Intestinal schistosomiasis and soil-transmitted helminthiasis in Ugandan schoolchildren: a rapid mapping assessment

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Abstract. Even with a national control programme in place, intestinal schistosomiasis continues to be a major public health problem in school-aged children and other community members in Uganda. This is especially the case in the environments around the Great Lakes, where disease transmission is high, such as Lake Victoria. Moreover, in the most remote areas, some schools might periodically miss large-scale drug administrations owing to inaccessibility. To provide contemporary monitoring and surveillance data, 27 schools along the lakeshore were surveyed with a rapid assessment protocol to determine both prevalence and intensity of Schistosoma mansoni and soil-transmitted helminth infections. In total, 25 (92.6%) of schools were positive for S. mansoni, with an average prevalence across the surveyed children of 42% and average infection intensity of 634 eggs per gram of faeces. Mean prevalence of Trichuris trichiura, Ascaris lumbricoides and hookworm was 12.9%, 9.3% and 2.4%, respectively. Results from questionnaire data revealed a high level of itinerancy among the children, and a total of 38.2% reported to have never received treatment for schistosomiasis, despite 96% living in districts targeted by the national control programme. A birthplace outside of Uganda was a significant predictor for increased risk of schistosomiasis infection (odds ratio (OR) = 9.6), and being resident at a school for less than a year was significantly associated with absence of praziquantel treatment (OR = 0.3). Univariate regression analysis showed a trend of increasing schistosomiasis towards the eastern region of Uganda, while semivariograms of infection prevalence demonstrated a range of spatial autocorrelation of ~78 km. Soil-transmitted helminth infections were more common in the Western region. Our results emphasise how social and demographic variables such as migration may affect epidemiological trends and confound the impact of existing treatment regimes.

Keywords: schistosomiasis, soil-transmitted helminthiasis, rapid epidemiological assessment, geospatial analysis, monitoring and evaluation, Uganda.

Introduction

Intestinal schistosomiasis is caused by the blood fluke Schistosoma mansoni and infections are acquired by contact with freshwater containing parasite larvae. The disease is hyper-endemic in the Great Lakes region of East Africa, owing to the favourable habitat for snails of the Biomphalaria genus, which are the intermediate host (Morgan et al., 2001), and it was first detected in north-west Uganda as early as 1902. Subsequent surveys have revealed the presence of the disease in other parts of the country, including Lake Victoria (Emmanuel and Doering, 2008). Lakeshore communities are typically dependent on water from these lakes for various daily activities, including cooking, bathing and washing clothes. Fishing is usually a major occupation of the men of these communities, further bringing them into contact with the water, and sometimes creating itinerant communities that move to follow productive fishing zones.
Since the 1950s, the Nile perch fishery in Lake Victoria has grown into a multi-million dollar business, and thus provides a lucrative means of earning cash rather than just subsistence. These circumstances dramatically increase infection risk with *S. mansoni* for people living close to the lakeshore (Stothard et al., 2005).

To combat schistosomiasis at the national level, efforts to control the disease are underway in some areas. Chemotherapy with praziquantel (PZQ) is currently the mainstay of control, which is available at a low cost (Doenhoff et al., 2008; Fleming et al., 2009). Uganda has had a national control programme in place since 2003, instigated in partnership with the Schistosomiasis Control Initiative (SCI) (Kabatereine et al., 2006b). This control programme first classified at-risk districts using maps and historical data, focusing on large lakes such as Victoria, Albert and Kyoga. Measures of risk are based on baseline infection surveys, malacological data and, more recently, satellite data which can be combined with other sources to create spatial risk maps in a geographical information system (GIS) software (Brooker et al., 2001; Kabatereine et al., 2004; Stensgaard et al., 2006; Brooker, 2007). Using these data, a pilot control programme of annual large-scale administration of PZQ to all primary schoolchildren within a selected sub-district was then rolled out. Schools were targeted for treatment based on the increased benefits of reducing infection burdens in children compared to adults and also due to the ease of providing treatment (Kabatereine et al., 2007; Zhang et al., 2007). The programme was expanded in 2004 to include treatment with albendazole against common soil-transmitted helminth infections such as hookworm, *Ascaris lumbricoides* and *Trichuris trichiura* (Kabatereine et al., 2006a). The 5-year initial run of the programme has now finished, ending SCI’s initial involvement (Fenwick et al., 2009); however, the government is committed to maintaining the programme, assisted now by new funding from the United States Agency for International Development (USAID; for details, see http://www.rti.org/page.cfm/Neglected_Tropical_Diseases) as well as SCI. Of the Lake Victoria districts of Uganda, Rakai was not included in the national control programme due to levels of prevalence and intensity of infection falling below the World Health Organization (WHO) guidelines for annual or biannual treatment (Kabatereine et al., 2004). Masaka and Mpigi districts were initially also excluded, but their status was revised in 2007 to be included.

