



# Health Considerations for HIV-Infected International Travelers

Daniel L. Bourque<sup>1,2</sup> · Daniel A. Solomon<sup>2,3</sup> · Paul E. Sax<sup>2,3</sup>

Published online: 12 April 2019

© Springer Science+Business Media, LLC, part of Springer Nature 2019

## Abstract

**Purpose of the Review** International travel continues to steadily increase, including leisure travel, travel to one's country of origin to visit friends and relatives, travel for service work, and business travel. Travelers with HIV may have an increased risk for travel-associated infections. The pre-travel medical consultation is an important means of assessing one's risk for travel-related health issues. The aim of this review is to provide an update on key health considerations for the HIV-infected traveler.

**Recent Findings** Like all travelers, the HIV-infected traveler should adhere to behavioral precautions, including safety measures with food and water consumption, safe sexual practices, and arthropod bite avoidance. HIV is a risk factor for venous thromboembolism and patients should be educated regarding this risk. Most pre-travel vaccines are safe and immunogenic in HIV-infected individuals, though live vaccines should be avoided in patients with low CD4 counts. Malaria chemoprophylaxis is strongly recommended in patients with HIV traveling to endemic areas and no significant interactions exist between the commonly used prophylactic anti-malarial agents and anti-retroviral therapy (ART). Travelers with HIV, particularly those who are not on ART or who have low CD4 cell counts, may have increased risk for tuberculosis, malaria, enteric infections, visceral leishmaniasis, American trypanosomiasis, and endemic mycoses such as histoplasmosis, talaromycosis, and coccidioidomycosis.

**Summary** The immune status of the HIV-infected traveler should be assessed prior to travel along with the duration, itinerary, and activities planned during travel in order to carefully consider individual risk for travel-related health issues.

**Keywords** HIV-international travel-infection

---

Daniel L. Bourque and Daniel A. Solomon are first co-authors.

---

This article is part of the Topical Collection on *Tropical, Travel, and Emerging Infections*

---

✉ Daniel L. Bourque  
dbourque@mah.harvard.edu

✉ Daniel A. Solomon  
dasolomon@bwh.harvard.edu

✉ Paul E. Sax  
psax@bwh.harvard.edu

<sup>1</sup> Division of Infectious Diseases and Travel Medicine, Mount Auburn Hospital, Cambridge, MA, USA

<sup>2</sup> Harvard Medical School, Boston, MA, USA

<sup>3</sup> Division of Infectious Diseases, Brigham and Women's Hospital, Boston, MA, USA

## Introduction

International travel has risen dramatically in the past two decades, especially to destinations in Africa, Asia, and the Middle East. In addition to leisure travel, there has been considerable growth in business travel, travel for service work, and travelers returning to their country of origin to visit friends and family [1]. International travel may place individuals at risk for infections due to pathogens endemic to the region visited. Individuals who travel internationally should take precautions to minimize the risk of travel-related infections and other complications. However, most travelers do not seek pre-travel consultation [2]. Furthermore, travelers who are returning to their country of origin to visit friends and relatives (VFR) seek pre-travel care less frequently and are at greater risk for travel-associated infections when compared to those traveling for tourism [3]. HIV-infected travelers are often VFR with destinations in countries where malaria, yellow fever, and other tropical diseases are endemic [4–6]. Travelers with HIV

may be at increased risk for travel-related infections and other adverse outcomes with the greatest risks for patients who are immunocompromised or not on antiretroviral therapy (ART). This review aims to provide a summary of the health considerations for HIV-infected international travelers, including the safety of travel-related vaccines, chemoprophylaxis, and the risk of travel-related infections and other health issues.

## Pre-travel Considerations

Pre-travel consultation for individuals with HIV should include a pre-travel health assessment and encompass general travel precautions in addition to a tailored discussion of how HIV may impact travel-associated risks.

## General HIV-Related Travel Guidance

Some countries have laws that may restrict entry for individuals with HIV. Laws vary in stringency by country and are variably enforced, so travelers with HIV are advised to speak with the destination country's consulate or embassy prior to travel.

Patients with HIV should travel with an adequate supply of ART for the duration of their trip to avoid lapse in treatment. Medication availability varies geographically, so long-term travelers (> 90 days) should consult with their clinicians regarding plans to obtain ART at their destination. Some travelers have friends or family members send medications monthly, others identify an alternative regimen available at their destination. All patients with HIV should identify available healthcare resources at their destination, preferably prior to travel.

HIV infection is an independent risk factor for venous thromboembolism (VTE). VTE is 2–10 times more common in HIV-infected individuals than the general population with the highest risk in patients with CD4 count < 200 cells/ $\mu$ L and a detectable viral load [7]. Primary prophylaxis of VTE with systemic anticoagulation is not recommended, but patients with HIV should be educated about the risk for blood clots and counseled regarding the importance of mobility and hydration when traveling especially on long flights.

## Malaria Chemoprophylaxis

Malaria chemoprophylaxis is strongly recommended in all HIV-infected individuals traveling to endemic regions. Pharmacokinetic (PK) data on the interactions between the different chemoprophylaxis agents and ART are limited. Ritonavir-boosted protease inhibitor (PI) combinations may induce glucuronidation of atovaquone leading to lower plasma concentrations, but the clinical significance of this reduction is unknown, and the combination atovaquone-proguanil

is generally considered safe [8]. A recent PK study of ART and chloroquine showed that Efavirenz (EFV) based ART reduces metabolism of chloroquine into desethyl-chloroquine, but did not result in an overall impact on plasma concentration of chloroquine [9]. Mefloquine is hepatically metabolized by CYP3A4, raising concern for significant interaction with PIs, but ritonavir has only a minimal effect on mefloquine pharmacokinetics despite strong inhibition of CYP3A4 [10]. There are no significant interactions between antiretrovirals and doxycycline. Based on available PK data, any of the above agents may be used for malaria prophylaxis and the choice should depend on whether the individual is traveling to an area with chloroquine-resistant malaria along with clinician discretion.

## Vaccines

Pre-travel vaccination is an essential component of safety in all travelers. HIV infection can interfere with vaccine efficacy as the immunologic response may be attenuated in the setting of immunosuppression. Antibody development to a vaccine depends on the patient's CD4 count and function and whether the given vaccine is T lymphocyte-dependent. A study of antibody formation in HIV-infected individuals stratified by CD4 count demonstrates this principle. Influenza vaccination is heavily dependent on the formation of antibodies against T lymphocyte antigens. For patients with CD4 counts < 100 cells/ $\mu$ L, almost no influenza antibodies were formed in response to the vaccine [11]. Even patients with CD4 cell count  $\geq$  200 cells/ $\mu$ L but below normal levels have demonstrated lower antibody response than HIV-uninfected individuals [12]. In contrast, after polysaccharide pneumococcal vaccination which is T lymphocyte-independent, normal antibody formation was observed even in patients with low CD4 counts [11].

