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A Review on Sporotrichosis and the Emergence of *Sporothrix brasiliensis* as a Pathogen

Alessandro F. Valdez[1,2] · Dario Corrêa-Junior[3] · Jhon Jhamilton Artunduaga Bonilla[1] · Daniel Zamith-Miranda[2] · Susana Frases[3,4] · Dayvison Francis Saraiva Freitas[5] · Rodrigo Almeida-Paes[4,6] · Leonardo Nimrichter[1,4] · Joshua D. Nosanchuk[2]

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Abstract

Purpose of Review This review explores sporotrichosis development as a disease in both cats and humans as well as options for diagnosis and treatment. This work also discusses the factors that might have culminated on the emergence of *Sporothrix brasiliensis* as the main etiological agent of this disease.

Recent Findings Sporotrichosis is currently an epidemic in Brazil with cats acting as the primary vector of the disease. And, although molecular diagnostic techniques have been recently developed, the disease remains largely unchecked evidencing the need for novel therapeutic options as well as a more effective public health response.

Summary It is becoming more evident that to manage and control sporotrichosis, a One Health approach needs to be globally adopted. In addition to that, global warming is creating increasingly favorable conditions to the emergence of fungal pathogens.

Keywords Sporotrichosis, *Sporothrix* · *Sporothrix brasiliensis* · *Sporothrix schenckii* · Global warming · Climate change

Introduction

Sporotrichosis, caused by the thermodimorphic fungi of the *Sporothrix* genus, is the most common subcutaneous mycosis worldwide. Although ubiquitous globally in the environment, most fungi from the *Sporothrix* genus do not usually cause human or animal infections [1]. Until recently, sapronotic transmission was the most common source of human sporotrichosis, especially in North America and Europe, with infection usually beginning after cutaneous trauma related to recreational or occupational activities such as gardening, farming, and mining, giving the disease the epithet of "Rose Gardener's Disease" [2].

Until the 1990s, *Sporothrix schenckii* was presumed to be the sole agent of sporotrichosis until recent data defined the pathogenic species as *Sporothrix globosa*, *Sporothrix brasiliensis*, *Sporothrix luriei*, and *Sporothrix schenckii* sensu stricto [1, 3]. Some of these species have high endemicity in countries such as China, Japan, Australia, India, South Africa, and Brazil [4]. In Asia, particularly in China, *S. globosa* is the causative agent in 99.3% of cases of human sporotrichosis [5]. *S. schenckii* is responsible for 94% of cases in Australia and South Africa, and it accounts for 89% of cases

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✉ Joshua D. Nosanchuk
josh.nosanchuk@einsteinmed.edu

1 Laboratório de Glicobiologia de Eucariotos, Departamento de Microbiologia Geral, Instituto de Microbiologia Paulo de Góes, Universidade Federal Do Rio de Janeiro, Rio de Janeiro, RJ 21941-902, Brazil

2 Departments of Medicine (Division of Infectious Diseases) and Microbiology and Immunology, Albert Einstein College of Medicine, Bronx, NY 10461, USA

3 Laboratório de Biofísica de Fungos, Instituto de Biofísica Carlos Chagas Filho, Universidade Federal Do Rio de Janeiro, Rio de Janeiro, RJ 21941-170, Brazil

4 Rede Micologia RJ, FAPERJ, Rio de Janeiro, RJ 21941-902, Brazil

5 Laboratório de Pesquisa Clínica Em Dermatologia Infecciosa, Instituto Nacional de Infectologia Evandro Chagas, Fundação Oswaldo Cruz, Rio de Janeiro, RJ 21040-360, Brazil

6 Laboratório de Micologia, Instituto Nacional de Infectologia Evandro Chagas, Fundação Oswaldo Cruz, Rio de Janeiro, RJ 21040-360, Brazil

in North and South America [6]. In Brazil, however, since the identification of a zoonotic sporotrichosis case transmitted from a cat to a human in 1998, sporotrichosis has emerged as a challenging epidemic in the region [7], with *S. brasiliensis* as the main etiological agent, accounting for 88% of cases reported in the south and southeast regions of the country, especially in Rio de Janeiro State, where the disease has been considered hyperendemic over the last two decades [3, 8•, 9, 10].