Despite these efforts to measure precisely geographical patterns of schistosomiasis risk, many of these variables have large margins of error, and may fall short of accurate “ground-truthing”, the description of the situation as actually observed in the field. For example, if a school is located in a generally low-transmission zone, it may be precluded from regular PZQ administration, despite actually being highly affected by schistosomiasis. As such, traditional monitoring at a local level is vital in ensuring efficiency and maximum cost-effectiveness within the control programme. It is within this context that as control is fully rolled out, further efforts at developing and executing new monitoring schemes are needed.

This paper presents data using a rapid assessment methodology to examine the prevalence and intensity of *S. mansoni* and soil-transmitted helminth infection in schools around the Ugandan perimeter of Lake Victoria. The aim was not only to shed light on the infection status of these schools, and thus simultaneously assess outreach of the national control programme within the six districts examined, but also to investigate some of the factors that might contribute to continued high levels of infection and/or re-infection with these intestinal parasites, such as place of origin, past treatment history and time of residence in a particular community.

**Materials and methods**

The data presented here were collected over a 3-week period in February and March of 2008. The methodology combined the use of short, structured interviews, examination of stool samples for intestinal worms and spatial analysis of collected data.
Study area and design

Twenty-seven schools were selected to form the sample sites for this study, from six districts bordering on Lake Victoria: Rakai, Masaka, Kalangala, Mukono, Mayuge and Busia (Fig. 1). All of these districts except Rakai had been included in the national schistosomiasis control programme. Within each district, the schools were chosen on an ad hoc basis, primarily to prevent selection interference which might be based on a priori performance or knowledge of the school on the part of the district health officer.

Within each school, in accordance with a modified WHO rapid assessment protocols (Montresor et al., 1998; Brooker et al., 2005, 2009), approximately 15 boys were selected by the head teacher from primary school classes 1, 2 and 3. This corresponded roughly to ages 6-10 years. Boys were typically targeted to optimize detection of *S. mansoni* infection in a particular school, as they are more likely to be infected than girls (Kapito-Tembo et al., 2009), but where there were not enough boys to make up the desired 15 children, girls were also accepted for the survey. Each child provided a single stool sample for processing.

Demographic and sociological surveys

Each child was asked five questions, in their local language, concerning their: (i) age; (ii) place of birth; (iii) length of time in the district; (iv) previous treatment with PZQ; and (v) knowledge of schistosomiasis. A copy of the survey sheet is available upon request from the lead author.

Parasitological examination of stool

Each child’s stool sample was used to make two Kato-Katz thick smears (Katz et al., 1972) which were first viewed in the field by trained Vector Control Division technicians and then cross-checked in the UK by the lead author. Egg count tallies for *S. mansoni*, *A. lumbricoides*, *T. trichiura* and
hookworm were made for each thick smear (with the realization that cross-check for hookworm eggs would not be possible). Where discrepancies between each count were found, the slide was re-read a further time, and the average of the three reads taken.

Eggs per gram of faeces (EPG) calculations were based on the average egg counts of both Kato-Katz thick smears and multiplied by 24. In accordance with WHO intensity categories (WHO, 2002) the infection was designated as “light” when EPG was <99, “moderate” if 100-399 and “heavy” if ≥400.

Statistical analysis

All statistical data were tabulated in Excel and analyses executed using version 2.8.0 of the R statistical package (Ihaka and Gentleman, 1996). For all models, adjusted odds ratios (ORs) are given along with 95% confidence intervals (CIs). Statistical significance in all cases was set at less than 0.05 probability.

The geometric means of all the egg counts per school were calculated, as were the arithmetic means of all positive egg counts per school (“1” was added to negative egg counts when calculating geometric means). As the egg count data were not normally distributed, an exact binomial was used to calculate prevalence and 95% CIs per school and per district (Armitage et al., 2001). A Fisher’s 2-way exact test was used to look for significant differences in prevalence of each school from the overall prevalence, and the significance of each school’s mean egg count values from those of the total was determined by comparing each 95% CI with those of the total. Any overlap of 95% CIs when comparing means was indicative of not a significant difference in value.

Univariate regression models were used to search for relationships within the data. The models were run with fixed effect parameters, and again, where appropriate, with a random effects model taking into account the schools as a confounding factor. The spatial autocorrelation of the data points was examined using omnidirectional and directional experimental semivariograms, also calculated in R. Covariance parameters were estimated using a parametric model and fitted with weighted least squares to compare between different experimentally-derived semivariograms for best fit.

The data were mapped for preliminary analysis using ArcGIS software, version 9.2 (ESRI, Redlands, CA, USA). Maps were redrawn using a Bamboo© writing tablet (Wacom, Saitama, Japan).

Informed consent and ethical considerations

Ethical approval was granted by National Health System Local Research Ethics Committee at St. Mary’s Hospital in London, and the Uganda Ministry of Health in Kampala. The head teacher at the school was asked for oral informed consent on behalf of the students to undertake the surveys and sample collection, and a teacher remained in the room with the pupils at all times. Each participating child was treated with PZQ and albendazole upon completion of the survey.

