Coccidioidomycosis and COVID-19 Co-Infection, United States, 2020

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We review the interaction between coronavirus disease (COVID-19) and coccidioidomycosis, a respiratory infection caused by inhalation of *Coccidioides* fungal spores in dust. We examine risk for co-infection among construction and agricultural workers, incarcerated persons, Black and Latino populations, and persons living in high dust areas. We further identify common risk factors for co-infection, including older age, diabetes, immunosuppression, racial or ethnic minority status, and smoking. Because these diseases cause similar symptoms, the COVID-19 pandemic might exacerbate delays in coccidioidomycosis diagnosis, potentially interfering with prompt administration of antifungal therapies. Finally, we examine the clinical implications of co-infection, including severe COVID-19 and reactivation of latent coccidioidomycosis. Physicians should consider coccidioidomycosis as a possible diagnosis when treating patients with respiratory symptoms. Preventive measures such as wearing face masks might mitigate exposure to dust and severe acute respiratory syndrome coronavirus 2, thereby protecting against both infections.

Persons with coronavirus disease (COVID-19) can have a wide range of symptoms, including cough, difficulty breathing, and fatigue (*1*). These symptoms are also common among patients with coccidioidomycosis (*2*), a primarily pulmonary disease caused by inhalation of *Coccidioides*, a soil-dwelling dimorphic fungi. These spores spread through the air, especially through wind erosion in dusty environments and dirt disrupting activities such as digging or construction. *Coccidioides* spores are found in hot and arid environments, including much of the southwestern United States, where coccidioidomycosis incidence has been increasing. Since 2016, California has recorded its highest incidences of coccidioidomycosis (*3*,*4*).

We reviewed epidemiologic and clinical literature on coccidioidomycosis and COVID-19 to identify subpopulations that might be at risk for co-infection and severe disease. We discuss how the COVID-19 pandemic might affect coccidioidomycosis diagnosis, surveillance, and clinical management. We also evaluate evidence that co-infection might contribute to severe COVID-19 or reactivation of latent *Coccidioides* infection. Our study informs healthcare providers, policymakers, and populations in regions to which coccidioidomycosis is endemic on potential interactions between this disease and COVID-19, encouraging protective measures and prompt diagnosis.

Methods

We searched peer-reviewed journals on PubMed, Google Scholar, Scopus, and Web of Science; preprints posted on medRxiv and bioRxiv; and reports from state health departments and correctional agencies for articles on risk factors for infection and disease severity, diagnosis, surveillance, and preventive measures for coccidioidomycosis and COVID-19. We assessed titles and abstracts for relevance to the risk factors, diagnostic issues, and complications of coccidioidomycosis and COVID-19 co-infections. We conducted searches published during April–December 2020 and did not exclude articles on the basis of publication date. We identified other relevant publications by backward citation searching. We analyzed 116 peer-reviewed articles, 4 preprints, and 28 reports.

Possible Risk Factors for Coccidioidomycosis and COVID-19

COVID-19 and coccidioidomycosis share certain risk factors for exposure, potentially increasing the risk for co-infection. In California and Arizona, the 2 states with the highest number of reported coccidioidomycosis cases, substantial overlap exists between

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county-level incidence of COVID-19 in 2020 and coccidioidomycosis in 2019 (Figures 1, 2).

Occupational Risks

Certain occupations pose increased risk for coccidioidomycosis. Because soil disruption and dusty environments promote dispersal of *Coccidioides* spores, coccidioidomycosis outbreaks frequently occur among workers in the construction and agricultural sectors (*8*,*9*,*10*). Of 47 coccidioidomycosis outbreaks reported during 1940–2015, a total of 25 (53%) were associated with occupational exposure, including 15 (60%) that were related to construction (*11*). An analysis of workers' compensation claims found that the incidence of coccidioidomycosis related to occupational exposure nearly quadrupled in California during 2000–2006, the highest rates seen among construction and agricultural workers (*12*).

Continued in-person work within the construction and agricultural sectors, which are considered essential occupations, also increases risk for COVID-19. In the United States, an estimated 8% of construction workers have had workplace exposure to the causative agent of COVID-19, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), at least monthly, and nearly 60% of the construction labor force has ≥1 risk factor for severe COVID-19 (*13*, *14*). Agricultural workers might also have heightened risk for COVID-19 because of high workforce turnover, shared transportation, and overcrowded living quarters that are often shared with other workers, multigenerational families, or both (*15*–*19*).

