation into wounds. Soil minerals introduced simultaneously may promote infection by suppressing local host defenses.[2] Microorganisms may be introduced into the respiratory tract via bioaerosols (dusts or mud particles from soil disturbances, windblown spores) or by direct ingestion of soil (geophagia) or indirect ingestion via contaminated food. Not covered here are other

Figure 1. Pictorial representation of factors determining distribution of bacterial and fungal microorganisms in soil. Side panels represent climatic, physical, and geobiochemical factors. Central panel: upper magnified circle represents soil particles, pore spaces, and bacteria in biofilm; lower magnified circle represents the rhizosphere and associated bacteria and fungi (and amoebae and nematodes).[1,3,4]



mechanisms of ingestion of soil microorganisms causing human disease, including airborne toxins (eg, endotoxin aerosolized by farming operations, mycotoxins), aeroallergens, and waterborne illnesses from soil contamination of water sources.[1]

Bacterial Infections

In addition to the "classic" infections, tetanus and botulism, soil-related bacterial infections include wound infections, gastroenteritis, and specific respiratory syndromes.

Tetanus and botulism are caused by the toxin-producing, anaerobic, spore-bearing, Gram-positive bacteria, *Clostridium tetani* and *Clostridium botulinum*, respectively.[10]

There are 4 clinical forms of tetanus. The generalized form is most common and is characterized by tonic contraction of the skeletal muscles and intense intermittent muscle spasms. Classic findings include trismus (in approximately 50%), stiff neck, opisthotonus, a "sardonic" smile, abdominal rigidity, and periods of apnea. More muscle groups become involved as the illness progresses and fractures of vertebrae or other bones may occur (sometimes triggered by relatively minor stimuli). Patients remain conscious during spasms and anxiety and pain may be significant. Signs of autonomic hyperactivity generally are present, and bradycardia and hypotension may lead to cardiac arrest.[11,12]

Localized tetanus occurs when circulating antitoxin prevents systemic spread of toxin. Localized, fixed, painful muscle contraction may result and last for weeks, followed by complete resolution. These occurrences may not be recognized as tetanus and are likely underdiagnosed.[11,12] Cephalic tetanus is rare and results from cranial nerve involvement following entrance of *C. tetani* into wounds of the head and neck. Initial findings (typically confusing) involve dysphagia, trismus, and focal cranial neuropathies. Patients may present with facial paralysis, dysphagia, otitis media, disruption of extraocular movements, and other eye findings. Neonatal tetanus follows infection of the umbilical stump of infants of unimmunized mothers; it presents during the second week of life with weakness and poor sucking and may result in developmental delay.[11,12]

There were 233 tetanus cases in the United States during the years 2001 to 2008, with a 13% fatality rate. Tetanus is a significant cause of death worldwide, especially in Asia, Africa, and South America. In 2006, 290,000 persons died of tetanus, of which 250,000 were neonatal deaths.[13]

C. tetani has a worldwide distribution in soil and dust (where spores can persist for years), feces (which can reinfect soil), and other agents (including contaminated heroin). The organism may be isolated from surface soils of school and hospital grounds, fields, roadsides, and along waterways.[14] Climate and soil pH relate to the increased prevalence of tetanus in tropical zones,[11] and clusters of infections may occur in developing countries after natural disasters such as earthquakes and tsunamis.[12] In the United States, half of tetanus cases follow known injuries, 45% result from infection of preexisting wounds, wounds of unknown cause, parenteral drug abuse, or animal-related injuries. No source is found for 5% of cases. Deep puncture wounds, crush injuries, and burns that include anaerobic environments particularly predispose to tetanus.[11]

Once in anaerobic tissue, *C. tetani* spores convert to the vegetative form, multiply, and produce the neurotoxin tetanospasmin, which migrates to the central nervous system via a peripheral nerve at the site of infection. There may be no apparent local infection. The incubation period ranges from 3 to 21 days; the farther the site of initial spore contact from the central nervous system, the longer the incubation period and the milder the disease.[11,12]

Botulism is a serious disease characterized by weakness, paresis, and paralysis, classically presenting with acute bilateral cranial neuropathies and symmetrically descending weakness. Infant botulism typically involves upper airway obstruction, constipation, feeding difficulties, weak cry, drooling, and hypotonia.[15] Two infants with suspected sepsis, apnea, and diarrhea were found to have infant botulism, possibly from contact with soil on the clothing of family members.[16]

