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Schistosomiasis in Zambia: an historical overview and review of the literature

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Summary

An historical overview and review of the literature on schistosomiasis in Zambia is presented. The review covers a period of approximately 130 years, from Livingstone's exploratory incursion in 1855 to present-day studies. Five species of mammalian schistosomes occur in Zambia and they are reviewed with respect to their distribution, snail hosts, human prevalence, and their role in producing morbidity and disease in man. The present status of infection and disease in Zambia, as well as considerations of transmission and control in the future, are discussed. The references cited represent 90–95% of the published literature on schistosomiasis in Zambia, excluding case reports.

Résumé

Un résumé historique et une revue de la littérature de la schistosomiase en Zambie sont présentés. La revue est d'une époque environ de 130 ans, de l'incursion exploratoire de Livingstone en 1855 aux études contemporaines. Cinq espèces de bilharzies mammaliennes se trouvent en Zambie et elles sont examinées quant à leur distribution, leurs hôtes limaces, leur fréquence chez l'être humain et leur rôle en produisant la morbidité et la maladie à l'homme. La situation à present de l'infection de la maladie en Zambie, de même que les considérations de la transmission et du contrôle à l'avenir, sont discutées. Les références sont citées et sont considérées représenter 90% à 95% de la littérature publiée de la schistosomiase en Zambia, à l'exception des rapports particuliers.

The opinions or assertions contained herein are the private ones of the author and are not to be construed as official or reflecting the views of the United States Department of Defense or the Uniformed Services University of the Health Sciences.

Introduction

> 'We left Cassanges on the 16th, a strong cold wind blowing from the West or N.W. Next morning many of my companions were laid up by fever. All complained of pains in the spine and head, we have all been obliged to remain three days waiting for them. Some complained of passing blood with urine. All are now recovering, and we hope to move on tomorrow.'

Thus wrote the missionary explorer David Livingstone in his journal entry of February 20, 1855 [1]. The illness that struck Livingstone's expedition can be attributed largely to malaria; however, the reference to haematuria in his African porters suggests infection with *Schistosoma haematobium*. Admittedly, the haematuria could have been due to other causes, including malaria, but it appears highly probable that the symptom reflects infection with urinary schistosomiasis. This may in fact represent the first report of this disease in Central Africa and Zambia. It has been suggested that Livingstone himself suffered from this affliction [2].

In subsequent years, it has been recognized that schistosomiasis is endemic to Zambia and exerts considerable impact on the health and well-being of its population. For example, more than 65,000 cases of the disease were treated in 1975 [3]. Moreover, the Ministry of Health estimated that, for the period from 1976 to 1980, 13 of every 10,000 Zambians were treated annually for schistosomiasis [4]. An article in

the 'Times of Zambia' quotes the Governor of the Southern District as follows: 'We have a problem as the disease has become endemic and in some parts over 70% of the population are suffering from it' [5].

In recent years, other more pressing health priorities have caused many aspects of the disease's distribution, transmission and morbidity to be neglected. This paper reviews the status of schistosomiasis in Zambia, with a view towards rectifying this neglect and stimulating interest in this area.

The country

The Republic of Zambia, formerly Northern Rhodesia, is situated in southern Africa between latitudes 8° and 18° south and longitudes 22° and 34° east, has a total area of 752,614 km^2 and a population of approximately 7.2 million with an estimated growth rate of 3.6% [6–8]. Politically, the country is divided into eight provinces, which are further subdivided into numerous districts (Fig. 1). The country is completely landlocked and is bordered by eight countries: Zaire, Angola, Namibia, Botswana, Zimbabwe, Mozambique, Malawi and Tanzania. Topographically the country consists of a large plateau ranging in altitude from 915 to 1200 m above sea level. There are 2250 km of inland waterways, several extensive areas of marsh and swamp, and five major lakes which provide ample habitats for the snail hosts. Most of the rivers and streams, except those of the eastern lobe, drain by means of the Zambezi River into the Indian Ocean. Those of the eastern lobe drain into the Atlantic Ocean via the Zaire River and its tributaries. The climate, modified by the altitude, is generally subtropical and has a defined rainy season from October to April.