Incarcerated Populations

Incarcerated persons have a high risk for exposure to *Coccidioides* spores and SARS-CoV-2. Prisons and other facilities, such as immigration detention centers, are often in isolated areas with high environmental dust concentrations that can place inmates at higher risk for *Coccidioides* infection (Appendix, https://wwwnc.cdc.gov/EID/article/27/5/20-4661-App1.pdf). In addition, crowding, unsanitary conditions, and poor ventilation in carceral environments contributes to the rapid spread of communicable respiratory diseases like COVID-19 (*20*). Researchers have documented COVID-19 outbreaks among fire-fighting crews composed of incarcerated persons (*21*); similarly, researchers documented 7 coccidioidomycosis outbreaks among such fire-fighting crews during 2000–2017 (*22*). During 1940–2015, a total of 5 (11%) reported coccidioidomycosis outbreaks were among incarcerated populations (*11*). During 2007–2011, a total of 19% of coccidioidomycosis cases in California were among incarcerated persons (*23*). More than 25% of California Department of Corrections and Rehabilitation facilities, including Lompoc Prison Complex (Lompoc, CA, USA), where a COVID-19 outbreak infected >1,000 persons (*24*), are in regions with high coccidioidomycosis incidence (*25*).

Researchers have documented several outbreaks of COVID-19 in carceral facilities (Appendix). During January 21–April 21, 2020, a total of 82% of reporting state and territorial health department jurisdictions reported confirmed COVID-19 cases among incarcerated or detained persons (including 4,893 reported

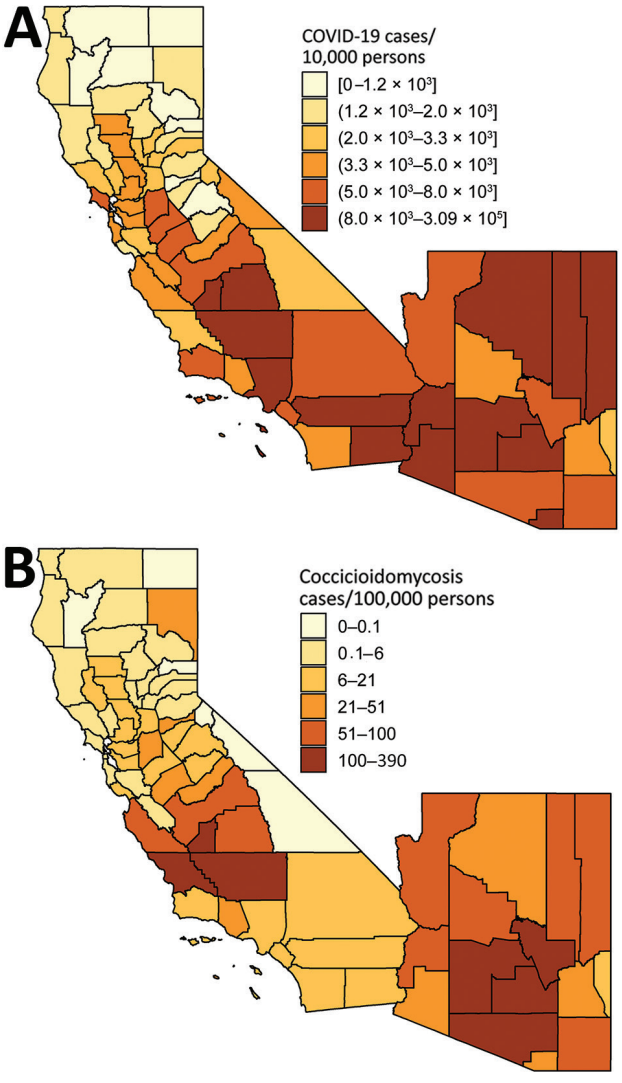

Figure 1. County-level incidence of (A) coronavirus disease (COVID-19) in 2020 and (B) coccidioidomycosis in 2019, California and Arizona. COVID-19 incidence reflects cumulative case count as of August 14, 2020 (*5*). Coccidioidomycosis incidence reflects annual incidence in 2019 (*6*,*7*). Shading indicates levels of incidence. Brackets indicate inclusive bounds; parentheses indicate exclusive bounds.

