A Review on the Epidemiology and Control of Schistosomiasis In Nigeria

Tariwari C .N. Angaye* 

1.Ecotoxicology Research Unit, Department of Biological Sciences, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.

ABSTRACT

Schistosomiasis is a vector-borne disease caused by parasites of the genus Schistosoma. The disease is transmitted by tiny aquatic snails (intermediate host), that sheds the parasite (cercariae) to its final host. The prevalence of schistosomiasis was investigated based on literature survey. Unfortunately, results shows that farmers and children in rural riverine settlements were most ravaged with the disease. In South-South Nigeria, a prevalence rate of 10.20 - 91.40% was recorded amongst Nomadic and Riverine settlements. In South-West Nigeria, the disease was more endemic amongst rural school children with prevalence rate ranging from 0.60 – 78.00%, while in South-East the prevalence was 4.10 – 55.20%, mostly amongst farmers and rural dwellers, who lack access to clean and portable water. In the North-West the reported prevalence rate was 18.37 – 74.00%, while the North-East and North-Central Nigeria had prevalence rates of 6.00 – 32.40 and 0.67 – 46.60% respectively. Consequent upon our findings, we therefore recommend public enlightenment of the rural populace on the etiology of the disease, provision of portable water, proper sanitary sewage disposal, as well as other preventive and control measures that will significantly reduce the incidence of the disease.

Keywords: Prevalence, Epidemiology, Schistosomiasis, Vector-borne disease

*Corresponding Author Email: maktaary@yahoo.com
Received 14 June 2016, Accepted 25 June 2016
INTRODUCTION

Schistosomiasis is a vector-borne disease caused by parasites of the genus Schistosoma; it is contracted when persons come in contact with infected river water harbouring cercariae-shedding snail\(^1\). The parasite penetrates the skin and migrates via the venous system to the portal vein of the intestine or the bladder where they eventually mature and lay eggs that scar tissues of the organs\(^2\), which eventually results to disease condition.

Schistosomiasis is ranked as the second most prevalent vector-borne disease (WHO 2002, 2010); second only to malaria\(^3,4\). The morbidity burden of the disease cannot be overemphasized, approximately 90% of human schistosomiasis lies in sub-Saharan Africa\(^5\). The disease is endemic in continents like; Africa, Asia, South America, the Middle East, and the Caribbean, Table 1\(^1\).

<table>
<thead>
<tr>
<th>Group</th>
<th>Species</th>
<th>Endemic Regions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mansoni</td>
<td>S. Edwardiense</td>
<td>Africa</td>
</tr>
<tr>
<td></td>
<td>S. Hippotami</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Mansoni</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Rodhaini</td>
<td>African</td>
</tr>
<tr>
<td>Haematobium</td>
<td>S. Bovis</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Curassoni</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Intercalatum</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Guineensis</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Haematobium</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Leiperi</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Margrebowiei</td>
<td>African</td>
</tr>
<tr>
<td>Indicum</td>
<td>S. Spindale</td>
<td>Africa/Asia</td>
</tr>
<tr>
<td></td>
<td>S. Nasale</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Indicum</td>
<td>Africa/India/Thailand</td>
</tr>
<tr>
<td>Japonicum</td>
<td>S. Japonicum</td>
<td>Africa/China</td>
</tr>
<tr>
<td></td>
<td>S. Mekongi</td>
<td>African</td>
</tr>
<tr>
<td></td>
<td>S. Malayensis</td>
<td>African</td>
</tr>
</tbody>
</table>

Source: Angaye et al.,\(^1\)

In Nigeria, schistosomiasis is most prevalent amongst children and farmers in rural communities already ravaged by other diseases; such as malaria and tuberculosis. In under-aged children, schistosomiasis infection induce a compromise in the body's immunity, thereby obliging other opportunistic infections\(^6,7\). Due to the overall morbidity and mortality burden of Schistosomiasis, multifaceted approaches in its control are desirable including the control of its intermediate host\(^8\).

On the other hand, control of schistosomiasis have become a major concern in tropical and subtropical regions over the past decades. For instance, chemotherapy of schistosomiasis only
Angaye et al., Journal of Medical and Health Research 2016(1):2 ISSN:abcd:0000

provides temporary abatement of morbidity rate as re-infection looms. Also, knowledge on the etiology and the epidemiology of the disease is inadequate. Hence, the need for this review.