The parasite

Central Africa is thought to be the 'cradle' of African schistosomes from which the species spread to other areas of the continent [9]. This hypothesis appears to be strengthened by the situation in Zambia where five of the 11 mammalian species of African schistosomes can be found. The principal species infecting man and initiating morbidity are *S. haematobium*

Fig. 1. Map of Zambia showing provincial boundaries and localities mentioned in the text. In the past, Chipata was known as Fort Jameson, Mansa as Fort Rosemary, and Kabwe as Broken Hill.

and *S. mansoni*. *S. haematobium* appears to have the greatest distribution and is responsible for the largest number of infections. The Zambian strain of *S. haematobium* from Kaloma was compatible with *Bulinus africanus* from South Africa and Tanzania, with *B. globosus* from Kenya and Zimbabwe, and with *B. wrighti* from South Arabia [10]. The distribution of *S. mansoni* appears to be more limited; however, the true extent of its range is not known.

In addition to the two major human species, there are several others which infect domestic animals and wild game predominantly: *S. mattheei*, *S. leiperi* and *S. margrebowiei*. *S. mattheei*, as in other areas of southern Africa, has been responsible for sporadic human infections [11–13]; however, it is mainly a parasite of cattle and sheep [14–17]. Although there are no reported cases of human infection by *S. margrebowiei* in Zambia, human infections have been reported in neighboring Botswana [18].

Dinnik and Dinnik [17] included Northern Rhodesia as part of the southern limits of *S. bovis*. They reported finding the species in 'an old cow slaughtered at Mbesuma Ranch in Chmsali District, the north-eastern part of Zambia close to the Tanzania border'. As this species has not been reported subsequently from Zambia, and considering that previous workers admitted difficulty in distinguishing the species from *S. leiperi*, whose range it overlaps, the endemicity of *S. bovis* in Zambia remains suspect.

S. leiperi and *S. margrebowiei*, first thought to be unique to Zambia, are recognized now as occurring over a larger portion of central Africa [19,20]. *S. margrebowiei* was described by Le Roux, in 1933, from cattle and a variety of wild game species in Southern Zambia [21]. Details of the parasite's life cycle were elucidated by Wright *et al.* [22]. The eggs of the species differ from all other African schistosomes as they are round. They are frequently described as lacking a spine and often appear so. However, Le Roux's original description and sketches clearly indicate the presence of a small lateral spine. The presence of the structure was verified further by Southgate and Knowles [23] who also described the morphology of the larval stages and the development of the adult worm.

In 1955 Le Roux discovered a new schistosome in an African antelope, the Sitatunga (*Tragelaphus speakei*) [24]. He named the species *S. leiperi* and designated the Central Research Station at Mazabuka as the type locality. Subsequent investigations by Pitchford and Wolstenholme [18] demonstrated that the principal hosts for the species were the Lechwe (*Kobus leche*) and the Puku (*Kobus vardoni*). A number of other antelope species and wild herbivores also serve as hosts [18,20]. *S. leiperi* is not thought to infect man and except for a spurious infection noted by Buckley [25] there is no evidence of human infection. Ross *et al.* [26] studied the isoenzymes of this species and compared their electrophoretic patterns with strains of *S. bovis*, *S. mattheei*, and *S. margrebowiei*. These studies have recently been extended by Southgate *et al.* [27] who included observations on the surface morphology of the adult worms using scanning electron microscopy. Development of the species in the rodent *Mesocricetus auratus* and the compatibility of the Zambian strain of the parasite with various snail hosts were compared. Results of these studies indicate that interspecific differences occur with respect to the size, shape and degree of spination that occurs in the cuticle of adult worms. *S. leiperi* developed more rapidly than *S. mattheei* in experimental hosts, but slower than *S. margrebowiei*. Although differences in isoenzymes were noted to occur among the species, additional populations must be studied so that intraspecific variation can be assessed properly. Reports prior to 1955, citing the presence of *S. spindale* in Zambia, actually refer to *S. leiperi*. Le Roux [28] corrected his earlier statements concerning the presence of the former species.

