ABO blood groups do not predict Schistosoma mansoni infection profiles in highly endemic villages of Uganda

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Introduction

- Schistosomiasis is a debilitating neglected tropical disease with over 240 million people infected globally. Despite over a decade of mass drug administration with praziquantel, hyperendemic hotspots persist.
- severity⁽¹⁾ and host blood group could be one of many elements leading to schistosome hotspot areas.
- Human blood groups include A, B, AB and O and either Rhesus positive (Rh+) or Rhesus negative (RH-).
- A and/or B antigens are thought to be advantageous in conferring resistance to different diseases because epitope sequences are more likely to be detected by the host immune system^(2, 3).
- The aim of this study was to quantify the effect of host blood type on *S. mansoni* infection dynamics over time and with praziquantel treatment

Methods

- A total of 630 school children (6 to 14 years old) were recruited from three primary schools in Mayuge District, Uganda.
- Three days of duplicate Kato-Katz thick smears were conducted at three timepoints in each school (Figure 1).



Figure 1: Timeline of Kato-Katz thick smear sampling (three days of duplicate Kato-Katz at each time point) and praziquantel treatment in the three study schools. Triangles indicate treatment administration to all sampled children after stool samples were collected and circles denote treatment administered to infected children only after sampling.

- Finger-prick blood samples were each spotted onto three glass slides.
- A single drop of anti-A and anti-B monoclonal sera was mixed with the blood on the respective A and B slides to determine blood group.
- An anti-D blend was dropped onto the D slides and mixed with a stir stick to detect Rhesus (Rh) factor.
- Each slide was examined for agglutination of blood by trained technicians (Figure 2).
- GLMMs were performed to assess the association between blood group, age, sex, body mass index (BMI), and S. mansoni infection intensity and prevalence over time.



Figure 2: Example of finger prick blood displaying agglutination for A+ blood group.

• Numerous associations have been described between host blood type and disease susceptibility or





Figure 3: S. mansoni infection prevalence, split by host blood type, pre-treatment, six months post treatment and three weeks after re-treatment. Blood groups are denoted by colour.

Discussion

- contradictory results.

- prevention measures for *S. mansoni*.

systematic review and meta-analysis. Journal of helminthology:1-10.

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Results

• The most commonly occurring blood group was O (49% in Bwondha, 39% in Bugoto and 39% in Musubi). • There was no significant difference between blood groups proportions between the schools (P=0.390). • Type AB occurred significantly less often than all other groups with 3%, 6%, and 1% in Bwondha, Bugoto and Musubi respectively.

• Only 4.8% of individuals from all three schools were Rh-. No individuals were Rh- in Musubi. • There was no significant association between blood group and *S. mansoni* prevalence (*P*=0.811). • Significant variation in prevalence occurred between the schools at each time point (P<0.001) (Figure 3). Infection intensity varied between schools at baseline and each follow up (P<0.001). • There was no association between blood group and *S. mansoni* infection intensity (*P*=0.818) (Figure 4).



Figure 4: Modelled outcome of GLMM showing no significant association between blood group and *S. mansoni* infection intensity but significant differences exist between the schools at each time point. Lines illustrate modelled effect and dots indicate raw data points.

• Research looking specifically at the influence of host blood type on S. mansoni, has previously focused on only individual timepoints and produced

• The results of this study showed no association between blood type and *S. mansoni* infection prevalence or intensity, clearance nor reinfection rates. • Most publications investigating the influence of host blood type on S. mansoni used the Kato-Katz method and only a single stool sample to determine S. mansoni infections, limiting determination of subtle host blood group effects on S. mansoni infection dynamics. • Our fully powered study indicates no association between host blood group and S. mansoni prevalence, intensity, clearance nor reinfection rate in these highly endemic Ugandan villages and that it is unlikely to be an important factor in maintaining transmission and disease in these communities. • The relationship between the school an individual attended and age, had a significant impact on infection profiles. • Continued work investigating individual and age-related immunity may be imperative to advancing current knowledge to improve treatment and

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