HISTORY AND ETIOLOGY OF SCHISTOSOMIASIS

History of Schistosomiasis

Morgan et al., 9, reported that the ancestries of the genus Schistosoma is still compounding. For instance, over the past decades it was documented that the genus originated from Africa, but DNA molecular sequencing suggests that the species (S. edwardiense and S. hippopotami) that infect the Hippopotamus (hippos) could be basal. Since hippos were endemic in Africa and Asia in the Cenozoic period the genus might have emerged as parasites of hippos. The original hosts for the South East Asian species were probably rodents. Beer et al 10, reported that based on the phylogenetic studies, it shows that the host snail genus evolved from the foregoing (as old as Gondwana), about 70 - 120 million years ago. The associated cluster to the Schistosoma is a genus of whose host is elephant (schistosomes-Bivitellobilharzia). The cattle, sheep, goat and cashmere goat parasite Orientobilharzia turkestanicum seems to be linked to the African schistosomes 11, 12, Species belonging to this group has since been transferred to the genus Schistosoma 12.

Schistosomiasis, also known as bilharzia, bilharziosis, and snail fever or, in the acute form as Katayama fever, was discovered by a German pathologist called Theodore Bilharz. He identified the eggs of parasite, Schistosoma haematobium in Egypt in 1851 during a post mortem. He wrote a paper in 1856 describing the parasite and named them Distoma haematobium. In 1856 Meckel von Helmsback created the genus Bilharzia, and in 1858 Weinland proposed the name Schistosoma (Greek: “split body) after the male worm morphology.

The generic name Schistosoma was later adopted by the International Commission for Zoological Nomenclature. Schistosoma mansoni had been identified by Bilharz but it was named by Louis Westenra Sambon, who named it after his teacher Patrick Manson. In 1899, all known species were placed in a sub-family by Stiles and Hassel and elevated to family status by Looss in 1899. Silva in 1908 corrected a grammatical error in the family name. In 2009, the genomes of S. mansoni and S. japonicum were decoded opening the way for new target treatments.

The genus Schistosoma may be paraphyletic in nature, however, they are a genus of dioecious trematodes commonly known as blood-flukes and bilharzias, they have numerous species which are sub-divided into four distinct groups; haematobium, mansoni, japonicum and indicum. The mansoni group have lateral projection on the egg, the haematobium group have terminal Projection on the egg while the Japonicum and Indicum have no Projections 13. Each Schistosoma species has

its own snail species as a vector; these snails require well-defined ecological conditions to thrive (Table 2). There are four basic groups belonging to the Schistosoma genus. These groups, their species and endemic regions as determined by the host is presented in Table 2.

Table 2: Schistosoma species showing, their intermediate/final host and endemic areas

<table>
<thead>
<tr>
<th>Schistosoma spp.</th>
<th>Intermediate/final host(s)</th>
<th>Endemic areas</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Schistosoma mansoni</em></td>
<td>Biomphalaria spp./Human</td>
<td>Africa, South America</td>
</tr>
<tr>
<td><em>S. haematobium</em></td>
<td>Bulinus spp./Humans</td>
<td>Africa, Middle East</td>
</tr>
<tr>
<td><em>S. intercalatum</em></td>
<td>other animals / Human</td>
<td>Africa</td>
</tr>
<tr>
<td><em>S. japonicum</em></td>
<td>Oncomelia sp. Domestic and some wild animals</td>
<td>China, East Asia, Philippines</td>
</tr>
<tr>
<td><em>S. mekongi</em></td>
<td>Neotricula aperta / humans/dog</td>
<td>Asia</td>
</tr>
<tr>
<td><em>S. guineensis</em></td>
<td>Bulinus forkalii</td>
<td>West Africa</td>
</tr>
<tr>
<td><em>S. malayensis</em></td>
<td>Rattus mulleri (zoonosis)</td>
<td>Asia</td>
</tr>
</tbody>
</table>

