

## b Urogenital schistosomiasis and HIV infection

Schistosomiasis is a parasitic infection of humans caused by *Schistosoma mansoni* and *S. haematobium*. Approximately 90% of the worldwide burden of schistosomiasis is found in Africa. Both species of *Schistosoma* occur in South Africa, however *S. haematobium* is more widely distributed and more prevalent. About 4 million South African children are estimated to be at risk, but the number of people infected is not known.

Urogenital schistosomiasis is a plausible risk factor for HIV acquisition and transmission in both sexes, and could enhance HIV disease progression. The biological basis for this hypothesis lies in the local mucosal disruption and inflammation brought about by urogenital schistosomiasis, and immunological mechanisms that hasten progression of HIV disease. Local mucosal disruption occurs through chronic inflammation in the tissue of the pelvic organs including the urinary bladder, lower ureters, cervix, vagina, prostate, and seminal vesicles. In females, there is damage to the epithelium with mucosa oedema, erosions, and ulcerations. The schistosome eggs elicit a local immune response, with accumulation of inflammatory cells that express CD4+ T-cell receptors, similar to sexually transmitted infections that lead to genital ulceration (syphilis and herpes simplex virus). Chronic schistosomiasis alters immune function and may increase susceptibility to HIV. Schistosomiasis results in preferential stimulation of Th2-type response, and CD4+ T-cells with this phenotype are more susceptible to infection and destruction by HIV. During infection with *Schistosoma* species, there is concomitant downregulation of the Th1-type response, important in initial control of HIV infection. In *S. mansoni* infections, monocytes and CD4+ T-cells have also been shown to display high densities of chemokine co-receptors for HIV, and these levels decreased after praziquantel treatment. Schistoso-

miasis raises viral loads as the upregulated chemokine co-receptors also promote cell-to-cell spread of HIV after initial infection. Praziquantel treatment may therefore slow progression of HIV disease.

In addition to biological evidence, epidemiological and treatment studies also suggest a relationship between schistosomiasis and HIV. Studies in Zimbabwe and Tanzania (>1000 subjects in total) showed significant associations between the diseases. Effect of praziquantel treatment of *S. mansoni* showed variable effects on HIV viral loads, but these studies were mostly observational, not controlled trials with control groups and randomisation. One randomised trial of praziquantel treatment of *S. mansoni* in HIV-positive subjects showed smaller increases in viral load compared to those in whom treatment was delayed, but this study was not blinded, so follow-up bias was possible. Appropriate longitudinal studies involving anti-schistosomal treatment integrated with HIV prevention interventions are required to confirm a causal relationship.

### Further reading

Kjetland EF, Ndhlovu PD, Gomo E, *et al.* Association between genital schistosomiasis and HIV in rural Zimbabwean women. *AIDS* 2006; 20(4): 593-600.

Kjetland EF, Leutscher PDC, Ndhlovu PD. A review of female genital schistosomiasis. *Trends Parasitol* 2012; 28(2): 58-65; abstract available at <http://www.ncbi.nlm.nih.gov/pubmed/22245065>

Mbabazi PS, Andan O, Fitzgerald DW, Chitsulo L, Engels D, *et al.* Examining the relationship between urogenital schistosomiasis and HIV Infection. *PLoS Negl Trop Dis* 2011; 5(12): e1396. doi:10.1371/journal.pntd.0001396

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