The snail intermediate hosts

The first and possibly one of the most extensive surveys on the snail intermediate hosts was conducted by Buckley (25) in the Luapula, Northern and Copperbelt Provinces. The snails were identified by Major M. Conolly, one of the leading authorities at the time on African freshwater snails. *Biomphalaria pfeifferi* and *Biom. tetragonostoma* (= *Biom. sudanica*) were identified as potential hosts of *S. mansoni*; however, only the former species was observed to emit cercariae similar to those of a

schistosome. Several species of bulinid snails were collected and tentatively identified as *B. africanus*, *B. globosus* and *B. natalensis*, but these could not be separated morphologically from one another with any degree of confidence. *B. tropicus* and *B. forskalii* were also found. Only *B. africanus* and/or *B. globosus* were considered to be potential hosts of *S. haematobium*. The snail fauna of Ndola and its surrounding area, which was also studied by Buckley, was examined by Schwetz [29]; he found that 1% (4/408) and 4.4% (3/69) of *Planorbis* and *Physopsis*, respectively, were shedding schistosome cercariae. No effort was made, however, to identify the snail species further. Some additional information on the snail hosts of the Northern Province was supplied by McCullough and Friis-Hansen [30], and a decade later Hira [31] studied the snail hosts found on the western shore of Lake Bangweulu. Hira identified *B. globosus* from sites between Samfya and Mansa but none of the snails proved to be infected. Offspring from these snails were exposed to miracidia, the cercariae obtained were then used to infect white mice, and *S. haematobium* were recovered. Several small biomphalarids thought to be *Biom. pfeifferi* were found, but again were not infected.

In the Southern Province, investigations on the snail hosts have centred on habitats in the vicinity of Siavonga on Lake Kariba. Hira [32–34] identified *Biom. pfeifferi* and *B. africanus* in Lake Kariba as the potential snail hosts. Although *Biom. pfeifferi* had been infected naturally and the infections were verified in experimental animals, *B. africanus* were not infected naturally nor could they be infected experimentally with miracidia obtained from ova excreted by a patient from Lusaka. It was only on epidemiological grounds that *B. africanus* could be considered a host in Lake Kariba; however, it should be noted that the failure of the experimental infections could have been caused by strain differences inherent in the Lusaka parasite. An extensive snail survey was conducted in and around Mazabuka in 1981 by Jelnes and Madsen of the Danish Bilharziasis Laboratory, and they noted the presence of *B. truncatus* which was identified on the basis of chromosome number and isoenzyme electrophoretic patterns. This species serves as an important snail host for *S. haematobium* in other parts of Africa, but had not previously been reported in Zambia. They also confirmed the presence of *Biom. rhodesiensis* in the area, which I also found to be common at the Nakambala Sugar Estate in 1985. This species was originally described in this area by Mandahl-Barth in 1958 [35] as a subspecies of *Biom. pfeifferi* and was raised to species level in 1960 [36]. Jelnes questioned that the species differs from *Biom. pfeifferi* as he could find no differences in their isoenzyme patterns.

Hira [37] also found snails which he identified as *B. africanus* and *B. globosus*, and also some which were intermediate between the two types, in and around Lusaka. These *africanus*-type species were considered to be the hosts of *S. haematobium* and had a natural infection rate of 2.8%. Both were also infected experimentally with local strains of the parasite. The form which served as the primary intermediate host could not be determined; however, *B. africanus* had the widest distribution. In the Lusaka area, as in other parts of the country, the snail host of *S. mansoni* was *Biom. pfeifferi*. The species was found to be infected naturally and, in experimental infections, 76.8% of the snails were positive [38]. The bulinid snails were generally found in permanent bodies of water, while *Biom. pfeifferi* tended to inhabit small streams and impoundment areas, but were not found in rivers [39]. In the Mkuski District of the Central Province, *B. africanus* and *Biom. pfeifferi* were again found to be the respective hosts of *S. haematobium* and *S. mansoni* [13]. *B. globosus* was also present, but was not infected naturally.

