Multihost Transmission of Schistosoma mansoni in Senegal, 2015–2018

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In West Africa, Schistosoma spp. are capable of infecting multiple definitive hosts, a lifecycle feature that may complicate schistosomiasis control. We characterized the evolutionary relationships among multiple Schistosoma mansoni isolates collected from snails (intermediate hosts), humans (definitive hosts), and rodents (definitive hosts) in Senegal. On a local scale, diagnosis of S. mansoni infection ranged 3.8%–44.8% in school-aged children, 1.7%–52.6% in Mastomys huberti mice, and 1.8%–7.1% in Biomphalaria pfeifferi snails. Our phylogenetic framework confirmed the presence of multiple S. mansoni lineages that could infect both humans and rodents; divergence times of these lineages varied (0.13–0.02 million years ago). We propose that extensive movement of persons across West Africa might have contributed to the establishment of these various multihost S. mansoni clades. High S. mansoni prevalence in rodents at transmission sites frequented by humans further highlights the implications that alternative hosts could have on future public health interventions.

The collective image of schistosomiasis in Africa remains that of a mainly human-driven disease; schistosomiasis inflicted a burden of >2.5 million disability-adjusted life-years in 2016 and required that ≈200 million persons be treated with preventive chemotherapy in 2017 (1). As pledged by the World Health Organization (2), the goal to eliminate schistosomiasis as a public health problem by 2030 can only be achieved through transdisciplinary programs that improve sanitation and hygiene and provide access to safe water sources, health education, and chemotherapeutic treatments for at-risk populations. Furthermore, answers on the host specificity of human schistosomes and the impact of multihost transmission on disease control strategies remain imperative (3). In Asia, vertebrate reservoirs for Schistosoma japonicum (largely ruminants, rodents, and other mammals) play a crucial role in perpetuating the transmission of this zoonotic parasite, even under strong multisectoral control pressures (4,5). Likewise, in the Caribbean and South America, where evidence supports the introduction of Schistosoma mansoni from West Africa via the transatlantic slave trade (6), rodent populations have become the main reservoirs of S. mansoni; transmission in this region can be maintained in absence of human activity (7,8).

The magnitude of Schistosoma zoonotic transmission, in which both domestic animals and wildlife are active participants, is yet to be determined in endemic countries across Africa. Sporadic investigations have attempted to answer whether schistosomes infecting humans are zoonotic and which, if any, other vertebrate species might be acting as definitive hosts (9-11). The emergence (or discovery) of hybridization events involving S. mansoni, Schistosoma haematobium, and other Schistosoma spp. in livestock and wildlife has raised the profile of these definitive hosts and the schistosomes they harbor (12,13). The interspecific interactions between Schistosoma spp. and the potential involvement of domestic and wild vertebrates in the transmission dynamics of these species might partially be a consequence of anthropogenic changes, loss

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of ecologic barriers, and movement of communities between endemic areas (12).

In 1986, the Diama Dam became operational and transformed the Senegal River Basin. The rice and sugarcane industries benefitted extensively from this change in land use, and the guaranteed freshwater supply favored the expansion of subsistence farming and livestock husbandry. In addition, communities attracted by employment opportunities migrated to the region, in particular to the town of Richard Toll and villages nearby the lake Lac de Guiers in northern Senegal (14,15). However, these anthropogenic changes in the area rapidly led to the first outbreaks of schistosomiasis in the early 1990s (16). As of April 2020, both intestinal schistosomiasis (caused by S. mansoni) and urogenital schistosomiasis (caused by S. haematobium and schistosome hybrids) remain endemic, with co-infections commonly observed across the Senegal River Basin (17). Records show a prevalence of 32%–40% for S. mansoni and 77%–81% for S. haematobium and schistosome hybrids in school-aged children and adults inhabiting towns surrounding Lac de Guiers and along the Senegal River (18,19). In this scenario, the role of animal hosts in the epidemiology of schistosomiasis is unclear. Wild rodents and humans seem to share the same Schistosoma species and hybrids at transmission foci (20,21). However, whether these schistosomes are truly multihost parasites or, in contrast, they have followed diverging evolutionary pathways indicative of definitive host specialization remains to be determined. Focusing on the regions of Richard Toll and Lac de Guiers, our objectives were to examine the evolutionary relationships and host use among Schistosoma isolates and the potential for rodent-to-human spillover.

