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Avian Schistosomes and Outbreaks of Cercarial Dermatitis

Petr Horák,[a] Libor Mikeš,[a] Lucie Lichtenbergová,[a] Vladimír Skála,[a] Miroslava Soldánová,[b] Sara Vanessa Brant[c]

Department of Parasitology, Faculty of Science, Charles University in Prague, Prague, Czech Republic[a]; Institute of Parasitology, Biology Centre of the Academy of Sciences of the Czech Republic, České Budějovice, Czech Republic[b]; Museum Southwestern Biology, Department of Biology, University of New Mexico, Albuquerque, New Mexico, USA[c]

SUMMARY

Cercarial dermatitis (swimmer's itch) is a condition caused by infective larvae (cercariae) of a species-rich group of mammalian and avian schistosomes. Over the last decade, it has been reported in areas that previously had few or no cases of dermatitis and is thus considered an emerging disease. It is obvious that avian schistosomes are responsible for the majority of reported dermatitis outbreaks around the world, and thus they are the primary focus of this review. Although they infect humans, they do not mature and usually die in the skin. Experimental infections of avian schistosomes in mice show that in previously exposed hosts, there is a strong skin immune reaction that kills the schistosome. However, penetration of larvae into naive mice can result in temporary migration from the skin. This is of particular interest because the worms are able to migrate to different organs, for example, the lungs in the case of visceral schistosomes and the central nervous system in the case of nasal schistosomes. The risk of such migration and accompanying disorders needs to be clarified for humans and animals of interest (e.g., dogs). Herein we compiled the most comprehensive review of the diversity, immunology, and epidemiology of avian schistosomes causing cercarial dermatitis.

INTRODUCTION

Cercarial dermatitis is a condition caused by both mammalian and avian schistosomes (Trematoda: Schistosomatidae). Which of those species is more prevalent in a dermatitis outbreak depends on where you are in the world and how humans and birds/mammals (and, by association, snails) come into contact with a particular type of aquatic environment. The name "cercarial dermatitis" is derived from the term "cercaria," the last larval stage developing in an aquatic snail. Cercaria is the infective stage that, after leaving the snail, searches for and invades a warm-blooded vertebrate host via skin penetration. Besides the official name, "cercarial dermatitis," many local terms are used ("sawah itch," "koganbyo," etc.), with the most widely used name being "swimmer's itch."

Schistosome cercariae were disclosed as the causative agent of cercarial dermatitis in the United States in 1928 (1). Since that time, numerous reports of cercarial dermatitis have been documented from different parts of the world. Global economic losses due to outbreaks of cercarial dermatitis are not known, as there is no systematic method of reporting either the number of cases or incurred economic losses in terms of recreation or person work hours. Furthermore, what data do exist that estimate local costs are usually not available to the public domain, but it is accepted that outbreaks can have considerable impacts on local, tourism-based economies in the areas of recreational lakes (2). For example, in the recreational area of Naroch Lake (Belarus), 4,737 cases of cercarial dermatitis were recorded between 1995 and 2006 (3). In addition, cercarial dermatitis may represent a debilitating oc-

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Address correspondence to Petr Horák, petrhorak@petrhorak.eu.

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cupational disease among rice farmers (4) and may incur costs in terms of lost person work hours. Older reports refer to 75% or more of the population experiencing the characteristic symptoms of "koganbyo" in the areas of Japan where the disease is most highly endemic (5). Recent reviews (6–9) agree that in some regions cercarial dermatitis has appeared as a new problem, either because the dermatitis was previously unknown (e.g., the U.S. Southwest and Chile) or because the number of reports of outbreaks increased (8, 10, 11). Consequently, cercarial dermatitis is now regarded as an emerging disease. Besides human schistosomes (*Schistosoma* spp.), no animal (e.g., avian) schistosomes have any other presently known pathogenic effects on humans. Thus, the use of animal models to study the potential risk of animal (avian) schistosomes to human health is invaluable.

The last decade has revealed diverse avian schistosome species and biology, as well as the snails that host them. These discoveries have outpaced the equally essential host-parasite biological, immunological, pathological, and epidemiological studies of species diversity in terms of incorporating the results of such studies into the current known diversity of schistosomes. Such studies are difficult and time-consuming, and consequently, only a few species have been adapted to experimental conditions. Nevertheless, such studies are crucial to understanding the current and future roles that these species might play in the frequency and distribution of cercarial dermatitis, as well as understanding how to break the life cycle to prevent outbreaks. What has been documented, and is detailed in the following sections, points to an understanding of the avian schistosome-host relationships and thus offers the foundation on which future studies will be modeled.

DIVERSITY OF SCHISTOSOMES CAUSING DERMATITIS

Considering only the named species in the literature, there are 4 schistosome genera from mammals and 10 from birds, with about 30 described species from mammals and about 67 from birds (12). The total is close to 100 species, with ~70% of them being avian schistosomes distributed around the world that may initiate cercarial dermatitis. The role of some of the species of avian schistosomes as dermatitis agents has not been studied sufficiently, as they are not often found in areas where people most commonly are in contact with water and snails. To discuss the distribution and diversity of schistosomes causing dermatitis within the phylogenetic framework of the family Schistosomatidae, we refer to Fig. 1 (12–14).

The basal clade of the family tree (Fig. 1, clade A) comprises the exclusively marine avian schistosomes *Austrobilharzia* (4 species) and *Ornithobilharzia* (2 species); the species shown in the tree are those for which there are genetic data. Species of these two genera are associated with outbreaks of dermatitis in shallow marine environments (15, 16). Infection often occurs in people who are swimming, playing in tidal pools, or working, for example, collecting tidal invertebrates in the sand (15, 17–27). Both of these genera have robust, large worms as adults and are common schistosomes of marine birds, particularly gulls. Species of *Austrobilharzia* are more often implicated as a cause of dermatitis outbreaks (25).

The next main clade includes the remaining schistosomes (Fig. 1, clades B, C, and D). Clades B and C are exclusively freshwater mammalian schistosomes. The largest clade of mammalian schistosomes includes the genus *Schistosoma*, with ~25 species (clade B). In particular, three of these species (*Schistosoma mansoni*, *Schistosoma haematobium*, and *Schistosoma japonicum*) cause one of the most devastating helminth diseases in humans, schistosomiasis, affecting about 220 million people, mainly in the tropical and subtropical latitudes around the world (WHO). All but one species (*S. mansoni*) occur exclusively in the Eastern Hemisphere. These species are not typically implicated in dermatitis outbreaks, yet there is a mild eruption of dermatitis following penetration by all schistosomes (28). Most reported cases of dermatitis caused by the genus *Schistosoma* are from parasites that infect domesticated work animals, such as cattle and buffalo, mainly in Asia. For example, in countries such as India and Nepal, the species *Schistosoma turkestanicum*, *Schistosoma nasale*, *Schistosoma indicum*, and *Schistosoma spindale* are often implicated in outbreaks of dermatitis (29–38). This relationship may not be a surprise, as bovids are the definitive host, and the people in these areas depend upon these animals for their livelihood in farming. Additionally, the snail host for the major species causing dermatitis (*S. nasale*, *S. indicum*, and *S. spindale*) is *Indoplanorbis exustus*, a widespread and abundant snail that is found mainly in Nepal and India, to the exclusion of *Biomphalaria* and *Bulinus*, snail hosts for a majority of the African transmitted species of *Schistosoma*.

The genus *Bivitellobilharzia* is considered a schistosome of elephants, but it has also been reported from wild rhinoceroses in Nepal (38–41). There are no known reports of cercarial dermatitis in humans from areas inhabited by African elephants (with the *Bivitellobilharzia loxodontae* schistosome), but in areas where domesticated Asian elephants are used, there have been cases of dermatitis in the mahouts, or elephant handlers, when the elephants are taken for bathing (e.g., in Sri Lanka [40]). In Nepal, *Bivitellobilharzia nairi* has thus far been found in wild, not domesticated, elephants (38). The snail host remains unknown but is likely a pulmonate snail (42). At least two species of *Schistosoma* from *Biomphalaria* snails infect the African hippopotamus, but these species have not been implicated directly in dermatitis outbreaks, despite the presence of humans working on lakeshores where there are hippopotamuses (43–45). Given the prevalence of human schistosomiasis in these areas, however, dermatitis caused by hippopotamus schistosomes may easily go undetected.

The small clade C (Fig. 1) has two species of mammalian schistosomes that, as far as we know, are found only in North America and are not frequently associated with dermatitis outbreaks, though they both produce a skin reaction (46–50). These two species are parasites of lymnaeid snails (often *Stagnicola elodes*), usually with raccoons and muskrats as mammalian hosts. *Schistosomatium douthitti* adults inhabit aquatic and semiaquatic rodents in more northern latitudes or at high elevations (48, 51, 52). *Heterobilharzia americana* has been reported from a wide range of mammalian hosts (rivaling *Schistosoma japonicum*), including horses, in the southern regions of North America (49, 53, 54).

Perhaps the most remarkable clade of schistosomes responsible for dermatitis is clade D, a large clade of avian schistosomes whose adults are long and threadlike (except *Dendritobilharzia* and *Bilharziella*) and that includes both freshwater and marine species. In particular, the genus *Trichobilharzia* has achieved notoriety as the primary etiological agent for dermatitis outbreaks around the world. The diversity of aquatic environments, host use, morphology, definitive host habitat, and cercarial behavior is unparalleled in any other group of schistosomes, and probably most other groups of trematodes (12, 14). Figure 1 includes a molecular phylogeny of all the known genera of schistosomes ex-

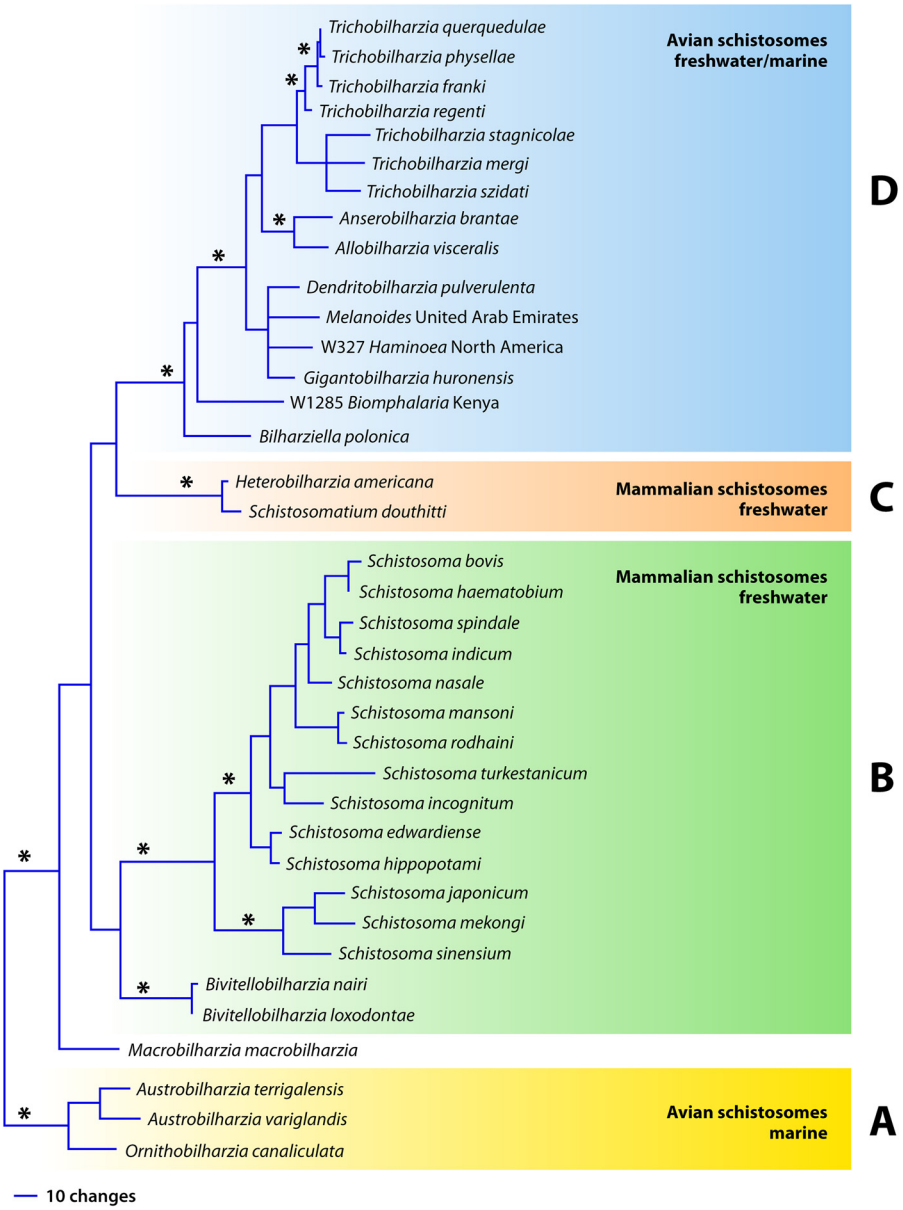

FIG 1 Phylogenetic tree showing generic and species positions based on Bayesian analysis of the nuclear ribosomal DNA 28S region (1,200 bp) of Schistosomatidae. Panels A to D refer to the clades discussed in the text. This tree is based on genetic data, not morphological data, and as such, there are more species that have been described morphologically than genetically. Asterisks denote significant posterior probabilities (>0.95).

cept one. *Jilinobilharzia* has not been reported since the original paper reporting it from the duck *Anas crecca* in northeastern China; its snail host remains unknown (55). Morphological characteristics and host use suggest that *Jilinobilharzia* belongs in the large clade of avian schistosomes (Fig. 1, clade D), perhaps even to *Trichobilharzia*.