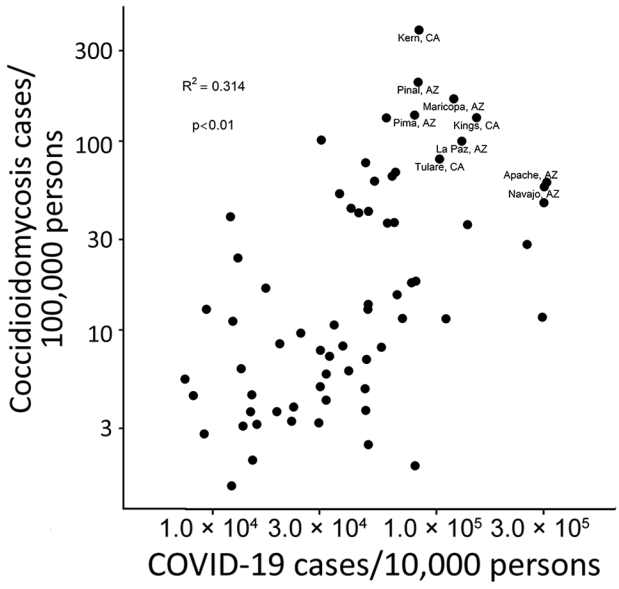

Figure 2. Scatterplot of county-level incidence of COVID-19 in 2020 and coccidioidomycosis in 2019, California and Arizona. R2 = 0.259; p<0.01.

cases and 88 deaths) or staff members (including 2,778 reported cases and 15 deaths) (*26*). COVID-19 outbreaks affecting >1,000 persons have occurred among incarcerated persons and staff working at carceral facilities in states from California to New York (Appendix).

Racial and Ethnic Minorities

Substantial racial and ethnic disparities exist in COVID-19 and coccidioidomycosis infection rates. Persons of Black and Latino heritage are at heightened risk for these infections. In California as of February 2021, Latino persons comprise 39% of the total population but account for 55% of COVID-19 cases (*27*). In the United States, COVID-19 incidence and death rates in counties with predominantly Black populations are significantly higher than in counties with predominantly white populations (*28*). In addition, Latino persons comprise 39% of the California population but 47% of its coccidioidomycosis patients; in the same state, non-Hispanic Black persons comprise 6% of the population but 9% of coccidioidomycosis patients (*3*).

Numerous societal inequities (including racism and discrimination, economic and educational disadvantages, and lack of healthcare access) contribute to higher pathogen exposure and infection rates among Black and Latino populations (*29*). In the context of the COVID-19 pandemic, social distancing might be more difficult for persons of low socioeconomic status because of their overrepresentation in essential occupations, elevated risk of living in densely populated homes and neighborhoods, and higher numbers of multigenerational households (*15*–*19*). For example, 55% of Latino and 48% of Black persons work in essential jobs, compared with 35% of White persons (*30*). Disparities in coccidioidomycosis rates might also be caused by the disproportionate numbers of Black and Latino persons who are incarcerated or work in occupations with high exposure risk. More than 50% of farm laborers, agricultural workers, and construction workers in California are Latino (*31*,*32*). In addition, Black and Latino persons are overrepresented in carceral facilities: in California, Black persons comprise 27% and Latino persons comprise 41% of jail and prison populations (*33*).

Exposure to Particulate Matter

Persons living in environments with high concentrations of dust, which is a major constituent of particulate matter ≤10 μm or ≤2.5 μm in diameter, might be at elevated risk for infection with *Coccidioides* and SARS-CoV-2 and severe illness from COVID-19. Exposure to particulate matter is a risk factor for illness and death from viral respiratory infections, including COVID-19 (Appendix). Exposure to outdoor particulate air pollution is also associated with *Coccidioides* infection because mineral dust can mobilize airborne spores (*34*,*35*). Coccidioidomycosis outbreaks have been linked to dust plumes generated by military exercises, agriculture, construction, archeology excavations, windstorms, and landslides (*36*–*43*). For example, in an outbreak affecting 89 persons at a solar farm, persons who reported being in a dust cloud had ≈6 times the odds of symptomatic coccidioidomycosis than those who were not in the dust cloud. Wetting the dirt before soil-disrupting activities, a common practice to reduce dust, decreased the odds of symptomatic infection by 58% (*44*). Because COVID-19 control measures encourage the use of outdoor spaces, persons might have increased exposures to mineral dust and other air pollutants during the pandemic.

Co-Circulation with SARS-CoV-2 Hampering Coccidioidomycosis Diagnosis

The diagnosis of coccidioidomycosis in areas with community transmission of COVID-19 might be challenging because the diseases cause similar symptoms, which might exacerbate existing delays in coccidioidomycosis diagnosis and treatment. Without antifungal treatment, coccidioidomycosis patients are at risk for severe illness, including disseminated disease, and for death (*45*). Promptly administering antifungal treatments reduces unnecessary use of antimicrobial drugs

and resolves symptoms more effectively (*45*). In addition, early case management, including assessing risk factors for severity, regular follow-up visits to monitor symptoms, regular testing to check antibody titer levels, and physical therapy, is crucial to mitigating severe disease (*46*).