Source: Angaye et al., 1

Life Cycle

Parasites of the genus schistosoma are unique dioecious trematodes with distinctive sexual dimorphism regarding both sexes. Matured cercariae penetrates the skin and migrate via the venous system (Figure 1). They parasitize and reproduce in their host blood capillaries (i.e mesenteries or plexus of the bladder). For instance, *Schistosoma mansoni* and *S. haematobium* parasitize the capillaries of the mesenteries and plexus of the bladder respectively. Furthermore, the formal and latter results to intestinal and urinary schistosomiasis. Countless eggs released by the adult worm gets to the bladder or the intestine, where they are passed out with faeces or urine into to fresh water (Figure 1). The eggs hatch into free swimming ciliated larvae called miracidia. The miracidia then pass through an intermediate snail host, and mature to the adult larval stage called cercariae which can infect a new mammalian host that have contact with such water by directly penetrating the skin.
Acute schistosomiasis

Acute schistosomiasis, also known as Katayama fever\textsuperscript{14}, is common in areas of high transmission rates. In some cases, patients usually present signs and symptoms within 14-28 days after infection\textsuperscript{15}. The symptoms are believed to be mediated by the immune complex, and the majority of cases begin with the deposition of an egg into host tissues\textsuperscript{15}. Symptoms common to this include fever, headache, generalized myalgia, right-upper-quadrant pain, and bloody diarrhoea. About 70\% of persons infected with \textit{S. mansoni} have respiratory symptoms but less frequently in those infected with \textit{S. haematobium}\textsuperscript{16-18}. There are mild cases of vessel and spleen enlargement, which occur in one third of cases; however, there may be radiologic evidence of interstitial pneumonitis.
Praziquantel works exclusively against adult worms, and therefore, repeated treatment or a prolonged course with 20 mg per kilogram of body weight per day has been used. Oxamniquine has also been recommended.

**Chronic Schistosomiasis**

When the host’s immune response to schistosome eggs a granulomatous reaction is evoked by the antigens secreted which causes acute schistosomiasis. King et al. reported that the intensity and duration of infection determine the amount of the antigen that will be released and the severity of chronic fibro-obstructive disease. Although the granulomas destroys the ova it results in fibrotic deposition in the host tissues, most granulomas develops at the sites of accumulation of the eggs which could either be the intestine, liver or bladder depending on the species of schistosome giving rise to urinary or intestinal schistosomiasis. Periovular granulomas have been found in many types of tissue, including the skin, lung, brain, adrenal glands, and skeletal muscle.

Inflammatory response may assist the migration of eggs into the lumen of the gut or urinary tract, which is thus responsible for intestinal or urinary schistosomiasis. This was observed in previous research demonstrating that t-cell–deficient in mice serum, and patients with advanced cases of the acquired immunodeficiency syndrome have significantly reduced egg output. Eggs are retained in the gut wall which induces inflammation, hyperplasia, ulceration, microabscess formation, and polyposis.

Children infected with schistosomiasis usually have diarrhoea and its presence correlates strongly with schistosomiasis. Occult or sometimes visible blood in the faeces is usual. Severe chronic intestinal disease may result in colonic or rectal stenosis. Colonic polyposis may be manifested as a protein-losing enteropathy. Inflammatory masses in the colon may even mimic cancer. The relation between colorectal cancer and schistosomiasis has been debated for decades. If there is an increase in the risk of colorectal cancer, it is small.

Periportal collagen deposits lead to the progressive obstruction of blood flow, portal hypertension, and ultimately varices, variceal bleeding, splenomegaly, and hypersplenism. Periportal fibrosis can be seen on ultrasonography, computed tomography, or magnetic resonance imaging and is characteristic of schistosomiasis. Schistosoma haematobium infection occasionally causes mild colonic or hepatic disease. Co-infection with both hepatitis B virus (HBV) or hepatitis C virus (HCV) and S. mansoni is associated with accelerated deterioration of hepatic function. Alcohol-induced cirrhosis, HBV, or HCV can coexist with clay-pipe-stem fibrosis. The combination of chronic schistosomiasis caused by S. mansoni and HBV infection may result in a
higher risk of hepatocellular carcinoma than that attributable to HBV alone. In contrast, there does not appear to be a significant interaction between HBV and S. japonicum infection.