At the base of clade D is an unresolved group of avian schistosomes, most of which have been implicated in dermatitis outbreaks and comprise the most diverse range of both bird and snail (9 families) host use (13, 40, 56–66). Current results based on all of the available sequence data in GenBank for the internal transcribed spacer (ITS) region indicate that there are about nine distinct lineages, only two of which are described: *Gigantobilharzia* and *Dendritobilharzia* (40, 64–69). Most of the lineages in this part of clade D have one to a few species and have been seen in only a few cases, many related to dermatitis outbreaks (12) (Table 1). Thus far, the literature suggests that species in this clade cause dermatitis in more local areas, whereas *Trichobilharzia* causes cercarial dermatitis globally. For example, in the San Francisco Bay area (California), one beach in particular has annual cases of dermatitis (64). The prevalence of dermatitis caused by schistosomes from *Valvata* or *Melanoides* snails (63, 65, 66) depends on how often people use areas where these snails release cercariae.

Species of *Trichobilharzia* (Fig. 1, clade D) are globally distributed and cause the majority of recreational and occupational reports of dermatitis found in the literature, especially in the temperate latitudes. In North America and Europe, where most of the research has been focused, outbreaks occur in recreational ponds

TABLE 1 Summary of general host use of known genera of schistosomes, reflecting current knowledge, habitat in the definitive host, and broad geographic locality

Genus	Snail host	Mammalian/avian host	Definitive host habitat	Locality	Aquatic habitat	Major areas for outbreaks
Austrobilharzia	Nassariidae, Batillaridae, Littoriniidae, Potamididae	Charadriiformes	Visceral	Global	Marine	Shallow marine areas, tidal pools
Ornithobilharzia	Batillaridae	Charadriiformes	Visceral	Global	Marine	Shallow marine areas, tidal pools
Macrobilharzia	Unknown	Suliformes (*Anhinga*)	Visceral	North America, Africa	Unknown	Unknown if causes dermatitis
Bivitellobilharzia	Unknown	Elephantidae, Rhinocerotidae	Visceral	Africa, Asia	Freshwater	Probably freshwater rivers
Schistosoma	Planorbidae, Lymnaeidae, Pomatiopsidae	Mammalia	Visceral, Nasal	Eurasia, Africa, South America	Freshwater	Mostly eutrophic ponds
Heterobilharzia	Lymnaeidae	Mammalia	Visceral	North America	Freshwater	Marshy areas
Schistosomatium	Lymnaeidae	Rodentia	Visceral	North America	Freshwater	Marshy areas
Bilharziella	Planorbidae	Anseriformes, Gruiformes, Ciconiiformes, Podicipediformes	Visceral	Europe	Freshwater	Eutrophic ponds
Species isolated from *Haminoea*	Haminoeidae	Charadriiformes, Pelicaniformes	Visceral	North America	Marine	Shallow marine areas, tidal pools
Gigantobilharzia[a]	Physidae	Passeriformes	Visceral	North America	Freshwater	Marshy areas, usually with cattails
Dendritobilharzia	Planorbidae	Anseriformes, Gruiformes, Pelicaniformes, Gaviiformes	Visceral	Global	Freshwater	Unknown if reports of dermatitis
Jilinobilharzia	Unknown	Anseriformes (Anatidae)	Visceral	China	Unknown	Unknown if reports of dermatitis
Allobilharzia	Unknown	Anseriformes (swans)	Visceral	Northern Hemisphere	Unknown	Unknown if causes dermatitis
Anserobilharzia	Planorbidae	Anseriformes (geese)	Visceral	Northern Hemisphere	Freshwater	Eutrophic ponds, reservoirs
Trichobilharzia	Lymnaeidae, Physidae	Anseriformes (Anatidae)	Visceral, nasal	Global	Freshwater	Eutrophic ponds, glacial lakes, reservoirs

[a] Since *Gigantobilharzia* is not a monophyletic genus, the information listed here is for *G. huronensis* only.

and reservoirs. These outbreaks have been reviewed extensively (7, 8, 70, 71). Species of *Trichobilharzia* have been reported to cause dermatitis from other areas as well, such as Rwanda-Burundi (72), South Africa (73, 74), New Zealand and Australia (75–79), Malaysia/Indonesia (80–82), Iran (65, 83–86), United Arab Emirates (66), Thailand (87), and China (88, 89) in the Eastern Hemisphere and Argentina (57, 90, 91), Chile (11), and El Salvador (92) in the Western Hemisphere. We are just beginning to better understand the significant disease components for dermatitis as a global problem.

Cercarial dermatitis is also recognized as an occupational hazard in many areas of the world, especially in areas where rice is grown (82, 93). Rice fields are areas where snails, domestic and wild ducks, cattle, and humans seasonally use the water, so the life cycle is maintained consistently (5, 84, 94–104). Species of *Trichobilharzia* are identified most often, though not exclusively (5). Rice fields are plowed by water buffalo and cattle in areas where *Indoplanorbis exustus* occurs, and hence, dermatitis may be caused by one of the species of *Schistosoma*, as noted above. Nonetheless, *Trichobilharzia* is still by far the most common etiological agent.

MOLLUSCAN AND AVIAN HOST SPECIFICITY

Schistosomes have colonized many families of snails as first intermediate hosts (12, 62). Mammalian schistosomes use 3 families of snails, compared to 15 families used by avian schistosomes, as summarized in Table 1. The majority of schistosome species are transmitted by the pulmonate snail families Physidae, Lymnaeidae, and Planorbidae (10, 70, 105). Interestingly, two snail families contain species (e.g., *Biomphalaria* and *Indoplanorbis* in the Planorbidae family and *Stagnicola* in the Lymnaeidae family) that can host both avian and mammalian schistosomes that cause dermatitis (40, 51, 70, 104, 106).

Previous papers have reviewed the details of host specificity in mammalian schistosomes (Fig. 1, clades B and C) (107, 108). Because avian schistosomes are the major group of schistosomes causing dermatitis, our own discussion focuses on their avian and snail hosts. For two reasons, these avian schistosome species comprise most of the dermatitis reports: first, many avian hosts seasonally migrate, consequently disseminating avian schistosomes as they fly (domestic ducks can serve as definitive hosts particu-

larly for *Trichobilharzia*); and second, some snail hosts are habitat generalists (e.g., *Physa* [syn. *Physella*] *acuta* and *Lymnaea stagnalis*) that are now globally distributed. As a result, the opportunities for birds and snails to come into contact across time and space are vast. For example, *P. acuta* (host to *Gigantobilharzia huronensis*, *Trichobilharzia physellae*, and *Trichobilharzia querquedulae*) thrives in both natural and altered environments, with a wide tolerance for water temperature and chemistry, including the conditions found in ponds, drainage ditches, rivers, marshes, and ephemeral water (109–111). In Europe, the lymnaeid snail *L. stagnalis* (host of *Trichobilharzia szidati*) has also been linked to cases of dermatitis in people who acquired it while working with aquaria (112, 113), providing evidence of the snail's ability to persist, in addition to its local global presence.

From an evolutionary perspective, the vagility and habitat specificity of most bird hosts, in concert with the availability of snails in aquatic habitats, are likely mechanisms for widespread host switching in snails, and thus for diversification of avian schistosomes (12, 114). Our knowledge of the current schistosome-snail associations indicates that once a schistosome is hosted by a particular species of snail, it possesses little ability to utilize more than a few species within that genus (e.g., *Radix*, *Stagnicola*, and *Physa*) (115). Two exceptions are *Dendritobilharzia pulverulenta*, which uses *Anisus vortex* in Europe (116) and *Gyraulus parvus* in North America (117), both of which are small, related planorbid snails, and *Trichobilharzia regenti*, which employs lymnaeid snails of the genus *Radix* in Europe and *Austropeplea tomentosa* in New Zealand (118). There is little evidence of schistosome species crossing snail families, naturally or experimentally (for an exception, see references 119 and 120). *Trichobilharzia franki* was reported to be widespread across Europe, but detailed molecular studies are showing that it may represent several species related to snail host use (70, 121, 122).

Cercarial dermatitis is caused not only by species of schistosomes from indigenous snails but also by those from invasive or introduced snails. For example, *Haminoea japonica* (originally from Japan but now off the California coast) and *Ilyanassa obsoleta* (originally from the east coast but now on the west coast of North America) are responsible for annual dermatitis outbreaks at marine swimming beaches (25, 64). In freshwater, *L. stagnalis* is found commonly in the northern Eastern hemisphere, yet in the northern Western hemisphere it is only locally common. When *L. stagnalis* is found to be infected, the infecting species is related to a common European species, *Trichobilharzia szidati* (70, 123). Interestingly, *P. acuta* is one of the most invasive pulmonate snails and can host at least four species of avian schistosomes in North America (10, 70), yet there are no reports of this snail hosting schistosomes in their invasive range (outside North America). It is also noteworthy that most of the schistosome species transmitted by physid snails have thus far been found only in North America (at least based on genetic comparisons) (e.g., *G. huronensis*, *T. physellae*, and one undescribed lineage of schistosome [10]). The lymnaeid snail genera *Stagnicola* (found in North America) and *Radix* (found in the Eastern Hemisphere) are the main snail hosts for most species of *Trichobilharzia*; in fact, thus far, *Radix* maintains most of the reported species diversity of *Trichobilharzia* (122). *Trichobilharzia regenti* has been recognized to cause dermatitis in Lake Wanaka in New Zealand, and it may have been introduced from Europe in wild duck breeds (*Anas platyrhynchos*) used for hunting. It is now found commonly in the endemic nonmigratory scaup *Aythya novaeseelandiae* and the snail *Austropeplea tomentosa* (118). Schistosomes seem to be specific to particular snail hosts at the species or genus level, but not as much to their avian or mammalian hosts, though loose specificity of definitive host use exists at higher taxonomic levels (Table 1) (e.g., *Schistosoma haematobium* in humans, *Bivitellobilharzia* in elephants, and *Allobilharzia* in swans).

The diversity of avian schistosomes found around the world is in no small part due to the ability of thousands of migratory birds to carry their parasites across several latitudes and longitudes, exposing commonly encountered snails. This propensity to migrate large distances distinguishes avian schistosomes from mammalian schistosomes in terms of distribution, diversification, and host use, perhaps with the exception of *Schistosoma mansoni* (in terms of long-distance migration only) (124). Yet the schistosome species found in a wide range of avian host orders (e.g., *Bilharziella* and *Dendritobilharzia*) (Table 1) are not the ones recurrently responsible for outbreaks and are also species that are not genetically diverse compared to other species (69, 125, 126). Currently, *D. pulverulenta* might be the most widespread single species of avian schistosome, crossing both the Northern and Southern Hemispheres (117, 125, 127, 128).