Materials and Methods

Small Mammal Trapping
During October–December 2017, we captured small mammals at 21 sites that represented Schistosoma spp. transmission foci frequented by humans and their livestock because they are access points to fresh water (Appendix Figure 1, https://wwwnc.cdc.gov/EID/article/26/6/20-0107-App1.pdf). These study sites were situated within or adjacent to villages on the shores of Lac de Guiers and were considered independent from each other for trapping purposes; the shortest distance between adjacent sites was ≈500 m, greater than the maximum home range of endemic species (22). We baited locally made wire-mesh live traps (26 × 10 × 10 cm) with peanut butter and placed them in lines of 14–22 traps at intervals of 5 m adjacent to bodies of water in riparian habitats where reeds (Phragmites sp. and Typha sp.) were the dominant vegetation. We set traps each evening before dusk and inspected them the following morning after dawn for 2 consecutive nights per study site. We calculated the relative abundance of trapped species (no. animals captured/no. active traps) per night for each trap site (23).

We euthanized small trapped mammals with an intraperitoneal injection of sodium thiopental (300 mg/kg body weight) and confirmed their deaths by cervical dislocation and the absence of pedal reflex. We recorded each animal’s species (based on morphologic identification), sex, age class, and anatomic measurements at postmortem examination (Appendix); dissected their thoracic and abdominal organs separately; and visually inspected these organs for helminths. We separated Schistosoma pairs, preserved them in separate vials containing 95% ethanol, and stored them at −20°C. We macerated dissected livers and large intestines of Schistosoma-positive hosts through 300 µm metal sieves using bottled spring water to hatch miracidia and then collected the free-swimming miracidia onto Whatman Indicating FTA Classic Cards (GE Healthcare Life Sciences, https://www.gelifesciences.com) for DNA storage and molecular analysis (24,25). We archived Schistosoma miracidia and adult worms in the Schistosomiasis Collection at the Natural History Museum (26).

Human and Snail Surveys
During October 2017–January 2018, as part of a large-scale program on the transmission dynamics of Schistosoma spp. across Senegal, we conducted a survey for parasites among randomly selected school-aged children (5–17 years of age, n = 290) and self-selected adults (18–78 years of age, n = 40) in the region of Richard Toll and Lac de Guiers. Each person provided 1 fecal sample; we diagnosed Schistosoma infections when eggs were observed in duplicate Kato-Katz thick smears (27). We processed each Schistosoma-positive fecal sample (30 g or the whole sample if <30 g) separately using the miracidial hatching technique (25) and pipetted the free-swimming miracidia onto Whatman Indicating FTA Classic Cards for DNA storage and molecular analysis (24). We archived Schistosoma miracidia in the Schistosomiasis Collection at the Natural History Museum (26).

During November 2015–April 2018, we sampled open freshwater sources within and nearby villages where we conducted surveys with human volunteers to identify snails acting as intermediate hosts of Schistosoma parasites. Throughout 5 surveys, we applied...
standardized protocols in malacology, determined species of collected snails, and identified cercarial shedding to diagnose infections (28). We pipetted free-swimming Schistosoma cercariae, which we identified using a morphologic key (29), onto Whatman Indicating FTA Classic Cards for DNA storage and molecular analysis (24). We archived Schistosoma cercariae in the Schistosomiasis Collection at the Natural History Museum (26).

**Molecular Analyses**

We extracted DNA of individual adult schistosomes using the DNeasy Blood and Tissue Kit (QIAGEN, https://www.qiagen.com) following the manufacturer’s instructions and extracted the DNA of miracidia and cercariae stored on Whatman Indicating FTA Classic Cards as previously described (30). We analyzed the following genomic regions because they are highly informative for phylogenetic identification and classification (31): the internal transcribed spacers (ITS) of the nuclear rDNA, the mitochondrial 12S rRNA gene, cytochrome c oxidase subunit 1 (cox1) and subunit 3 (cox3) genes of the mitochondrial DNA (mtDNA), and NADH dehydrogenase subunit 4 (nad4) and subunit 3 (nad3) genes of the mtDNA. We amplified these regions using 25-µL reactions containing 2.5 µL of 10× buffer, 200 µM of dNTPs, 0.5 µM of each primer, 0.2 units of KOD XL DNA Polymerase (EMD Millipore Corporation, https://www.emdmillipore.com), and 2 µL of DNA template (Appendix Tables 1 and 2). We purified and sequenced PCR products using Eurofins Genomics (https://www.eurofinsgenomics.com) and then edited and assembled contigs using CodonCode Aligner 8.0.1 (https://www.codoncode.com/index.htm). We aligned the noncoding ITS and 12S regions using MAFFT v7 (32) with automated selection of parameters and aligned the protein-coding mtDNA genes (i.e., cox1, cox3, nad4, and nad3) with respect to their amino acid translations using MACSE (33) as implemented in CodonCode Aligner 8.0.1. Molecular sequences from the S. mansoni samples are deposited in GenBank (accession nos. MN593375–434).