**EPIDEMIOLOGY AND CONTROL OF SCHISTOSOMIASIS**

**Epidemiology of Schistosomiasis**

Schistosomiasis, is a debilitating disease with a global prevalence of 4-5%. It is common in continents like Africa, Asia and South America. Epidemiological studies had shown that, schistosomiasis is second only to malaria as the most devastating parasitic disease in tropical countries, in terms of morbidity and mortality burden. Ugomoiko et al., reported that Nigeria has the most cases of human schistosomiasis which is widespread in both rural and urban communities and approximately 20 million Nigerians mostly children need to be treated for schistosomiasis. The morbidity burden of the disease cannot be overemphasized, it affects about 4 - 5% of the world population and approximately 90% of human *schistosomiasis* lies in sub-Saharan Africa.

As presented in Table 3, the incidence of schistosomiasis in the South-South region was highest in some rural riverine settlements in Ndokwa-East LGA with a prevalence levels rising from 21.9 to 91.4% from 2003 to 2005. On the other hand, Cross-Rivers state recorded the lowest prevalence of 10.2%. Notwithstanding, nomadic settlements in Rivers State (Eleme and Oyigbo), recorded significant prevalence rates of 81.3% in Eleme and 76.2% in Oyigbo, as well as Odau Community with 83.3%. Also in Ewan town of Akoko Edo LGA of Edo State prevalence rate of 43.30% was recorded. Generally, the South-south region recorded significant levels of schistosomiasis amongst under-aged children and farmers due to poor sanitary practice and lack of access to clean and potable water.

Table 4, presents the prevalence of schistosomiasis in South-East Zone Nigeria. The highest prevalence was found in Ebonyi State with prevalence rate of 55.7%. Similarly, in Eboyi State Ohaukwu (47.9%) and Onicha (11.0%) were reported, while the lowest prevalence was in Anambra state 7.9%. Notwithstanding, higher rates were reported in Orumba North 12.8% and South 19.8%, and Agulu Town and Lake with 4.10 – 55.2% and 11.8% respectively. About 26% of school children were found to be infected in Anambra State, South-Eastern Nigeria.

Also, in Ebonyi State 124 (8.97%) *Bulinus globosus* and *B. truncatus* were infected with schistosome cercariae parasite. In Imo State the disease was reported with prevalence rates ranging from 14.2 – 44.9%. Abia States had prevalence of 10.0%, with prevalence of 42.3% in erstwhile Bendel State.
In South-western Region (Table 5), 21% of school children, 18.4% of local dry cleaners and 15.8% of local vehicle washers were found to be infected in studied population in Ibadan[^49]. The highest prevalence was reported in Ijoun of Yewa North LGA of Ogun State with prevalence of 85.5%[^50], while Oyan dam and Apojula community recorded 52.4 and 9.5% respectively[^51]. Also prevalence rate of 58.1% was recorded in pre-school children from ages of 1 - 6 years[^52]. In Ogbese of Ekiti State, prevalence of 75.6% was recorded[^53], as well as Lagos State in Igbokuta village with 78.00%[^54], Oyo State with 46.6%[^55], Ondo State with 30.50%[^56], and Osun State with the lowest prevalence of 0.6%[^57].
### Table 3: Prevalence of schistosomiasis in South-South Zone Nigeria

<table>
<thead>
<tr>
<th>S/N</th>
<th>States</th>
<th>Endemic areas</th>
<th>Prevalence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Delta State</td>
<td>Some Riverine settlement in Ndokwa-East LGA</td>
<td>2003 (21.9%)</td>
<td>Nwanbueze and Opara, [35]</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2005 (91.4%)</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Cross River State</td>
<td>-</td>
<td>10.2%</td>
<td>Adie et al., [36]</td>
</tr>
</tbody>
</table>
| 3.  | Rivers State  | Fulani herdsmen in Eleme and Oyigbo Odau Community | 81.3% in Eleme 76.2% in Oyigbo 83.3% | Chinwe and Agi, [37]  
|     |              |                                            |                    | Agi and Okafor [38]                            |
| 4.  | Edo State    | Enwan, Akoko Edo LGA                       | 43.3%              | Imarenezor et al., [39]                        |