Biom. angulosa is another potential host of *S. mansoni* and has been reported by Mandahl-Barth [40] from the Chozi River and from Chambezi Wantipa near Mbesuma.

The snail hosts of non-human schistosomes have also received some attention. Thus, Wright *et al.* [22] demonstrated that both *B. forskalii* and *B. scalaris* were natural hosts of *S. margrebowiei* in Zambia. However, Southgate and Knowles [23], were not able to infect *B. forskalii* experimentally with this schistosome, nor could they infect any of a series of snails belonging to the *africanus* complex. They succeeded in infecting diploid species of the *truncatus–tropicus* complex, including species from Zambia, and later demonstrated a natural infection in several *B. tropicus* collected from

Lockinvar National Park [41]. Southgate *et al.* [27] infected Zambian *B. globosus* from Chilanga with strains of *S. leiperi* from both Botswana and Zambia. On epidemiological grounds, Hira [11] suggested that *B. africanus* serves as the host for *S. mattheei*; however, direct or experimental proof is still lacking.

Prevalence and distribution

As noted previously, the earliest reference to schistosomiasis in Central Africa was in Livingstone's journal in 1855. There is no evidence to suggest that he associated the haematuria observed in his African porters with *S. haematobium* infection or that he was aware of its occurrence in this part of Africa. At a later date (June 1885), he attributed the symptom to the high iron content of the water drunk from Lake Dilolo and the Lotembwa River [2]. No other reports are known from this period of early Western exploration, which suggests that neither the signs nor symptoms of schistosomiasis were known to the populace. Similarly, the disease was not reported from about 1888, when Cecil Rhodes first showed interest in the region, to 1911, when the north-eastern and north-western regions of Rhodesia were first united under the name Northern Rhodesia. It is obvious, however, that the medical establishment was aware of the disease, for in the 'Annual Report on Health, Northern Rhodesia for 1912', there was a section heading for Bilharzia, though no cases were recorded. In the Report of 1913–1914, the Medical Officer for the Fundu District, Dr A. F. Wallace noted, 'A large number of natives are found infected with either ankylostomiasis or schistosomiasis'. He reports on examining ten individuals, two of whom were infected with *Ascaris*, one with both *Ascaris* and ankylostomiasis, three with *S. mansoni*, and four were found to be negative. This appears to be the first time that *S. mansoni*, as such, is reported from Northern Rhodesia. Scant attention appears to have been paid to schistosomiasis in the Annual Reports of 1915–1923, although individual Medical Officers noted the occurrence of cases from time to time. This paucity in reporting cases may be explained, in part, by a comment made by Dr Stanley Colyer, the Medical Officer from the Fort Rosebury District, in the Annual Report of 1916–1917, 'Endemic haematuria is a comparatively common disease. As there is no cure cases are not taken into the hospital.' Thus, if there are no hospital admissions, there are no hospital statistics to report. It is of interest to note that the Annual Reports from 1912 to 1923, as found in the National Archives of Zambia, were typewritten reports with letters or sections submitted by the various district medical officers. Beginning in 1925–1926, the Annual Reports were printed in London and this corresponded with the administration of the region being relinquished by the British South Africa Company to the British Colonial Office.

A perusal of the 'Annual Medical Reports' from 1925 to 1938 [42], during which time Northern Rhodesia was a Crown Protectorate, demonstrates an increasing awareness of schistosomiasis. The following excerpts illustrate the changing level of awareness in the medical community.