The *Sporothrix* Genus and the Emergence of *S. brasiliensis*

The recent emergence of some of these species, *S. brasiliensis* in particular, has shifted the historic paradigm of sapronotic sporotrichosis transmission, with zoonotic and enzootic infection becoming increasingly common in Brazil [1]. Previous contact with decaying plant material is commonly reported in *S. globosa* and *S. schenckii* infections, and sapronotic transmission of *S. brasiliensis* is possible [1]. *S. brasiliensis* is generally transmitted via bites, scratches, or direct contact with cutaneous lesions of infected cats [1, 4, 11, 12]. While dogs are generally not considered a significant source of human infections caused by *S. brasiliensis*, *Sporothrix* spp. has been isolated from the oral cavity and conjunctival mucosa of dogs, allowing for dog-to-human transmission [1]. Overall, sporotrichosis can be transmitted through various routes, including sapronotic, zoonotic, and animal horizontal transmission (Fig. 1).

Cat-transmitted sporotrichosis (CTS) has been reported in the American and Asian continents mostly as isolated cases and small outbreaks [11]. Isolated cases of feline sporotrichosis have also been reported in Spain, Japan, and Germany; however, there has been no evidence or report of cat-to-human transmission in these countries [13–15]. The situation is different in South America, especially in Brazil, where CTS emerged in the state of Rio de Janeiro in 1998 and currently remains hyperendemic and has begun to spread to other states and South American countries [16•, 17, 18]. Argentina, Paraguay, and Chile are among those countries where CTS has recently emerged [1, 19–21]. In 2022, in the UK, the first three cases of CTS due to *S. brasiliensis* were reported involving mother and daughter of a Brazilian family and the veterinarian of their cat with sporotrichosis, apparently only developed after three years living in the UK [22].

There are a few different factors that could explain the changes in the disease profile and recent emergence of *S. brasiliensis* and CTS epidemic such as environmental factors (changes in temperature and humidity), evolution, and urbanization coupled with changes in human culture and the instinct cat behavior, as well as an inadequate public health response [21].