Botulism occurs in sporadic cases and outbreaks occur worldwide.[13] An average of 110 cases occur across the United States per year, of which 70% are infant cases and 25% are food borne. It is caused by neurotoxins that affect a peripheral neuromuscular junction and autonomic synapses, preventing the release of acetylcholine.[15] *C. botulinum* (and its resistant spores) is widely distributed in soil and water. Different toxin types are found in different geographic areas: Type A, commonly found in the western United States, may prefer "virgin soils," which are neutral or alkaline and low in organic

content, whereas type B (eastern United States) may prefer cultivated soil. Usually, *C. botulinum* toxin is ingested through food that has been contaminated by the bacterium, resulting in growth and toxin production. Spores may resist boiling (which promotes anerobiasis) for several hours; pressure cooking is required to kill spores.[15]

Wound botulism is clinically similar to food-borne disease except for the gastrointestinal prodrome. In the United States, the soil-related, non–heroin-associated form is most commonly associated with deep trauma in young men. The type A toxin recently has been reported in wounds acquired in Ecuador and may be underreported in developing countries.[17]

Gas gangrene may be caused by one of several species of *Clostridium*, the spores of which are distributed worldwide in the soil. The clostridia usually are present in combination with aerobic bacteria or anaerobic streptococci. Gas gangrene typically follows infection of deep wounds that have been contaminated by soil or feces.[10]

Clostridium perfringens is ubiquitous in soil, from which it may be ingested into the gastrointestinal tract.[10] It is associated with a variety of human diseases,[18] including classic food poisoning. Evidence suggests, however, that most cases of gastrointestinal disease caused by *C. perfringens* have their source from food contaminated by other humans or by animal feces rather than directly from the soil.[19]

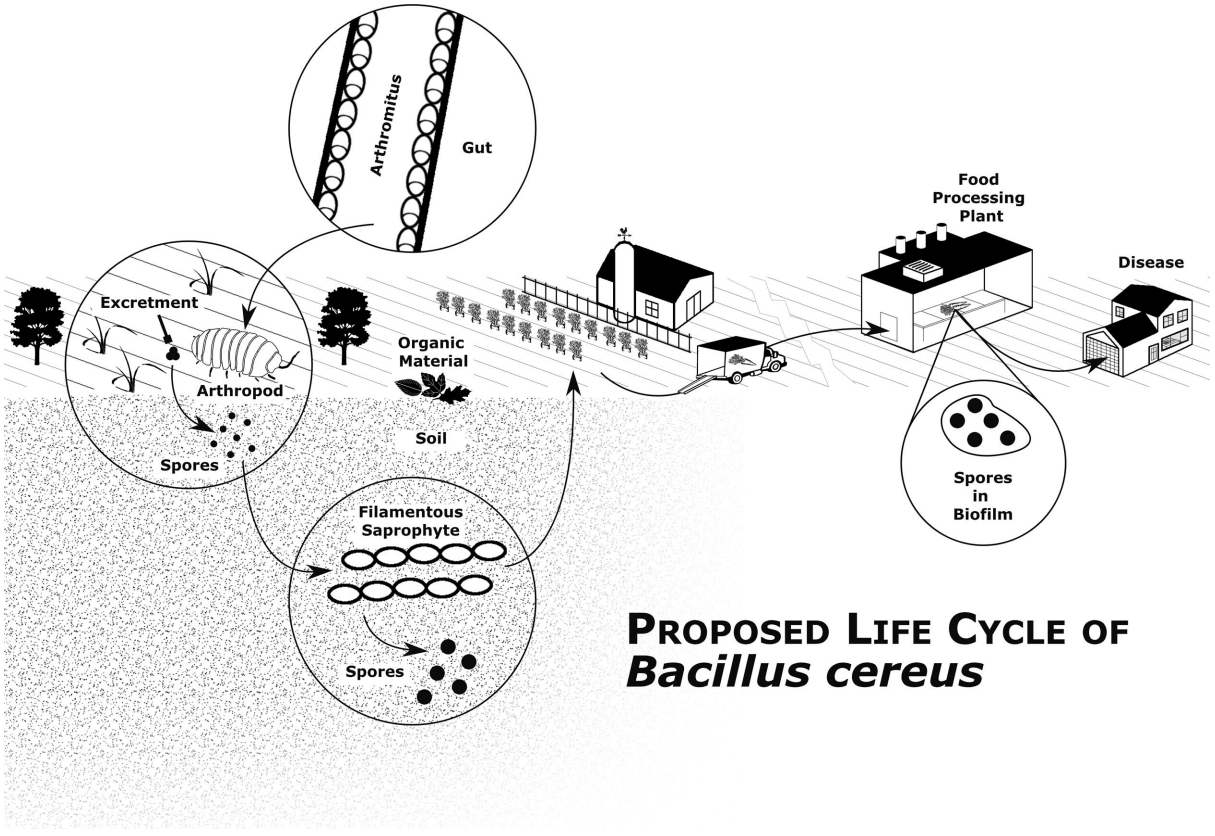
Anthrax, primarily a disease of herbivores, is caused by the Gram-positive, spore-forming rod *Bacillus anthracis*.[20] Readers are referred to current literature regarding detailed descriptions of natural and bioterrorism-related anthrax presentations. In brief, cutaneous anthrax involves a pruritic papule on exposed skin, which ulcerates with surrounding vesicles, followed by black eschar. Inhalation anthrax begins with a nonspecific respiratory illness that may be confused with a variety of common respiratory illnesses,[21] followed by a (usually fatal) sudden second phase of severe respiratory distress. Ingestion of food containing *B. anthracis* results in an abdominal form of gastrointestinal anthrax, an oropharygeal form, or both. Meningitis may complicate any form of anthrax, but this rarely occurs.