The presence of circulating HIV RNA also has an important impact on vaccine response, independent of CD4 count. Decreased antibody production in the presence of circulating virus has been demonstrated with killed vaccines including influenza and hepatitis B vaccines and live vaccines such as varicella [12–14], and further highlights the importance of adherence to ART in order to optimize immunologic response. Conversely, vaccines may also have an effect on HIV activity. Vaccination has been linked to transient upregulation of HIV replication in some studies [15, 16]. There are no studies suggesting any negative effect on HIV disease progression, and the potential risk of increased HIV replication should not change clinical practice.

Despite the attenuated antibody response, killed and recombinant vaccines are safe in all HIV-infected individuals, and still have clinical effectiveness making them an important component of preventive care. A systematic review of influenza vaccination in immunocompromised patients showed

that serologic immune response was lower in HIV-infected individuals, but the rate of influenza-like illness was comparable to vaccinated immunocompetent patients [17]. Because the risk of infection is especially high in the setting of a low CD4 count, killed vaccines should not be delayed while awaiting immune reconstitution in patients with imminent risk such as travelers. The most recent NIH guidelines support the use of killed vaccines in this population with no changes from HIV-uninfected individuals [18]. This includes, but is not limited to, the following common vaccines considered during the pre-travel evaluation: influenza, tetanus, diphtheria, hepatitis A, hepatitis B, rabies, Japanese encephalitis, and meningococcus (see Table 1).

Live vaccines, in contrast, are not recommended in patients with CD4 count < 200 cells/ $\mu$ L due to the lack of safety data, but for patients with CD4 count  $\geq$  200 cells/ $\mu$ L, there are several live vaccines that should be considered during the pre-travel evaluation. Yellow fever vaccine is safe for patients with CD4 count  $\geq$  200 cells/ $\mu$ L and can be administered to patients traveling to endemic regions. A systematic review of observational studies that included 450 HIV-infected patients who received a yellow fever vaccine revealed no serious adverse events in this population [19••]. Measles, mumps, and rubella (MMR) were also shown to be safe in patients with CD4 count  $\geq$  200 cells/ $\mu$ L [20]. Varicella has been shown to be safe and effective in multiple studies of HIV-infected children [14, 21, 22], and pre-travel vaccination with the live varicella vaccine is recommended for susceptible HIV-infected adults with CD4 count  $\geq$  200 cells/ $\mu$ L [23]. One study assessed the safety of the live oral typhoid vaccine, Ty21a, in HIV-infected patients and found no severe adverse events [24]

in the 39 HIV-infected participants, which included six subjects with CD4 count < 200 cells/ $\mu$ L. However, more data are needed to determine the safety of Ty21a in HIV-infected patients. Therefore, the Vi polysaccharide vaccine is currently preferred over the live oral vaccine. For HIV-infected patients for whom polio vaccination is recommended, the inactivated polio vaccine is recommended.

### Prevention of Sexually Transmitted Infections (STIs)

Many individuals are sexually active while traveling and should be counseled about the global epidemiology of STIs, and the increased risk of STIs in travelers [25]. Rates of STIs vary widely by region, but are particularly high in many low- and middle-income countries [26]. Sex tourism is common in travelers and represents a growing industry in many parts of the world. Travelers should be cautioned about the high burden of STIs in commercial sex workers [27]. Individuals with HIV who are not on ART are at increased risk for acquisition of STI and for more severe manifestations of infection, and patients should be counseled about STI prevention strategies. Individuals with high-risk sexual practices who are HIV-uninfected should be educated about the role for HIV pre- and post-exposure prophylaxis to decrease the risk of HIV acquisition [28, 29].

### Infectious Risks While Traveling

Understanding the duration of travel, travel itinerary, planned activities, and type of accommodations is essential in

**Table 1** Vaccine recommendations in HIV-infected adults prior to travel by CD4 T cell counts

Vaccine	CD4 cell count > 200 cells/ $\mu$ L	CD4 cell count < 200 cells/ $\mu$ L
Measles, Mumps, Rubella (MMR)	Recommended	Contraindicated
Varicella	Recommended	Contraindicated
Yellow fever vaccine	If indicated	Contraindicated
Typhoid Vi polysaccharide	If indicated	If indicated
Ty21a Typhoid	Contraindicated	Contraindicated
Cholera (CVD 103-HgR)	Contraindicated	Contraindicated
Inactivated influenza	Recommended	Recommended
Hepatitis A	If indicated	If indicated
Hepatitis B	Recommended	Recommended
Inactivated polio vaccine	If indicated	If indicated
Meningococcal (MenACWY-D)	Recommended	Recommended
Japanese encephalitis (JE-VC)	If indicated	If indicated
Rabies	If indicated	If indicated
Td/Tdap	Recommended	Recommended
Pneumococcal conjugate (PCV13)	Recommended	Recommended
Pneumococcal polysaccharide (PPSV23)	Recommended	Recommended

assessing the risks of travel-associated infections. Counseling HIV-infected travelers should include advice on general behavioral precautions, including food and water precautions, insect repellent use, and avoiding other high-risk activities [30].

Travelers with HIV who are on ART with normal CD4 cell counts are not considered to be at increased risk of travel-associated infections compared to the general population of travelers. However, HIV-infected travelers who are not on ART or who have CD4 cell counts below 200 cells/ $\mu$ L are at increased risk for both typical travel-associated infections and opportunistic infections as highlighted below.

## Malaria

The global burden of malaria remains high, with an estimated 216 million cases in 2016 [31]. HIV-infected individuals who are not on ART have higher risk for malaria infection, and have demonstrated higher burden of parasitemia and higher incidence of severe infection and death than HIV-uninfected individuals [32, 33]. Moreover, acute malaria infection causes an increase in the HIV viral load and decrease in the CD4 cell count [34, 35].

ART has an important impact on a patient's risk for malaria as well as the efficacy of treatment in malaria-infected individuals. PIs have in vitro activity against *Plasmodium falciparum* though the clinical implications of this antiparasitic effect are unknown [36–38]. A study performed in Uganda where malaria is endemic randomized HIV-infected children to NNRTI-based ART or lopinavir-ritonavir (LPV/r) based ART and demonstrated a 41% reduction in the incidence of malaria in the lopinavir-ritonavir arm [39]. While direct antiparasitic effect of the PI may have played a role, the decreased incidence in the LPV/r arm was primarily attributed to a reduction of recurrence after treatment with artemether-lumefantrine suggesting the impact of ART on the efficacy of anti-malarial medications was a more important factor.