Results

Infection with S. mansoni

Prevalence of S. mansoni infection, geometric mean of the overall EPGs and the arithmetic mean EPG for infected children were calculated per school/site and overall, for each the Western and Eastern regions (Tables 1 and 2, respectively). Total mean prevalence was 41.7% (95% CI = 37.1-46.3%), geometric mean of all EPG values was 7.30 (95% CI = 7.17-7.44) and the arithmetic mean of infected cases was 634.3 EPG (95% CI = 466.3-802.3). At the unit of the school, there were six schools with significantly higher overall prevalence, 10 schools with significantly higher geometric or arithmetic mean EPGs, five schools with lower prevalence and 19 schools with one or both of its mean egg counts lower than the total average. Four
Table 1. Prevalence and infection intensities of *S. mansoni* in schools in the Western three districts of Uganda surveyed: Masaka, Kalangala and Rakai. Geometric means were calculated for all egg counts, whereas arithmetic means were calculated from positive counts only.

| School (Map ID)  | District | No. examined | No. positive | Prevalence in % (95% CI) | Geometric mean (95% CI) | Arithmetic mean (95% CI) |
|------------------|----------|--------------|--------------|--------------------------|-------------------------|--------------------------|
| Bukakata (2)     | Masaka   | 15           | 6            | 40.0 (16.3-67.7)          | 7.31 (5.99-8.64)        | 130.0§ (33.7-226.3)      |
| S0.273267, E32.026517 |          |              |              |                          |                         |                          |
| Bridge of Hope (9) | Kalangala | 18           | 5            | 27.8 (9.7-53.5)           | 2.14§ (1.18-3.10)       | 230.4 (0.0-595.4)        |
| S0.322220, E32.284466 |          |              |              |                          |                         |                          |
| Bubeke (13)      | Kalangala | 18           | 3            | 16.7 (3.6-41.4)           | 0.65§ (0.10-1.20)       | 24.0§ (0.5-47.5)         |
| S0.320250, E32.357500 |          |              |              |                          |                         |                          |
| Bugoma (3)       | Kalangala | 16           | 9            | 56.3 (29.9-80.2)          | 10.23* (9.06-11.41)     | 148.0§ (39.7-256.3)      |
| S0.257183, E32.066383 |          |              |              |                          |                         |                          |
| Bunyama (10)     | Kalangala | 13           | 4            | 30.7 (9.1-61.4)           | 2.34§ (1.30-3.37)       | 54.0§ (29.5-78.5)        |
| S0.363533, E32.294883 |          |              |              |                          |                         |                          |
| Buyange (11)     | Kalangala | 13           | 0            | 0.0§ (0.0-24.7)           | 0.00§ (0.00-0.00)       | NA                      |
| S0.351583, E32.3571533 |         |              |              |                          |                         |                          |
| Jaana (12)       | Kalangala | 16           | 6            | 37.5 (15.2-64.6)          | 5.35§ (4.06-6.63)       | 328.0 (0.0-664.7)        |
| S0.233800, E32.3575083 |        |              |              |                          |                         |                          |
| Kazira (5)       | Kalangala | 19           | 2            | 10.5§ (1.3-33.1)          | 0.45§ (0.00-0.96)       | 36.0§ (12.5-59.5)        |
| S0.322849, E32.193616 |          |              |              |                          |                         |                          |
| Kibanga (8)      | Kalangala | 16           | 5            | 31.3 (11.0-58.7)          | 2.52§ (1.56-3.49)       | 72.0§ (20.5-123.5)       |
| S0.323950, E32.284250 |          |              |              |                          |                         |                          |
| Mulabana (6)     | Kalangala | 17           | 9            | 52.9 (27.8-77.0)          | 6.41 (5.37-7.44)        | 118.7§ (0.0-255.3)       |
| S0.438650, E32.227900 |          |              |              |                          |                         |                          |
| St Kizito (4)    | Kalangala | 18           | 4            | 22.2 (6.4-47.6)           | 1.20§ (0.48-1.92)       | 48.0§ (0.0-96.0)         |
| S0.300666, E32.141233 |          |              |              |                          |                         |                          |
| St Therza (7)    | Kalangala | 18           | 5            | 27.8 (9.7-53.5)           | 2.43§ (1.48-3.37)       | 86.4§ (62.4-110.4)       |
| S0.417300, E32.228283 |          |              |              |                          |                         |                          |
| Goma (1)         | Rakai    | 18           | 1            | 5.5§ (0.1-27.3)           | 0.26§ (0.00-0.70)       | 60.0§ (only 1 inf.)      |
| S0.915335, E31.767300 |          |              |              |                          |                         |                          |
| Total for Western region | - | 232       | 67           | 28.9§ (23.1-35.2)         | 2.47 (2.20-2.73)        | 180.5§ (100.5-260.6)     |
| Overall total    | -        | 456          | 190          | 41.7 (37.1-46.3)          | 7.30 (7.17-7.44)        | 634.3 (466.3-802.3)      |