The most derived clade, or most recently evolved clade, in clade D has three genera: *Allobilharzia*, *Anserobilharzia*, and *Trichobilharzia* (Fig. 1). *Allobilharzia*, from swans, and *Anserobilharzia*, from geese, both have a circumpolar distribution (69, 70, 105, 129, 130). *Anserobilharzia brantae*, which is common in North America (in the Canada goose [*Branta canadensis*] and the snow goose [*Chen caerulescens*]) but also found in Europe (in the greylag goose [*Anser anser*]), has been identified in at least one outbreak of dermatitis in the United States (10). The area was a eutrophic municipal lake/pond with a dense population of *Gyraulus parvus* snails and Canada geese. The third genus, *Trichobilharzia*, the most species-rich genus in the family, is found almost exclusively in ducks. It should be noted that there are several species of *Trichobilharzia* reported from other avian families (*Trichobilharzia corvi* from passeriforms, along with other species [131, 132]), but based on the morphology of adults and eggs and snail host use (when known), these probably represent new genera, or these avian hosts are not the primary (competent) hosts or might be aberrant cases (70, 78). Within the genus *Trichobilharzia*, several clades are specific to certain groups of ducks: for example, *Trichobilharzia stagnicolae* and *Trichobilharzia mergi* are found in mergansers (Merginae) (70, 133), *T. querquedulae* in the "blue-winged duck" clade (*Anas clypeata*, *Anas discors*, and *Anas cyanoptera*) (70, 134), *T. physellae* in an ecological group of diving ducks that includes ducks of the Aythinae and Merginae, and a common, undescribed species of *Trichobilharzia*, species A, in *Anas americana* (70). It is not yet clear which duck groups (phylogenetic or ecological) are more specific for *T. franki* or *T. szidati*. Interestingly, *T. regenti* has probably been reported from the most diverse duck species (135) and does not appear to have a preferred host within the Anatidae. In North America, *T. stagnicolae* and *T. physellae* are most often identified in dermatitis outbreaks (70).

INTRAMOLLUSCAN DEVELOPMENT OF AVIAN SCHISTOSOMES

As noted above, *Trichobilharzia* is the most diverse schistosome genus and has most often been implicated in outbreaks of cercarial dermatitis. As a result, studies on the avian schistosome-snail in-

termediate host relationship have focused primarily on species of the genus *Trichobilharzia*. Additionally, long-term laboratory maintenance of *T. szidati* and *L. stagnalis* enabled the experiments that uncovered the intimate molecular interactions of avian schistosomes and their snail hosts. (The *Trichobilharzia ocellata* organism used as an experimental model in European laboratories for the last few decades is identical to *T. szidati*, and the latter name is preferred and is used here [123]. If *T. ocellata* is used in the text body, then it refers to the non-European isolates of the parasite.)

After hatching from eggs in an aquatic environment, schistosome miracidia search for and invade an appropriate snail host species. This behavior must be accomplished quickly, as miracidia have a temperature-dependent limited life span of around 20 h, as reported for, e.g., *T. stagnicolae* (136). Studies on the miracidial behavior of *T. szidati* have shown a progression of steps from host finding to penetration and migration. Miracidia respond to environmental stimuli, such as light or gravity, that direct them to the microhabitat occupied by the host snails (137). Snails release various chemical compounds that form an "active space" around them and serve as chemoattractants for the miracidia. Miracidia recognize macromolecular glycoconjugates, termed miracidium-attracting glycoproteins (MAGs) or miraxones, that consist of a protein core and carbohydrate chains linked O-glycosidically via *N*-acetyl-D-galactosamine and serine/threonine (138, 139). The attractant for miracidia is encoded in these carbohydrate moieties. Upon entering the "active space" of the snail, miracidia modify their movement by increased random turns within the increasing attractant gradient and by a turn-back form of swimming within the decreasing attractant gradient (140). This mode of orientation (chemokinesis), observed, e.g., in *T. szidati* or *T. franki* (115, 138), results in the first contact of the miracidia with the snail, which is then followed by repeated investigation/probing of the snail surface and, finally, miracidial attachment (140). Penetration of the snail surface follows, although the factors contributing to this process, such as the components of miracidial penetration glands, remain largely unknown.

After penetrating the surface epithelium of the snail, miracidia of avian schistosomes transform into mother sporocysts that give rise to daughter sporocysts, which migrate to the snail hepatopancreas, where production of the final larval stage, the cercariae, takes place (13). The prepatent period lasts about 3 to 10 weeks, depending on several factors, such as the miracidial dose or temperature (13, 141). Infection by avian schistosomes may lead to alterations of the snail internal defense system (IDS), metabolism, and endocrine functions. Such alterations have been studied widely in *L. stagnalis*, the intermediate host of *T. szidati* (142–145).

The snail IDS is based solely on innate immune mechanisms composed of humoral and cellular limbs. Lectins are essential humoral components, whereas hemocytes represent the main effector cells (146, 147). Both limbs of the *L. stagnalis* IDS appear to be activated and then suppressed during early and late stages, respectively, of a *T. szidati* infection (143). *In vitro*, hemocytes failed to encapsulate and destroy *T. szidati* sporocysts (148). It has been suggested that parasite excretory-secretory products participate in the modulation of hemocyte activities (149). Disruption of hemocyte signaling pathways, such as protein kinase C (PKC) and extracellular signal-regulated kinase (ERK) pathways, may also influence hemocyte activities (150–152). For example, carbohydrates known to be present on larval surfaces of *T. szidati* and *T. regenti*, such as D-galactose and L-fucose (153–156), affected hemocyte PKC and ERK signaling in *L. stagnalis*, which suggests an immunosuppressive role (157, 158). However, experiments investigating the direct effect of *Trichobilharzia* larvae on hemocyte signaling have not been performed. Alterations of humoral defense components also occur during infections by avian schistosomes, and at least two molecules, molluscan defense molecule (MDM) and granularin, have been investigated in this respect (159, 160). Both molecules, produced in *L. stagnalis* by granular cells of connective tissue, are related to phagocytic activity of hemocytes. MDM enhances phagocytosis of hemocytes, and the expression of a corresponding gene for MDM is downregulated in *L. stagnalis* infected with *T. szidati* (159, 161). In contrast, treatment of hemocytes with granularin decreases phagocytic activity, and the encoding gene is upregulated in parasitized snails (160, 161).

In the snail host, avian schistosomes also interfere with metabolism, such as causing abnormal body growth, and endocrine functions, such as causing a reduction of egg laying (161). In *L. stagnalis*, these processes are regulated by neuroendocrine cells in the central nervous system (CNS) (162, 163). *Trichobilharzia szidati* releases an undescribed substance that induces the snail host to produce schistosomin (a peptide of 8.7 kDa consisting of 79 amino acids) from its connective tissue and hemocytes (145). Schistosomin acts as a neuropeptide that interferes with some hormones, such as calfluxin, a neuropeptide that stimulates the influx of Ca^{2+} into the mitochondria of albumen gland cells (164, 165). As a consequence, ovulation and egg laying are inhibited in the snail. Excitability of neuroendocrine cells (light green cells [LGCs]) responsible for growth (162) increases in response to schistosomin (166). As a result, the body size of infected snails may become considerably larger than that of uninfected snails (142, 161). Other neuropeptides (FMRFamide-related peptides) are also upregulated during infection of *L. stagnalis* by *T. szidati*, and these peptides, via inhibition of neuroendocrine cells, may be responsible for the suppression of snail metabolism and reproduction (167). All these changes in infected snails may provide energy resources and space that can be exploited by the schistosomes for development (145, 161).

The survival rate of infected snails releasing schistosome cercariae as the agent of cercarial dermatitis varies among species. A limited number of studies have focused on survival rates of avian schistosome-infected versus uninfected snails. As an example, 90% of *L. stagnalis* snails infected experimentally with a single miracidium of *T. ocellata* (North American isolate) were alive at 28 weeks of age (three infected snails were alive for 19 months), whereas all uninfected snails were dead (168). In contrast, *L. stagnalis* or *Planorbarius corneus* snails naturally infected with *T. szidati* or *Bilharziella polonica*, respectively, lived a shorter time, on average, than the corresponding uninfected individuals (169, 170).

VERTEBRATE HOST FINDING AND PENETRATION

Cercariae emerging from the snail intermediate host are the infective stage to the definitive host and are also the stage responsible for causing cercarial dermatitis. A cercaria is a multicellular larva comprised of an oblong body and a slender tail that is bifurcated (furcocercous) at the posterior end (Fig. 2). Cercariae of schistosomes leave their snail hosts actively. For this purpose, they employ a pair of specialized unicellular escape glands that are obvious in mature cercariae within sporocysts. Once the cercariae have emerged, only their ducts lined by microtubules are visible, sug-

FIG 2 Furcocercaria of *Trichobilharzia regenti* with protruded acetabulum (arrow), lateral view. Bar, 200 μm. (Courtesy of J. Bulantová, reproduced with permission.)

gesting a release of granular gland content likely containing histolytic enzymes during their migration through the snail tissue (171).

Once in the water, schistosome cercariae express a complex pattern of behaviors that is composed of movement cycles that are repeated in defined frequencies (172). In general, they express negative geotaxy and positive phototaxy, which result in the concentration of cercariae just beneath the water surface, where appropriate definitive hosts may occur (173). Surface tension of the water enables clinging of cercariae via their ventral sucker (acetabulum) (173). This resting phase is interrupted by phases of active swimming (173). The effect of light on the behavior of swimming cercariae was studied in detail for *Trichobilharzia szidati*. Cercariae are able to react to a moving shadow stimulus (produced by a potential host) by a burst of forward swimming (body first) away from the source of light, to deeper levels, where they can encounter the feet of duck definitive hosts. A shadow stimulus applied in the active phase inhibits swimming and prolongs the following passive phase (173). Two types of photoreceptors, located near the dorsal surface of the body, were described for this species: a pair of lens-covered pigment cup ocelli and a special type of unpigmented, rhabdomeric photoreceptors, composed of three cells arranged in a three-dimensional (3-D) configuration. The lens-covered pigment cup ocelli probably serve to detect the direction of incoming light and to control the direction of swimming in relation to a light source, while the unpigmented photoreceptors serve as monitors for light intensity (174). The pigmented ocelli are also present in other genera of avian (*Bilharziella*, *Dendritobilharzia*, *Gigantobilharzia*, and *Austrobilharzia*) and mammalian (*Schistosomatium* and *Heterobilharzia*) schistosomes but are absent in *Schistosoma*.

Moving shadows also trigger a readiness for cercarial attachment to substrates, further stimulated by thermal and chemical host cues (172). As for the latter signals, compounds in the host skin (ceramides and cholesterol) stimulate enduring contact of cercariae of *T. szidati* with the host skin (173, 175). There is variability among schistosome species in responses to light, shadow, physical, and chemical cues, such that for different species, some of the signals may not work or may include some additional ones, such as touch, water turbulence, and/or additional chemical compounds (172, 176). The variation among the different avian schistosome species reflects the diversity in biology and ecology of schistosomes and their adaptations to the spectra of avian hosts (172, 176).

Invasion of the bird or mammal skin is initiated by the cercariae receiving the proper signal. Surprisingly, there were few differences between the avian *T. szidati* and human *S. mansoni* organisms in their pattern of invasive behavior toward living human skin; most cercariae did not penetrate the skin immediately after attachment but performed a leech-like creeping which lasted 0 to 80 s for *T. szidati* and 15 s to 5.58 min for *S. mansoni* (177). Such behavior guided the cercariae to skin wrinkles or hair follicles, where most penetration sites were located (178) (Fig. 3). Penetration behavior and production of secretions stimulate neighboring cercariae to use the same entry site on the skin (177, 178). Invasion of the skin is facilitated by secretions of cercarial penetration glands released from openings at the apex of the muscular head organ by spasmodic contractions of cercarial body musculature (179). The head organ performs concurrent thrust movements against the skin surface while the cercaria is firmly attached by the ventral sucker (176, 178, 180). Signals for skin invasion seem to be universal for schistosomes—fatty acids, especially polyunsaturated fatty acids containing 18 carbons and two or three *cis* double bonds (linoleic and linolenic acids), which are bound in cell membranes and occur as free molecules on the surface of human and bird skin (176).