### Table 4: Prevalence of schistosomiasis in South-East Zone Nigeria

<table>
<thead>
<tr>
<th>SN</th>
<th>States</th>
<th>Endemic Areas</th>
<th>Prevalence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Ebonyi State</td>
<td>-</td>
<td>55.7%</td>
<td>Njoku, [40]</td>
</tr>
<tr>
<td>2.</td>
<td>Ebonyi State</td>
<td>Ohaukwu and Onicha</td>
<td>Ohaukwu (47.9%) Onich (11.0%)</td>
<td>Uneke et al., [41]</td>
</tr>
</tbody>
</table>
| 3.  | Anambra State| Umuikwu-Anam Orumba North and South LGA Agulu Town Near Agulu Lake | 12.8 - 19.8% 4.1 - 55.2% 11.8% | Ezeagwuna et al., [42]  
|     |              |                                            |                    | Ekojindu et al., [45]                          |
| 4.  | Imo State    | -                                          | 14.2 - 44.9%       | Nnoruka, [46]                                  |
| 5.  | Abia State   | Amagodu, Abriba hamlet Erstwhile Bendel LGA | 10.0% 42.3% | Okolie, [47]                                   
|     |              |                                            |                    | Alozie and Anosike, [48]                       |

### Table 5: Prevalence of schistosomiasis in South-West Zone Nigeria

<table>
<thead>
<tr>
<th>SN</th>
<th>States</th>
<th>Endemic areas</th>
<th>Prevalence</th>
<th>References</th>
</tr>
</thead>
</table>
| 1.  | Ogun State   | Ijoun, Yewa North LGA Oyan Dam, Apojula community in Abeokuta Pre-school children (1-6 years) in Ilewo-Orile and its environs | 82.5 % 52.4% haematuria, 9.5% ova in urine 58.10% | Sowole and Adegbite, [50]  
|     |              |                                            |                    | Akinwale et al., [51]                           |
| 2.  | Ekiti State  | Ogbese Ekiti                                | 75.60%             | Ologunde et al., [53]                           |
| 3.  | Lagos State  | Igbokuta Village, Ikorodu North Local Government | 78.00 % | Oluwasogo and Fagbemi, [54]                      |
| 4.  | Oyo State    | Some School Children in Ibadan             | 46.6%              | Nwanbueze et al., [55]                          |
| 5.  | Ondo State   | 18 LGAs                                    | 30.5%; most endemic area was Owena River/dam | Odiabo et al., [56]                             |
| 6.  | Osun State   | 3 to 14 years Pupils in Ile-Ife            | 0.6%               | Ojurongbe et al., [57]                          |
Table 6: Prevalence of schistosomiasis in North-West Zone Nigeria

<table>
<thead>
<tr>
<th>SN</th>
<th>States</th>
<th>Endemic areas</th>
<th>Prevalence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Zamfara State</td>
<td>Abarma in village Gusau</td>
<td>74.0%</td>
<td>Bala et al., [58]</td>
</tr>
<tr>
<td></td>
<td>Jigawa State</td>
<td>Kazaure LGA</td>
<td></td>
<td>Adebayo, [59]</td>
</tr>
<tr>
<td>2.</td>
<td>Sokoto State</td>
<td>Wurno Rural Area</td>
<td>37.7%</td>
<td>Bello et al., [60]</td>
</tr>
<tr>
<td>3.</td>
<td>Sokoto State</td>
<td>Wamakko town</td>
<td>38.3%</td>
<td>Mohammed et al., [61]</td>
</tr>
<tr>
<td>4.</td>
<td>Kaduna State</td>
<td>Birnin-Gwari LGA</td>
<td>20.3%</td>
<td>Alhassan et al., [62]</td>
</tr>
<tr>
<td>5.</td>
<td>Kaduna State</td>
<td>Samaru stream and ABU dam in Zaria</td>
<td>S. mansoni 20.31%; S. haematobium 18.37%</td>
<td>Ayanda, [63]</td>
</tr>
<tr>
<td>6.</td>
<td>Kano State</td>
<td>Tomas and Rimin Gado</td>
<td>26.6 and 36.8% respectively</td>
<td>Betteron et al., [64]</td>
</tr>
</tbody>
</table>