A follow-up PK study of malaria treatment in HIV-infected children on ART showed that selection of ART has a highly significant impact on plasma concentrations of antimalarial medications and clinical outcomes. EFV reduced the plasma concentration of both artemether and lumefantrine whereas LPV/r increased lumefantrine plasma concentrations resulting in fourfold higher odds of recurrent malaria in patients on EFV compared to LPV/r-based ART [40]. In another study, EFV decreased the serum concentration of dihydroartemisinin (DHA), the active component of AL, by 50% [41, 42]. Furthermore, subjects with co-administered EFV and AL, serum levels of lumefantrine on day 7, which are associated with clearance of infection, were significantly reduced [41]. Similar findings have been demonstrated when AL is combined with other non-nucleoside reverse transcriptase inhibitors (NNRTI) [43]. In addition, the CYP3A4 inhibitors, ritonavir and

cobicistat, may increase the serum concentration of lumefantrine, quinine, and quinidine and thus increasing the risk of toxicities, such as QT prolongation [44]. Modified dosing regimens of anti-malarial medication may need to be considered in some HIV-malaria co-infected patients and close clinical follow-up for malaria recrudescence following treatment is warranted for travelers on EFV or other NNRTI-based regimens who are infected with malaria. Individuals on PIs or integrase inhibitor (INSTI) based regimens should not be at increased risk for treatment failure with artemisinin-based combination therapy though may still be at increased risk for toxicities associated with lumefantrine and quinine (see Table 2). Because INSTI-based regimens are now preferred in most patients, clinicians may consider switching patients to dolutegravir- or bictegravir-based ART if malaria needs to be treated.

## Enteric Infections

Traveler's diarrhea (TD) is one of the most common travel-related illnesses and it is important that all travelers take precautions with food and water consumption to minimize the risk of TD. The etiology of traveler's diarrhea varies based on geographic region though includes bacterial pathogens such as enterotoxigenic and enteroaggregative *Escherichia coli*, *Campylobacter*, *Salmonella*, and *Shigella*, viral pathogens such as rotavirus and norovirus, and, less commonly, intestinal protozoa [45]. HIV-infected individuals with CD4 counts less than 200 cells/ $\mu$ L have an increased risk of infection due to bacterial enteropathogens and intestinal protozoa [46–48]. There is an increased incidence of *Campylobacter* infections in HIV-infected patients and higher risk for more severe illness, including bacteremia [49, 50]. Numerous studies have demonstrated an increased burden and severity of disease from non-typhoidal *Salmonella* in HIV-infected patients, which can present as two distinct syndromes, enteritis and bacteremia [51–53]. In addition, several studies have shown that there is increased susceptibility to shigellosis in HIV-infected patients, which appears to be predominantly due to sexual transmission in men who have sex with men [54–56]. In general, antibiotic prophylaxis for travelers' diarrhea (TD) is not recommended but should be considered in HIV-infected patients with a low CD4 count traveling to regions that are considered high risk for TD [57]. Prompt self-treatment with an antibiotic, typically azithromycin, for severe TD should be recommended to the HIV-infected traveler.

Though data on cholera infection in patients with HIV is limited, HIV was associated with a heightened risk of cholera in one recent study performed in Haiti [58]. While there is an oral cholera vaccine (live attenuated *V. cholerae* O1 CVD 103-HgR) approved for travelers in the USA, its safety has not been determined in immunocompromised individuals [59]. A clinical trial in Mali assessed the safety and

**Table 2** Impact of different ART classes on antimalarial agents

Antimalarial agent	Class of antiretroviral therapy					
	Nucleoside reverse transcriptase inhibitors (NRTIs)	Non-nucleoside reverse transcriptase inhibitors (NNRTIs)	Protease inhibitors (PIs)	Integrase strand inhibitors (INSTIs)	Cobicistat*	
Artemether/Artesunate	No relevant interactions	No relevant interactions	No relevant interactions	No relevant interactions	No relevant interactions	
Lumefantrine	No relevant interactions	Decreased concentration <sup>^</sup>	Increased concentration	No relevant interactions	Increased concentration	
Quinine/Quinidine	No relevant interactions	Decreased concentration <sup>^</sup>	Increased concentration <sup>+</sup>	No relevant interactions	Increased concentration	
Amodiaquine	No data	Hepatotoxicity	No data	No data	No data	
Primaquine	No data	No data	No data	No data	No data	
Atovaquone-Proguanil	No relevant interactions	Decreased concentration of proguanil <sup>++</sup>	Decreased concentration of proguanil <sup>^</sup>	No relevant interactions	Decreased concentration of proguanil <sup>^</sup>	
Doxycycline	No relevant interactions	No relevant interactions	No relevant interactions	No relevant interactions	No relevant interactions	
Mefloquine	No relevant interactions	No relevant interactions	Potential increased concentration <sup>+++</sup>	No relevant interactions	Potential increased concentration	
Chloroquine	No relevant interactions	No relevant interactions	Potential increased concentration <sup>+++</sup>	No relevant interactions	Potential increased concentration	

\* Not a direct acting antiretroviral agent; used in some regimens to increase concentration of ART by inhibiting hepatic metabolism

<sup>^</sup> Potentially compromised antimalarial activity

<sup>+</sup> Increased risk of toxicity including QT prolongation, electrolyte abnormality, cinchonism

<sup>++</sup> No significant clinical effect proven

<sup>+++</sup> Ritonavir only, not well studied

immunogenicity of a single dose of live oral cholera vaccine CVD 103-HgR in HIV-infected individuals and there were no increase in adverse events in individuals with HIV. However, the serological responses in HIV-infected, particularly those with CD4 counts less than 500 cells/ $\mu$ L, were significantly reduced [60]. Furthermore, given the low overall risk of cholera in travelers visiting endemic countries, the vaccine is favored only in individuals with high risk exposures such as aid and refugee workers traveling to endemic countries [61].

Enteric fever due to *Salmonella enterica* serotype Typhi and Paratyphi is a common cause of acute febrile illness in returning travelers [62]. Data on whether enteric fever is more common or severe in HIV-infected patients are conflicting. One study from Tanzania demonstrated significantly lower rates of bacteremia due to *Salmonella* Typhi in HIV-infected adolescents and young adults presenting with fever, suggesting a possible protective effect of HIV [63]. However, a cohort study from Peru demonstrated an increased risk of infection with *Salmonella* Typhi and Paratyphi in patients with HIV, with some cases presenting with severe diarrhea and colitis [64]. Another study of patients with typhoid fever in Tanzania demonstrated increased mortality and higher rates of intestinal perforation in HIV-infected patients with CD4 counts less than 200 cells/ $\mu$ L [65, 63]. Finally, an analysis from South Africa conducted from 2003 to 2013 and included 855 patients with typhoid fever showed that HIV infection was associated with increased mortality due to typhoid fever with an odds ratio of 11.3 [66]. Considering these data, food and water precautions should be strictly followed and when appropriate vaccination with the intramuscular Vi polysaccharide vaccine should be considered.