For *T. szidati*, the tail is shed within 0 to 105 s after the onset of penetration, sometimes during creeping. This shedding seems to be generated at least partially by contractions of a muscular collar at the body-tail junction, which plays a role in the closure of the cercarial hind body after tail shedding. Cercarial penetration occurs in a nearly surface-parallel direction, while the spined ventral sucker supports squeezing of the cercarial body into the opening in the skin caused by histolytic gland secretions. Full penetration of living human skin was achieved within a mean of 4 min (83 s to 13.3 min), which was significantly faster than the case for the human parasite *S. mansoni* (6.58 min, on average) (177). Faster penetration of avian schistosome cercariae might be a consequence of these parasites' adaptation to lower concentrations of fatty acids in duck skin; therefore, reaction to higher concentrations in human skin may induce faster invasion (177, 178). Another explanation may be that different histolytic enzymes are used for penetration (see below). Skin penetration success rates

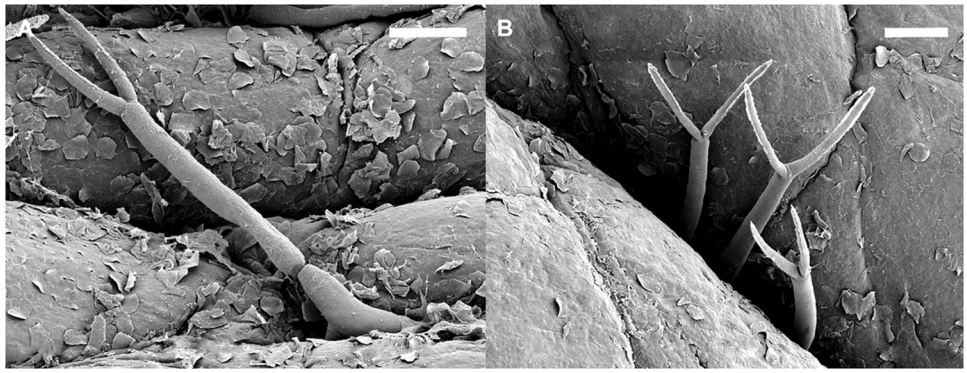

FIG 3 Scanning electron micrographs of cercariae of *Trichobilharzia regenti* penetrating the skin of a duck leg. (A) An individual larva entering the skin; the tail is still preserved. (B) Tails of three cercariae penetrating the skin in a group. Bars, 100 μm. (Courtesy of J. Bulantová, reproduced with permission.)

may vary greatly. In experiments with *T. szidati* and human volunteers, the highest penetration success rate was 49% underneath the forearm (181).

The ultrastructure and chemical composition of avian schistosome cercarial penetration glands and their secretions have been poorly studied relative to the case for human schistosomes (*Schistosoma*). In fact, most of our knowledge is based on only two species of *Trichobilharzia*. There are five pairs of unicellular penetration glands: three pairs are located behind the ventral sucker (postacetabular glands), and two pairs are located around the ventral sucker (circumacetabular or preacetabular glands). These glands are filled with secretory vesicles that are released through the gland processes at the surface of the head organ. A 3-D model of *T. regenti* acetabular glands shows that they occupy more than one-fourth of the cercarial body volume (postacetabular glands, ca. 15%; and circumacetabular glands, ca. 12%). Differences were observed in the appearance of granular material/secretory vesicles contained in the glands, pH value, and the ability to bind various dyes and fluorescent markers (180, 182).

In *T. szidati*, proteolytic activity was detected in cercarial gland secretions induced by linoleic acid. This activity was linked with an orthologue of a chymotrypsin-like serine peptidase, named cercarial elastase and characterized from *S. mansoni* and *S. haematobium* cercariae. A protein on blots of *T. szidati* cercarial secretions as well as in histological sections of penetration glands immunologically cross-reacted with antibodies against elastases of *S. mansoni* and *S. haematobium* (183, 184). However, in another study, the reaction of antibodies raised against elastase of *S. mansoni* was observed neither with the penetration glands of *T. szidati* nor with the cercarial secretions on Western blots (180). In contrast, high activities of cysteine peptidases were noticed in induced cercarial secretions and homogenates for *T. szidati* and *T. regenti* (180, 185). In addition, the presence of cercarial elastase in the latter species was not confirmed by screening of a cDNA library (186). On the other hand, a papain-like cysteine peptidase, termed cathepsin B2, was found in the postacetabular penetration glands of *T. regenti*. This enzyme was shown to cleave proteins of the host skin, similar to the case with *S. japonicum* (187, 188). Its expression was even higher in intravertebrate stages (schistosomula and adults), suggesting that there are multiple roles of this enzyme during the life cycle (189). It seems that the use of particular peptidase families for skin penetration and tissue invasion is diverse among schistosomes (190) and may confer host specificity.

In the postacetabular penetration glands of *T. szidati* and *T. regenti* cercariae, a lectin(s) specific for β-1,3- and β-1,4-linked saccharide chains and their sulfated derivatives is present, though its biological function is still unknown (180, 191). It is interesting that there are high concentrations of calcium in the circumacetabular glands of both species (180). Several hypotheses suggest that the role(s) of calcium in the glands (including those of *S. mansoni*) may be to regulate gland peptidase activity, stimulate glycocalyx removal, interact with connective tissue proteoglycans, regulate host blood coagulation, or polymerize the adhesive substance from postacetabular glands. However, none of these hypotheses (except for a regulation of peptidase activity) has been confirmed adequately (180, 192, 193). Following contact with the host skin or a linoleic acid-coated surface (L. Mikeš, unpublished data), cercariae start to expel small amounts of gland content during the creeping movement, which is "printed" as the cercariae touch the surface—these "kissing marks" are made of a sticky substance. In *S. mansoni*, this substance is a product of the postacetabular glands and is composed of neutral and acidic mucosubstances (194). This product might serve adhesive or enzyme-directive functions. Similar material is produced by cercariae of *Trichobilharzia* spp. yet differs in chemical composition (180). Finally, the production of three types of eicosanoids by *T. szidati* cercariae is stimulated by linoleate (195). Eicosanoids may have a role in host invasion (vasodilatation), and their involvement in immune evasion was proven by the inhibition of superoxide production by human neutrophils (195).

During cercarial penetration, dramatic changes of surface structures and metabolism lead to the transformation of the cercaria to a schistosomulum. The thick glycocalyx that served as a protective layer for the free-living cercaria is shed, as its carbohydrate-rich composition is a target of the host complement cascade. In the schistosome species studied so far, the glycocalyx is markedly rich in fucose residues (154, 196–198). There is an obvious loss of saccharide moieties at the surfaces of transformed schistosomula of *T. szidati* and *T. regenti*, leading to reduced immunoreactivity and attractiveness for fucose-specific lectins (154, 155, 199, 200). Also, similar to the case for human schistosomes and members of other families of blood flukes, the trilaminar surface membrane of the outer cercarial tegument gradually changes to the doubled heptalaminar membrane of the schistosomulum, which has a protective function against the host immune system (199). In *T. regenti* stimulated by linoleate *in vitro*, shedding of the

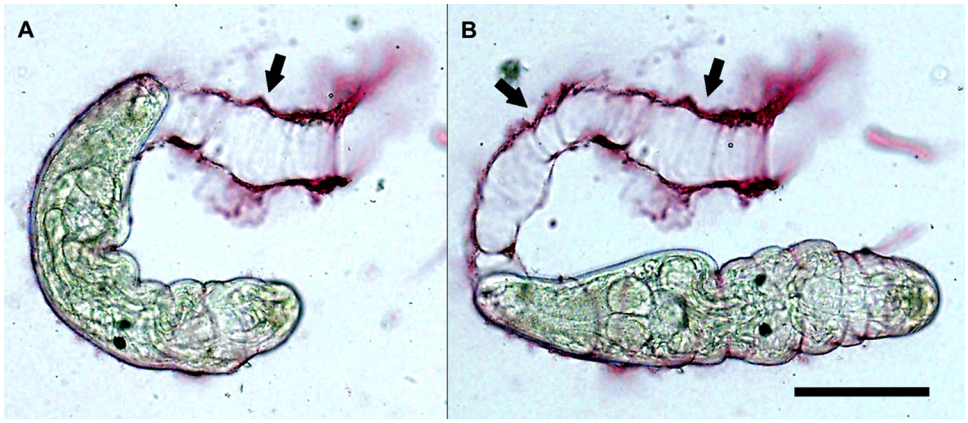

FIG 4 Living *Trichobilharzia regenti* cercaria *in vitro*, shedding its glycocalyx upon stimulation by linoleate. The sticky products of the penetration glands, stained with lithium carmine, adhere to the surface of the body, and as the cercaria crawls forward by periodical constrictions (A) and extensions (B), the glycocalyx and bound secretions are removed from the surface in a sleeve-like manner. Arrows indicate detached sleeve-like remnants of glycocalyx and gland products. Bar, 100 μm. (Courtesy of J. Chaloupecká, reproduced with permission.)

glycocalyx starts at the anterior of the cercaria, surrounding the openings of penetration glands (J. Chaloupecká and L. Mikeš, unpublished data). Sticky products of the glands adhere to the surface of the body, and as the cercaria crawls forward, the glycocalyx, with the gland products, is shed in a sleeve-like manner, until it is detached at the end of the hind body (Fig. 4). Transformed cercariae/schistosomula then lose their osmotic resistance toward water and become dependent on isosmotic conditions of the host (Chaloupecká and Mikeš, unpublished data). Whether any compounds of gland secretions take part directly (e.g., enzymatically) in the process of glycocalyx shedding is still unclear.

It should be mentioned that cercariae of avian schistosomes readily penetrate other (soft) tissues, and peroral infections of birds (definitive hosts) and mice (accidental hosts) with cercariae of *T. regenti* and *T. szidati* have been confirmed (L. Kolářová, K. Blažová, V. Pech, and P. Horák, unpublished data). This phenomenon is also known for mammalian schistosomes of the genus *Schistosoma* (201–203). It is not clear, however, how much these peroral infections contribute to the transmission of schistosomes under natural conditions. Due to the features of the esophageal mucosa, the penetration of *Trichobilharzia* cercariae does not require the penetration glands to be emptied completely, and their tails may be preserved for some time. The stimuli triggering this penetration behavior remain unknown.

PATHOGENICITY OF AND IMMUNE REACTIONS AGAINST AVIAN SCHISTOSOMES

Survival and Migration in Avian Hosts

Once the cercariae have transformed into schistosomula and reached their final location, it is not clear how long avian schistosomes live in their definitive hosts. For example, in experimental infections of ducks, adults of *Trichobilharzia parocellata* were found at 86 days postinfection (p.i.) (204). Whereas experimental infections with *T. szidati* and *T. regenti* last for about 3 to 5 weeks (13, 205), the adult worms of a Canadian isolate of *T. ocellata* were found in the liver at 370 days p.i. (206). Data describing the schistosome life span and length of time for egg release in a bird host will be necessary in considering the epidemiology of dermatitis. Nonetheless, avian schistosomes have a preference for two major habitats within the avian host: the visceral venous system (mesenteric, renal, cloacal, and portal vessels) and the nasal passages (except for *Dendritobilharzia*, which is found in the arterial system) (13).

Migration and localization have been characterized for a few visceral schistosomes and only one (*T. regenti*) of the eight nasal schistosomes (205, 207). Visceral schistosomes in birds have a migration pattern similar to those of *Schistosoma* spp. in mammals. After skin penetration, schistosomula of *T. szidati* navigate toward deeper skin layers by following dark and higher concentrations of D-glucose and L-arginine (208, 209). Once a blood capillary is found, the worms penetrate it and migrate to the heart and lungs. In the lungs, the worms enter free air space and then reenter the blood system (206, 210). Finally, visceral blood vessels (usually portal and mesenteric veins) are the preferred habitat (13). However, there are at least two exceptions, as follows: (i) the adults of *T. szidati*/*T. ocellata* leave the blood system and enter the layers of the host intestinal wall and mucosa (206, 211) and (ii) *Dendritobilharzia pulverulenta* prefers the arterial system of its hosts, where it is found in the lower dorsal aorta and the femoral arteries (212). For the nasal schistosome *T. regenti*, migration is dramatically different. Schistosomula leave the skin and then seek and penetrate peripheral nerves (Fig. 5) to migrate to the spinal cord and brain of their host. From there, the adult worms appear intra- and extravascularly in the nasal mucosa (207).