One reason for the underdiagnosis of coccidioidomycosis is low testing rates. For instance, a study in Tucson, Arizona, estimated that 15%–44% of community-acquired pneumonia cases could be attributed to coccidioidomycosis (*47*), but only 2%–13% of community-acquired pneumonia cases were tested for coccidioidomycosis (*48*). Valdivia et al. (*47*) found that half of patients had ≥2 clinic visits before being tested for coccidioidomycosis. Low sensitivities of coccidioidomycosis tests might further contribute to delays in diagnosis (Appendix). Given such diagnostic constraints, the median time between seeking healthcare and coccidioidomycosis diagnosis was estimated to be 23 days in Arizona (*49*).

The COVID-19 pandemic might contribute to further delays in coccidioidomycosis diagnosis. Both diseases can cause dry cough, muscle aches, headache, fatigue, and difficulty breathing; however, patients with COVID-19 tend to have a more acute progression of symptoms than those with coccidioidomycosis (*50*; Appendix references *51*–*54*). Although pulmonary specialists and primary care physicians in regions to which coccidioidomycosis is endemic are probably aware of the diagnosis and treatment of this fungal infection, physicians in other regions might be less familiar with the diagnosis. Attributing coccidioidomycosis symptoms to COVID-19, whether presumed or laboratory-confirmed, might preclude coccidioidomycosis diagnosis in patients with monoinfections or co-infections. In addition, underutilization of healthcare services during the COVID-19 pandemic might result in further delays in the testing and diagnosis of coccidioidomycosis (Appendix reference *55*).

Risk Factors for Severe Disease

Although most cases of coccidioidomycosis or COVID-19 are mild respiratory illnesses, either infection can cause severe disease and death (Appendix). Risk factors associated with severe coccidioidomycosis or COVID-19 often overlap, prompting concerns of elevated death rates associated with co-infections or serial infections. Patients with SARS-CoV-2 and *Coccidioides* co-infection might be at higher risk for severe disease; however, whether synergistic effects might exist requires further data. Overlapping risk factors associated with severe disease caused by coccidioidomycosis or COVID-19 include older age, diabetes mellitus, immunosuppression, Black/African American ancestry, and smoking (Appendix references *56*–*70*). Although the long-term pulmonary effects of COVID-19 remain unknown, early data suggest that the virus might cause lung damage (Appendix reference *71*), resulting in elevated long-term risk for severe coccidioidomycosis.

Age

Older persons have heightened risk for severe disease caused by either infection. In the United States, 62% of COVID-19 hospitalizations and 80% of deaths were among patients >65 years of age (Appendix reference *72*). Similarly, older persons, especially those >65 years of age, with coccidioidomycosis have higher risk for severe pulmonary disease. Rates of coccidioidomycosis-associated death increase with age. These trends might be partially explained by the higher prevalence among older adults of preexisting conditions and immunosuppression, which are risk factors for severe COVID-19 and coccidioidomycosis (Appendix references *56*–*64*).

Diabetes

Diabetes is also associated with severe progression of either disease (Appendix references *56*,*63*–*68*). A study of COVID-19 patients found that those with diabetes had a higher risk for severe pneumonia and organ damage (Appendix reference *73*). The study also showed that patients with diabetes were more susceptible to a SARS-CoV-2–induced cytokine storm, which can cause rapid deterioration and death (Appendix reference *73*). In addition, patients with diabetes are more likely to have relapsing coccidioidomycosis (risk ratio [RR] 3.39, 95% CI 1.65–6.46) or cavitary lung disease (RR 2.94, 95% CI 1.63–4.90) than those without diabetes (Appendix reference *74*). Furthermore, among coccidioidomycosis patients with diabetes, those with higher serum glucose levels are more likely to have disseminated coccidioidomycosis, the most severe form of the disease, than those with lower levels (Appendix reference *74*). The exact mechanisms through which diabetes influences the progression of coccidioidomycosis and COVID-19 are not well understood but might be related to impaired innate and adaptive cellular immunity (especially T-cell function) or the effects of a hyperglycemic environment on microorganism virulence (Appendix reference *75*).