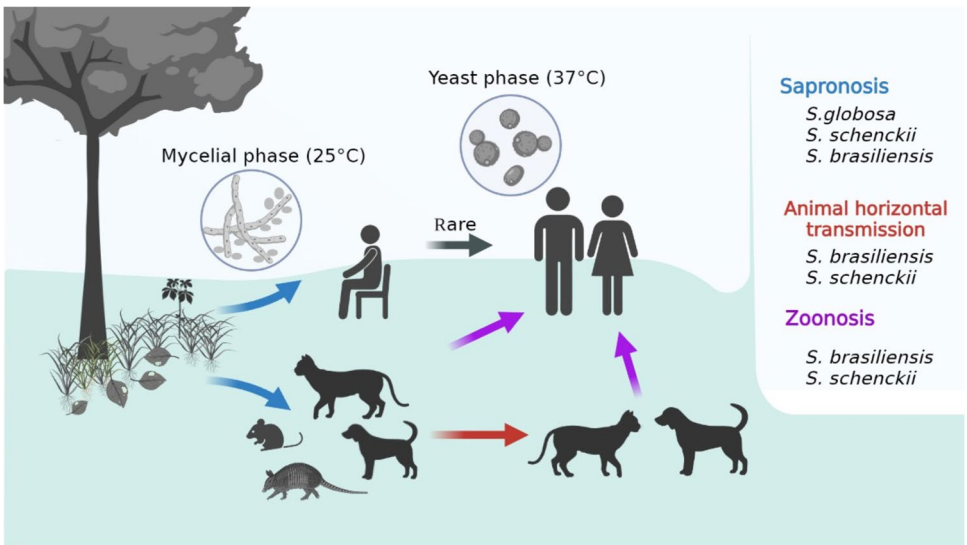

Fig. 1 Sporotrichosis transmission routes. *Sporothrix* spp. are commonly found in soil and decomposing plant matter in a mycelial form, which can lead to infections in humans and animals by sapronotic transmission (blue arrows). This occurs when filamentous propagules are traumatically inoculated into the skin. Deep scratches or contact with exudate from the cutaneous lesions of ill cats can transmit the infection horizontally to other animals (red arrows), cats being the most susceptible. Likewise, the infection can reach humans by zoonosis (purple arrows) where high load of yeast is inoculated through scratches, bites, and secretions from cats with sporotrichosis. Human–human transmission is extremely rare, but transmission is possible when daily interactions involve direct contact with injuries (dark gray arrow). The most involved species according to the routes of transmission are shown on the right side of the figure. Original figure, created with BioRender.com

Different approaches can help us analyze those factors and help us understand the problem. From a public health point of view, it is important to assess what has been done (or not) in the last two decades, to put sporotrichosis in check in its hyperendemic region. The geographic expansion of CTS cases is partially related to the fact that fungal infections are generally neglected with limited public health policies around the world [21, 23, 24]. Notably, *Sporothrix* was left off the 2022 World Health Organization Fungal Priority Pathogen List [25]. In Brazil, sporotrichosis is a known problem, but its rise and spread have been poorly addressed leading to an uncontrolled situation [21]. The expansion of the disease is a direct result of socioeconomic difficulties coupled with scarce or inadequate health services. A good example of this is that it took 16 years for the government of Rio de Janeiro, the major CTS endemic area, to implement an animal sporotrichosis control program, with free diagnosis and treatment for animals, and, even then, control measures have remained relatively inefficient with compulsory notification not universally performed and a general absence of educational campaigns to inform the population, leading to an ever-growing number of human and animal cases [21].

Other valid approach to understand why *S. brasiliensis* emergence is happening now is to explore the environmental and evolutionary point of view, and for that it is necessary to evaluate what differentiates *S. brasiliensis* to its counterparts and how that correlates to environmental changes in the last two decades. A collection of requirements exists for fungi to act as a primary pathogen [26, 27]. Among those are the ability to invade/bypass host barriers, evade/withstand the immune system, acquire nutrients in the human tissue, and, more importantly for our discussion, the ability to grow at or above host temperature [26, 27]. Not coincidentally, *S. brasiliensis* exhibits high thermotolerance and can effectively infect both humans and cats, which have a slightly higher body temperature ranging from 38 to 39 °C, whereas other members of the *Sporothrix* genus like *S. globosa* do not thrive well above 35 °C [28]. In addition, recent studies with clinical isolates suggest that *S. brasiliensis* has the capacity to undergo microevolutions within its host, increasing virulence in vivo over the course of infection [29–32]. These facts alone could help us explain why *S. brasiliensis* was so successful not only as a primary pathogen, but in its adaptation as a cat pathogen, which in turn collaborates to the spread of the disease, but we believe the correlation between its emergence and thermotolerance is not that simple and passes through environmental factors such as climate change.

S. brasiliensis grows in soil and decaying plant material, with warmer and damp conditions creating more opportunities for the fungus to proliferate and infect humans and animals. These environmental conditions justify why the majority of cases reported are concentrated in regions with tropical weather [33]. Nevertheless, in the last two decades, the number of cases reported in the hyperendemic regions seems disproportional, leading to a belief that global warming triggered the emergence of the thermotolerant species [19, 34–37].