Travel may also place individuals at increased risk for intestinal protozoa, such as *Giardia duodenalis*, *Entamoeba histolytica*, *Cryptosporidium*, and other coccidian parasites [62, 67]. It has been well documented that coccidian parasites, including *Cryptosporidium*, *Cystoisospora belli*, and *Cyclospora*, can cause chronic diarrhea in healthy adults though they are much more common in immunocompromised patients with HIV in whom chronic infection is often accompanied by weight loss and malnutrition [67]. Microsporidia are another group of intestinal protozoa that can be acquired during travel, and are a well-characterized cause of chronic diarrhea in individuals with CD4 counts less than 100 cells/ $\mu$ L [67].

Intestinal helminths including *Strongyloides*, *Ascaris*, and hookworm are uncommon in travelers [68]. Nonetheless, they remain important considerations in travelers who return with gastrointestinal complaints and a peripheral eosinophilia. Multiple reports have shown increased rates of strongyloidiasis in HIV-infected patients, though these studies only included individuals living in endemic regions [69, 70]. One study from Uganda demonstrated an increased prevalence of hookworm infection in HIV-infected individuals which correlated

with decreased CD4 counts, suggesting hookworm infection may impact immune status in HIV-infected patients [71]. It is unclear if ascariasis is more frequent in HIV-infected patients. However, it has been shown that in patients with HIV and *Ascaris lumbricoides* co-infection, anti-helminthic therapy with albendazole led to improved CD4 counts, suggesting that ascariasis may impact HIV progression [72, 73]. Additional studies have also shown that individuals with HIV and helminth co-infection have an improvement in HIV viral loads and/or CD4 counts when treated with anti-helminthic therapy, suggesting helminth infection contributes to progression of HIV disease [74]. While helminth infections may be uncommon in short-term travelers, they are important considerations for the long-stay traveler or the expatriate with HIV infection.

## Tuberculosis

Tuberculosis (TB) remains a leading cause of morbidity and mortality in HIV-infected individuals, and patients with HIV are at increased risk for reactivation of latent tuberculosis infection (LTBI) [75–77]. TB risk appears to associate closely with the incidence rate of the region of travel [78]. In a prospective study of HIV-uninfected long-term travelers, there was a 1.8% rate of TB infection or 3.5 per 1000 person-months of travel, most of which were LTBI [79]. VFR travelers appear to have increased risk of acquiring TB during travel [80]. The TB risk of the respective destinations should be assessed in all travelers with HIV, including the immune status of the HIV-infected traveler. For HIV-infected travelers to high incidence countries, pre-travel assessment for LBTI with a tuberculin skin test or an interferon-gamma release assay is recommended with follow-up testing after travel has been completed. In cases of seroconversion, treatment for LTBI should be considered.

## Leishmaniasis

There are over sixteen *Leishmania* species that cause human disease with three major syndromes: cutaneous, mucocutaneous, and visceral leishmaniasis. Cutaneous and mucocutaneous leishmaniasis do not appear to be impacted by HIV infection, though there are reports of severe or refractory cases as well as possible visceralization with extracutaneous involvement in HIV-infected patients [81, 82]. On the other hand, visceral leishmaniasis (VL) is an important opportunistic infection in patients with HIV [83]. In VL endemic regions, HIV-infected individuals have significantly higher rates of VL compared to the general population and VL has been observed to contribute to HIV progression [84]. For instance, VL due to *Leishmania infantum* is endemic to the Mediterranean basin. In Spain, which has the highest burden of VL in this region, up to 37% of individuals with VL have HIV co-infection [83, 85]. In Ethiopia, there have been reports

of HIV/VL co-infection rates of up to 48.5% [86]. Therefore, in regions where VL is endemic, the traveler with HIV should take steps to avoid sand fly bites, including the use of insect repellent and bed net use. In addition, it is important to consider that VL may also be transmitted via sexual intercourse and needle sharing [87]. VL should be considered in the HIV-infected traveler to endemic regions who presents with a persistent febrile illness.

### Trypanosomiasis

*Trypanosoma cruzi*, the cause of American trypanosomiasis or Chagas disease, is rare in travelers. Chagas disease is found throughout Latin America and parts of the Caribbean and transmitted by triatomine bugs [88]. In a recent GeoSentinel survey of 42,173 ill returning travelers, there was one documented case of acute Chagas in a long-term traveler to Mexico [80]. In HIV-infected individuals, chronic Chagas can reactivate with low CD4 counts and typically manifests as an acute meningoencephalitis and brain abscess (Chagoma) [89]. It is worth noting that these cases are typical in individuals living in endemic regions who become infected with *T. cruzi* during childhood and subsequently acquire HIV. While the risk of acquiring *T. cruzi* infection while traveling to Latin America is low, it is reasonable to recommend sleeping under pyrethroid-impregnated bed nets when staying in thatched-roofed or mud-walled dwellings [90].

Human African trypanosomiasis (HAT), or African sleeping sickness, is transmitted by the bite of a tsetse fly and caused by *Trypanosom brucei rhodesiense* in East Africa and *Trypanosom brucei gambiense* in West and Central Africa. HAT is uncommon in travelers though most commonly seen in travelers visiting safari parks in East Africa [80]. Limited data exist on the interaction of HIV and HAT, though one study from West Africa showed that there was no association between the two [91].

### Schistosomiasis

Schistosomiasis is relatively common in travelers and is associated with an increased risk for HIV infection [80]. Multiple studies have provided evidence that there is an increased risk of HIV acquisition in females with genital schistosomiasis [92–94]. Increased HIV viral loads have been observed at the time of seroconversion in women with genital schistosomiasis, though it is unclear if schistosomiasis leads to progression of HIV disease [93]. There is no evidence to suggest that HIV infection increases susceptibility to schistosomiasis but avoiding freshwater exposure in regions where schistosomiasis is endemic should be recommended to all patients.

### Arboviral Infections

Travel may increase the risk of exposure to various arboviral infections, including dengue, Zika, chikungunya, yellow fever, and Japanese encephalitis. Mosquito bite avoidance and the use of insect repellents are key preventative measures in preventing arboviral infections and should be recommended to all travelers with destinations in endemic regions. In addition, effective vaccines for yellow fever and Japanese encephalitis are widely used for travelers. The live recombinant tetravalent dengue vaccine (CYD-TDV) is currently licensed in 20 countries for use in individuals living in endemic regions with prior dengue infection though not recommended for travelers [95]. It is unclear if arboviral infections may impact the course of HIV disease or have worse outcomes. There is limited data on the impact of HIV and Zika infection, including whether there are more adverse pregnancy outcomes in women infected with HIV [96]. A retrospective study from Thailand did not find worse outcomes in chikungunya infection in patients with HIV and it did not appear that chikungunya had an impact on HIV disease [97]. A matched case-control study in Singapore found that patients with HIV were more likely to develop severe manifestations of dengue [98]. However, another report from Mexico showed no difference in outcomes after dengue infection between HIV-infected and -uninfected patients [99].