Immunosuppression

Although immunosuppressive steroids such as dexamethasone have reduced inflammatory lung

damage in patients with severe COVID-19 (Appendix reference *76*), emerging evidence suggests that persons with a history of prolonged immunosuppression might be at higher risk for severe COVID-19. A study of 17 million adults in the United Kingdom found higher risks for death among COVID-19 patients who have hematologic malignancies, who are taking immunosuppressant drugs for organ transplants, or who have other causes of immunosuppression (Appendix reference *77*). Immunosuppressed patients with cancer or solid organ transplants might be at higher risk for severe COVID-19 (Appendix reference *78*). Coccidioidomycosis patients with suppressed immune responses, such as patients with hematologic malignancies, HIV, or organ transplants, also have higher risk for disseminated disease (Appendix references *61*–*63*).

Black/African American Ancestry

Black persons have higher rates of severe COVID-19 and disseminated coccidioidomycosis than do White persons. Growing evidence indicates higher risk for severe COVID-19–associated disease and death among Black than White persons living in the United States (Appendix). A study of coccidioidomycosis in military personnel found dissemination rates to be 10 times higher among Black than White persons (Appendix reference *79*). Similarly, a study in Kern County, California, found that patients with disseminated coccidioidomycosis were 4.6 times more likely to be Black than patients with mild disease (Appendix reference *56*). The observed racial and ethnic disparities in severe COVID-19 and coccidioidomycosis are probably driven by structural inequities that systematically disadvantage persons of color in the forms of reduced healthcare access and exposure to environmental stressors that increase risk for conditions such as diabetes, obesity, and hypertension, which are associated with severe disease (*29*). For coccidioidomycosis, whether any biological basis for this association exists is unclear but might be related to immunogenic differences in T-cell function (Appendix references *56*,*69*,*70*).

Smoking

Recent history of cigarette smoking has been linked to higher risk for severe disease from either infection. A systematic review and meta-analysis found that smokers with COVID-19 were significantly more likely (RR 2.4, 95% CI 1.43–4.04) to be admitted to an intensive care unit, need mechanical ventilation, or die compared with nonsmokers (Appendix reference *80*). A case–control study in Kern County found that patients with severe or disseminated coccidioidomycosis were more likely to have smoked cigarettes in the previous 6 months compared with patients with mild coccidioidomycosis (Appendix reference *56*).

Possible Effects of Co-Infection on Disease Progression

Severe COVID-19

Underlying respiratory illness is a major risk factor for severe COVID-19 (Appendix references *60*,*64*). The Centers for Disease Control and Prevention reported that among COVID-19 patients in the United States with available data on concurrent conditions, 9.2% had a chronic lung disease such as chronic obstructive pulmonary disease, asthma, or emphysema; chronic lung disease was the most common concurrent condition after diabetes (Appendix reference *81*). The prevalence of chronic lung disease is higher among hospitalized patients (15%) and highest among patients in the intensive care unit (21%) (Appendix reference *81*). Several studies of COVID-19 patients in China have also shown elevated rates of death and severe disease among those with underlying chronic respiratory conditions (Appendix references *64*,*82*,*83*). Acute coccidioidomycosis is often self-limiting, but ≈3%–5% of patients have a chronic pulmonary form of the illness (Appendix references *84*,*85*). The evidence that chronic lung disease increases risk for severe COVID-19 suggests that patients with chronic pulmonary coccidioidomycosis might be predisposed to severe COVID-19.

Coccidioidomycosis Reactivation

Infection with COVID-19 might reactivate disease in a coccidioidomycosis patient whose illness has progressed to a chronic but inactive state. After an initial *Coccidioides* infection resolves, the fungus can remain in the lungs in a latent state and become reactivated under certain conditions (Appendix references *86*–*93*). Coccidioidomycosis reactivation has been reported among pregnant women, especially those who previously had disseminated coccidioidomycosis (Appendix reference *94*). Patients with organ transplants, which usually require immunosuppressive medications, also have higher rates of coccidioidomycosis reactivation (Appendix references *87*–*92*). SARS-CoV-2 infection has been associated with immune dysregulation, including lymphopenia (Appendix reference *95*), which might lower the host's ability to regulate *Coccidioides* infection (Appendix reference *96*). Although no studies have reported coccidioidomycosis reactivation in COVID-19 patients

as of February 2021, emerging evidence suggests that COVID-19 infection might accelerate the reactivation of latent tuberculosis (L. Pathak, unpub. data, https://www.biorxiv.org/content/10.1101/2020.05.06.077883v2). In addition, dexamethasone, a medication recommended for patients with severe COVID-19, increases the risk for severe coccidioidomycosis (Appendix references *97*,*98*).