> [1927, p. 11] 'It is impossible to state with any accuracy to what extent this disease is prevalent but its incidence is thought to be slight except in the populations living in proximity to the borders of Portuguese East Africa and in the Kasempa District.'
>
> [1928, p. 18] 'Dr Acheson at Fort Jameson reports 17 cases were treated there, of whom 15 were young children, and states that reliable natives affirm that the disease is prevalent in the district.'
>
> [1931, p. 43] 'It cannot be expected that any great reduction in the incidence of this disease can be looked for until the population is educated to the danger of fouling the water supplies.'
>
> [1932, p. 16] 'European public appear not to appreciate that throughout the Territory bathing in rivers and streams is associated with risk of infection with this disease.'
>
> [1934, p. 14] 'A considerable amount of field work will have to be done before the true incidence and importance of this disease in this territory is understood.'
>
> [1937, p. 6] 'Chief interest regarding helminthic disease centres round bilharziasis . . . Each year seems to make it clearer (a) that the infection is much commoner than has been realized; (b) that the infection is frequently present without causing recognized symptoms or complaint; and (c) the present textbook accounts of bilharzia as a clinical entity give a

very incomplete picture of the disease and might well lead to many cases being overlooked.'

The 1947 Report lists the cases recorded, on a yearly basis, from 1939 to 1946. During this interim, 4649 cases were recognized officially with 12 deaths. Only 63 of these cases occurred in Europeans.

Zambian surveys designed to evaluate the prevalence or incidence of schistosomal infection and/or disease fall into one of four categories: (i) those which investigate general populations cross-sectionally; (ii) those which investigate selected groups of a general population, e.g. schoolchildren; (iii) those which are based on hospital or clinic populations; and (iv) those which use a selected hospital population to elucidate morbidity or a specific clinical or pathological condition, e.g. post-mortem studies or patients with bladder cancer.

General populations

The first survey conducted on general populations was that of Buckley [25]. He reported the prevalence in Luapula Province and in a small part of the Northern Province, observed the distribution of potential snail hosts and attempted to correlate transmission with ecological parameters. The survey encompassed an arc of the Province which was bounded by the Chambezi and Luapula Rivers and included the region around Lake Bangweule. Urine and stool specimens were examined from 2617 and 2557 individuals for *S. haematobium* and *S. mansoni*, respectively. The overall prevalence of *S. haematobium* was 14.7%, but ranged in individual villages from 0 to 60%. Haematuria was found associated with infection in children more frequently (41.4%) than in adults (26%). The mean prevalence for *S. mansoni* was approximately 7% with a range 0–61%. Both parasite species were absent from the Bangweule region. This proved to be the only survey that was done during the period when Northern Rhodesia was a Crown Protectorate. Blair [43] did publish a review on Northern Rhodesia; however, he presented no original data and reiterated Buckley's data plus some additional information gleaned from a limited number of government publications.

The next major survey of a general population was conducted by Blair in 1959 [44]. He examined 10,633 individuals who were resettled on the northern bank of the Zambezi River as a consequence of the flooding of Lake Kariba. The prevalence of *S. haematobium* in these individuals ranged from 2.3 to 8.8% with a mean of 6.8%. For *S. mansoni* the range was from 0 to 16.2% with a mean of 2.4%. It is of interest to note that in 1968, 10 years after completion of the Kariba Dam, the mean prevalence for *S. haematobium* in lake-shore populations was approximately 35% and for *S. mansoni* it was 7.76% [32]. In children (5–14 years) the mean rates were 68.8% and 15.6% for *S. haematobium* and *S. mansoni*, respectively. This dramatic increase in infection, which to some extent was unexpected, amply illustrates why schistosomiasis is often called a 'man-made' disease.

In other studies on general populations, Hira [37–39] examined children from urban and suburban settings in and around Lusaka and found *S. mansoni* rates ranging from 9.6 to 21.8% and *S. haematobium* rates from 24.9 to 41.1%. Fine [45] cited data from a variety of general population subgroups (villages, antenatal clinics, prisons, factories) and noted an overall prevalence of 16% for *S. haematobium* in a total population of 4868. Using his own data and that of others he concluded that there was a national prevalence of 10% for urinary schistosomiasis and 2% for the intestinal form. The only survey, however, that has been national in scope and might truly be representative for the population at large is that of the National Food and Nutrition Programme, in which data was collected from 1969 to 1973 in rural areas of all the provinces except Luapula [46]. Urine samples submitted by 5877 people showed an infection rate for *S. haematobium* of 16.6%. The highest prevalence rate was in the Southern Province and lowest in the Northern Province. These regional rates varied from 0.6% to 64.5%, the latter being found in individuals from the Gwembe Valley of the Southern Province [47,48]*. Peak prevalence