The estimated annual number of human anthrax cases worldwide is 2000 to 20,000.[20] The nonuniform distribution of *B. anthracis* in soil may be largely determined by its preference for black soils, which are rich in organic matter and calcium (which promotes spore viability).[22] It also has been proposed that spores accumulate in low-lying areas during rainfall, followed by exposure of grazing animals during dry periods.[23] Because of the concentration of spores needed, it seems that human acquisition of anthrax directly from soil is very unlikely. Rather, host animals are required for spore germination and anthrax is naturally acquired from infected meat and animal products such as goat hair.[20,23]

Bacillus cereus gastroenteritis is an important food-borne disease worldwide, including an estimated 63,400 annual cases in the United States.[24] This entity may be confused with other common bacterial or viral infections or toxin ingestions.[25,26] Pathogenicity is aided by a variety of toxins. Emetic toxin (a peptide) induces nausea and vomiting a few hours after ingesting a meal contaminated with the toxin. A diarrheal syndrome results from protein enterotoxins produced in the small intestine. This bacterium is found naturally in decaying organic matter, in and out of soil, fresh and salt water, plants, dusts, fomites, and the intestinal tract of invertebrates. Spores germinate within an insect or animal host or on contact with organic matter, entering the soil via the droppings of an animal host or upon the host's death. Saprophytic growth in soil, including transition from a single cell to a multicellular form, then ensues. Cells and spores may then contaminate plant material and enter food processing areas. *B. cereus* spores resist extreme environmental conditions including heat, freezing, drying, and radiation. The hydrophobic spore surface allows attachment to food and processing equipment, where biofilm formation may further protect forms of the organism[25–27] (Figure 2). *B. cereus* also has been associated with pulmonary infections mimicking anthrax (perhaps by inhalation of contaminated dust), invasion of the oral cavity or upper respiratory tract in immunosuppressed patients, bone and soft tissue and central nervous system infections, and endophthalmitis (often from a foreign body contaminated with soil or dust).[25]

Listeria monocytogenes causes an estimated 1591 cases of gastroenteritis in normal humans per year in the United States,[24] in addition to meningitis and focal infections in the immunocompromised and serious infections in pregnant women and babies. It is ubiquitous in soil and other material such as sewage, silage, groundwater, and vegetation. *L. monocytogenes* does not form spores and can with-

Figure 2. Proposed life cycle of *Bacillus cereus*. *B. cereus* spores germinate within an animal host and enter soil through droppings or carcasses or they germinate on contact with organic material. Saprophytic growth occurs in soil. Cells/spores contaminate plant material and food processing areas (where spores persist, especially in biofilms) then cause disease after human consumption.[25–27]



stand environmental stressors such as pH change, salinity, low temperatures, and metal ions. Like *B. cereus*, it may persist in contaminated food-processing environments. Thus, some cases of listeriosis may involve infection via the soil–food processing–oral route. Suited for both saprophytic life and life within human cells, a specific regulatory protein and the type of available carbon source may regulate transition.[28]