Table 7: Prevalence of schistosomiasis in North-East Zone Nigeria

<table>
<thead>
<tr>
<th>S/N</th>
<th>States</th>
<th>Endemic areas</th>
<th>Prevalence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Adamawa State</td>
<td>Mayo-Belwa LGA</td>
<td>32.4%</td>
<td>Bala et al., [65]</td>
</tr>
<tr>
<td>2.</td>
<td>Borno State</td>
<td>Konduga LGA</td>
<td>24.3%</td>
<td>Biu et al., [66]</td>
</tr>
<tr>
<td>3.</td>
<td>Borno State</td>
<td>Maiduguri Metropolis</td>
<td>14.5%</td>
<td>Joseph et al., [67]</td>
</tr>
<tr>
<td>4.</td>
<td>Yobe State</td>
<td>Boarding students in Potiskum Metropolis</td>
<td>6.0%</td>
<td>Bigwan et al., [68]</td>
</tr>
<tr>
<td>5.</td>
<td>Taraba State</td>
<td>Bali Town</td>
<td>15.5%</td>
<td>Monday et al., [69]</td>
</tr>
</tbody>
</table>

Table 8: Prevalence of schistosomiasis in North-Central Zone Nigeria

<table>
<thead>
<tr>
<th>S/N</th>
<th>States</th>
<th>Endemic Areas</th>
<th>Prevalence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Benue State</td>
<td>Buruku and Katsina-Ala</td>
<td>41.5%</td>
<td>Houmsou et al., [71]</td>
</tr>
<tr>
<td>2.</td>
<td>Plateau State</td>
<td>Gwong and Kabong</td>
<td>2.07%</td>
<td>Dawet et al., [72]</td>
</tr>
<tr>
<td>3.</td>
<td>Kogi State</td>
<td>Ibaaji LGA (Niger-Benue Basin)</td>
<td>18.7%</td>
<td>Ejima and Odaibo, [75]</td>
</tr>
<tr>
<td>4.</td>
<td>Plateau State</td>
<td>Jos North LGA</td>
<td>4.6%</td>
<td>Goselle et al., [74]</td>
</tr>
<tr>
<td>5.</td>
<td>Nasarawa State</td>
<td>Keffi Town in Keffi LGA</td>
<td>30.5%</td>
<td>Ishaleku et al., [77]</td>
</tr>
<tr>
<td>6.</td>
<td>Nasarawa State</td>
<td>Shabu, Lafia North</td>
<td>11.5%</td>
<td>Alaku et al., [76]</td>
</tr>
<tr>
<td>7.</td>
<td>Plateau State</td>
<td>Apati and Laranto</td>
<td>0.67%</td>
<td>Okpala et al., [73]</td>
</tr>
<tr>
<td>8.</td>
<td>Benue State</td>
<td>Ogbadibo LGA</td>
<td>46.6%</td>
<td>Mbata et al., [70]</td>
</tr>
</tbody>
</table>

Table 6 presents schistosomiasis prevalence in North-western Region Nigeria, the highest prevalence was reported in Abarma village of Gusau in of Zamfara State with prevalence of 74.0% \(^{58}\). Some localities of Kazaure LGA of Jigawa State recorded a prevalence of 50.00% in Kazaure, 38.00% in Kirkina and 12.00% in Dandi \(^{59}\). Also prevalence rates of 37.7 \(^{60}\) and 38.3% \(^{61}\) were recorded in Wurno and Wamakko settlements of Sokoto State respectively. In Kaduna State, prevalence of 20.3% was recorded in Birin-Gwari LGA \(^{62}\), as well as Samaru stream (20.31%) and ABU dam (18.37%) \(^{63}\). Also, Tomas and Rimin Gado of Kano State had prevalence of 26.6 and 36.8% respectively \(^{64}\).

In North-eastern Region Nigeria (Table 7), Mayo-Belwa LGA of Adamawa State had the highest prevalence of 32.40%, was reported by Bala et al., \(^ {65}\). In Borno State Konduga LGA and Maiduguri Metropolis recorded prevalence rates of 24.3% and 14.5% respectively \(^ {66}\). Also Records of Maiduguri metropolis indicated prevalence of 14.5% \(^ {67}\). Amongst boarding students of Potiskum Metropolis in Yobe State prevalence rate of 6.0% was recorded \(^ {68}\). Also in Bali Town of Taraba State, prevalence rate of 15.5% was recorded \(^ {69}\).