Pathology Caused by Visceral Species in Birds and Mammals

Most pathological studies of avian schistosomes in the avian host have been detailed for experimental birds, though there are a few reports from wild birds. As for accidental mammalian hosts, only experimental infections have been evaluated. The schistosomula migrate through the heart and lungs of birds and mammals, and only in the avian host do they reach their final destination. Migration through host lungs has been shown to cause damage (16, 210, 213). Infections of duck and mouse lungs by *T. szidati* are accompanied by hemorrhages in the periphery of the lungs (210). Migration of schistosomula in the lungs of ducks leads to formation of lymphocytic lesions and an influx of macrophages, heterophils,

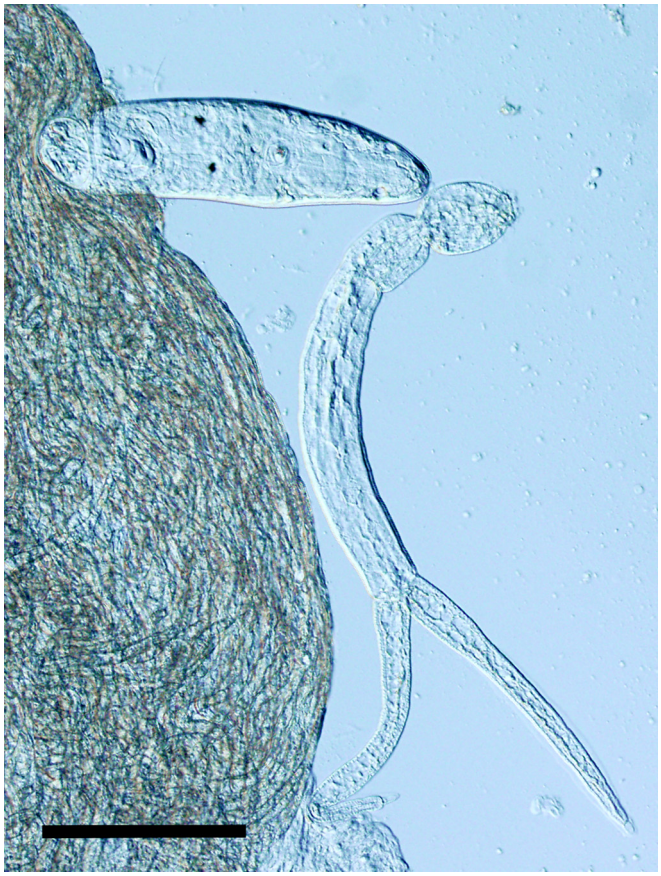

FIG 5 Cercaria of *Trichobilharzia regenti in vitro*, penetrating a peripheral nerve isolated from a duck. The tail is already detached, and the head organ burrows into the nerve. Bar, 200 μm. (Courtesy of J. Bulantová, reproduced with permission.)

and eosinophils into the afflicted tissue (210). However, in mouse lungs, schistosomula do not evoke a specific inflammatory reaction; only alveolar congestion and edema are observed (210). Damage to the lung tissue in general, and formation of alveolar congestion in particular, might be caused by schistosomula that leave the blood system and localize extravascularly in the alveolar walls. Their inability to reenter the blood system might be linked to their relative size and loss of orientation in a noncompatible mouse host (210). Similarly, migrations to mammalian lungs and accompanied hemorrhages have been observed in hamsters, guinea pigs, rabbits, and rhesus monkeys experimentally exposed to three species of *Trichobilharzia* (213). Pulmonary infections of chickens and pigeons with the marine schistosome *Ornithobilharzia canaliculata* led to the development of lesions in the arterial and venous vascular systems (lymphocytic endarteritis, periarteritis, and segmental proliferation of the vascular endothelium), hyperplasia of smooth muscles in tertiary bronchi, and thickening of alveolar septa. Cellular infiltrates consisted mainly of histiocytes, heterophils, and lymphocytes (16). In contrast to the case with visceral schistosomes, infections of duck and mouse lungs with the nasal species *T. regenti* probably represent an ectopic localization of schistosomula (207, 214, 215).

As for patent infections (exclusively in birds), major pathology is caused by granulomas around eggs and only partly by adult worms. Obliterative endophlebitis caused by adult schistosomes (probably *Trichobilharzia filiformis*) in the intestinal veins of mute swans (*Cygnus olor*) has been recorded (216). The intestinal surface showed various stages of villous atrophy with intestinal mucosal lesions, associated with infiltration of the lamina propria of the jejunum and ileum by lymphocytes and plasma cells and by a smaller number of heterophils and eosinophils. Eggs were observed multifocally in the lamina propria of the small and large intestines, and their presence triggered mild to severe granulomatous reactions (216). Eggs laid by the adult worms of *Austrobilharzia variglandis* in experimentally infected chickens caused edema, cellular infiltration, and hyperplasia of smooth muscle of the muscularis externa of the intestine. Around the eggs, mononuclear cells formed early granulomas with a few eosinophils and heterophils, followed by accumulation of giant and epithelioid cells (217).

Similarly, examination of Atlantic brant geese (*Branta bernicla hrota*) infected with *Trichobilharzia* sp. (probably *Anserobilharzia brantae*, based on current taxonomy) revealed the development of granulomas around eggs located in the colon (218). Granulomas were also observed in the duodenum and small intestine of pigeons infected with *Ornithobilharzia canaliculata* (16). Three species of ducks infected with *T. physellae* showed no associated tissue reaction in the vicinity of adults located in mesenteric veins, but a granulomatous reaction around the eggs was detected occasionally in the mucosa and submucosa of the intestine (219). On the other hand, the most serious lesions and fibroplasia of the portal triads and adjacent parenchyma were observed in the livers of those ducks and were attributed to mature *T. physellae* (219). In the case of pigeons infected with *O. canaliculata*, granulomatous lesions surrounding collapsed eggs were observed in the liver parenchyma (16). Intestinal pathology may be accompanied by poor nutritional conditions, as noticed in pigeons infected with *O. canaliculata* (16). Exceptionally, a more serious manifestation of the infection in naturally infected wild ducks was reported where the adults and eggs of *T. physellae* caused partial to complete paralysis of the cervical, wing, and leg muscles, foul-smelling diarrhea, and half-closed pasted eyelids (219).

Pathology Caused by Nasal Species in Birds and Mice

Histological examination of the nasal tissue of birds showed the extra- and intravascular locations of adult worms of *T. regenti* (220), with their first appearance on day 13 p.i. (205). Immature eggs appeared from day 15 p.i., and at day 19 p.i., eggs were fully developed and observed extravascularly in the nasal mucosa, with the maximum number of eggs seen on day 22 p.i. (221). The area surrounding the eggs was infiltrated by numerous eosinophils, heterophils, histiocytes, and multinucleated giant cells and a few plasma cells and mononucleated cells (220). The formation of granulomas around the eggs was noted from day 22 p.i. (221). Miracidia that hatched directly in the host tissue were surrounded by lymphocytes, eosinophils, and heterophils, without granuloma formation (221). While an infected bird is drinking/feeding, only the miracidia leave the tissue to enter the water, which represents an exceptional mode of transmission among schistosomes (205). The presence of adults only did not initiate an influx of immune cells to their vicinity, but the presence of large worms and eggs caused the development of focal hemorrhages throughout the nasal mucosa (207, 221). Probably the more devastating aspect of the pathology of this species is the effect on the CNS of hosts (birds

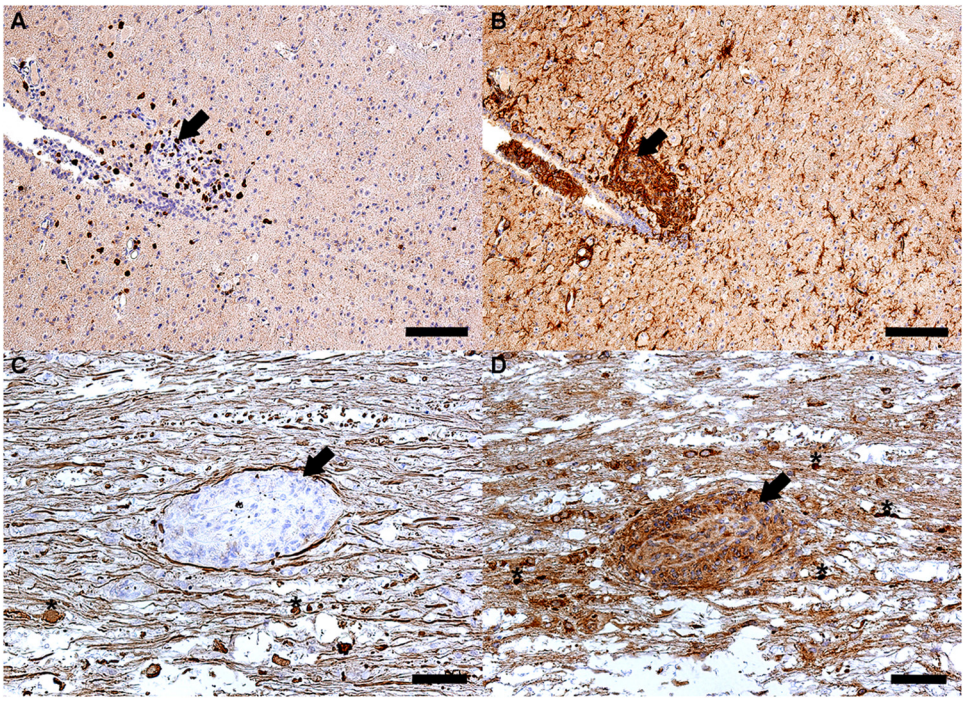

FIG 6 Destruction of a schistosomulum (arrows) in the thoracic part of the spinal cord of a BALB/c mouse at 21 days p.i. (longitudinal sections). (A and B) Inflammatory lesion consisting of $CD3^+$ lymphocytes (dark spots) (A) and microglia cells (brown-stained ramified cells) (B) that were detected by use of anti-mouse $CD3^+$ and anti-mouse Iba-1 antibodies, respectively. Nuclei of other cells were stained blue by hematoxylin. (C and D) Tissue around the schistosomulum contains damaged axons. Axonal damage was accompanied by formation of spheroids (asterisks) in the site of axonal disruption and was visualized immunohistochemically by use of anti-mouse nonphosphorylated and phosphorylated neurofilament antibodies (SMI-311 and SMI-312, respectively) (C) and anti-mouse β-amyloid precursor protein antibodies (D). Bars, 100 μm (A and B) and 50 μm (C and D). (The figure was created by L. Lichtenbergová.)

and experimental mammals). As stated above, cercariae penetrate the skin and migrate to the nasal passages via the CNS rather than the circulatory system.

CNS infections of ducks and mice by *T. regenti* may lead to the development of various transient or permanent neuromotor symptoms, such as weak to severe leg paralysis and balance/orientation disorders (207, 214, 215). In avian hosts, schistosomula in the CNS initiated an accumulation of inflammatory cells, such as eosinophils and heterophils, that represented the most abundant cell infiltrates, yet minimal damage to nervous system cells was detected (220). In a few cases, however, damage to the CNS was observed in birds with visceral schistosomes. For example, granulomatous encephalitis in mute swans, caused by *Dendritobilharzia* sp., has been described (222, 223). Schistosome eggs found in the cerebrums and cerebellums of naturally infected swans were surrounded by giant cells, macrophages, lymphocytes, and, to a lesser extent, heterophils and fibroblasts (223). The presence of *Dendritobilharzia* eggs in the CNS represents an ectopic localization.

Because of the pathology caused by *T. regenti*, and thus the implications for human health, most experimental work has been done in mice rather than birds. For up to 3 days p.i., migration of schistosomula through the murine nervous tissue did not evoke inflammation or tissue damage, and all detected parasites were intact (215, 224). A host reaction to the infection was visible on days 6 and 7 p.i. The presence of parasites led to the accumulation of immune cells, predominantly microglia, macrophages, and neutrophils and, to a lesser extent, $CD3^+$ lymphocytes (215, 224). Proliferating astrocytes formed "glial scars" at the sites of previously migrating schistosomula (215). Ongoing infection was associated with a more intense inflammatory reaction in white and gray matter of the spinal cord. Microglia, macrophages, neutrophils, eosinophils, and $CD3^+$ lymphocytes participated in the formation of inflammatory lesions surrounding the disintegrating schistosomula, and damage to the axons was detected (215) (Fig. 6). The localization of schistosomula outside the solid tissue, in the subarachnoidal space of the spinal cord and the brain and in the cavity of the 4th ventricle of the brain, led neither to damage nor to inflammation of the adjacent nervous tissue (215). It seems that schistosomula located in the cavities of CNS were able to delay destruction by the immune cells. Nevertheless, most of the worms were eliminated by 21 days p.i. (215). Challenge infections triggered a strong immune response, which efficiently and rapidly eliminated the schistosomes (215, 224).