**Phylogenetic Approach**

We concatenated the 12S rRNA gene and the 4 protein-coding mtDNA genes of each S. mansoni specimen (i.e., adult worms and miracidia from rodents, miracidia from humans, and cercariae from snails), as well as those from S. mansoni specimens previously collected from Hubert’s multimammate mice (Mastomys huberti) and Nile grass rats (Arvicanthis niloticus) in Senegal (21). In addition, we also obtained and concatenated the respective sequences from publicly available genomes of S. mansoni previously isolated from school-aged children in Uganda (34) and Schistosoma rodhaini from an undetermined intermediate host in Burundi (6). In brief, we downloaded an S. mansoni reference genome (GenBank accession no. SAMEA2272516) from WormBase ParaSite (35) and aligned the 5 specified mitochondrial genes with those of S. mansoni from Uganda and S. rodhaini from Burundi using BWA-MEM version 0.7.17 (Li H, unpub. data, https://arxiv.org/abs/1303.3997v2). For each sample, we used the Genome Analysis Toolkit (36) tools HaplotypeCaller version 3.6.0 to perform variant calling and FastaAlternateReferenceMaker version 3.6.1.0 to replace reference bases with single-nucleotide polymorphisms at variation sites.

We implemented maximum-likelihood analyses in RAxML version 8.2 (37) and Bayesian inference analyses in MrBayes 3.2.6 (38). Across 4 partitions (noncoding positions and protein-encoding first, second, and third codon positions), we selected the generalized time-reversible substitution model with rate heterogeneity for both maximum-likelihood and Bayesian inference. Bootstrap resampling was automatically arrested within the maximum-likelihood analysis. We performed Bayesian inference analysis using 2 independent Markov chain Monte Carlo runs including 4 chains and 10 million generations, sampling every 10,000 generations, and discarding the first 25% of trees as burn-in (Appendix).

We analyzed the temporal structure of the data by using Bayesian inference analysis and specifying independent Hasegawa-Kishino-Yano substitution models with rate heterogeneity across the 4 partitions, a coalescent constant population tree prior with default settings, and a strict clock model in BEAST 2.5.1 (39). We based divergence dating on previous estimates of mutation rates (8.1 × 10⁻⁷ substitutions/site/year) per generation time (0.2 years) that were determined by using whole-genome S. mansoni sequences (6). We inferred the resulting uniform clock rate prior of 4.05 × 10⁻⁷ substitutions/site/year. We computed 2 independent Markov chain Monte Carlo runs including 10 million generations, sampling every 1,000 generations, and discarding the first 10% of trees as burn-in. We inspected convergence and effective sample size values ≥200 using Tracer version 1.7.1 (https://beast.community/tracer) and generated the maximum clade credibility tree using TreeAnnotator version 2.5.1 (https://beast.community/treeannotator). We tested the association between phylogenetic clustering and geographic structure of S. mansoni isolates in BaTS (40) by implementing 1,000
null replicates, 5 discrete states, and an initial burn-in period of 10% (Appendix).

Ethical Considerations
We obtained informed written consent from all human participants or their legal guardians. All infected persons were treated with praziquantel 40 mg/kg either at school or at home. After explicit consent from local authorities and land owners, we targeted our small mammal trapping activities on animal populations classified as least concern by the International Union for the Conservation of Nature Red List. We recorded the trapping of nontarget animals (i.e., unidentified birds and anuran amphibians) and immediately released them at their point of capture. The examined animals were treated in accordance with published guidelines on animal welfare and the use of wildlife in research (41). All investigations were approved by the Comité National d’Ethique pour la Recherche en Santé de Senegal (reference no. SEN15/68), the Imperial College Research Ethics Committee of the United Kingdom (reference no. 03.36), and the Clinical Research Ethical Review Board of the Royal Veterinary College of the United Kingdom (reference nos. 2015-1322 and 2016-1505).