Areas for Future Research

Cloth Masks

Although cloth masks are a critical control method for COVID-19 (Appendix), studies have not examined the efficacy of cloth masks for filtering *Coccidioides* arthroconidia. At 2–5 μm in diameter, *Coccidioides* arthroconidia are substantially larger than SARS-CoV-2; this size difference might lead to differing levels of filtration effectiveness (Appendix references *99*,*100*). One study found that cloth masks containing tightly woven cottons can filter 98% of particles in the 300 nm–6 μm range (Appendix reference *101*), yet such results are difficult to extrapolate to specific particles such as *Coccidioides* arthroconidia (Appendix reference *102*). It is also difficult to extrapolate results to other cloth masks, which vary widely in their filtration properties. Furthermore, leakage from improperly fitting masks can reduce efficacy of particle filtration by up to 50% (Appendix reference *101*). The effects of leakage on disease prevention might differ on the basis of infectious dose; although a single *Coccidioides* spore might confer infection, the infectious dose of SARS-CoV-2 is probably higher. California therefore requires employers with worksites in regions to which coccidioidomycosis is endemic to provide respiratory protection filters rated at least N95 to workers if dust cannot be controlled; no mask recommendation exists for the general public (Appendix reference *103*).

Climate

Transmission of SARS-CoV-2 and *Coccidioides* spores might be influenced by climatic conditions, such as temperature and humidity, that can affect pathogen survival and transport. For example, high humidity can suppress aerosol transmission of respiratory pathogens such as influenza and respiratory syncytial virus (Appendix references *104*–*110*). Early research in Wuhan, China, suggested that SARS-CoV-2 might be transmitted more efficiently in less humid environments (Appendix references *111*–*113*; W. Luo, unpub. data, https://www.medrxiv.org/content/10.1101/2020.02.12.20022467v1). Although the influence of temperature and other climatic conditions on transmission and seasonality of SARS-CoV-2 currently might be outweighed by the large size of the susceptible population, the introduction of a vaccine could result in patterns of population immunity that enable climate to play a larger moderating role (Appendix reference *114*). Because relative humidity plays a major role in regulating atmospheric dust concentrations, high atmospheric moisture can limit the dispersal of *Coccidioides* spores, potentially suppressing coccidioidomycosis transmission. For example, under wind conditions strong enough to mobilize dust, increases in relative humidity were associated with decreasing atmospheric dust concentrations (Appendix reference *115*).

Disparities in Surveillance

The extent of socioeconomic, demographic, racial, and other disparities in COVID-19 and coccidioidomycosis is probably greater than reflected in administrative data sources. For example, analyses from hard-hit regions have indicated that high rates of excess death probably reflect a large burden of unreported SARS-CoV-2 infection (Appendix reference *116*; J. Felix-Cardoso, unpub. data, https://www.medrxiv.org/content/10.1101/2020.04.28.20083147v1). Although testing coverage for SARS-CoV-2 is increasing, infections will probably continue to be undercounted in certain regions and populations because of factors such as disparate healthcare access, reagent shortages, and varied willingness to get tested. Undocumented or migrant farmworkers at high risk for exposure to *Coccidioides* spores are mostly uninsured, ineligible for healthcare benefits, or unable to afford healthcare (Appendix reference *117*,*118*). The disparities seen in rates of illness and death caused by COVID-19 and coccidioidomycosis might have many contributing factors, including barriers to affordable, high-quality, and accessible healthcare; occupational exposures; mass incarceration; residential segregation; discrimination; and differential rates of concurrent conditions. Understanding these disparities is critical for attracting the attention and resources needed to remedy inequities in exposures, care-seeking, and illness and death caused by coccidioidomycosis and COVID-19.

Conclusions

Public health professionals, healthcare providers, and populations in areas to which coccidioidomycosis is endemic should be aware of the overlap in risk factors for coccidioidomycosis and COVID-19. Because prompt diagnosis is critical for effective management of coccidioidomycosis and the COVID-19 pandemic might exacerbate existing delays,

healthcare professionals should know how to identify these diseases and potential co-infection. Agricultural and construction workers, firefighters, Black and Latino persons, persons with diabetes, elderly persons, incarcerated persons, and migrant or undocumented farmworkers might be at increased risk for coccidioidomycosis and COVID-19. Employers and public health officials should mitigate exposure to dust and SARS-CoV-2 by promoting the use of face masks and social distancing practices.