§ = values of intensity or prevalence of infection which are significantly lower than for all of the schools combined; * = values of intensity or prevalence of infection which are significantly higher than for all of the schools combined.
Table 2. Prevalence and infection intensities of *S. mansoni* in schools in the Eastern three districts of Uganda surveyed: Busia, Mayuge and Mukono. Geometric means were calculated for all egg counts, whereas arithmetic means were calculated from positive counts only.

| School (Map ID) | GPS coordinates | District | No. examined | No. positive | Prevalence in % (95% CI) | Geometric mean (95% CI) | Arithmetic mean (95% CI) |
|----------------|----------------|----------|--------------|--------------|--------------------------|-------------------------|--------------------------|
| Maduwa (27)    | N0.253067, E33.988933 | Busia    | 18           | 2            | 11.1§ (1.4-34.7)         | 0.68§ (0.00-1.41)        | 234.0 (0.0-645.6)         |
| Majanji (26)   | N0.263167, E33.984750 | Busia    | 17           | 0            | 0.0§ (0.0-19.5)          | 0.00§ (0.00-0.00)        | NA                       |
| Bugoto (23)    | N0.318117, E33.627033 | Mayuge   | 17           | 10           | 58.8 (32.9-81.6)         | 10.56* (9.46-11.68)      | 121.2§ (38.6-203.8)       |
| Bumba (24)     | N0.031083, E33.644650 | Mayuge   | 18           | 13           | 72.2* (46.5-90.3)        | 29.80* (28.68-30.92)     | 228.9§ (68.6-389.2)       |
| Jagusi (20)    | N0.156183, E33.566017 | Mayuge   | 18           | 4            | 22.2 (6.4-47.6)          | 1.52§ (0.70-2.35)        | 66.0§ (43.5-88.5)         |
| Kaaza (21)     | N0.111767, E33.602150 | Mayuge   | 17           | 10           | 58.9 (32.9-81.6)         | 18.96* (17.58-20.33)     | 488.4 (63.7-913.1)        |
| Sagitu (25)    | N0.003033, E33.658600 | Mayuge   | 17           | 14           | 82.4* (56.6-96.2)        | 84.96* (83.80-86.13)     | 456.0 (178.4-733.6)       |
| Serinyabi (22) | N0.052350, E33.605833 | Mayuge   | 17           | 12           | 70.6* (44.0-89.7)        | 31.63* (30.47-32.80)     | 214.0§ (94.7-333.3)       |
| Busagazi (16)  | N0.240300, E33.137083 | Mukono   | 17           | 9            | 52.9 (27.8-77.0)         | 7.65 (6.20-9.11)         | 406.0 (40.0-772.0)        |
| Kasimizi (19)  | N0.185583, E33.215217 | Mukono   | 18           | 18           | 100.0* (81.5-100.0)      | 1337.43* (1336.81-1338.04) | 2165.7* (1430.6-2900.7)   |
| Kimi (14)      | S0.086383, E32.652200 | Mukono   | 18           | 8            | 44.4 (21.5-69.2)         | 9.94* (8.58-11.30)       | 549.0 (61.1-1036.9)       |
| Kisu (15)      | N0.014767, E32.767167 | Mukono   | 16           | 14           | 87.5* (61.7-98.4)        | 309.89* (308.49-311.28)  | 1842.0* (843.9-2840.1)    |
| Makonge (17)   | N0.270217, E33.205883 | Mukono   | 16           | 5            | 31.3 (11.0-58.7)         | 5.86 (4.40-7.31)         | 552.0 (230.5-873.5)       |
| Namatale (18)  | N0.173200, E33.183967 | Mukono   | 17           | 12           | 70.6* (44.0-89.7)        | 102.16* (100.50-103.83)  | 1698.6 (384.4-3012.7)     |
| **Total for Western region** | - | - | 224 | 123 | **54.9§ (48.1-61.5)** | **19.79§ (19.38-20.20)** | **891.9 (643.8-1139.9)** |
| **Overall total** | - | - | 456 | 190 | **41.7 (37.1-46.3)** | **7.30 (7.17-7.44)** | **634.3 (466.3-802.3)** |

§ = values of intensity or prevalence of infection which are significantly lower than for all of the schools combined; * = values of intensity or prevalence of infection which are significantly higher than for all of the schools combined.
schools had mixtures of significantly higher and lower results, when compared to the total sample. These data were then used to map the prevalences and mean arithmetic intensity of infection together onto the locations of each of the schools (Fig. 2). Dividing the geographic range of the surveyed schools into “Eastern” and “Western” regions based on longitude, the districts of Rakai, Kalangala and Masaka were considered “Western”, whereas Mukono, Mayuge and Busia were “Eastern”. The Western districts had, on average, significantly lower prevalence, geometric mean EPGs and arithmetic mean of positive egg counts compared to the overall total, whereas the Eastern districts had significantly higher prevalence and geometric EPG. Geometric means of EPGs for each school were also plotted against prevalence, showing an asymptotic relationship (Fig. 3). Place of birth data was also included in this analysis (see next section for further demographic results).