Table 8 presents schistosomiasis prevalence in North-central Region Nigeria, the highest prevalence was reported in Ogbadibo LGA of Benue State with prevalence of 46.60% \(^ {70}\). Also, Bukuru and Katsina Ala of Benue state recorded prevalence of 41.5% \(^ {71}\). Gwong and Kabong in Plateau State had 2.70% \(^ {72}\), Apata and Laranto with 0.67% as well as Jos North with 4.60% \(^ {73}\), while Jos North reported 4.6% \(^ {74}\). In Ibaji LGA (Niger-Benue Basin) of Kogi State, prevalence rate of 18.70% was reported \(^ {75}\). Also in Shabu of Lafia North and Keffi in LGAs of Nassarawa State prevalence rates of 11.5% was reported \(^ {76}\), and 30.5% \(^ {77}\) were recorded, respectively.

**Diagnosis**

The detection of schistosome eggs in faeces or urine is diagnostic of schistosomiasis. The extent of shedding of eggs may fluctuate widely, hence, as much as three specimens may be required in some patients. *S. mansoni* or *S. japonicum* eggs may be observed in stool specimens of 2-10 mg with or without suspension in saline. The use of formalin-based techniques for sedimentation and concentration may increase the diagnostic yield \(^ {78}\). Such techniques are useful in patients with few eggs, as in a returned traveller. Miracidic-hatching test has been used extensively by public health workers in China to rule out *S. japonicum* infection \(^ {78}\). It is initiated by the concentration of ova from faeces through a nylon tissue bag and suspension in distilled water.

Furthermore, miracidia that hatch from ova are visualized microscopically, and their presence is diagnostic of infection. For patients with a typical clinical presentation but negative urine and faeces specimens, a biopsy of bladder or rectal mucosa must be used for diagnosis. These are the most
sensitive diagnostic procedures available. The rapid, simple, and inexpensive Kato–Katz thick-smear stool examination requires 40-50 mg of faeces which is widely used in field studies, and national control programs to determine the burden of eggs in faeces. Several population-based studies have demonstrated that mean egg burdens correlate with the mean severity of disease. However, it is generally unnecessary to quantify the egg burden in order to provide clinical care.

Antibody detection is useful in a few specific circumstances, but its use is limited because antibodies persist after parasitologic cure. A positive serologic test may be diagnostic in patients in whom there are no eggs, such as those with Katayama fever. Furthermore, serologic testing is useful in field studies for defining regions of low-level endemicity where individual patients have low egg burdens. Serologic testing may also be useful in determining whether infection has re-emerged in a region after an apparently successful eradication program.

Commercially-available imuno-diagnostic kits are not sensitive as multiple faecal examinations and are less specific. Detection of circulating adult-worm and egg antigens is a promising technique that may eventually supersede traditional diagnostic methods. A recent development is an immunoblot assay for the detection of adult-worm antigen, which reportedly has 95% sensitivity and 100% specificity. Additional supportive laboratory evidence of schistosomiasis might include evidence of peripheral-blood eosinophilia, anaemia (iron-deficiency anaemia, anaemia of chronic disease, or macrocytic anaemia), hypoalbuminemia, elevated urea and creatinine levels, and hypergammaglobulinemia. Splenomegaly develops in some patients with pancytopenia.

Biochemical markers of hepatic fibrosis are currently a focus of research. Serum levels of procollagen peptide (types III and IV), the P1 fragment of laminin, hyaluronic acid, and fibrosin may be elevated in patients with severe hepatic fibrosis and can decrease after praziquantel treatment. Persistent elevation of these levels after parasitologic cure suggests the presence of coinfection with HBV or HCV. The measurement of the N-terminal propeptide of type III procollagen, combined with the C-terminal propeptide of type IV procollagen and collagen VI, can be used to predict the risk of progressive hepatic fibrosis. A biopsy of the liver may be necessary in some patients with coinfection. Liver involvement in patients with schistosomiasis is often suggested by the characteristic appearance of the organ on abdominal imaging.

**Control of Schistosomiasis**

Chemotherapy method of controlling schistosomiasis had witnessed landmark success; however it only provides temporary abatement of human parasite burden because of rapid re-infection rates subsequent to drug intervention. Praziquantel, a pyrazinoisoquinoline derivative, is the mainstay of therapy.
treatment and a critical part of community-based schistosomiasis control program since its discovery in the mid-1970s \(^8^9\). Its safety and efficacy have ensured its widespread use. It is absorbed well but undergoes extensive first-pass hepatic clearance. Praziquantel is metabolized by the liver, and its (inactive) metabolites are excreted in the urine, the drug's precise action on immature stage of the parasite is ineffective \(^8^8, 90-92\).