Gram-negative enteric pathogens are occasionally acquired from soil rather than from water or via the fecal-oral route. Any soil organism may potentially enter water or an aerosol; thus soil is often the origin of water-borne infections. Enteric pathogens may enter soil after contamination by sewage or other human or animal waste, and in developing countries, via untreated domestic wastewater disposal. Salmonella species resist freezing and drying, and some coliforms are thermotolerant. Soil moisture and adsorption to clay particles also promote survival of enteric pathogens.[29]

A large outbreak of *Campylobacter* has been associated with mountain bike racers "swallowing mud" during a race in British Columbia, without known environmental contamination of the track.[30] An outbreak of *Escherichia coli* 0157 disease has been convincingly associated with soil (likely via hands contaminated with mud) from a Scottish scout camp that had been previously grazed by sheep.[31] Indistinguishable strains of *E. coli* 0157 also were isolated from a diseased child and from a recently manured garden where the child had played.[32] Soil may be a reservoir for *E. coli* 0157 in part because of its ability to replicate within the common soil protozoan *Acanthamoeba*.[33]

Melioidosis is caused by the aerobic Gram-negative rod *Burkholderia pseudomallei*. After percutaneous inoculation, inhalation, or ingestion from the environment, disease may range from asymptomatic infection to acute mild to severe pneumonia, disseminated septicemia, or both, including shock and death. Multiple abscesses may occur in virtually

any organ, but commonly in skin (with ulcers) and soft tissue or lung. A chronic disease that mimics tuberculosis may occur. Similarly, latent *B. pseudomallei* may reactivate years later.[34]

Melioidosis is most commonly reported in Southeast Asia and northern Australia but also is found in other parts of the world, including the tropical and subtropical Americas. Thailand has the highest incidence rate (up to 3000 cases per year). Cases were seen to increase after the 2004 Asian tsunami.[13,34] It is not uniformly distributed within endemic areas and seems to occur in space-time clusters. Whether this is because of differential distribution of *B. pseudomallei*, differences in local strain virulence, differential exposure (heavy occupational or recreational soil contact), or differences in the susceptibilities of those exposed (diabetes and renal disease may predispose to infection) is unclear.[35] *B. pseudomallei* is thought to primarily inhabit rice paddies, still waters, and subsurface moist, tropical soils. The organism seems to obtain nutrition from rotting organic matter and via protozoal invasion and is capable of persisting for years in soil. Infected humans and other animals likely transmit the organism to new environments.[35,36]

Several species of *Legionella*, a fastidious Gram-negative rod, cause pneumonia (and, rarely, extrapulmonary disease) and are associated with Pontiac fever, a febrile, nonpneumonic, influenza-like illness. The organisms exist in biofilms in the environment, often in concert with other microorganisms, and survive and multiply within free-living amoebas.[7] The amoeba may encyst during exposure to harsh environments to further promote survival of the parasitic *Legionella*. The primary route of infection by *Legionella pneumophila* is through inhalation of aerosolized contaminated water sources; however, there are reports of soil as an additional environmental source.[37] Globally, *Legionella longbeachae* accounts for up to 5% of cases of Legionnaires disease, which is being increasingly diagnosed and is as common as *L. pneumophila* in Australia, New Zealand, and Japan. This species may be found and persist in a variety of soils.[37–41] *L. longbeachae* is not commonly isolated from water; instead, disease seems to be associated with potting mixes, composts, and soil products (particularly those subjected to high heat and moisture).[42]

Similarly, it has been hypothesized that soil may serve as a source of *Mycobacterium leprae* and other mycobacterial infections.[43–46] Finally, soil-acquired primary cutaneous *Nocardia* infection may be seen after trauma in gardeners and other outdoor or agricultural workers.[47]