Fig. 2. Summary map denoting the approximate locations of each school surveyed, and the prevalence and intensity of S. mansoni infection within each sampled school. It is visually apparent that both infection prevalence and intensity of intestinal schistosomiasis is not uniform across the surveyed area.

Fig. 3. Bubble graph showing the prevalence of S. mansoni infection at each school against the respective geometric mean EPG. The area of each bubble represents the proportion of children who attend school in their district of birth.
Infections with soil-transmitted helminths

At a district level, soil-transmitted helminths were also widespread, but showed strong heterogeneity (Table 3). Four out of six districts (66.7%) were negative for hookworm, and the overall prevalence was low (2.4%; 95% CI = 1.2-4.3%). Only one district (Busia) was negative for both *A. lumbricoides* and *T. trichiura*, and average prevalence in the other districts was 9.3% (95% CI = 6.8-12.4%) and 12.9% (95% CI = 9.9-16.3%), respectively. Soil-transmitted helminth infections, stratified by different infection intensity classes, are presented in Table 3.

Table 3. Prevalence and intensity of infection with hookworm, *A. lumbricoides* and *T. trichiura*, by district.

| School (Map ID) | % (95% CI) | Light | Mod. | Heavy | % (95% CI) | Light | Mod. | Heavy | % (95% CI) | Light | Mod. | Heavy |
|----------------|------------|-------|------|-------|------------|-------|------|-------|------------|-------|------|-------|
| Busia          | 0.0 (0.0-10.0) | 0   | 0   | 0     | 0.0 (0.0-10.0) | 0   | 0   | 0     | 0.0 (0.0-10.0) | 0   | 0   | 0     |
| Kalangala      | 3.4 (1.2-7.2) | 5   | 0   | 1     | 11.2 (7.0-16.7) | 17  | 3   | 0     | 11.2 (7.0-16.7) | 19  | 1   | 0     |
| Masaka         | 0.0 (0.0-21.8) | 0   | 0   | 0     | 20.0 (4.3-48.1) | 3   | 0   | 0     | 33.3 (11.8-61.6) | 5   | 0   | 0     |
| Mayuge         | 0.0 (0.0-3.5) | 0   | 0   | 0     | 3.8 (1.1-9.6) | 3   | 1   | 0     | 10.6 (5.4-18.1) | 8   | 3   | 0     |
| Mukono         | 0.0 (0.0-3.7) | 0   | 0   | 0     | 9.1 (4.2-16.6) | 9   | 0   | 0     | 12.1 (6.4-20.2) | 12  | 0   | 0     |
| Rakai          | 27.8* (9.7-53.5) | 5   | 0   | 0     | 33.3* (13.3-59.0) | 5   | 1   | 0     | 55.6* (30.8-78.5) | 8   | 2   | 0     |
| **Total**      | 2.4 (1.2-4.3) | 10  | 0   | 1     | 9.3 (6.8-12.4) | 37  | 5   | 0     | 12.9 (9.9-16.3) | 52  | 6   | 0     |

*= values of intensity or prevalence of infection which are significantly higher than for all of the schools combined.

Table 4. Variation in survey responses to demographic and treatment history questions, by district.

| District (N) | District of birth (%) | Length of stay (%) |
|--------------|-----------------------|--------------------|
|              | This | Other L.V. | Other Uganda | KE / TZ | <1 year | 1-3 yrs | < whole life |
| Busia (36)   | 91.7 | 0.0   | 8.3   | 0.0   | 2.8   | 2.8    | 2.8   | 91.7 |
| Kalangala (189) | 40.8  | 43.9  | 14.8  | 0.5   | 15.7  | 0.8    | 21.5  | 62.0 |
| Masaka (14)  | 57.1 | 28.6  | 14.3  | 0.0   | 0.0   | 27.3   | 18.2  | 54.5 |
| Mayuge (105) | 44.8 | 23.8  | 20.0  | 11.4  | 18.6  | 10.8   | 22.5  | 48.0 |
| Mukono (92)  | 59.8 | 9.8   | 28.2  | 2.2   | 4.4   | 15.4   | 14.3  | 57.1 |
| Rakai (18)   | 66.7 | 27.8  | 5.5   | 0.0   | 5.6   | 11.1   | 27.8  | 55.6 |
| **Total (454)** | 51.1 | 27.8  | 17.8  | 3.3   | 5.5   | 17.9   | 15.0  | 59.4 |

L.V. = Lake Victoria; TZ = Tanzania; KE = Kenya.
lake. Similarly, length of residence in a particular area varied considerably, with 23.4% of children having only lived in that district for less than 3 years, and 5.5% less than 1 year (Table 4). Treatment history also varied widely across the six districts surveyed, from 0% to almost 100% (Fig. 4); overall, 61.8% (95% CI = 57.1-66.2%) of children reported having received PZQ at some point in their lives. Treatment history was closely correlated with knowledge of the disease (Fig. 4, R² = 0.88).