The combination of endothermy and a complex immune system has been identified as the cause for the relatively high resistance of mammals against fungal infections [34]. Furthermore, the correlation between thermotolerance and virulence in fungal pathogens has been broadly discussed in the literature, as mammals are capable to maintain high body temperatures in comparison to environmental temperatures, which creates a thermally restrictive ambient for the majority of fungi, with all common fungal pathogens sharing the characteristic of being thermotolerant [26, 34–37].

In 2010, Garcia-Solache and Casadevall hypothesized that global warming is playing a key role in promoting the emergence of new fungal diseases in mammals by both increasing the geographic range of current pathogenic species and also selecting species for adaptive thermotolerance [34]. The emergence and spread of *S. brasiliensis* in Brazil and South America can be supported by both mechanisms described above. The rise in temperature favors evolution and adaptation of a thermotolerant *Sporothrix* species, while secondary effects of global warming like the increased rainfall totals help create the perfect environmental conditions to *S. brasiliensis* to thrive [38]. This, in combination with the large number of stray cats roaming the streets of Rio de Janeiro and other urban areas, created the "perfect storm" scenario to facilitate the emergence and spread of *S. brasiliensis*.

Pathogenesis

After traumatic inoculation from the environment or a cat, which enables *Sporothrix* propagules like conidia and mycelial fragments or yeast cells, respectively, to enter the host, the disease usually limits itself to the skin, subcutaneous tissue, and adjacent lymphatic vessels, accounting for the majority of cases reported [39•, 40]. Less frequently, but not rare in the cat transmission scenario, multiple skin lesions may arise from multiple inoculations (scratches and bites), or at the other extreme, skin or mucous lesions may arise after a non-traumatic contact with cats with sporotrichosis (touching exudates or being exposed to cat sneezing) [9, 39•, 41]. Atypical clinical presentations can also occur, especially in immunocompromised individuals or in the rare event of fungal conidia being inhaled from the environment, ranging from pulmonary sporotrichosis to disseminated and meningeal forms of the disease [40, 42–44]. Unfortunately,

the Brazilian epidemic has had an increase in the number of disseminated infections [10].

The disease has a fairly broad incubation period, ranging from a few days to a few months [45]. Clinical manifestations begin with the development of a small papule or pustule at the inoculation site, which evolves into a nodule, ulcerating or not, involving the skin and usually with additional similar nodular-ulcerative lesions along the ascending lymphatic vessels [45]. This ascending distribution defines the classic lymphocutaneous form and, when there is a single lesion, it is classified as the fixed or localized cutaneous form with systemic symptoms typically absent [45]. The atypical clinical presentations, either extra-cutaneous or systemic, occur following contiguous or hematogenous spread from the primary affected site or from pulmonary infection, both cases more commonly observed in immunocompromised individuals, especially the hematogenous spread [9, 45]. It is worth mentioning that, besides the immune status of the patient, the clinical manifestations of sporotrichosis can vary based on several factors, including the virulence, inoculum site and size, and thermal tolerance of the strain [19].

The concept of virulence refers to the ability of a microbe to cause damage in a host, with injury resulting from the microbial processes, host immune response, or both, leading to a state of disease when homeostasis is disturbed [46]. The *Sporothrix* genus exhibits several virulence factors such as glycoproteins, secreted proteins, extracellular vesicles, thermotolerance, melanin production, ergosterol peroxide, dimorphism, and the ability to form biofilm in both filamentous and yeast forms [28, 47]. The genus produces a heat shock protein, namely, the chaperone HtpG or HSP90, which is crucial for preserving the yeast form of the fungus [48]. In 2009, Arrillaga-Moncrieff et al. performed a comparative study in the murine model that indicated that the potential virulence of the main human pathogenic species of the *Sporothrix* genus varies [49], with *S. brasiliensis* being considered the most virulent, *S. globosa* the least virulent, and *S. schenckii* exhibiting an intermediate virulence phenotype, findings that are also supported by epidemiological data [8•, 19, 49].