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Coccidioidomycosis and COVID-19 Co-Infection, United States, 2020

Appendix

Appendix Results

Coronavirus Disease Among Construction and Agricultural Workers

Construction, agriculture, and wildland firefighting are considered essential occupations under coronavirus disease (COVID-19) shelter-in-place guidelines for California and Arizona (*119*). Continued in-person work in these sectors poses challenges to maintaining physical distance and limiting contacts, resulting in higher risks for infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) (*120,121*). For instance, ≈8% of persons in construction occupations across the United States are exposed to SARS-CoV-2 ≥1 time a month (*121*), and nearly 60% of the construction labor force in the United States has ≥1 factor (>65 years of age or preexisting medical condition) that is associated with higher risk for severe illness from COVID-19 (*122*). Outbreaks of COVID-19 have been documented at multiple construction sites across the United States (*123–126*), including a cluster of 10 confirmed cases and >30 exposures at a construction site in Santa Clara, California (*126*), and 75 confirmed cases among a construction crew in Salt Lake City, Utah (*125*).

Agricultural workers might also have heightened risk for COVID-19 because of high workforce mobility, shared transportation, and overcrowded living quarters, often shared with other workers, multigenerational families, or both (*127–129*). One study estimated that >133,000 agricultural workers across the United States had tested positive for COVID-19 by September 1, 2020 (*130*), and media reports have documented clusters of COVID-19 at farms in >17 states (*131*). Some of the largest COVID-19 clusters among agricultural workers have been documented in California counties to which coccidioidomycosis is endemic, including Merced (1 cluster involving 392 COVID-19 cases and 8 deaths) (*132*), Ventura (3 clusters with 201, 35, and 28 COVID-19 cases) (*133–135*), and Monterey (1 cluster involving 247 COVID-19 cases) (*136*).

COVID-19 within Carceral Facilities

Crowding, unsanitary conditions, and poor ventilation in prison environments is known to contribute to rapid spread of communicable respiratory diseases (*137*), including influenza and tuberculosis (*138–141*). As the introduction of 1 case of influenza into a prison setting has been found to be sufficient to spark a large outbreak (*139*), concerns surrounding the spread of COVID-19 among detainees and staff members at correctional facilities are high (*142*,*143*). During January 21–April 21, 2020, 82% (32/37) of reporting state and territorial health department jurisdictions reported confirmed COVID-19 cases among incarcerated or detained persons or staff members (*144*). As of April 21, 2020, a total of 4,893 cases and 88 deaths among incarcerated and detained persons and 2,778 cases and 15 deaths among staff members in these 37 jurisdictions have been reported to the Centers for Disease Control and Prevention (CDC) (*144*). Large COVID-19 outbreaks have been documented among incarcerated persons and staff working at carceral facilities in Lompoc Prison Complex in Lompoc, California (n = 1,111 cases) (*145*), San Quentin State Prison in California (n = 2,221) (*145*), Rikers Island in New York, New York (n = 1,711) (*146*), the Cook County Jail in Chicago, Illinois (n = 1,040) (*147*), and Marion Correctional Institution in Marion, Ohio (n = 2,168) (*148*). COVID-19 outbreaks have also been documented at training camps for fire-fighting crews comprising incarcerated persons in northern California (*149*).

Coccidioidomycosis within Carceral Facilities

In 1 review, 5/47 (11%) reported coccidioidomycosis outbreaks were among incarcerated populations (*150*). During 2007–2011, 19% of coccidioidomycosis cases in California were among incarcerated persons (*151*). Over a quarter of California Department of Corrections and Rehabilitation facilities, including Lompoc Prison Complex, where a COVID-19 outbreak of >1,000 cases occurred (*147*), are in regions with high coccidioidomycosis incidence (*152*). One study showed that *Coccidioides* spores were detected in 15% of air samples taken outside Avenal State Prison (*153*) in Kings County, California, where the incidence of coccidioidomycosis during 2007–2011 was nearly 6 times higher than that of the nearby city (2,195 vs. 411 cases/100,000 population) and 14 times higher than the surrounding county (155 cases/100,000) (*151*). Pleasant Valley State Prison in Fresno County, CA, recorded an incidence of 3,323 cases/100,000 persons in 2005, or 415 times higher than the incidence of the surrounding county (8 cases/100,000 persons) (*154*). Other prisons in endemic areas, such as those in 3 Kern County

cities of Delano, Wasco, and Taft, have reported incidence rates ≈2 times that of the surrounding county (*151*).