Univariate regression modeling

Univariate fixed effect models were used to test for predictors of infection status (Table 5). For schistosomiasis, sex and age were not significant predictors of infection status, and nor was previous treatment history. Significant risk factors were place of birth outside Uganda (OR = 9.6; 95% CI = 2.1-43.7; P = 0.003) compared to baseline of local birth, and region of current residence; children in the Western region were only a third as likely to be infected with schistosomiasis compared to children in the Eastern region (OR = 0.33; 95% CI = 0.23-0.49; P <0.001). Soil-transmitted helminth infections showed a reverse relationship for this factor, with an overall increased likelihood of infection in children from the Eastern region (OR = 2.85; 95% CI = 1.75-4.64; P <0.001). An infection was also negatively associated with treatment with PZQ (OR = 0.49; 95% CI = 0.31-0.78; P = 0.003).

A random effects model controlling for variation

Table 5. Results of univariate fixed model logistical regression analyses of infection with *S. mansoni* and/or soil-transmitted helminths.

| Variable (Baseline; Factor) | *S. mansoni* | *A. lumbricoides* | *T. trichiura* | Soil-transmitted helminths overall |
|-----------------------------|--------------|-------------------|---------------|---------------------------------|
| Infection with *S. mansoni* (No; Yes) | NA | NA | 1.20 (0.64-2.28) | 0.569 | 1.51 (0.87-2.63) | 0.142 | 1.46 (0.93-2.30) | 0.10 |
| Place of Birth (Local) | | | | | | | | |
| Other Lake district | 0.74 (0.46-1.18) | 0.208 | 1.19 (0.54-2.64) | 0.664 | 0.66 (0.32-1.37) | 0.261 | 0.86 (0.49-1.52) | 0.608 |
| Other Uganda district | 1.30 (0.76-2.20) | 0.336 | 1.36 (0.56-3.28) | 0.498 | 1.07 (0.51-2.27) | 0.850 | 1.08 (0.57-2.02) | 0.818 |
| Other country | 9.60 (2.11-43.68) | 0.003 | 2.93 (0.75-11.39) | 0.121 | 3.22 (1.03-10.11) | 0.044 | 2.53 (0.86-7.49) | 0.093 |
| Length of residence (Whole life) | | | | | | | | |
| <1 year | 0.97 (0.39-2.44) | 0.951 | 1.19 (0.54-2.63) | 0.664 | 0.31 (0.04-2.43) | 0.267 | 0.74 (0.21-2.62) | 0.635 |
| 1-3 years | 1.67 (0.97-2.90) | 0.066 | 1.36 (0.56-3.28) | 0.498 | 0.97 (0.44-2.18) | 0.949 | 1.75 (0.93-3.29) | 0.085 |
| 3 years - <whole life | 1.70 (0.94-3.06) | 0.081 | 2.93 (0.75-11.39) | 0.121 | 2.09 (1.02-4.30) | 0.045 | 1.76 (0.90-3.47) | 0.100 |
| PZQ treatment (No; Yes) | 1.21 (0.82-1.79) | 0.347 | 0.30 (0.15-0.60) | <0.001 | 0.72 (0.41-1.26) | 0.245 | 0.49 (0.31-0.78) | 0.003 |
| Sex (Male; Female) | 1.16 (0.57-2.37) | 0.687 | 4.24 (1.89-9.51) | <0.001 | 1.30 (0.52-3.26) | 0.578 | 2.09 (1.03-4.27) | 0.042 |
| Age (continuous; +1) | 1.02 (0.95-1.08) | 0.632 | 0.95 (0.85-1.06) | 0.345 | 1.06 (0.97-1.16) | 0.194 | 1.03 (0.96-1.11) | 0.446 |
| Region (East; West) | 0.33 (0.23-0.49) | <0.001 | 2.63 (1.31-5.27) | 0.007 | 1.84 (1.04-3.25) | 0.037 | 2.85 (1.75-4.64) | <0.001 |
Table 6. Results of univariate fixed model logistical regression analyses of incidence of treatment with praziquantel (PZQ).

| Variable                        | Treatment with PZQ | OR (95% CI) | P-value |
|---------------------------------|--------------------|-------------|---------|
| Place of Birth (Local)          | 0.7 (0.5-1.2)      | 0.192       |
| Other Lake district             | 0.5 (0.3-0.9)      | 0.015       |
| Other Uganda district           | 2.0 (0.5-7.3)      | 0.294       |
| Length of residence (Whole life)| 0.3 (0.1-0.7)      | 0.006       |
| <1 year                         | 0.6 (0.3-1.1)      | 0.092       |
| 1-3 years                       | 1.2 (0.6-2.4)      | 0.522       |
| 3 years - < whole life          |                    |             |
| Knowledge of bilharzia (No; Yes)| 25.3 (15.2-41.8)   | <0.001      |
| Age (continuous; +1)            | 1.2 (1.1-1.3)      | <0.001      |
| Region (East; West)             | 0.6 (0.4-0.8)      | 0.004       |