Fungal Infections

Excepting dermatophytes, many environmentally acquired fungal infections were apparently uncommon until the emergence of immunosuppresive agents and drugs in the second half of the 20th century. Overall, relatively few endemic fungi cause human disease, and they seem to be pathogenic for mammals without a requirement for such a host during their life cycle. Mammalian body temperatures and layered immune systems seem to inhibit a majority of potentially pathogenic soil fungal species (and may have contributed to the proliferation of mammals and the extinction of dinosaurs). The ability to survive in warm-blooded, slightly alkaline animal environments may be the result of harsh selective factors in soil environments.[48,49] Similar to infection with the bacterium, *Burkholderia pseudomallei* (discussed above), the chance of infection by soil fungi, and its outcome, are determined by geographic, soil, and environmental factors that determine the presence of the particular fungus, local strain virulence, active (eg, digging) or passive (eg, inhalation of windborne dust) soil exposure, and susceptibility of the host.

Most commonly, soil-related endemic fungi cause primary pulmonary disease, with the potential for dissemination, or primary skin disease in normal or immunocompromised hosts. Clues to fungal disease include exposure to endemic areas (with or without a specific history of soil contact), immunocompromise, nosocomial exposure,[50] and pneumonia, which is unresponsive to appropriate empiric antibacterial therapy.

Coccidioidomycosis ("valley fever") may represent up to 29% of community acquired pneumonia cases (150,000 annually) in endemic areas. It is more apt to be severe in elderly, pregnant, African-American, diabetic, and immunosuppressed patients or those who smoke. Progressive disease may include respiratory failure, chronic pneumonia, and dissemination to other organs, including skin, bones/joints, and the central nervous system. Many infections are inapparent.[51]

Coccidioides, a dimorphic (mycelium/spherule) fungus endemic to southwestern North America and portions of Central and South America, has been associated with alkaline, highly salinic, sandy

soils and extremes of temperature. The ability to survive such harsh conditions may allow successful competition with other soil microorganisms.[52] Precipitation followed by high temperatures and drought promotes growth and arthroconidia formation. Wind or excavation then results in inhalation of arthroconidia (the "blow and grow hypothesis"), and pulmonary infection may occur.[53] Epidemics have followed dust storms,[54] excavation,[55] and earthquake,[56] some covering an extensive area. [57] Thus, coccidioidomycosis could be considered a "classic" soil-acquired infection; however, a recent comparative genomic analysis suggests that Coccidioides are not residents of soil, but rather of mammalian host carcasses within the soil.[58]

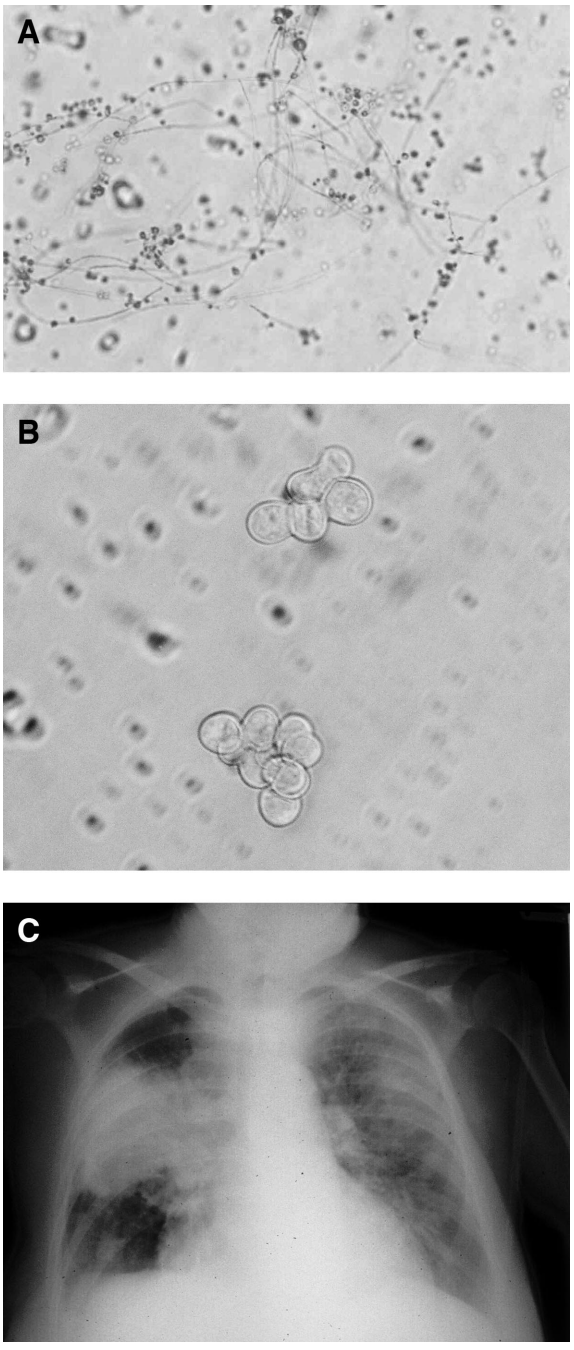
Pulmonary blastomycosis has a wide differential diagnosis and may be asymptomatic or present as mild, moderate, or severe acute pneumonia. The latter may be complicated by acute respiratory distress syndrome. Subacute to chronic infiltrates, cavitary lung disease, or both may occur instead. In addition, acute or chronic dissemination of *B. dermatitidis* to the skin, brain, genitourinary system, bone, or any other organ system may result.[59,60]