In humans, sporotrichosis is categorized into cutaneous, mucosal, and extracutaneous forms based on the location of the lesions [39•, 50, 51]. Apart from the cutaneous presentations already described, *S. brasiliensis* has been associated with ocular involvement, disseminated disease, central nervous system (CNS) disease, and hypersensitivity reactions [52–54].

As for cats, which are the primary animal host and vector for human infection, multiple ulcerated skin lesions associated with enlarged lymph nodes and respiratory signs are the most common clinical manifestations of sporotrichosis [55]. The incubation period after infection ranges from three to 30 days [56]. While disseminated sporotrichosis cases in humans are more prevalent among individuals with immunosuppressive conditions, such an association was not found for feline sporotrichosis, as cats diagnosed with feline immunodeficiency virus (FIV) or feline leukemia virus (FeLV) did not exhibit an increased incidence of sporotrichosis [57]. For currently unknown reasons, *S. brasiliensis* is not easily controlled by the cat immune system, compared to response typical in humans and certain other mammals, such as dogs, leading to high fungal burdens in these susceptible animals.

Diagnosis and Treatment

In a context of hyperendemicity, especially considering areas with little access to mycology laboratories, the diagnosis of sporotrichosis is based on clinical-epidemiological probability. However, ideally, the isolation of the fungus from skin lesion scraping, exudate, or biopsy is the primary microbiologic diagnostic method for sporotrichosis. Other clinical specimens are used according to the affected sites. Biological materials from human skin lesions and other tissue samples present challenges in visualizing small yeast cells due to the low fungal load causing direct microscopic examination (DME) to be ineffective. Giemsa-stained purulent lesion imprints, biopsies, or aspirates enhance sensitivity [8•], and histopathological examinations with periodic acid-Schiff (PAS) or Gomori-methenamine silver (GMS) staining detect granulomas with epithelioid cells, yeast cells (rare in immunocompetent humans), and, in some cases, asteroid bodies [45]. In cases of extracutaneous or disseminated forms, the ideal clinical sample to isolate the fungus for definitive diagnosis will depend on the sites of *Sporothrix* infection and can include deep biopsies, sputum, cerebrospinal fluid, and blood cultures, for example. Stains like Gram, Giemsa, PAS, and GMS are helpful in these cases. Molecular techniques are considered ideal for epidemiological studies and correctly classify all species. The calmodulin (CAL), β-tubulin, and elongation factor (EF) genes are the main targets used in these techniques [16•, 58, 59]. It is also worth to mention that immunologic tools have been developed in the last two decades, and antibody detection through enzyme-linked immunosorbent assay (ELISA) is available for the presumptive diagnosis of both feline and human sporotrichosis [60–62]. Furthermore, nested PCR or quantitative PCR can be used to detect *Sporothrix* DNA in clinical samples with lower fungal burden such as the cerebrospinal fluid [63••].

Itraconazole is the drug of choice for treating sporotrichosis in humans and animals, but treatment is usually prolonged, especially in cats, with a treatment duration typically spanning from 3 to 6 months or even longer for severe or low-responsive cases [17, 64, 65]. A dosage of

Table 1 Compounds that exhibited remarkable in vitro activity against *Sporothrix* spp