Table 6. Results of univariate fixed model logistical regression analyses of incidence of treatment with praziquantel (PZQ).

at the school level was added to the univariate analysis for factors which were considered independent of school, normally distributed and not highly skewed (age, length of residence and region). This model revealed increased age as a predictor for an infection with *A. lumbricoides* (OR = 0.91; 95% CI = 0.84-0.99; P = 0.037), and an *S. mansoni* infection as a predictor for soil-transmitted helminths (OR = 1.79; 95% CI = 1.07-2.97; P = 0.027) but no other changes in significance occurred.

Treatment was also tested for covariates among the data (Table 6). The model statistically confirmed the significance of knowledge of the disease as a correlate of PZQ treatment (OR = 25.3; 95% CI = 15.2-41.8; P < 0.001). It also showed that children who had been resident in an area for less than 1 year were significantly more likely not to have ever received treatment (OR = 0.3; 95% CI = 0.1-0.7; P = 0.006). Children in the Eastern region were more likely overall to have received treatment compared to in the Western region (OR = 0.6; 95% CI = 0.4-0.8; P = 0.004).

Spatial analysis

A number of experimentally derived variograms were calculated, their lines tested for best fit to the data and two of best models (Figs. 5a and 5b) selected. The first (Fig. 5a) was omnidirectional, 10 bin and fitted with a “sphere” correlation model, and the other (Fig. 5b) was directional for 60°, and also with a “sphere” correlation model.

The omnidirectional and 60° model showed spatial autocorrelation existing within the data up to a range of 0.7 and 0.8 decimal degrees, respectively. As the study region falls directly across the equator, this corresponds to spatial autocorrelation of between 77.9 and 89.1 km.
Discussion

This survey has found that intestinal schistosomiasis is still ubiquitous around the Lake Victorian shoreline of Uganda, with prevalence and intensity of infection significantly higher in the Eastern region. Eleven schools, which were in districts covered by large-scale administration of PZQ, had prevalences of over 50%, considered “high prevalence” by WHO (WHO, 2002), and infected cases were on average classified as “heavy” according to WHO guidelines (WHO, 2002). It should be noted, however, that measures of intensity based on single stools have been shown to vary from day-to-day as well as within samples, with the result that often prevalence is underestimated and intensity overestimated (Kongs et al., 2001; Utzinger et al., 2001; Mutapi et al., 2003; Bergquist et al., 2009). Other rapid diagnostic methods, such as SEA-ELISA tests or circulating cathodic antigen (CCA) tests, may prove useful as an additional tool in rapid assessment surveys (Stothard, 2009; Stothard et al., 2009). The graph of geometric mean of EPGs against prevalence for each school showed a negative binomial (asymptotic) shape, which is consistent with expected observations indicating overdispersion of heavy infection in a few individuals (Anderson and May, 1991).

The spatial analysis of our data showed two main spatial trends. First, regionally, there was a significant difference in the prevalence of S. mansoni between the Eastern and Western regions of the lakeshore. The high prevalence and intensity of infection found in the Eastern region corroborates with previous schistosomiasis surveys, such as those undertaken by Stothard et al. (2005) at the inception of the national control programme. Second, locally there appeared to be evidence for spatial autocorrelation at roughly a 75-85 km scale. This was an unexpected finding, as although climatic conditions are influential at that kind of scale, micro-distribution of suitable snail habitats would suggest that autocorrelation, if present at all, would operate on a much smaller spatial scale. Previous work on the spatial distribution of S. mansoni has shown limits of autocorrelation that are much lower, which perhaps reflect snail micro-distribution, or perhaps socio-economic factors that also operate on a small scale (Xu et al., 2004; Raso et al., 2005). The error margins on the semivariograms should also be considered; a larger sample size of schools would be recommended in future surveys to add resolution to these data.

Most noticeably, the results of the survey showed high levels of migration throughout the Lake Victoria region of Uganda, and thus may influence the geographical patterns observed. This aspect of human behaviour is often overlooked in parasitological surveys, which are more concerned with the current state of infection in a particular locality, although migration is commonly cited as a significant factor in the spread of other infectious diseases (Lurie et al., 2003; Weiss and McMichael, 2004). The implications of these findings are manifold. First, although the low sample size (n = 15) weakens the finding, the significantly higher chance of children born outside Uganda being infected with S. mansoni could hint at a major obstacle facing the success of a national control programme emphasizing PZQ treatment; if children are coming in from countries without large-scale drug administration, they could provide a continuous source of parasite population in the communities to which they move. They, along with in-country migrants, may also be at higher risk of missing annual treatments, through frequent changes in school. This hypothesis is strengthened by the finding that children who had been resident in a location for less than a year were significantly less likely to have ever received treatment, and the P-value for the group who had been resident between one and three years was also approaching significance. Frequent movement may also act as a mixing effect among communities, thus contributing to the spatial autocorrelation shown in the data. To emphasise this point, a follow-up survey at two schools (Kisu and Kimi), conducted 10 months after the initial field work, only succeeded in re-finding two of the 34 children pre-
viously surveyed at these sites, demonstrating once again the high level of itinerancy among lakeshore communities.