The action of the drug appears to cause tetanic contractions and tegumental vacuoles, causing worms to detach from the wall of the vein and die \(^1^5\). Optimal therapy requires two to three doses of 20 mg per kilogram given six to eight hours apart with food. Community-based control programs usually treat patients with a single dose of 40 mg per kilogram. Higher doses are often used against *S. japonicum* (a total dose of 60 mg/kg). Re-examination of feces or urine one month after treatment is recommended in order to assess efficacy.

Praziquantel reliably cures 60-90% of patients and substantially decreases the worm burden and produce egg in those who are not cured. Patients who continue to shed viable eggs should be retreated with the same dose; the second treatment is usually successful \(^1^5\). Hepatic fibrosis from *S. mansoni* infection, \(^9^3, 9^4\), and *S. japonicum* infection (Li, *et al.*, 2000, Richter *et al.*, 2000) and the urinary tract disease from *S. haematobium* infection \(^9^3, 9^5\) may improve after successful treatment if reinfection is avoided.

The efficacy of praziquantel is unaltered in patients who are coinfected with HIV type 1 (HIV-1). \(^9^6\). Schistosomiasis that is caused by *S. mansoni* does not influence the load of HIV-1 \(^9^7\). Corticosteroids may be useful adjuvant treatment for cerebral disease associated with features of surrounding edema apparent on radiology or for severe Katayama fever \(^9^8\). Oxamniquine is the only alternative to praziquantel for *S. mansoni* infection but has limited availability. Metrifonate is an alternative drug for *S. haematobium* infection but is no longer available commercially. It is necessary to make all these drugs available in case of widespread.

Praziquantel may not be the best choice for chemoprophylaxis because of its short half-life (1 - 1.5 hours) and because it cannot kill schistosomula (the migrating larvae) that are 3-21 days old \(^1^5\). Artemether, which is well known for its antimalarial activity, does kill schistosomula during the first 21 days in the body. Therefore, it should kill all immature schistosomula if it is given every two weeks \(^9^9\). In a trial involving residents of an area in southern China where *S. japonicum* is endemic, the drug was administered every 15 days throughout the transmission season at a dose of 6 mg per kilogram \(^9^9\). Acute cases were prevented and new infections were less than half as frequent as in the control group, and those that did occur were of lower intensity.
Artemether is also potent against the other schistosome species that infect humans. Combining artemether with praziquantel appears to produce a synergistic killing of adult worms. The prospects seem good for prophylaxis with artemether in high-risk groups in areas where schistosomiasis is endemic, such as flood relief workers, tourists, and fishermen. The doses required are lower than those required for treatment of malaria, but it is unlikely that artemether would be used in areas where malaria is endemic because such use might lead to the selection of artemether-resistant *Plasmodium falciparum*. Resistance to praziquantel may be emerging after nearly 20 years of intensive use.

Hycanthone resistance in *S. mansoni* is well documented. In regions of Egypt and Kenya where there has been heavy exposure to praziquantel, there are reports of *S. mansoni* and *S. haematobium* infections that are not responsive to multiple courses of treatment. There is some laboratory evidence suggesting that these drug-tolerant worms may have altered tegumental architecture, which could limit the effectiveness of the drug. So far, however, patients in many communities have undergone multiple courses of treatment over a period of 10 or more years without a demonstrable loss of efficacy.

**CONCLUSION**

The prevalence of Schistosomiasis in Nigeria cannot be overemphasized. The disease is endemic amongst rural settlements aligning coastal area, with high incidence amongst farmers and under-aged children. These settlements lack access to basic amenities (especially potable water). Hence making river water the basic source of water. The poor sanitary disposal of sewage might have encouraged the breeding of the parasites. We therefore recommend the public enlightenment of rural populace on the disease and Government intervention in the provision of basic amenities.

**REFERENCES**


---

**JMHR is**

- Peer-reviewed
- monthly
- Rapid publication

Submit your manuscript at: jmhr@epixpub.com