Compound	Strains tested	[a]MIC_{50}	Mechanism of effect	Reference
Pentamidine	*S. brasiliensis* (10), *S. globose* (2), *S. mexicana* (4), and *S. schenckii* sensu stricto (3) clinical isolates	0.06–0.25 µg/mL	Not fully understood, but may inhibit DNA topoisomerases and impacts cell surface	[81]
Terpinen-4-ol (T-OH)	*S. brasiliensis* (6), *S. globose* (3), *S. Mexicana* (3), and 3 *S. schenckii* sensu stricto clinical isolates	4–32 µg/mL	Interferes with ergosterol biosynthesis	[82]
α- and β-2,3-dihydrofuranaphthoquinones	*S. brasiliensis* CBS 133,006 and *S. schenckii* ATCC 3228	2–32 µM	Impacts redox cycles, induces DNA strand breaking, results in ROS accumulation	[83]
Pentathiepin-based inhibitors	*S. brasiliensis* (14 isolates from feline sporotrichosis)	0.5–˃8 µg/mL	Not described	[91]
Silver salts of Keggin-type heteropolyacid (Ag-HPA)	*S. schenckii* ATCC 32285 and 7 isolates from feline sporotrichosis	8–128 µg/mL	Impacts both cell wall and vacuolization; induces a cytoplasm disorder and membrane detachment	[92]
MMV002817 (iodoquinol) and MMV102872	*S. brasiliensis* CBS 133006 *and S. schenckii* ATCC 32286; human (6) and feline (2) isolates of *Sporothrix brasiliensis* and *schenckii*	0.12–0.5 µM	Inhibits ergosterol biosynthesis; impacts enzymatic activity and metabolic pathways	[93]
Ibuprofen	*S. schenckii* ATCC 16345 and ATCC 32286; *S. schenckii* and *S. brasiliensis* (10 clinical isolates)	128–512 µg/mL (alone) (16–256 µg/mL when combined with AMB)	Causes plasma membrane damage and ROS accumulation	[90]
Acylhydrazone derivatives (BHBM, D13, SB-AF-1002)	*S. brasiliensis* ATCC 5110/MYA4823 and *S. schenckii* ATCC 1099–18/MYA4821	0.12–0.5 µg/mL	Impacts vesicular transport, cell budding, and cell cycle progression	[86]
Essential oil of *Origanum majorana* Linn. (marjoram)	*S. brasiliensis* isolate from feline sporotrichosis	2.25–18 mg/mL	Binds ergosterol, causing pores in the membrane	[84]
Buparvaquone	*S. brasiliensis* (CBS 133006, ATCC MYA 4823, and ATCC MYA 4824) and 17 clinical isolates from feline sporotrichosis	0.005–0.16 µg/mL	Causes plasma membrane damage impacting mitochondrial activity, ROS and neutral lipid accumulation	[89]
Silver chitosan nanocomposites	*S. brasiliensis* ATCC 5110/MYA4823 and *S. schenckii* ATCC 1099–18/MYA4821	0.06–0.25 µg/mL	Causes discontinuity of the plasma membrane, disruption of cytoplasmic organelles, and cell wall disaggregation	[87]
NAC-SNO NPs and SNO-MPs	*S. schenckii 1099–18, S. globosa CFP1021, and S. brasiliensis CFP0551*	5 mg/mL*	Provides a sustained release of nitric oxide	[88]
Hydroethanol extract derived from the leaves of *S. terebinthifolius*	*S. brasiliensis* CBS 120339, *S. schenckii* IPEC 36277, *S. globose* IPEC 27135, and 5 *S. brasiliensis* clinical isolates	0.25–1 µg/mL	Not described	[85]
1,4-Naphthoquinone derivates	*S. brasiliensis* CBS 133006 and *S. schenckii* ATCC 32286	1–16 µM	Results in ROS accumulation, mitochondrial disturbances, and damage to plasma membranes	[94]

[a]*MIC_{50}* minimum inhibitory concentration considered the lowest compound concentration that produced 50% inhibition compared to the yeast control

*The concentrations tested in this study (5–10 mg/mL) had inhibitory activity greater than 50% of inhibition

100 to 400 mg per day is used for treating sporotrichosis in humans as it results in clinical improvement in nearly all patients and has an acceptable incidence of adverse effects, ranging from 10 to 40%, mostly mild [66–68]. Cats, on the other hand, are often reported as low or unresponsive to itraconazole [69] and little is known about predictors of the treatment response; however, the occurrence of respiratory signs and lesions on nasal mucosa and skin with high fungal loads was associated with treatment failure [70]. If patients are unresponsive to itraconazole, potassium iodide solution or terbinafine can be used as oral treatments for localized cutaneous forms [17]. Antifungal agents that act on ergosterol biosynthesis, such as polyenes, azoles, and allylamines, are also effective treatments. Amphotericin B, a polyene antifungal, is reserved for severe cases, but it can be cardiotoxic and nephrotoxic [71, 72].