*There is a discrepancy between the data presented in the FAO report and that given in the two papers published by Wenlock. The former states (p. 29) that 16.8% of 6115 persons examined were infected with bilharzia. Wenlock [47,48] states that only 5877 were examined and 16.6% were infected.

occurred between the ages of 10 and 14, with the rate reaching a plateau at 25 years of age, before gradually decreasing after 30 years [49]. Stool examinations were also done on 4920 individuals; however, only hookworm ova were sought and schistosome ova were not reported. This was a tragic oversight.

From 1976 to 1982 the Tropical Diseases Research Centre (TDRC) at Ndola conducted a number of surveys in the Copperbelt and Northern Provinces [50]. In the Kabinga area of the Northern Province, 11 villages were studied and 121 of the 528 persons examined (22.9%) were found to be infected with *S. haematobium*. Infection rates varied from 0 to 58.3% and not only demonstrated great variability, but that at some sites in the Northern Province infection rates may be quite high. This is in contrast to most previous studies in this province. Likewise, in the Kampumbu area of the Northern Province, eight villages had prevalence rates ranging from 45.3 to 77.2%. The greatest prevalence occurred in the 10–14 year age group (55.2%). The Kampumbu study is of particular significance, since it is the only survey in which quantitative egg counts are given as an indication of intensity of infection. Overall the intensity was low.

School populations

School surveys are popular in that one has a captive population which is reasonably easy to sample. They also, in most instances, reflect the group at greatest risk, that which plays the major role in transmission, and that which is most likely to develop morbidity. However, if they are used as the basis for projecting regional or national prevalence, an inflated image is apt to emerge.

Fine [51] was one of the first to study schoolchildren and reported a prevalence of 12.6% in 548 children examined in Kitwe. Subsequently, Bhagwandeen and Borg-Grech [52] reported the prevalence in three Lusaka schools and noted an overall rate of 34%. They stated, perhaps unwarrantedly, that 'one in every three school-going Zambian children in Lusaka suffers from bilharziasis'. The Zambian Flying Doctor Service conducted a survey on 11 schools in remote areas of the country during 1970 and 1971 [53]. Stool specimens were collected from 265 students and urine specimens from 497. No students were found to be infected with *S. mansoni*, whereas 133 (26.8%) had *S. haematobium*. The prevalence of *S. haematobium* ranged from 0% at a school near Lake Bangweulu to 69% at a school in the Northern Province bordering on Tanzania. No correlation could be observed between height, weight, haemoglobin levels or school performance in those excreting ova or those not excreting ova at the time of the examination. These results appear to be in agreement with Fine's [54] study of an outbreak of schistosomiasis in a Kitwe primary school in which 60% of 333 children were found to be infected. Infected and non-infected children did not differ significantly with respect to weight, height, percent haemoglobin, blood pressure, clinical abnormalities or mental brightness. It should be noted that mental function tests were not given and brightness of students was a subjective evaluation by teachers. The intensity of infection was also not known. Later in 1975, Fine [45] published a summary of prevalence rates in various schools from the Copperbelt, Central and Lusaka Provinces. A mean prevalence of 27% was noted for *S. haematobium* in 5111 specimens. Additional studies were conducted in the Central Province by Hira and Patel [13] in schools of the Mkushi District and by Watts [55] in Kabwe. The former investigators collected 847 urine samples from students in five schools and 240 stool samples from schoolchildren residing in one village. Infection rates for *S. haematobium* ranged from 3 to 68.4% in the various schools, with a mean of 28.9%. The mean for *S. mansoni* was 16.2%. Watts [55] examined 1208 children from nine primary schools in the Mwaschistompola Demonstration District and noted that 17% were positive for *S. haematobium*. No *S. mansoni* could be found in the 60% that submitted a stool specimen. Poor sanitary conditions (one toilet for 80 or more students) and the proximity of impounded water used for swimming and fishing were considered to be contributing factors responsible for disease transmission. Schoolchildren from villages along the western shore of Lake Bangweulu were surveyed by Hira [31]. Infection rates in the 871 students comprising this study ranged from 3 to 35.3% for urinary tract infections and about 1.5% for those with the intestinal form. These rates were somewhat higher than