In immunodeficient SCID mice, primary infections as well as reinfections did not evoke a significant skin reaction, and the schistosomula often escaped from the skin to the CNS (224), where migrating schistosomula caused axonal damage and an influx of immune cells (215). In comparison to the case with immunocompetent mice, the schistosomula survived longer in the CNS, probably due to the absence of T and B lymphocytes (215, 224), cells that may represent important effectors in destruction of schistosomula. Larger numbers of schistosomula in the CNS and their extended time of migration via nervous tissue resulted in a higher rate of occurrence of paralysis of immunodeficient SCID mice (215).

The above-mentioned damage to the nervous tissues of birds and mice demonstrates that not only the eggs but also the other stages of schistosomes are responsible for major pathology. In this particular case, just the schistosomula (migrating juveniles) of *T. regenti* can be regarded as the most pathogenic stage of the parasite.

Skin Immune Response and Cercarial Dermatitis

Skin immune reactions of birds to the penetration of avian schistosome cercariae are insufficiently described. In any case, birds do respond to penetrating cercariae, as shown in a histological observation of chicken skin infected by *Ornithobilharzia canaliculata*. Severe infiltrations of the dermis by histiocytes and heterophils and aggregation of lymphocytes around dilated capillaries in the dermis were recorded (16). Dead and destructed schistosomula surrounded by heterophils and histiocytes were found in the epidermises of chickens at 12 h p.i. At 24 h p.i., lymphocytes, histiocytes, and heterophils still persisted in the dermis, but the number of immune cells decreased (16).

Immunohistopathology of cercarial dermatitis in humans recognized three phases of cellular responses (leucocytic, lymphocytic, and histiocytic) against *Trichobilharzia* larvae (225). However, because studies on humans are rarely performed (112, 225, 226), a mouse model was established to provide more-detailed studies on immunohistopathology. Primary infection of mice with *T. regenti* causes an acute inflammatory reaction with edema and vasodilatation (227). Parasites located in the dermis are surrounded by large inflammatory cellular foci that are formed by neutrophils, macrophages, mast cells, major histocompatibility complex class II (MHC II) antigen-presenting cells, and a small number of $CD4^+$ lymphocytes (227). Repeated infections cause perivasculitis, folliculitis, and substantially more influx of the same cell types that are noted after primary infection (227). Extensive skin inflammation leads to the formation of large abscesses and subsequently to dermal and epidermal necrosis. Sites of previous cercarial penetration are characterized by intraepidermal pustulae and parakeratosis (227).

In vitro culture of skin biopsy specimens from primary mouse infections by *T. regenti* revealed a release of the acute-phase cytokines interleukin-1β (IL-1β) and IL-6 and an increased production of IL-12 (227). Larger amounts of IL-12 correlated with elevated production of gamma interferon (IFN-γ) in the cell culture supernatant (antigen-stimulated lymphocytes) from the skin draining lymph nodes (227). IFN-γ and IL-12 are associated with Th1 cell differentiation, and IL-1β and IL-6 are important in Th17 polarization (228). Although the role of Th17 in host tissue immunopathology has been described for some infections by helminths (e.g., human schistosomes) (228), participation of Th17 cells in the processes associated with skin penetration by avian schistosomes needs to be clarified.

Reinfections of mice with *T. regenti* were accompanied by edema that developed as a consequence of local vascular permeability caused by histamine released from activated mast cells (227). Histamine has a regulatory function in the Th1 and Th2 polarization of the immune response (229). Mast cells also rapidly released a large amount of IL-4, which has been detected in the supernatants of skin biopsy specimens from *T. regenti*-reinfected mice (227). Like mast cells, basophils degranulate and release IL-4 as a response to the presence of *T. regenti* antigens (230). Elevation of total serum IgE levels implies that histamine and IL-4 production by mast cells and basophils occurs in an IgE-dependent manner (227, 230). Dominance of the Th2 response was also supported by an elevation of antigen-specific IgG1 antibodies and a decrease of IgG2b antibodies (Th1 associated) in the sera of mice reinfected with *T. regenti* (230). Cercarial dermatitis in reinfected mice is therefore Th2 polarized, with a response comprised of an early type I hypersensitivity reaction and late-phase skin inflammation (227).

It has been shown in several cases that mammals (including humans) are unsuitable hosts for avian schistosomes, such that the worms cannot mature and reproduce (except for *Austrobilharzia variglandis*, which is able to reach sexual maturity in the lungs of *Meriones unguiculatus* gerbils [231]). Nevertheless, cercarial dermatitis is probably not the only interaction of avian schistosomes and mammals. Dermatitis develops as an immune (allergic) reaction of the already sensitized person; it represents a powerful protection of the body against worms in the skin. However, in a naive (nonsensitized) or immunodeficient experimental host, at least some worms survive, leave the skin, and migrate throughout the body (213, 232). Mild to severe consequences of such migration may appear (see above); most importantly, *T. regenti* is neurotropic and can cause damage to the central nervous system (155, 207, 214, 215, 220, 224). To date, no information about migration of avian schistosomes in human bodies is available. It therefore seems that laboratory animals (mice, rats, etc.) are indispensable for assessing all risks associated with infections of mammals (humans) by avian schistosomes.

DETECTION AND IDENTIFICATION OF AVIAN SCHISTOSOMES

Prior to or during the dermatitis season, especially following a dermatitis outbreak, a standard protocol for the detection of schistosome cercariae involves the collection of and screening for cercarial emergence in snails. Usually, in a laboratory, individual snails are placed in a beaker/small wells with clean water and exposed to a lamp to stimulate shedding of cercariae. Subsequently, cercariae are collected and identified under a light microscope. If no cercariae emerge from the snails, there are three options: the snails can be dissected to find the schistosomes, the snail tissue can be pooled and molecular techniques applied to detect a schistosome infection, and/or additional snails can be collected from affected and surrounding areas (233, 234). Detection of microorganisms directly from water samples is developing rapidly, but among schistosomes, these techniques have been optimized for human schistosomes (235, 236). Methods thus far to detect avian schistosomes, *Trichobilharzia* in particular (237, 238), involve concentrating the cercariae from a water sample and using a PCR assay to detect a single cercaria in plankton (0.5 g) and snail tissues (0.25 g) (239). Using definitive host-seeking behavior, cercariae can be lured to a trap that contains linoleic acid, a known stimulus for cercarial penetration (240). Irrespective of a dermatitis outbreak, identifying which adult worms can be found in the habitat can be completed by examination of feces for eggs by using sedimentation/flotation (241) or Kato-Katz fecal smear methods (242) and/or postmortem examination of the arterial/venous system, nasals, liver, or kidney of the host. For nasal schistosome species, lavage of the nasal cavity of birds represents a method of choice. After rinsing out the nasal cavity with saline or water, freely moving miracidia (and partly the liberated eggs) can be detected in the wash fluid (13). The above-described examination of snails

and birds for avian schistosomes is thoroughly summarized in reference 243.

Examination of water or the snail or bird host is important for species delineation. Not only is there a highly diverse population of schistosomes, especially avian schistosomes, that are responsible for dermatitis, but these species differ markedly in their morphology and pathogenicity in birds and experimental animals (12, 13). Identification of schistosomes, particularly avian schistosomes, is challenging. Traditional methods using morphology (adults mostly, but also eggs and cercariae) are usually not sufficient to separate species, particularly within a genus, as these characteristics can be missing or minute, and their recognition often depends on the experience of the observer. However, some characteristics have been found to be informative for rough species identification, such as the position of internal organs and tegument spination of adults (13), morphology of eggs (244), or distribution of sensory papillae on cercariae (68, 198).

In addition to the morphological features, there are some nonmorphological criteria that can be used to differentiate species, for example, the behavior of cercariae (245), compatibility with the snail hosts, and organ/tissue affinity of adults in birds (135). If eggs and adults from a host are examined, it is not always clear which adults/eggs are conspecific or which match cercariae from snails unless experimental trials are conducted. Experimental infections with cercariae from snails to obtain adult worms for morphological assessment are ideal, but these are time-consuming and often yield low prevalences of infection. Because of the reported species, morphological, and pathogenic diversity of avian schistosomes (12, 13), from an epidemiological perspective on dermatitis, there is a need for species delineation and host use, and molecular techniques have proven a necessary and excellent tool for more rapid identifications (63, 70, 135). Such techniques have allowed us to genetically connect larval stages from snails to adult stages from birds, greatly advancing the epizootology and epidemiology of avian schistosomes in particular (10, 40, 123).

Molecular identification of an avian schistosome provides clues to the host source and biology and possible targets for control. There have been steady efforts and testing of markers that have revealed some consistency and validity for species identification of schistosomes (123, 246, 247). Molecular phylogenetics has had a major impact on the taxonomy and discovery of new lineages of avian schistosomes, particularly the major etiological agents of cercarial dermatitis, i.e., species of *Trichobilharzia* (10, 40, 63, 67, 69, 70, 121–123, 125, 129, 135, 248–251). Most of the effort has been conducted using three gene regions. To date, the nuclear ribosomal DNA D1-D2 regions of 28S, the internal transcribed spacer (ITS) region, and the mitochondrial *cox1* gene have been tested as molecular markers for systematics (40, 63, 67, 69, 70, 105, 123, 248, 249, 251), epidemiology (252), and diagnostics (121, 253). Sequencing of gene regions such as the nuclear ITS region and the mitochondrial *cox1* gene not only has linked life cycle stages but also has suggested that our recognition of the diversity of avian schistosomes continues to grow (10, 12, 40, 65–67, 69, 70, 105, 129, 133, 135).

While the molecular identification of avian schistosomes is still in the early stages, these markers have thus far proved successful in attributing most samples to a known species. At the level of (infra)populations, genetic diversity of *T. szidati* cercariae from 7 snails collected at 3 localities in Russia was recently shown by use of randomly amplified polymorphic DNA (RAPD) (254). At this time, neither microsatellite markers nor next-generation sequencing protocols have been developed that would allow detection of specific populations or host strains. The development of genome-wide sequencing protocols for population genetic analyses would greatly aid in identifying the epidemiological determinants of cercarial dermatitis outbreaks. Although genetic identifications have provided a framework for circumscribing species and for rapid detection, caution must be exercised, since genetic identification alone is not sufficient in the absence of data on disease dynamics and morphology (255, 256). A species designation should ideally reflect all data available, i.e., host species, location, and morphological characteristics of adult worms and eggs. In addition, comparative molecular analyses must be performed to obtain reliable and convincing results of species identification.

Most importantly, specimens and other data, e.g., genetic data, should be archived in a permanent museum collection or an archivable Web-based database for data resulting from a specimen, such as the sequence archive GenBank. Voucher samples of any life cycle stage of these schistosomes (or any parasite and host) should be preserved and deposited in a permanent museum collection (257–259). This is imperative for several reasons: the most important is for the question that has not yet been asked. In the event that the parasite sample does not match known species or has odd features, further work may be necessary. Access to images and measurements, plus an additional sample(s), will be used in further sequencing. Moreover, documentation of any species of schistosome coming through a clinic is an important record that can contribute to epidemiological studies, especially if it is associated with people affected by dermatitis.

CLINICAL FEATURES, DIAGNOSIS, TREATMENT, AND PROPHYLAXIS OF HUMAN INFECTIONS

As for the clinical symptoms and signs, penetration of avian schistosome cercariae into mammalian (human) skin may initiate an immediate prickling sensation that persists for approximately 1 h (4, 260). The development and intensity of the subsequent allergic reaction depend on the number and duration of previous cercarial contacts, as well as individual susceptibility (4, 7, 260). The primary contact with cercariae may lead to either an imperceptible (7, 261) or mild skin reaction, with the development of small and transient macules, maculopapules, or inconspicuous papules of about 1 to 2 mm after 0.5 to 2 days p.i. A delayed reaction in the form of small papules can be observed in some persons as late as 8 days p.i. (7, 238).