Results
A total of 1,618 traps were set over the course of 27 consecutive nights, and 195 M. huberti mice, 42 A. niloticus rats, and 14 Crocidura shrews were trapped and examined (Appendix Figure 2). We detected Schistosoma trematodes in 16 (8.2%) M. huberti mice (Appendix Table 3), specifically in the mesenteric vessels (in 81.2% of infected mice) and the portal system (in 68.7% of infected mice). On a local scale, 1.7%–52.6% of M. huberti mice were infected with S. mansoni (Table 1). In contrast, we did not observe Schistosoma infections in A. niloticus rats and Crocidura shrews or at the dissection of the urogenital systems of any animals trapped during this study (Appendix Table 3). Miracidial hatching was successful for 8 of 16 infected M. huberti mice. No association was found between Schistosoma infection prevalence and M. huberti mice sex or age (Appendix). All adult schistosomes and miracidia from infected rodents were identified as S. mansoni on the basis of molecular analyses.

A total of 290 school-aged children were examined by duplicate Kato-Katz thick smears, and 37 (12.8%) had S. mansoni infections. We performed miracidial hatching on a randomly selected subset of Schistosoma-positive fecal samples and collected miracidia from samples from 4 infected persons. Molecular analysis confirmed the identification of S. mansoni. In contrast, none of the 40 adults examined by duplicate Kato-Katz thick smears had S. mansoni infections. On a local scale, 3.8%–44.8% of school-aged children were infected with S. mansoni. Infections were identified by using molecular tools. On a local scale, 1.8%–7.1% of B. pfeifferi snails were infected with S. mansoni (Table 1).

The dataset including ITS, 12S rRNA, and the protein-coding mtDNA sequences (i.e., cox1, cox3, nad4, and nad3) of S. mansoni from school-aged children, rodents, and B. pfeifferi snails (Table 2) showed no intraspecific variability within the ITS alignment (914 bp), whereas variability was present within the 12S (760 bp, polymorphism ≤0.52%) and concatenated mtDNA (2,874 bp, polymorphism ≤0.77%) gene alignments. Intraspecific single-nucleotide

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Table 1. *Schistosoma mansoni* infection rate and intensity by host and study site, Senegal, 2015–2018*

<table>
<thead>
<tr>
<th>Study site</th>
<th><em>Mastomys huberti</em> mice</th>
<th><em>Arviccanthis niloticus</em> rats</th>
<th>School-aged children</th>
<th><em>Biomphalaria pfeifferi</em>, snails, no. infected/total no. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. infected/total no. (%)</td>
<td>Median (range) infection intensity</td>
<td>No. infected/total no. (%)</td>
<td>Median (range) infection intensity</td>
</tr>
<tr>
<td>Didjiery†</td>
<td>0/12</td>
<td>NA</td>
<td>0/69</td>
<td>NA</td>
</tr>
<tr>
<td>Ganket</td>
<td>2/4 (50.0)</td>
<td>18.5 (5–32)</td>
<td>0/4</td>
<td>NA</td>
</tr>
<tr>
<td>Gueo</td>
<td>10/19 (52.6)</td>
<td>14 (2–64)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Keur Momar</td>
<td>1/19 (5.3)</td>
<td>2</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Sarr</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mbane†</td>
<td>0/60</td>
<td>NA</td>
<td>0/34</td>
<td>NA</td>
</tr>
<tr>
<td>Merina Guwel</td>
<td>1/12 (8.3)</td>
<td>2</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Nde†</td>
<td>1/60 (1.7)</td>
<td>2</td>
<td>0/11</td>
<td>NA</td>
</tr>
<tr>
<td>Ndombo</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Richard Toll†</td>
<td>0/10</td>
<td>NA</td>
<td>1/73 (1.4)</td>
<td>4</td>
</tr>
<tr>
<td>Temeye†</td>
<td>8/43 (18.6)</td>
<td>4 (2–35)</td>
<td>0/4</td>
<td>NA</td>
</tr>
<tr>
<td>Thiag†</td>
<td>0/4</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

*Only study sites where infected hosts were detected are included. Infection intensities were calculated by using eggs per gram of fecal samples for school-aged children and number of adult worms in rodents (M. huberti and A. niloticus). Infection intensity was not quantified for *B. pfeifferi* snails (intermediate host). NA, not applicable.