In this study, treatment history was not found to be significantly associated with reduced prevalence of schistosomiasis, potentially due to a number of reasons. First, perhaps annual treatment is not enough, and in high transmission zones, biannual treatment may be necessary. It is also worth recalling that due to low perceived thresholds of prevalence and intensity of *S. mansoni*, Rakai has not been included in Uganda’s national treatment programme for large-scale drug administration in schools, and Masaka only included as of 2007. This may partially account for the findings of this survey of lower treatment incidence in the Western region. Second, our findings suggest that perhaps coverage across districts is not as thorough as it could be, and that certain isolated or difficult-to-access schools are being missed by treatment interventions. The study was designed specifically to target schools on an *ad hoc* basis as an “ambush” strategy, to prevent visiting only schools which are commonly surveyed or well-known by local health officers. By doing so, some of the more overlooked schools may have been sampled here, which correspondingly had lower incidences of treatment, irrespective of their prevalence levels. Third, other factors may be involved which were not considered in this survey, such as differences in socio-economic status, health education, local access to health centres and water contact behaviours (Kloos, 1995; Akogun and Akogun, 1996; Kloos et al., 1997; Raso et al., 2005). One factor which was investigated and proved significant in some cases was place of birth, which should be explored further, perhaps with a targeted survey focusing on the Eastern region and aiming to compare schistosomiasis prevalence and intensity in local schoolchildren with migrants. The finding that recent migrants are less likely to have received treatment immediately suggests a potentially improvement to the treatment methodology; in areas of high community motility, schools could be encouraged to treat all new registrants upon starting at that school, to reduce the chance of missing treatment if that child is going to move again. This might also benefit children who register at a school, but subsequently fail to attend regularly. Finally, the local geographical heterogeneities seen in the data suggest that district-wide or even sub-county level analyses are too broad for effective and efficient targeting of schistosomiasis control in schoolchildren, and thus more detailed observation methods are required for fine-scale reporting and monitoring. Rapid assessment protocols, such as that used in this study, are effective for covering numbers of schools in a short amount of time, and using limited resources, and thus are ideally suited for this level of coverage (Brooker et al., 2009).

The prevalence of soil-transmitted helminth infections were much lower than those of *S. mansoni* overall, and were also geographically distributed differently. The distribution of soil-transmitted helminth infections went exactly opposite to that of *S. mansoni*, with significantly lower prevalences of infection in the Eastern region as compared to the Western region, although this effect was not seen at the district level. The regional scale corresponds to previous studies which also indicated a higher prevalence of certain *A. lumbricoides* in the south-west of Uganda (Kabatereine et al., 2005). This study also found a high incidence of soil-transmitted helminths on islands in Lake Victoria, known to be hot-spots for transmission but which are difficult to reach from the mainland by drug administration programmes due to the prohibitive logistical and access costs. The implications of these findings are that monitoring at different scales could produce different conclusions as to the significance of a particular parasite in a given community, district or region (Brooker et al., 2009), and perhaps lend further weight to calls to increase funds for mobilisation of treatment to less readily accessible communities.

Although treatment with albendazole was not specifically surveyed for in this study, it can be reasonably assumed to be closely correlated with PZQ administration, as the two were combined in the national control programme in 2003. As such, it was
encouraging to find a strong positive relationship between history of PZQ treatment and lack of soil-transmitted helminth infection, as this could be seen as an indication of the success of treatment interventions. However, treatment levels were lower in the Western region of the country, which is also where soil-transmitted helminths showed the highest prevalence; these areas should become of greater importance for future treatment interventions using albendazole.

In conclusion, it has been shown that this rapid epidemiological assessment has been effective in mapping variation in the distribution of *S. mansoni* and soil-transmitted helminths and revealed some potentially unique demographic trends at a local level. However, inter-site heterogeneity demonstrated that it may not be viable to extrapolate local findings to the district level. Similarly, distributions of schistosomiasis and soil-transmitted helminthiasis may not follow the same patterns for distributions, complicating efforts at delivering efficient coordinated treatment for both infections simultaneously; this has implications for the way in which national treatment programmes should target risk areas for future anthelminthic drug administration. Furthermore, high levels of movement of some lakeshore communities may confound attempts at complete treatment coverage. Further study, explicitly considering migration as a risk factor and possibly comparing its value as a predictor for risk across different regions of East Africa, should be a priority on the research agenda.

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