### Fungal Infections

The HIV-infected traveler may have an increased risk of acquiring infection from endemic mycoses, including histoplasmosis, coccidioidomycosis, and talaromycosis. Histoplasmosis is endemic throughout the Americas, particularly Midwestern USA, Central America, and the northern part of South America [100]. A recent modeling study found that the incidence of histoplasmosis is equal to that of tuberculosis in patients with HIV in Latin America and that histoplasmosis is associated with greater mortality [100]. Patients with CD4 counts less than 150 cells/ $\mu$ L are at greatest risk for disseminated histoplasmosis [101]. Therefore, patients with low CD4 counts traveling to endemic regions should be counseled to avoid activities that increase risk for exposure to *Histoplasma capsulatum* including spelunking, work that includes disturbing soil or renovating old buildings [101]. *Talaromyces marneffe* is endemic to Southeast Asia, north-eastern India, and southern China and is a common opportunistic pathogen in this region [102]. While individuals living in endemic areas are at greatest risk, travel-related cases of talaromycosis have been reported [103, 104]. Coccidioidomycosis, found in the southwest USA and northern Mexico, Argentina, and other parts of South America, is another important mycosis in HIV-infected patients with CD4 counts less than 250 cells/ $\mu$ L [105]. Given the increased

incidence of infection in patients with low CD4 counts, risk of exposure to these fungal pathogens should be considered as part of the pre-travel assessment of HIV-infected individuals.

## Conclusion

With a rise in international travel, there is a heightened awareness of travel-related health issues. Pre-travel medical consultation is recommended for all travelers with HIV so pre-travel guidance can be tailored to an individual's personal risk factors in addition to the itinerary and planned activity during travel. HIV-infected individuals who are not on ART and/or have CD4 counts below 200 cells/ $\mu$ L are at the greatest risk for travel-related infections whereas patients who are stable on ART are generally not considered to be at increased risk over the general population. Clinicians should use the immune status of the HIV-infected patient to guide pre-travel vaccination, and to educate patients about their individual risk for both typical and opportunistic infections relevant to their destination.

## Compliance with Ethical Standards

**Conflict of Interest** Daniel L. Bourque, Daniel A. Solomon, and Paul E. Sax declare no conflict of interests.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

## References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Crotti R, Misrahi T. The Travel & Tourism Competitiveness Report 2017. Paving the way for a more sustainable and inclusive future. World Economic Forum: Geneva, Switzerland. p. 2017.
2. Hill DR. The burden of illness in international travelers. *N Engl J Med*. 2006;354:115–7.
3. Angell SY, Cetron MS. Health disparities among travelers visiting friends and relatives abroad. *Ann Intern Med*. 2005;142:67–72.
4. Elfrink F, van den Hoek A, Sonder GJB. Trends and characteristics among HIV-infected and diabetic travelers seeking pre-travel advice. *Travel Med Infect Dis*. 2014;12:79–83.
5. Salit IE, Sano M, Boggild AK, Kain KC. Travel patterns and risk behaviour of HIV-positive people travelling internationally. *CMAJ*. 2005;172:884–8.
6. Sherrard AW, McCarthy AE. Travel patterns and health risks for patients infected with HIV. *Travel Med Infect Dis*. 2009;7:291–5.
7. Bibas M, Biava G, Antinori A. HIV-associated venous thromboembolism. *Mediterr J Hematol Infect Dis*. 2011;3:e2011030.

8. Dooley KE, Flexner C, Andrade AS. Drug interactions involving combination antiretroviral therapy and other anti-infective agents: repercussions for resource-limited countries. *J Infect Dis*. 2008;198:948–61.
9. Ippolito MM, Jacobson JM, Lederman MM, Winterberg M, Tarning J, Shapiro TA, et al. Effect of antiretroviral therapy on plasma concentrations of chloroquine and desethyl-chloroquine. *Clin Infect Dis*. 2018; doi:<https://doi.org/10.1093/cid/ciy405>. **An important study assessing the impact of antiretroviral therapy on commonly used antimalarial medications.**
10. Khaliq Y, Gallicano K, Tisdale C, Carignan G, Cooper C, McCarthy A. Pharmacokinetic interaction between mefloquine and ritonavir in healthy volunteers. *Br J Clin Pharmacol*. 2001;51:591–600.
11. Kroon FP, van Dissel JT, de Jong JC, van Furth R. Antibody response to influenza, tetanus and pneumococcal vaccines in HIV-seropositive individuals in relation to the number of CD4+ lymphocytes. *AIDS*. 1994;8:469–76.
12. Fuller JD, Craven DE, Steger KA, Cox N, Heeren TC, Chernoff D. Influenza vaccination of human immunodeficiency virus (HIV)-infected adults: impact on plasma levels of HIV type 1 RNA and determinants of antibody response. *Clin Infect Dis*. 1999;28:541–7.
13. Mehta N, Cunningham CK, Flynn P, Pepe J, Obaro S, Kapogiannis BG, et al. Impaired generation of hepatitis B virus-specific memory B cells in HIV infected individuals following vaccination. *Vaccine*. 2010;28:3672–8.
14. Levin MJ, Gershon AA, Weinberg A, Song L-Y, Fentin T, Nowak B, et al. Administration of live varicella vaccine to HIV-infected children with current or past significant depression of CD4(+) T cells. *J Infect Dis*. 2006;194:247–55.
15. Glesby MJ, Hoover DR, Farzadegan H, Margolick JB, Saah AJ. The effect of influenza vaccination on human immunodeficiency virus type 1 load: a randomized, double-blind, placebo-controlled study. *J Infect Dis*. 1996;174:1332–6.
16. Staprans SI, Hamilton BL, Follansbee SE, Elbeik T, Barbosa P, Grant RM, et al. Activation of virus replication after vaccination of HIV-1-infected individuals. *J Exp Med*. 1995;182:1727–37.
17. Beck CR, McKenzie BC, Hashim AB, Harris RC. University of Nottingham Influenza and the ImmunoCompromised (UNIIC) Study Group, Nguyen-Van-Tam JS. Influenza vaccination for immunocompromised patients: systematic review and meta-analysis by etiology. *J Infect Dis*. 2012;206:1250–9.
18. adult\_oi.pdf. Available: [https://aidsinfo.nih.gov/contentfiles/lvguidelines/adult\\_oi.pdf](https://aidsinfo.nih.gov/contentfiles/lvguidelines/adult_oi.pdf)
19. Barte H, Horvath TH, Rutherford GW. Yellow fever vaccine for patients with HIV infection. *Cochrane Database Syst Rev*. 2014; CD010929. **An extensive meta-analysis summarizing the key data regarding the safety and immunogenicity of yellow fever vaccine in HIV-infected patients.**
20. Stermole BM, Grandits GA, Roediger MP, Clark BM, Ganesan A, Weintrob AC, et al. Long-term safety and serologic response to measles, mumps, and rubella vaccination in HIV-1 infected adults. *Vaccine*. 2011;29:2874–80.
21. Son M, Shapiro ED, LaRussa P, Neu N, Michalik DE, Meglin M, et al. Effectiveness of varicella vaccine in children infected with HIV. *J Infect Dis*. 2010;201:1806–10.
22. Bekker V, Westerlaken GHA, Scherpbier H, Alders S, Zaaier H, van Baarle D, et al. Varicella vaccination in HIV-1-infected children after immune reconstitution. *AIDS*. 2006;20:2321–9.
23. Rubin LG, Levin MJ, Ljungman P, Davies EG, Avery R, Tomblyn M, et al. 2013 IDSA clinical practice guideline for vaccination of the immunocompromised host. *Clin Infect Dis*. 2014;58:e44–100.
24. Banda R, Yambayamba V, Lalusha BD, Sinkala E, Kapulu MC, Kelly P. Safety of live, attenuated oral vaccines in HIV-infected Zambian adults: oral vaccines in HIV. *Vaccine*. 2012;30:5656–60.