†For rodents, values include data previously reported (21).
polymorphisms within protein-coding mtDNA genes represented nonsynonymous amino acid substitutions in 4.4% (35/796) of codons, and saturation at model-corrected genetic distances was not detected (Appendix Figure 3). Maximum-likelihood and Bayesian inference analyses of the concatenated 12S and mtDNA gene sequences yielded consensus trees with congruent topologies, including different multihost S. mansoni lineages (Appendix Figure 4). The presence of multiple, well-supported S. mansoni clades within Senegal, 4 of which included samples collected from both humans and rodents, was confirmed by a phylogenetic analysis constructed by using a strict molecular clock (Figure, panel A; Appendix Figure 5). Different S. mansoni lineages prevalent in the Senegal River Basin diverged between 0.13 (95% highest posterior density interval [HPDI] 0.11-0.16) and 0.02 (95% HPDI 0.01-0.03) million years ago (MYA). Using uniform clock rate prior (4.05 × 10⁻⁸ substitutions/site/year), we determined that divergence between the sampled S. mansoni parasites from Uganda and Senegal occurred ≈0.19 (95% HPDI 0.15-0.23) MYA, whereas the speciation of S. rodhaini may have occurred ≈0.14 (95% HPDI 0.95-1.35) MYA. The association index, parsimony score, and monophyletic clade metrics of S. mansoni within Senegal were not significant in BaTS (p>0.05). These findings strongly support the null hypothesis of random phylogenetic trait associations and, therefore, that S. mansoni clades are not associated with the geographic structure on a local scale (Figure, panel B).

**Discussion**

In this study, we provide direct evidence of the zoonotic nature of S. mansoni in West Africa, revealing a potential ecologic cause for human reinfection after chemotherapeutic treatment. Our phylogenetic approach demonstrated that S. mansoni lineages responsible for intestinal schistosomiasis in humans also exploit rodent populations as reservoirs at transmission sites frequented by humans; prevalence could be as high as 52.6% in M. huberti mice at these sites. Therefore, we exclude the presence of an independent sylvatic life cycle and host specialization for S. mansoni in the Senegal River Basin. The phylogenetic similarity between parasite isolates collected from humans, rodents, and freshwater snails indicates that host use has not played a prominent role in the evolutionary pathway of S. mansoni in this region. Similar results were obtained during the analysis of specimens from different regions and hosts within the geographic distribution of S. mansoni, suggesting that murine isolates did not constitute monophyletic assemblages (42).

This lack of a geographic structure for S. mansoni on the local scale might be caused by disease foci of recent origin or the rapid dissemination of S. mansoni across the Senegal River Basin, which probably
occurred as a result of the land-use changes associated with the Diama Dam construction and transport infrastructure development (14–16). Furthermore, 3 decades of endemicity and the extensive movement of communities from within Senegal and other countries of West Africa could have substantially contributed to *S. mansoni* lineage diversification and gene flow in the Lac de Guiers region (14,43). The different *S. mansoni* clades detected herein might have diverged between $0.13 \pm 0.03$ MYA and $0.02 \pm 0.01$ MYA, firmly corroborating the hypothesis of their ramification from a common precursor during ancestral times. Multiple introduction events of various parasite populations could indicate that *M. huberti* mice and other rodent populations inhabiting periaquatic ecosystems act as competent alternative hosts for *S. mansoni* in many endemic areas across sub-Saharan Africa. The mainly nocturnal activity of *M. huberti* mice (vs. diurnal activity of *A. niloticus* rats) (22) may support the presence of different *S. mansoni* chronotypes characterized by differing circadian rhythms of cercarial emergence (44). Therefore, the risk for infection among local communities might not be limited to just the warmest hours of the day (diurnal transmission) but also extend to the early morning and late afternoon (crepuscular transmission). The high excretion rates of *S. mansoni* eggs by *M. huberti* mice during experimental infections (median intensity 720 eggs/g fecal sample) (45) and field observations (median intensity 262 eggs/g fecal sample) (46) are a warning about the potential contamination of freshwater bodies by parasitized rodents.