Repeated infections cause a more pronounced cutaneous reaction followed by diffuse edema and development of erythematous papules or papulovesicles (4, 261). More specifically, the first transitory macules (up to 10 mm) and primary itching can appear as soon as 4 to 20 min after exposure. Thereafter (1 to 15 h p.i.), macules are replaced by papules (about 3 to 8 mm), and an intense itching (secondary itching) is experienced. In addition, erythema and edema may occur in the afflicted area for a few days. Vesicles of about 1 to 8 mm may form on papules at 2 to 3 days p.i. and may rupture as a result of scratching. As a consequence, bacterial superinfection may result in formation of pustules. Papules usually regress and disappear at 4 to 10 days p.i., leaving a pigmented spot (about 1 to 4 mm) on the skin for weeks (7, 225, 238); however, in some cases, the symptoms may persist for about 20 days p.i. (7, 238, 262, 263). Every macula/papula is a reaction against the penetrating cercaria(e) and thus represents the part of the body in

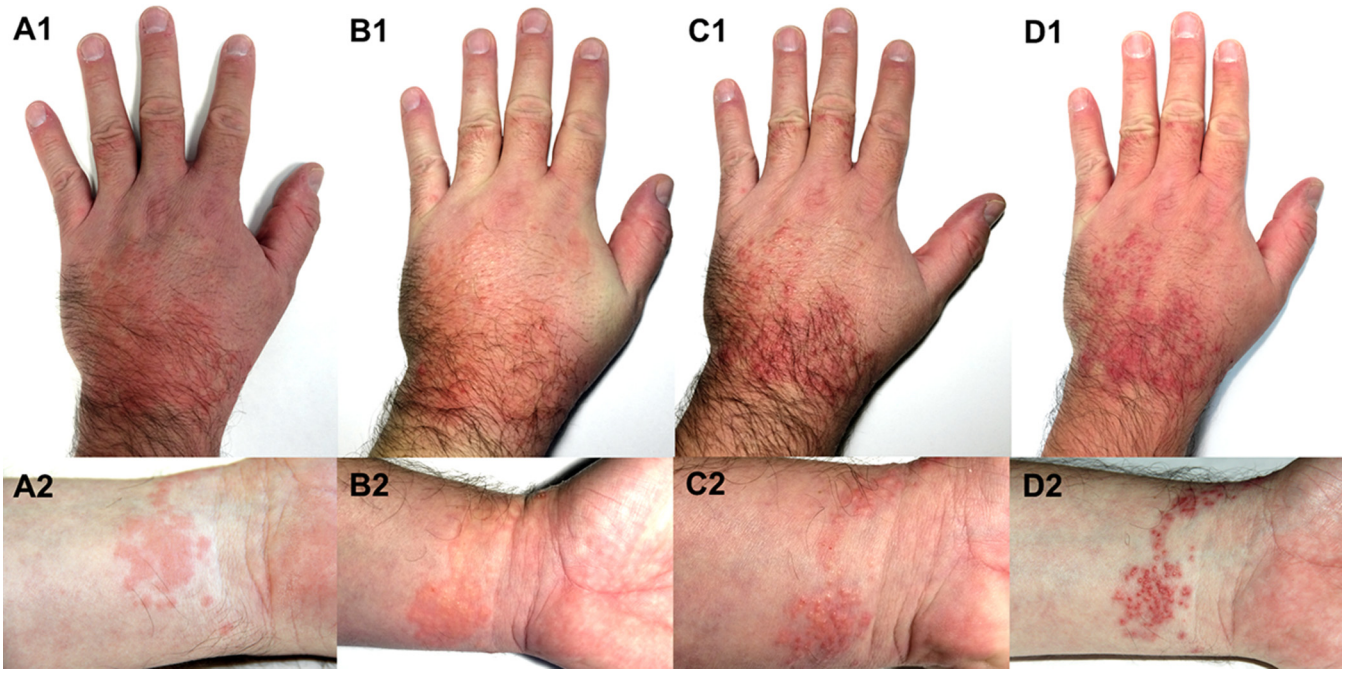

FIG 7 Development of cercarial dermatitis on the dorsal (1) and ventral (2) parts of the left hand of a sensitized volunteer infected experimentally by *Trichobilharzia szidati* (the whole hand was immersed into a beaker containing water with cercariae). Images were taken at 1 (A), 2 (B), 3 (C), and 4 (D) days postexposure. Formation of macules (A) and papules with vesicles (B to D) can be seen. No penetration/reaction was recorded for the palm. Noticeable swelling of the hand is shown mainly in panel B1. (The figure was created by H. Kulíková and P. Horák.)

direct contact with cercariae (Fig. 7). An attack by many cercariae may be accompanied by generalized reactions, such as limb and lymph node swelling, nausea, diarrhea, and fever (7, 238).

Diagnosis is rather problematic. Skin reactions to cercariae from freshwater or marine environments may resemble insect bites, bacterial dermatitis, contact dermatitis, or skin reactions against nematocysts of larval cnidarians (sea anemones, thimble jellyfish, etc.) (7). Anamnestic data (suggesting recent contact with water reservoirs) and maculopapular skin eruption on the body parts that were in contact with water are important indicators. Direct proof of schistosome infection may be shown by skin biopsy of the papulae no later than 48 h p.i. Individual papulae should be excised/shaved off under local anesthesia, put in Bouin's fixative, cut into 10-μm sections, and stained with hematoxylin-eosin (225, 226). As for basic laboratory tests, increased eosinophil counts and elevated levels of total IgE may indicate an attack by avian schistosomes (7, 230, 238). Specific immunological/serological assays (skin test, "Cercarienhüllenreaktion" [a precipitation reaction surrounding the cercarial body in the presence of a specific antibody], indirect fluorescent-antibody test [IFAT], enzyme-linked immunosorbent assay [ELISA], and complement fixation test) to detect penetration by avian schistosomes are not sufficiently specific or sensitive (7, 238). Exceptionally, some reaction has been obtained with sera of dermatitis patients and a heterologous antigen—*Schistosoma mansoni* cercariae—used in Cercarienhüllenreaktion and IFAT (263, 264). Selection of a reliable (recombinant) antigen or primer for serological or DNA-based tests, respectively, is in progress (our unpublished data).

Therapy for afflicted areas includes only symptomatic (not causal) treatment of the condition in the form of soothing agents. For example, water chestnut planters in India use mustard oil to relieve the itching and rash in mild cases of dermatitis (265). In serious cases, application of systemic antihistamines (tablets or gels, e.g., hydroxyzine) or mild corticosteroids (e.g., 0.1% triamcinolon cream or 1% hydrocortisone ointment) may be considered (7, 226, 262, 266).

There are several recommendations to protect individuals. Wearing rubber waders or gloves or neoprene diving suits is 100% reliable, although not always appropriate or realistic. Upon contact with cercariae in water, the number of penetrating larvae can be reduced if action is taken within seconds to a few minutes (see the part on vertebrate host penetration above). For example, thorough toweling and exposure of skin to the sun are recommended for bathers immediately after leaving water. In the case of an accidental exposure in the laboratory, skin can be washed with 70% ethanol or warm water (as much as one can tolerate) and soap (our personal experience). Several chemicals have been tested as barriers to cercarial penetration. For example, LipoDEET, a long-acting liposome formulation of DEET (*N*,*N*-diethyl-*m*-toluamide), a common, safe, and available insect repellent, has been used successfully to prevent penetration of *S. mansoni* (267). However, a cream formulation of DEET was poorly effective against *T. szidati* penetrating living human skin (181). Two other formulations were effective: (i) SafeSea lotion against jellyfish stings, where the effective compound may be H1-antihistamine diphenhydramine; and (ii) niclosamide in a dosage as low as 0.1% in water-resistant sunscreens. We hypothesize that sunscreens based on plant oils (nonmedicated) will trigger cercarial penetration due to a high content of unsaturated fatty acids. In this regard, a negative effect in terms of human protection was observed for dimethicones (polydimethylsiloxanes and silicone oils), which are common ingredients in many skin care products (181).

ECOLOGICAL FACTORS INFLUENCING THE OCCURRENCE OF AVIAN SCHISTOSOMES AND CERCARIAL DERMATITIS

Due to climate changes and land alterations, the seasonal window for parasite transmission may become longer, in addition to changes in the behavioral and physiological patterns of parasite hosts. For example, the accelerated growth of both snail and trematode larval populations (71) or changes in phenology of aquatic migratory birds to sedentary (268–270) can increase chances for outbreaks of cercarial dermatitis in both space and time.

Global Warming and Eutrophication

At least in temperate climates, schistosome cercariae occur seasonally once the ambient temperature is high enough for snail activity and thus cercarial emergence (271). Also, snails infected during late summer can survive through the winter season and serve as a source of cercariae in spring (13, 272, 273). Global warming is predicted to have a positive effect on trematode intramolluscan stages, because their development is strongly temperature dependent, leading to increased cercarial emission rates. For example, a shorter prepatent period in relation to higher temperatures was demonstrated in experimental infections with *T. szidati* (141, 274). A slight rise in temperature will increase developmental rates and transmission success (number of cercariae available to find the definitive host). For example, a 5-fold increase in cercarial emergence with a 10°C increase in temperature was recorded for *Trichobilharzia* sp. from *Radix peregra* (275). In addition, a link has been suggested between the rise of 0.8°C within the last century and the increased frequency of cercarial dermatitis and *Trichobilharzia* prevalence at higher latitudes in Europe (9, 276). What does not change is the life span of cercariae, which is limited to the restricted source of reserve glycogen (cercariae do not take up any food). In this regard, temperature strongly affects cercarial survival. For example, the half-life of *T. szidati* was 16 h at 16°C but only 7.8 h at 25°C (169), and the life span of *Trichobilharzia arcuata* was 72 h at 4°C but only 48 h at 26°C (277). Infected snails change their thermal microhabitat selection, e.g., *Lymnaea stagnalis* infected with *T. szidati* prefers a colder water temperature than that preferred by uninfected snails (19.97°C versus 25°C). This may be an adaptation for either the snail or the schistosome, because at these temperatures larval development is slower and the rate of cercarial emission is lower, leading to less tissue damage in the snail (169, 170).

Eutrophication promotes excessive plant growth and decay, causing alterations in the dynamics of freshwater communities and thus leading to an increased risk of acquiring cercarial dermatitis (11, 278–280). Greater biomass of primary producers is associated with faster development and growth of snails due to the increase in food availability (280–282). Dense snail populations may increase the probability that a miracidium will find a snail and lead to a higher prevalence of infection. In addition, aquatic birds are attracted to such nutrient-rich environments, also contributing to an increase in parasite transmission (283). Using data from a mark-recapture study of *L. stagnalis* in eutrophic fishponds, rapid trematode recruitment into the snail populations was demonstrated (284). Maximum annual rates of colonization/recruitment for *T. szidati* were shown to reach up to 300%, such that the odds of trematode establishment in an individual snail were 3 times per year versus once in 10 years in other, mainly marine trematode-snail systems (284, 285).

Large snail populations with a high prevalence of avian schistosomes have been reported for several localities in Europe (71, 286–288), North America (289, 290), and Australia (291). Usually, natural preserves support diverse and abundant populations of potential hosts of schistosomes, so these areas may serve as hot spots for outbreaks of cercarial dermatitis. Although eutrophication contributes to infection risk, cases of cercarial dermatitis or findings of avian schistosomes have also been reported from oligotrophic or mesotrophic systems (8). In addition, other abiotic and biotic factors and human-induced habitat alterations may influence the occurrence of schistosomes and cercarial dermatitis, such as altered hydrology conditions with water-level fluctuation, ice cover, acidification, or dam constructions (268, 292, 293), anthropogenic pollutants (268, 294–297), biodiversity change in terms of introducing nonindigenous species that may affect endemic parasites (298), host susceptibility or resistance (9), predation upon trematode free-swimming larval stages by fish and other aquatic animals (295, 299, 300), or interspecific competition of parasites within the same snail host (301–303).