25. Matteelli A, Schlagenhauf P, Carvalho AC, Weld L, Davis XM, Wilder-Smith A, et al. Travel-associated sexually transmitted infections: an observational cross-sectional study of the GeoSentinel surveillance database. *Lancet Infect Dis*. 2013;13:205–13.
26. Ward BJ, Plourde P. Travel and sexually transmitted infections. *J Travel Med*. 2006;13:300–17.
27. Carr N. Sex in tourism: reflections and potential future research directions. *Tourism Recreation Res Routledge*. 2016;41:188–98.
28. Updated Guidelines for Antiretroviral Postexposure Prophylaxis After Sexual, Injection-Drug Use, or Other Nonoccupational Exposure to HIV—United States, 2016. Available: <https://www.cdc.gov/hiv/pdf/programresources/cdc-hiv-npep-guidelines.pdf>
29. Brett-Major DM, Scott PT, Crowell TA, Polyak CS, Modjarrad K, Robb ML, et al. Are you PEPped and PrEPped for travel? Risk mitigation of HIV infection for travelers. *Trop Dis Travel Med Vaccines*. 2016;2:25.
30. Freedman DO, Chen LH, Kozarsky PE. Medical considerations before international travel. *N Engl J Med*. 2016;375:247–60.
31. World Health Organization World malaria report 2017. World Health Organization; 2018.
32. Whitworth J, Morgan D, Quigley M, Smith A, Mayanja B, Eotu H, et al. Effect of HIV-1 and increasing immunosuppression on malaria parasitaemia and clinical episodes in adults in rural Uganda: a cohort study. *Lancet*. 2000;356:1051–6.
33. French N, Nakiyingi J, Lugada E, Watera C, Whitworth JA, Gilks CF. Increasing rates of malarial fever with deteriorating immune status in HIV-1-infected Ugandan adults. *AIDS*. 2001;15:899–906.
34. Mermin J, Lule JR, Ekwaru JP. Association between malaria and CD4 cell count decline among persons with HIV. *J Acquir Immune Defic Syndr*. 2006;41:129–30.
35. Kublin JG, Patnaik P, Jere CS, Miller WC, Hoffman IF, Chimbiya N, et al. Effect of plasmodium falciparum malaria on concentration of HIV-1-RNA in the blood of adults in rural Malawi: a prospective cohort study. *Lancet*. 2005;365:233–40.
36. Skinner-Adams TS, McCarthy JS, Gardiner DL, Hilton PM, Andrews KT. Antiretrovirals as antimalarial agents. *J Infect Dis*. 2004;190:1998–2000.
37. Nsanzabana C, Rosenthal PJ. In vitro activity of antiretroviral drugs against plasmodium falciparum. *Antimicrob Agents Chemother*. 2011;55:5073–7.
38. Parikh S, Gut J, Istvan E, Goldberg DE, Havlir DV, Rosenthal PJ. Antimalarial activity of human immunodeficiency virus type 1 protease inhibitors. *Antimicrob Agents Chemother*. 2005;49:2983–5.
39. Achan J, Kakuru A, Ikilezi G, Ruel T, Clark TD, Nsanzabana C, et al. Antiretroviral agents and prevention of malaria in HIV-infected Ugandan children. *N Engl J Med*. 2012;367:2110–8.
40. Parikh S, Kajubi R, Huang L, Ssebuliba J, Kiconco S, Gao Q, et al. Antiretroviral choice for HIV impacts antimalarial exposure and treatment outcomes in Ugandan children. *Clin Infect Dis*. 2016;63:414–22.
41. Huang L, Parikh S, Rosenthal PJ, Lizak P, Marzan F, Dorsey G, et al. Concomitant efavirenz reduces pharmacokinetic exposure to the antimalarial drug artemether-lumefantrine in healthy volunteers. *J Acquir Immune Defic Syndr*. 2012;61:310–6.
42. Byakika-Kibwika P, Lamorde M, Mayito J, Nabukeera L, Namakula R, Mayanja-Kizza H, et al. Significant pharmacokinetic interactions between artemether/lumefantrine and efavirenz or nevirapine in HIV-infected Ugandan adults. *J Antimicrob Chemother*. 2012;67:2213–21.
43. Kakuda TN, DeMasi R, van Delft Y, Mohammed P. Pharmacokinetic interaction between etravirine or darunavir/ritonavir and artemether/lumefantrine in healthy volunteers: a two-panel, two-way, two-period, randomized trial. *HIV Med*. 2013;14:421–9.
44. Taylor WRJ, White NJ. Antimalarial drug toxicity: a review. *Drug Saf*. 2004;27:25–61.
45. Steffen R, Hill DR, DuPont HL. Traveler's diarrhea: a clinical review. *JAMA*. 2015;313:71–80.
46. Angulo FJ, Swerdlow DL. Bacterial enteric infections in persons infected with human immunodeficiency virus. *Clin Infect Dis*. 1995;21(Suppl 1):S84–93.
47. Stark D, Barratt JLN, van Hal S, Marriott D, Harkness J, Ellis JT. Clinical significance of enteric protozoa in the immunosuppressed human population. *Clin Microbiol Rev*. 2009;22:634–50.
48. Sanchez TH, Brooks JT, Sullivan PS, Juhász M, Mintz E, Dworkin MS, et al. Bacterial diarrhea in persons with HIV infection, United States, 1992–2002. *Clin Infect Dis*. 2005;41:1621–7.
49. Kownhar H, Shankar EM, Rajan R, Vengatesan A, Rao UA. Prevalence of Campylobacter jejuni and enteric bacterial pathogens among hospitalized HIV infected versus non-HIV infected patients with diarrhoea in southern India. *Scand J Infect Dis*. 2007;39:862–6.
50. Tee W, Mijch A. Campylobacter jejuni bacteremia in human immunodeficiency virus (HIV)-infected and non-HIV-infected patients: comparison of clinical features and review. *Clin Infect Dis*. 1998;26:91–6.
51. Gordon MA, Banda HT, Gondwe M, Gordon SB, Boeree MJ, Walsh AL, et al. Non-typhoidal salmonella bacteraemia among HIV-infected Malawian adults: high mortality and frequent recrudescence. *AIDS*. 2002;16:1633–41.
52. Dhanoa A, Fatt QK. Non-typhoidal Salmonella bacteraemia: epidemiology, clinical characteristics and its' association with severe immunosuppression. *Ann Clin Microbiol Antimicrob*. 2009;8:15.
53. Preziosi MJ, Kandel SM, Guiney DG, Browne SH. Microbiological analysis of nontyphoidal Salmonella strains causing distinct syndromes of bacteremia or enteritis in HIV/AIDS patients in San Diego, California. *J Clin Microbiol*. 2012;50:3598–603.
54. Mohan K, Hibbert M, Rooney G, Canvin M, Childs T, Jenkins C, et al. What is the overlap between HIV and shigellosis epidemics in England: further evidence of MSM transmission? *Sex Transm Infect*. 2018;94:67–71.
55. Baer JT, Vugia DJ, Reingold AL, Aragon T, Angulo FJ, Bradford WZ. HIV infection as a risk factor for shigellosis. *Emerg Infect Dis*. 1999;5:820–3.
56. Toro C, Arroyo A, Sarria A, Iglesias N, Enriquez A, Baquero M, et al. Shigellosis in subjects with traveler's diarrhea versus domestically acquired diarrhea: implications for antimicrobial therapy and human immunodeficiency virus surveillance. *Am J Trop Med Hyg*. 2015;93:491–6.
57. Riddle MS, Connor BA, Beeching NJ, DuPont HL, Hamer DH, Kozarsky P, et al. Guidelines for the prevention and treatment of travelers' diarrhea: a graded expert panel report. *J Travel Med*. 2017;24:S57–74.
58. Richterman A, Cheung HC, Meiselbach MK, Jerome G, Ternier R, Ivers LC. Risk factors for self-reported cholera within hiv-affected households in rural Haiti. *Open Forum Infect Dis*. 2018;5:ofy127.
59. Cabrera A, Lepage JE, Sullivan KM, Seed SM. Vaxchora: a single-dose oral cholera vaccine. *Ann Pharmacother*. 2017;51:584–9.
60. Perry RT, Plowe CV, Koumaré B, Bougoudogo F, Kotloff KL, Losonsky GA, et al. A single dose of live oral cholera vaccine CVD 103-HgR is safe and immunogenic in HIV-infected and HIV-noninfected adults in Mali. *Bull World Health Organ*. 1998;76:63–71.
61. Wittlinger F, Steffen R, Watanabe H, Handszuh H. Risk of cholera among Western and Japanese travelers. *J Travel Med*. 1995;2:154–8.