COVID-19 and Particulate Matter

Persons living in environments with high concentrations of dust, which is an important constituent of particulate matter with diameter ≤10 μm (PM10) or ≤2.5 μm (PM2.5), might be at elevated risk for infection with *Coccidioides* and SARS-CoV-2, as well as increased severity of COVID-19 infection. Exposure to PM10 and PM2.5 has been recognized as a risk factor for disease and death from viral respiratory infection (*155*), including severe acute respiratory syndrome coronavirus (*156*). Macrophages laden with fine particles might have reduced ability to induce immune responses leading to increased disease severity (*157*,*158*), and PM2.5 has been shown to exacerbate underlying health conditions such as diabetes and chronic lung disease (*159*), that can complicate the course of viral respiratory infections. Evidence from several countries suggests that both acute and chronic exposure to fine particulate matter is associated with increased COVID-19 disease and death (*160*). For example, 1 study found that a 1 $\mu g/m^3$ increase in long-term exposure to PM2.5 was associated with an 8% increase in the COVID-19 death rate in counties across the United States (*161*). In a study of 120 cities in China, elevated particulate matter in the previous 2 weeks was associated with a 2.2% increase in daily confirmed COVID-19 cases (*162*). In Italy, the number of days in the previous 4 years that had exceeded regulatory limits for atmospheric pollutants such as PM2.5 and PM10 was significantly associated with increased COVID-19 cases (*163*).

Diagnostic Tests for Coccidioidomycosis

Laboratory confirmation is necessary to distinguish coccidioidomycosis from other conditions (*45*), and serologic detection of anticoccidiodal antibodies is the most common method to diagnose coccidioidomycosis infection, partly because of the low sensitivity (≈46%–67%) of culture-based methods in respiratory samples (*164–166*). Newer serologic assays for IgM antibodies have sensitivities ranging from 68% to 88% (*167*,*168*), but IgM antibodies are not typically detectable until 7–21 days after symptom onset, and IgG antibodies even later (*164*,*165*,*169*). As a result, a coccidioidomycosis diagnosis might occur ≥1 month after symptom onset. The high rate of false negatives (≤32%) in coccidioidomycosis testing (*165*) further complicate the situation. Given the low sensitivities, experts recommend repeated testing if the original test is negative and symptoms persist (*98*,*170*). COVID-19 might make patients less

likely to return to their healthcare providers for a second test due to fear of exposure to SARS-CoV-2 in medical facilities.

Prevalence of Severe Disease in COVID-19 and Coccidioidomycosis Patients

Among coccidioidomycosis patients, ≈5% develop severe chronic infections, and 1% progress to disseminated disease in which the infection spreads beyond the pulmonary system (*68*). Disseminated disease can lead to meningitis, bone and skin lesions, swollen joints, hospitalization, and death (*56*). An estimated 80% of COVID-19 cases are mild (no or mild pneumonia), 15% are severe (severe pneumonia and respiratory distress), and 5% are critical (respiratory failure, septic shock, organ dysfunction/failure) (*82*).

COVID-19 Risk among Black/African American Persons

Growing evidence points toward higher risk for severe disease and death from COVID-19 among Black persons living in the United States. Compared with White persons, the age-adjusted COVID-19 death rate on August 4, 2020 was 3.7 times higher among those who identify as Black (80.4 deaths/100,000 persons vs. 35.9 deaths/100,000 persons) (*171*). This trend is reflected across coccidioidomycosis endemic states, such as California and Arizona. In Arizona, the age-adjusted death rate from COVID-19 among Black populations is 2.1 times higher than among White populations. In California, the age-adjusted death rate from COVID-19 among Black populations is 3.0 times higher than among White populations (*171*).

COVID-19 and Cloth Masks

The CDC has recommended that all Americans wear cloth face coverings in public settings to reduce transmission of SARS-CoV-2 (*99*). Although cloth masks are inadequate for filtering out the SARS-CoV-2 virus (which is only 70–90 nm in size) and do not protect the wearer from inhaling viral particles (*172*), masks can protect wearers from virus suspended in large droplets and can prevent wearers from spreading droplets by coughing, sneezing, or breathing. Mask use is recommended for the entire population because persons are believed to be most infectious before the onset of COVID-19 symptoms (*173*). Models suggest that if 80% of the American population wore cloth face masks consistently, COVID-19 transmission would decrease significantly, even if lock-down restrictions were loosened (D. Kai, unpub. data, https://arxiv.org/abs/2004.13553v1), but 1 national survey found that only 60% of respondents follow CDC mask recommendations (*174*).

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