Sporothrix species show the potential to develop in vitro resistance to conventional antifungals. *S. schenckii* [73] and *S. globosa* [74] have developed in vitro resistance to itraconazole in human infections, *S. schenckii* in felines [75, 76], and *S. brasiliensis* in dogs [77]. Recently, some cases of human *S. brasiliensis* infection caused by strains with reduced in vitro susceptibility to azoles, terbinafine, and/or amphotericin B have been described. These cases require extended treatment times and may develop sequelae [78]. The unequivocal correlation with clinical unresponsiveness and in vitro resistance remains to be proven, since host factors play an important role in the outcomes. Antifungal resistance in *Sporothrix* is related to the fungus' ability to produce melanin, low genetic diversity, possibly due to the abnormal number of chromosomes, and single nucleotide polymorphisms in cytochrome P450 or other genes related to antifungal resistance [79, 80]. Sporotrichosis has a well-established therapeutic protocol, but the need for long courses of treatment and the occurrence of feline and human cases unresponsive to the treatment of choice or even with relapses, maybe related to resistant strains or low drug absorption and metabolism, highlight the importance of the development of novel antifungal drugs and therapeutic options, preferably more efficient and less toxic.

Significant research efforts have explored various therapeutic options for treating sporotrichosis. Over the past several years, these efforts have focused on diverse alternatives such as synthetic organic chemical compounds [81–83], essential oils [84], plant extracts [85], metal complexes [86, 87], nitric oxide (NO) releasing particles [88], and repositioned drugs [89, 90]. These compounds have exhibited promising results in vitro, as evidenced in Table 1. Moreover, some of these compounds show antifungal efficacy in animal models of sporotrichosis, further highlighting their potential as viable therapeutic options.

Conclusions

It is important to highlight that a One Health approach needs to be adopted globally to prevent and control the epidemic of *S. brasiliensis* and CTS. This means that surveillance, prevention, and treatment efforts should cover not only people and animals but also the environment, which is particularly crucial in densely populated urban areas where the risk of transmission is higher [95].

The possibility for gaining a better understanding of the transfer of both pathogenic and non-pathogenic microorganisms between humans, animals, and the environment is also of fundamental importance. This is achieved by analyzing evidence of the interactions that take place within this ecosystem [96]. Furthermore, global warming concerns are often focused elsewhere and the potential for its effects on fungal pathogens is broadly overlooked. A warmer climate has the potential to create favorable conditions to thermotolerant species [34], which can lead to a change in their geographic distribution. Adaptation to higher temperatures also has the potential to create conditions that could turn environmental species into mammal pathogens, as for every 1 °C gained in body temperature between 30 and 42 °C, approximately 6% of fungal species are currently excluded as potential pathogens [34, 37], meaning that global warming is particularly dangerous because it narrows down the thermal gradient between environmental conditions and mammalian temperatures [34].

Taking all the facts together, *S. brasiliensis* poses an especially challenging threat to control. We believe that only a One Health approach, coupled with increased research to enhance our understanding of the disease and to create alternative diagnostics and therapeutics, and implementation of educational campaigns for both health professionals and general population can stop the further spread of sporotrichosis.

Author Contribution AFV, DCJ, JJAB, DZM, SF, DFSF, and RAP wrote the main manuscript text. AF, JAB, and LN prepared Table 1 and Fig. 1. JDN and LN were responsible for conceptualization. All authors reviewed the manuscript.

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Declarations

Ethical Approval Not applicable.

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- •• Of major importance

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