62. Harvey K, Esposito DH, Han P, Kozarsky P, Freedman DO, Plier DA, et al. Surveillance for travel-related disease—GeoSentinel surveillance system, United States, 1997–2011. *Morb Mortal Wkly Rep Surveill Summ.* JSTOR. 2013;62:1–23.
63. Crump JA, Ramadhani HO, Morrissey AB, Saganda W, Mwako MS, Yang L-Y, et al. Invasive bacterial and fungal infections among hospitalized HIV-infected and HIV-uninfected adults and adolescents in northern Tanzania. *Clin Infect Dis.* 2011;52:341–8.
64. Gotuzzo E, Frisancho O, Sanchez J, Liendo G, Carrillo C, Black RE, et al. Association between the acquired immunodeficiency syndrome and infection with *Salmonella typhi* or *Salmonella paratyphi* in an endemic typhoid area. *Arch Intern Med.* 1991;151:381–2.
65. Chalya PL, Mabula JB, Koy M, Kataraiya JB, Jaka H, Mshana SE, et al. Typhoid intestinal perforations at a university teaching hospital in Northwestern Tanzania: a surgical experience of 104 cases in a resource-limited setting. *World J Emerg Surg.* 2012;7:4.
66. Keddy KH, Sooka A, Smith AM, Musekiwa A, Tau NP, Klugman KP, et al. Typhoid fever in South Africa in an endemic HIV setting. *PLoS One.* 2016;11:e0164939.
67. Okhuysen PC. Traveler's diarrhea due to intestinal protozoa. *Clin Infect Dis.* 2001;33:110–4.
68. Freedman DO, Weld LH, Kozarsky PE, Fisk T, Robins R, von Sonnenburg F, et al. Spectrum of disease and relation to place of exposure among ill returned travelers. *N Engl J Med.* 2006;354:119–30.
69. Assefa S, Erko B, Medhin G, Assefa Z, Shimelis T. Intestinal parasitic infections in relation to HIV/AIDS status, diarrhea and CD4 T-cell count. *BMC Infect Dis.* 2009;9:155.
70. Feitosa G, Bandeira AC, Sampaio DP, Badaró R, Brites C. High prevalence of giardiasis and strongyloidiasis among HIV-infected patients in Bahia, Brazil. *Braz J Infect Dis.* 2001;5:339–44.
71. Morawski BM, Yunus M, Kerukadho E, Turyasingura G, Barbra L, Ojok AM, et al. Hookworm infection is associated with decreased CD4+ T cell counts in HIV-infected adult Ugandans. *PLoS Negl Trop Dis.* 2017;11:e0005634.
72. Blish CA, Sangaré L, Herrin BR, Richardson BA, John-Stewart G, Walson JL. Changes in plasma cytokines after treatment of ascaris lumbricoides infection in individuals with HIV-1 infection. *J Infect Dis.* 2010;201:1816–21.
73. Walson JL, Otieno PA, Mbuchi M, Richardson BA, Lohman-Payne B, Macharia SW, et al. Albendazole treatment of HIV-1 and helminth co-infection: a randomized, double-blind, placebo-controlled trial. *AIDS.* 2008;22:1601–9.
74. Walson JL, Herrin BR, John-Stewart G. Deworming helminth co-infected individuals for delaying HIV disease progression. *Cochrane Database Syst Rev.* 2009; CD006419.
75. Getahun H, Gunneberg C, Granich R, Nunn P. HIV infection—associated tuberculosis: the epidemiology and the response. *Clin Infect Dis Oxford University Press.* 2010;50:S201–7.
76. Pawlowski A, Jansson M, Sköld M, Rottenberg ME, Källenius G. Tuberculosis and HIV co-infection. *PLoS Pathog.* 2012;8:e1002464.
77. WHO|Global tuberculosis report 2018. World Health Organization; 2018; Available: [https://www.who.int/tb/publications/global\\_report/en/](https://www.who.int/tb/publications/global_report/en/)
78. Jung P, Banks RH. Tuberculosis risk in US Peace Corps Volunteers, 1996 to 2005. *J Travel Med.* 2008;15:87–94.
79. Cobelens FG, van Deutekom H, Draayer-Jansen IW, Schepp-Beelen AC, van Gerven PJ, van Kessel RP, et al. Risk of infection with *Mycobacterium tuberculosis* in travellers to areas of high tuberculosis endemicity. *Lancet.* 2000;356:461–5.
80. Leder K, Torresi J, Libman MD, Cramer JP, Castelli F, Schlagenhaut P, et al. GeoSentinel surveillance of illness in returned travelers, 2007–2011. *Ann Intern Med.* 2013;158:456–68.
81. Nicolás M, Gimenez-Arnau A, Camarasa JG. Cutaneous leishmaniasis in AIDS. *Dermatology.* 1995;190:255–6.
82. Lartey M, Adusei L, Hanson-Nortey L, Addy J. Coinfection of cutaneous Leishmaniasis and HIV infection. *Ghana Med J.* 2006;40:110–2.
83. Alvar J, Aparicio P, Aseffa A, Den Boer M, Cañavate C, Dedet J-P, et al. The relationship between leishmaniasis and AIDS: the second 10 years. *Clin Microbiol Rev.* 2008;21:334–59 table of contents.
84. Lopez-Velez R, Perez-Molina JA, Guerrero A, Baquero F, Villarrubia J, Escribano L, et al. Clinicoepidemiologic characteristics, prognostic factors, and survival analysis of patients coinfecting with human immunodeficiency virus and *Leishmania* in an area of Madrid, Spain. *Am J Trop Med Hyg.* 1998;58:436–43.
85. Gil-Prieto R, Walter S, Alvar J, de Miguel AG. Epidemiology of leishmaniasis in Spain based on hospitalization records (1997–2008). *Am J Trop Med Hyg.* 2011;85:820–5.
86. Diro E, Lynen L, Ritmeijer K, Boelaert M, Hailu A, van Griensven J. Visceral Leishmaniasis and HIV coinfection in East Africa. *PLoS Negl Trop Dis.* 2014;8:e2869.
87. Burza S, Croft SL, Boelaert M. Leishmaniasis. *Lancet.* 2018;392:951–70.
88. Pérez-Molina JA, Molina I. Chagas disease. *Lancet.* 2018;391:82–94.
89. Bern C. Chagas disease in the immunosuppressed host. *Curr Opin Infect Dis.* 2012;25:450–7.
90. Diaz JH. Recognizing and reducing the risks of Chagas disease (American trypanosomiasis) in travelers. *J Travel Med.* 2008;15:184–95.
91. Meda HA, Doua F, Laveissière C, Miezan TW, Gaens E, Brattegaard K, et al. Human immunodeficiency virus infection and human African trypanosomiasis: a case-control study in Côte d'Ivoire. *Trans R Soc Trop Med Hyg.* 1995;89:639–43.
92. Kjetland EF, Ndhlovu PD, Gomo E, Mduluzi T, Midzi N, Gwanzura L, et al. Association between genital schistosomiasis and HIV in rural Zimbabwean women. *AIDS.* 2006;20:593–600.
93. Downs JA, Dupnik KM, van Dam GJ, Urassa M, Lutonja P, Kornelis D, et al. Effects of schistosomiasis on susceptibility to HIV-1 infection and HIV-1 viral load at HIV-1 seroconversion: a nested case-control study. *PLoS Negl Trop Dis.* 2017;11:e0005968.
94. Brodish PH, Singh K. Association between *Schistosoma haematobium* exposure and human immunodeficiency virus infection among females in Mozambique. *Am J Trop Med Hyg.* 2016;94:1040–4.
95. Dengue vaccine: WHO position paper, September 2018 - Recommendations. *Vaccine.* 2018; doi:<https://doi.org/10.1016/j.vaccine.2018.09.063>
96. João EC, da Ferreira O, C Jr, Gouvêa MI, de Teixeira M, LB, Tanuri A, Higa LM, et al. Pregnant women co-infected with HIV and Zika: outcomes and birth defects in infants according to maternal symptomatology. *PLoS One.* 2018;13:e0200168.
97. Kositpantawong N, Charoenmak B, Siripaitoon P, Silpapojakul K. 1724 Clinical presentations and interactions of the Chikungunya viral infection in HIV patients during the Chikungunya epidemic in Southern Thailand. *Open forum infectious diseases.* Oxford University Press; 2014;1: S462.
98. Pang J, Thein T-L, Lye DC, Leo Y-S. Differential clinical outcome of dengue infection among patients with and without HIV infection: a matched case-control study. *Am J Trop Med Hyg.* 2015;92:1156–62.
99. Espinoza-Gómez F, Delgado-Enciso I, Valle-Reyes S, Ochoa-Jiménez R, Arechiga-Ramírez C, Gámez-Arroyo JL, et al. Dengue virus coinfection in human immunodeficiency virus-1-