Blastomycosis is caused by *Blastomyces dermatitidis*, a dimorphic (mycelia/yeast) fungus (Figure 3) found in eastern North America and parts of India and Africa. Annual incidence of the disease ranges from <1 to 100 per 100,000 in endemic areas. Except for rare inoculation cutaneous disease, the organisms enter the body via inhalation of conidia from the environment (wind or excavation dust, digging, direct contact) into the lungs.

B. dermatitidis occurs in soil and near-soil environments (often associated with waterways, animal excreta, sandy soils); an animal reservoir has not been established. It also seems to be a survivor of harsh or changing environmental conditions and may utilize a wide variety of substrates.[61,62] It has been proposed that the ability to grow and sporulate in carbon-poor, high-ammonia environments may be a key to its competitive success.[63] Preceding precipitation and environmental temperatures may impact the occurrence of *B. dermatitidis*.[64]

The related dimorphic fungus, *Histoplasma capsulatum*, is found worldwide in temperate zones, often in or near river valleys.[65,66] Evidence of exposure is found in up to 80% of residents of the eastern and central United States.[13] Like blastomycosis, histoplasmosis is often asymptomatic, is a primary pulmonary disease, and may disseminate to

Figure 3. *Blastomyces dermatitidis*: Example of endemic dimorphic fungal pathogen causing primarily pulmonary infection. A: Infectious mycelial (mold) forms of *B. dermatitidis* grown on Sabouraud dextrose agar at 20°C (magnification ×400). B: Yeast forms of *B. dermatitidis* grown on brain-heart infusion agar at 37° (magnification ×400). C: Chest radiograph illustrating 2 of several nonspecific radiographic patterns of pulmonary blastomycosis. A dense opacity in the right mid-lung fields and patchy infiltrates in the left lung are seen in this adult woman.



a variety of organ systems (the liver, spleen, bone, lymph nodes, central nervous system, and others). *Histoplasma* may cause opportunistic infections. The intensity of acute pneumonia likely correlates with the amount of inhaled conidia. Pulmonary masses, granulomas, fibrosis, or pericarditis may result. Those with underlying lung disease may suffer progressive chronic (cavitary) pulmonary histoplasmosis.[51,65]

Climate associations include 67% to 87% relative humidity, 90 to 127 cm of annual precipitation, and mean soil temperatures of 22 to 29°C. The fungus lives in soil that is generally acidic, moist, and has high nitrogen content enriched by bird and bat guano,[65] perhaps aided by its ability to assimilate uric acid and related compounds.[67] Conidia are inhaled (sometimes as point-source or extended outbreaks) after soil disturbance by construction or excavation, by contact with caves or soil around potted plants, or through proximity to bird roosts.[65,68–71]

Paracoccidioidomycosis is a disease of scattered areas of Central and South America where exposure prevalence varies from 0.3 to 1000 per 100,000 inhabitants.[72] It may be subclinical, and clinical infection typically follows a long latent period. Juvenile disease is a serious illness in both sexes of normal children and immunocompromised adults and includes hepatosplenomegaly and lymphadenopathy and often fever, malaise, weight loss, skin and bone lesions, and mild respiratory symptoms. Chronic adult infection, predominantly in men, is a progressive pulmonary and multiorgan systemic disease that particularly involves skin and mucus membranes. The precise ecological niche of *Paracoccidioides brasiliensis* is undefined. There is an association with armadillos, and the macroecology seems to include forests, disturbed vegetation or crops, nearby waterways, and shaded, sandy or clay, mildly acidic soil.[61,73] Disease incidence may relate to prior humidity and El Niño activity.[74]