previous studies from the area; however, it was thought that the infections were probably acquired at sites other than the actual lake. A series of surveys was initiated by the TDRC [50] from 1976 to 1979 in 72 schools located in the Copperbelt Province. Urine samples from 12,347 individuals were collected and 13.6% were found to be positive for *S. haematobium*. However, only a 0.9% infection rate was observed in 5782 stool samples examined for *S. mansoni*. The highest prevalence for this parasite in any of the schools was 2.8%.

Hospital populations

Several efforts have been made to use data obtained from hospital admissions and post-mortem series as indicators of regional or national prevalence. Although such data may be of value in assessing the morbidity or economic impact of the disease, selection bias excludes their use as a means of estimating either true prevalence or incidence. At the Luampa Mission Hospital, for example, Henderson [56] examined stool specimens from 436 patients selected at random. An incidence of 57.6% was noted and it was concluded that '*Schistosomiasis mansoni* is a very prevalent condition in this area and that it presents a serious health problem'. Indeed it is questionable if the data obtained warrants the conclusion. A more extensive approach was that of Fine [45] who cited laboratory data collected by the Ministry of Health from 27 hospitals located throughout the country during the years 1966–1968. A total of 309,277 urine specimens and 195,487 stool specimens were examined and 11.3% of the former and 2% of the latter were found positive for schistosome ova. Although considerable variation occurred among the hospitals, the mean infection rates each year were remarkably consistent. In the same report, he reviewed the results of an earlier study [57] on 1200 post-mortem examinations carried out at the Kitwe Hospital in 1968. In this series, a prevalence rate of 9% was found for *S. haematobium* and age-specific rates peaked in the 16–20 year cohort.

Clinico-pathological studies and morbidity assessment

Clinical and pathological manifestations and associated morbidity have been well documented in Zambian patients with schistosomiasis, particularly in those infected with *S. haematobium*. An editorial which appeared during 1974 in the *Medical Journal of Zambia*, noted that the commonest cause of haematemesis in Zambia was due to bleeding esophageal varices resulting from schistosomal portal hypertension [58]. Fine [57] remarked that in his experience the incidence of schistosomiasis was three times greater (45%) in patients with bladder cancer than in the population at large. Similarly, Carruthers [59] observed that in 86 consecutive cases of bladder neoplasms seen at the University Teaching Hospital (UTH), 69% were squamous cell carcinomas, and that 70% of these contained the schistosome ova. Bhagwandeen [60] reviewed all cases of bladder cancer seen during a 5-year period at the UTH in Lusaka. He noted that, at this time, carcinoma of the bladder was the third most important malignancy found in Zambia and represented about 9% of all of the malignancies seen at the Department of Pathology. Of the 217 cases of bladder cancer that he reviewed, 65% were associated with concomitant schistosomiasis, and 75% of these were squamous cell carcinomas. More recently, Elem and Purohit [61] have shown that while 45% of cadavers with non-malignant bladders had urinary schistosomiasis, 96% of those with malignancies were infected. Quantitative egg counts were performed on digests of tissues from these cases, and intensities as great as 200,000 eggs/g of tissue were observed. It is of interest to note, however, that calcification of the bladder in the malignant group was only slightly greater (38%) than in the non-malignant group (35%). In one-third of their patients, infection was of moderate–low intensity with less than 10,000 eggs/g of tissue. They surmised that factors other than intensity of infection were responsible for initiation of malignancy. Cervical carcinoma constitutes one-third of all cancers seen in Zambian females, and in one study of 274 cases, 24.4% were found associated with schistosomiasis [62].