- infected patients on the West Coast of Mexico. *Am J Trop Med Hyg.* 2017;97:927–30.
100. Adenis AA, Valdes A, Cropet C, McCotter OZ, Derado G, Couppie P, et al. Burden of HIV-associated histoplasmosis compared with tuberculosis in Latin America: a modelling study. *Lancet Infect Dis.* 2018;18:1150–9.
  101. Sarosi GA, Johnson PC. Disseminated histoplasmosis in patients infected with human immunodeficiency virus. *Clin Infect Dis.* 1992;14(Suppl 1):S60–7.
  102. Vanittanakom N, Cooper CR Jr, Fisher MC, Sirisanthana T. *Penicillium marneffei* infection and recent advances in the epidemiology and molecular biology aspects. *Clin Microbiol Rev.* 2006;19:95–110.
  103. Castro-Lainez MT, Sierra-Hoffman M, Llompart-Zeno J, Adams R, Howell A, Hoffman-Roberts H, et al. *Talaromyces marneffei* infection in a non-HIV non-endemic population. *IDCases.* 2018;12: 21–24.
  104. Walsh TJ, Groll A, Hiemenz J, Fleming R, Roilides E, Anaissie E. Infections due to emerging and uncommon medically important fungal pathogens. *Clin Microbiol Infect.* 2004;10(Suppl 1):48–66.
  105. Brown J, Benedict K, Park BJ, Thompson GR 3rd. *Coccidioidomycosis: epidemiology.* *Clin Epidemiol.* 2013;5: 185–97.
- Publisher's Note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.