Penicillium marneffei, the only dimorphic *Penicillium*, may cause a fatal systemic mycosis in patients infected with HIV in Southeast Asia. It is associated with soil exposure during the rainy season and bamboo rats, although the nature of association (common source exposure versus vector) is unclear.[61,66,75,76]

Sporotrichosis is a rare, subacute to chronic mycosis caused by the dimorphic fungus *Sporothrix schenckii*. It is primarily a local lymphocutaneous infection in healthy individuals that is usually the result of direct inoculation from the environment (thorns, splinters, cuts, etc.). Days to weeks after inoculation, a papule forms at the site, becomes nodular, and may ulcerate. More nodules then appear along the proximal lymphatic distribution. Extensive cutaneous and systemic dissemination often occurs in untreated patients with underlying disease (diabetes, alcoholism, AIDS). Subacute or chronic pulmonary disease may result after dissemination or inhalation of conidia.[66,77] *S. schenckii* is found worldwide in soil, vegetation, sphagnum moss, decaying wood, and hay. Exposure may occur during normal outdoor occupational or recreational activities. Zoonotic transmission and outbreaks occur.[66,77–79]

A variety of nondimorphic, soil-related yeasts and molds can cause significant local or systemic human infection, particularly in those who are immunocompromised.[50] These include Rhizopus and Mucor (sinus, pulmonary, gastrointestinal, wound) and a variety of traumatic infections.[80,81] The latter include the fungal causes of mycetoma, or "Madura foot," the etiologic agents of which inhabit tropical and subtropical regions typified by extremes of wet and dry conditions and temperature swings. This entity is most common among those working in fields, where infection is acquired in a manner similar to sporotrichosis. Mycetoma starts as a slowly progressive, painless subcutaneous nodule, usually on the foot, and progresses to a classic triad of induration, draining sinuses, and discharging granules that are collections of (in this case) fungal hyphae. Hands and other body parts may be the primary site. If unabated, deep tissue or visceral organ invasion may occur.[82,83]

Infections caused by *Aspergillus* species (excludes allergic brochopulmonary aspergillosis) are usually in immunocompromised patients and include invasive pulmonary aspergillosis (cough, dyspnea, possible fever, chest pain, hemoptysis, wheezing); pulmonary or sinus fungus balls; chronic pulmonary aspergillosis (cavitary, fibrosing, subacute); sinusitis; endocarditis; and other superficial or disseminated forms.[84] Most infections occur after inhalation of conidia. *Aspergillus* is ubiquitous in air, and the sources of spores include soil, decaying vegetation, and dust. Huge spore dispersal may follow disasters such as hurricanes. The relative importance of soil as a source of infection compared with

plants, flowers, building materials, water, and hospital environments is unclear.[9,85] No specific virulence factors have been identified for *Aspergillus*. The ability of the organism to live in a wide variety of environments, including those as harsh as a rock surface, has led to the hypothesis that its ability to "opportunistically" metabolize wide-ranging substances and inhabit multiple niches also allows this fungus to inhabit the unique environment that is the human body.[9]

Conclusions

A variety of bacterial and fungal microorganisms are capable of departing a soil environment to cause serious focal or systemic infection. Specific evolved virulence factors or the ability to grow in diverse, sometimes harsh, microenvironments may promote human infection. Questions regarding travel and soil exposure, by direct contact or ingestion, inoculation, or dust or aerosol inhalation, should be included in the history of any patient with syndromes consistent with tetanus, botulism or anthrax, traumatic wounds, recalcitrant skin lesions, gastroenteritis, and nonresponsive, overwhelming, or chronic pneumonia. Prompt recognition of tetanus and botulism, supportive intensive care, tetanus immune globulin or botulism antitoxin therapy, respectively, and adjunctive antibiotic therapy may significantly improve outcomes in affected patients. Prompt, directed antimicrobial therapy for anthrax, wound infection, and systemic fungal disease may be life-changing. *Bacillus* and *Listeria* gastroenteritis is usually self-limited in immunocompetent people, but investigation of their source(s) may be an important public health measure.

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