Recreational Activities and Cercarial Dermatitis

The data in the literature suggest that lakes represent high-risk areas for cercarial dermatitis, as they are attractive for a large number of people, usually for recreational purposes (8). Cercarial dermatitis develops as a result of sensitization of the human immune system (232, 260), and repeated exposures to cercariae influence the occurrence and intensity of subsequent infections. For example, longer exposure in water increases cercarial penetration via frequent water visits and more time spent in shallow water (4, 304–306). While gender does not influence the risk of infection (4, 304, 305, 307), some studies found a higher risk among children of less than 15 years of age, since they tend to spend more time in shallow water (280, 308, 309). There are rare cases of nonsensitive individuals that may be due to host desensitization (310) or individual nonsusceptibility/nonattractivity to cercariae (4, 311).

Locally, the highest risk of infection usually occurs in shallow, warm, and vegetation-rich shore areas, where the snails accumulate and release cercariae (308, 309). Cercariae of avian schistosomes are concentrated just beneath the water surface (see the information on host finding), so swimming in deeper water may reduce the infection probability (280). However, snail preferences for habitat differ: *Lymnaea stagnalis* is found mostly in patches of aquatic vegetation, feeding on periphyton; *Radix* spp. accumulate in vegetation-free areas, on stones or muddy sediments; and physid snails prefer detritus as a source of food (312, 313). In addition, cercariae of avian schistosomes can be transported by wind and water currents for several kilometers (2, 304, 305). Therefore, taking the local situation into account is essential for risk assessment, because conditions unsuitable for one snail species may provide an ideal habitat for another.

Season and time of day were shown to have considerable effects. Most cases of cercarial dermatitis correlate with high air and water temperatures during the summer months, when schistosome development in snails is amplified and emission rates of cercariae are stimulated by sunlight (4, 11, 279, 280, 304, 305, 309, 314). Production can reach several thousands of cercariae/snail/day (169, 300, 315), and these emission rates may even counterbalance the loss of cercarial capability to survive and infect hosts at high temperatures (see above). Irrespective of the season, outbreaks of cercarial dermatitis have been reported in geothermally heated lakes and ponds in Iceland (71). Time of day proved to be another risk factor. Production of schistosome cercariae is usually discontinuous—particular species

may differ in circadian rhythms of cercarial release. This is usually attributed to the peak activity of the preferred definitive hosts. Thus, *Trichobilharzia* and some other genera infecting aquatic birds leave the snail host during the light period of the day, mainly in the morning hours (2, 4, 280, 305). Increased illumination, temperature, and snail locomotory activity trigger cercarial emergence (169, 211, 316, 317). Patterns of cercarial emergence can be changed almost immediately by reversing the dark-light regimen, suggesting that light has a decisive influence on the direction of the process (317, 318). The number of cercariae released from snails may vary greatly depending on the parasite/host species and the phase (age) of infection; large species of snails represent a higher risk, as more cercariae are produced per snail and released into the environment (10, 275, 315). This may also explain why some species of avian schistosomes that use smaller species of snails are not often detected during an outbreak.

Control Measures Related to the Ecology of Avian Schistosomes

In order to reduce the risk of cercarial dermatitis, either water use during peak cercarial emergence should be avoided or the life cycle of avian schistosomes should be interrupted. Interruption of the schistosome life cycle has been tried on a number of occasions. One option is to reduce the prevalence of adult avian schistosomes in birds by eliminating ducks from high-risk areas. However, public acceptance of regular hunting or repelling of birds (ducks) at recreational lakes during summer months is uncertain. Another option is to treat birds with the antihelminthic drug praziquantel. Birds are captured in the field by use of modified dive traps and subsequently treated with praziquantel and then released. Recapture results showed a significant reduction of infections in mallards (319–321). Furthermore, in the subsequent year, there was a marked decline in prevalence of avian schistosomes in snails (320). However, treatment of the entire duck population is time-consuming and labor-intensive.

Reduction or elimination of snail populations is another strategy of cercarial dermatitis control by interrupting the life cycle. In the past, application of commercial molluscicides, such as niclosamide or copper sulfate, was common (319). However, repeated and unlimited treatments had a deleterious effect on animal communities. In addition, after repeated exposures, some snails may become resistant to copper sulfate or avoid the treatment by burrowing into the mud (322, 323). Manual collection of snails (309, 324) or destruction of their habitats by local removal of littoral vegetation (2) represents a less harmful approach to snail control. Large-scale destruction of snail populations by use of heavy machines along the shores of Annecy Lake (France) and Cultus Lake (Canada) led to substantial reductions of cercarial dermatitis (2). As for the biological control of snail populations, use of molluscivorous fish or prawns as natural snail predators is promising for long-term control (325–328), although it represents a risk for native flora and fauna if nonindigenous organisms are introduced.

Free-swimming miracidia and cercariae may represent a promising target for life cycle interruption. Snail finding and recognition by miracidia are based markedly on recognition of host molecules called miraxones (see the information on intramolluscan development), which led to a proposal to use miracidial traps containing miraxones (329). Unfortunately, due to the high level of diversity of avian schistosomes and presumed variability of snail recognition (139), this proposal proved unrealistic. Similarly, cercarial traps based on species-specific host-finding behavior (see the information on vertebrate host finding) might provide local protection (330). For example, fatty acid-stimulated transformation to the schistosomulum stage could be initiated in these traps, leading to the loss of resistance against a hypo-osmotic water environment (178, 331, 332). As far as trophic interactions of organisms are concerned, predation by small aquatic animals may represent a promising means of biological control. Schistosome miracidia and cercariae may serve as prey for larval aquatic insects, crustaceans, oligochaetes, shrimps, and fish (271, 333–338). In particular, the annelid *Chaetogaster limnaei sensu lato*, living commensally or parasitically on the shell surface or in the mantle and pulmonary cavities of freshwater snails, may act as an efficient predator and prevent penetration of miracidia (339, 340) or feed on cercariae as they are released from the snail (341, 342).

Finally, competitive interactions among trematode larval communities within an individual snail can substantially influence establishment and survival of schistosome intramolluscan stages. In terms of interspecific interactions, particular species of trematodes can be dominant, usually by producing competitively aggressive rediae, or subordinate because they have less aggressive sporocysts and no redial stage. Schistosomes belong to the latter group, and their sporocysts are eliminated via the predatory interactions of redia-producing trematodes, e.g., echinostomes (301, 343). Yet schistosomes may exert a dominant effect in some cases. The avian schistosome *Trichobilharzia brevis* persists in coinfections with two dominant echinostomes, *Echinostoma audyi* and *Hypoderaeum dingeri* (343), and can cause developmental suppression of the latter species. The nonpredatory exclusion of *H. dingeri* by *T. brevis* has also been shown experimentally (344). Intertrematode competition was recently identified as an important determinant in transmission of avian schistosomes. For example, competitive exclusion was estimated to result in an 18.0% reduction of *Trichobilharzia szidati* in *Lymnaea stagnalis* (345). Interestingly, *T. szidati* may represent a subordinate species, yet it frequently cooccurs in snails with other trematodes. Therefore, *T. szidati* may be either an obligate secondary invader of snails with a compromised immune system or a schistosome that can coexist in double infections, as was demonstrated for other species of *Trichobilharzia* and *Austrobilharzia* (302, 343, 346).

PERSPECTIVES

The more we know about avian schistosome diversity and distribution, the better we are positioned to understand their evolutionary history and potential for the future dynamics of dermatitis outbreaks. These days, we have practical means for their identification and can better gauge the likelihood that some of these species may emerge in new contexts to cause unexpected problems. An effort to describe the avian schistosome species diversity in their hosts in the circumpolar regions, where most migratory birds spend the summer (and young birds become infected), seems to be a critical component to understanding the epidemiology of cercarial dermatitis. In addition, the condition itself and the host-parasite interaction require further characterization at the molecular level. At least three examples of future applications can be mentioned. (i) A more detailed knowledge of cercarial penetration mechanisms may help in the prevention of dermatitis by introducing new formulations containing inhibitory molecules. (ii) Characterization of biologically active secretions produced by avian schistosomes may help us to understand the molecular basis of tissue pathology in avian and mammalian hosts.

(iii) Newly designed primers or carefully selected antigens may be developed for reliable antibody- or DNA-based diagnostic tools. Such tools may be used to screen people or animals with health problems following recent water contact to assess the risk associated with exposure to cercariae of avian schistosomes.

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Petr Horák, M.Sc., Ph.D., is a Professor of Parasitology and Head of the Department of Parasitology at the Faculty of Science, Charles University in Prague, Czech Republic. He obtained his parasitology/biology degree from the Charles University in Prague. The President of the Czech Republic appointed Petr Horák as a Professor of Parasitology in 2005. Professor Horák has presented and published on many topics, dealing with parasitic helminths in general and cercarial dermatitis/avian schistosomes in particular. His teaching activities lie in general parasitology and the biology of helminths. His research is focused on functional morphology of helminths, trematodes, and avian schistosomes (systematics, life cycles, and host-parasite interactions). Details of his personal data can be found at http://www.petrhorak.eu.

Libor Mikeš, M.Sc., Ph.D., is an Assistant Professor of Parasitology at the Department of Parasitology, Faculty of Science, Charles University in Prague, from which he received his master's and doctoral degrees. His international experience includes, e.g., stays at the Keele University, United Kingdom, and the Institute for the Biotechnology of Infectious Diseases, UTS, Sydney, Australia. Dr. Mikeš is currently the President of the Czech Society for Parasitology. His basic academic interests lie in the general biology of helminths (with a special focus on the trematode superfamilies Schistosomatoidea and Diplostomoidea). He is also involved in teaching molecular and cellular interactions between parasites and hosts and biochemical parasitology, and he leads several field courses in parasitology. His research and publication activities are focused mainly on the biochemistry of helminths (proteomics, peptidases of trematodes and monogeneans, and glycobiology of larval trematodes) and immune reactions of hosts against avian schistosomes.

Lucie Lichtenbergová, M.Sc., Ph.D., is a Postdoctoral Fellow at the Department of Parasitology, Faculty of Science, Charles University in Prague. She finished her master's study at the Masaryk University in Brno, Czech Republic, and obtained her Ph.D. from the Charles University in Prague, Czech Republic. Since her doctoral study, she has focused on the research of interactions of avian schistosomes and their natural avian and accidental mammalian hosts. Immunology and histopathology are her centers of interest.

Vladimír Skála, M.Sc., is a Ph.D. student at the Department of Parasitology, Faculty of Science, Charles University in Prague, Czech Republic. His master's degree was obtained at the same institution, and his master's thesis focused on the morphology and development of the monostome fluke *Notocotylus attenuatus* (Digenea: Notocotylidae). At present, he is investigating the defensive activity of hemocytes of two lymnaeid snails transmitting avian schistosomes and the immunomodulation caused by trematodes in their snail hosts. His research is carried out in collaboration with Anthony J. Walker from Kingston University, London, United Kingdom. Vladimír has presented the results of his research at several national and international scientific meetings.

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Miroslava Soldánová, M.Sc., Ph.D., received a Ph.D. in parasitology at the Faculty of Science, University of South Bohemia, České Budějovice, Czech Republic, in 2011. Simultaneously, she started as a Research Assistant at the Institute of Parasitology, Biology Centre, Academy of Sciences of the Czech Republic, where she currently continues as a Postdoctoral Fellow in the research team focused on trematode ecology and systematics. Her 1-year postdoctoral fellowship (2012–2013) at the University of Duisburg-Essen (Faculty of Biology, Aquatic Ecology, Germany) focused on the impact of eutrophication on the composition of trematode communities in large lake ecosystems in the Ruhr River, screening of freshwater snails in relation to trematode infection with avian schistosomes, and assessment of potential risks of cercarial dermatitis for humans. Her fields of interest comprise parasite ecology (trematode community structure and organization in freshwater molluscs), environmental parasitology (use of parasite communities in molluscs as bioindicators), and trematode taxonomy.

Sara Vanessa Brant, Ph.D., is an Assistant Research Professor and Senior Collections Manager of the Museum of Southwestern Biology, Division of Parasites, at the University of New Mexico. She obtained her parasitology/biology M.S. and Ph.D. from the University of Nebraska-Lincoln. Her main research focus is the evolutionary and ecological diversity of trematodes, in particular those in the blood fluke families (Schistosomatidae, Spirorchiidae, Sanguinicolidae). Her recent efforts have been funded to address global avian schistosome diversity and their host interactions. She is also involved in efforts to motivate and promote the importance of placing both parasite and host vouchers in permanent museum collections for future studies.