In our study, fully resolved spatial and temporal dynamics could not be determined. Future incorporation of *S. mansoni* sequences from multiple endemic regions across West Africa and Africa as a whole might help decipher the origin and radiation pattern of the various lineages observed in the Richard Toll and Lac de Guiers areas. Furthermore, the temporal estimates of *S. mansoni* evolution displayed herein should be interpreted with caution. The molecular clock calibration relied on previous estimates of the mutation rate and generation time calculated by using whole-genome *S. mansoni* data across its known geographic distribution (6). However, our reconstruction of the divergence between *S. rodhaini* and *S. mansoni* (1.14 $\pm$ 0.20 MYA) differs from previous dating (0.13 $\pm$ 0.02 MYA and 2.80 $\pm$ 0.19 MYA) (6,42). This conundrum highlights that further evidence is needed to characterize the evolutionary history within the genus *Schistosoma*. The application of a single calibration method in divergence dating remains subject to time-dependent bias.
if not integrated by ancestral DNA, fossil records, or biogeographic events (47).

The zoonotic *S. japonicum* in Asia illustrates the pivotal role that animal reservoirs and multihost dynamics have as drivers of pathogen transmission and human reinfection, even after decades of multifaceted interventions (4,5). With the presence of multiple multihost *S. mansoni* lineages characterized by different divergence times circulating across the Senegal River Basin, our results support a similar scenario for *S. mansoni* in sub-Saharan Africa. Therefore, the parasite should be acknowledged as zoonotic, and public health campaigns must be planned considering the availability of alternative hosts (including wildlife, although *S. mansoni* prevalence in wildlife reservoirs can markedly vary) when transmission is maintained despite repeated interventions. The implementation of coprologic and DNA-based diagnostics within nonlethal sampling schemes can directly facilitate targeted surveillance where rodents might be contributing to the transmission of *S. mansoni*, other *Schistosoma* spp., and hybrids. However, the results of our study and previous surveys in endemic settings of Senegal (21) and Corsica, France (48), support the role of rodents as accidental (rather than maintenance) hosts of the *Schistosoma* hybrids responsible for urogenital schistosomiasis. Furthermore, although evidence suggests that rodents could be competent hosts of *Schistosoma bovis* (typically a schistosome of ruminants) across sub-Saharan Africa (11,21), we did not isolate any during this survey.

In conclusion, the multihost transmission dynamics of *S. mansoni* promote the recruitment of various definitive hosts spatially and temporally overlapping at transmission sites in the region of Lac de Guiers. In sub-Saharan Africa, the role of nonhuman vertebrates in the epidemiology of *Schistosoma* species and hybrids has yet to be fully determined, considering these could be spillover hosts incapable of maintaining transmission by themselves. However, our study supports that rodents have the potential to act as true reservoirs of *S. mansoni* and influence the evolution of this parasite (i.e., by providing opportunities for host switching and genetic exchange), which could thwart attempts to control or interrupt transmission of *S. mansoni* in human populations (3,12). Nevertheless, the presence of zoonotic pathogens in their animal reservoirs should not be considered synonymous with human disease risk, but rather a measure of underlying transmission potential, which is itself mediated by many additional intersecting ecologic and social drivers (19,49). The extent to which rodents contribute to the zoonotic transmission of *S. mansoni* and *Schistosoma* hybrids remains a question to be further developed by epidemiologic surveys, mathematical modelling, and genomics. As we move our efforts from disease control toward interruption of *S. mansoni* transmission and local elimination, the implementation of alternative hosts in disease dynamics will be crucial and threaten to undermine future chemotherapeutic-focused interventions on local scales. Cross-disciplinary initiatives between the natural resource and public health sectors, including the long-term establishment of regional expertise, can be used to guide preventive measures not only for schistosomiasis but also for other rodentborne zoonoses across Africa and beyond.

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Dr. Catalano completed his doctoral studies in the Department of Pathobiology and Population Sciences, Royal Veterinary College, London, United Kingdom, during the publication of this research. His research interests focus on disease dynamics at the human–wildlife interface and biodiversity conservation initiatives.

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