The extent and seriousness of the morbidity produced by *S. haematobium* is controversial and appears to differ with the experience of individual investigators. For example, Fine [57] observed that over a period of 3 years at the Kitwe General Hospital, only 43 of 4000 biopsies had bilharzial lesions. As a conse-

quence of this study, he states that, except for carcinoma of the bladder, 'My experiences with surgical bilharzia do not impress me with the numbers or the seriousness of the lesions produced . . .'. In a subsequent study on hydronephrosis in a post-mortem series he commented that 'bilharzia does not seriously contribute to advanced hydronephrosis' [63]. Umerah [64] studied 68 patients with hydronephrosis secondary to schistosomiasis and concluded that, except when complicated by infection, cancer or calculosis, renal function was relatively good even in advanced cases. On the other hand, Elem and Vandal [65] studied 100 consecutive cases of urinary schistosomiasis by cytoscopic, urographic and histological techniques, and concluded that 'urinary bilharziasis in Zambia is associated with a high morbidity and a significant mortality'. Elem [66] also noted, in a subsequent series of 75 patients, that eight showed grade 3 vesico-ureteric reflux. However, he was not able to associate the presence of urinary calculi with schistosomiasis [67]. Attili *et al.* [68] reported the occurrence of nodules, initiated by schistosome eggs, in the genitalia of patients seen in the STD clinic at UTH. These lesions were difficult to differentiate from those normally associated with venereal diseases, and diagnoses could only be made by histological examination of tissue biopsies.

Grove [69] found that rectal biopsies were helpful in diagnosing *S. haematobium* infections, but that 'timed urine specimens' were of little value [70]. Kakoma *et al.* [71] demonstrated that heterophile antibodies to rabbit erythrocytes could be used to distinguish between infected and non-infected individuals under field conditions. The test could be used for both *S. haematobium* and *S. mansoni*.

Intestinal schistosomiasis is not as prevalent as the urinary form of the disease and, consequently, has been studied to a lesser extent. In Zambia, as in other areas endemic for *S. mansoni*, the disease is frequently associated with both pipestem fibrosis, portal hypertension, and splenomegaly. Both Bhagwandeen [72] and Carruthers and Sinha [73] presented accounts of schistosomal portal fibrosis as seen in Zambia, and Bhagwandeen has given a detailed histological account of the process [60,72]. Schistosomiasis and malaria appear to be the two major causes of massive splenomegaly in Zambia, and schistosomiasis accounted for 11.9% in a series of 344 patients seen in Ndola [74] and 29.5% of 44 cases studied in Lusaka [75]. IgG levels appear to be elevated considerably in Zambian patients with massive splenomegaly associated with schistosomiasis, and tends to differentiate these patients from those whose splenomegaly is associated with malaria [76]. Although IgM levels were also elevated in splenomegalic patients with *S. mansoni*, they were not as high as those found in individuals suffering from the Tropical Splenomegaly Syndrome caused by malaria. Sukwa *et al.* [77] obtained data on morbidity of *S. mansoni* by interviewing a rural community in Northern Zambia. A total of 928 individuals were screened, with 581 (63.2%) found to be infected with *S. mansoni*. A group of symptoms were solicited from the population and analysed with respect to their specificity, sensitivity and predictive value in determining *S. mansoni* infection. It was found that blood in the stool, bloody diarrhoea, and watery diarrhoea were significantly ($P < 0.001$) associated with infection and could be used by rural health workers to make an early diagnosis in the absence of a parasitological examination. It was also demonstrated that morbidity was associated directly with intensity of infection, as measured by egg load [78].

Efforts to equate infection with either mental alertness in children [53], with physico-chemical parameters [53,54], or with productivity of workers [79] have been equivocal. Some of the studies can be faulted as being non-objective and subject to bias and, to date, a satisfactorily designed study has not been conducted in Zambia.

Present status and future considerations

It is as difficult today as it was in the past to arrive at an estimate of national prevalence for schistosomiasis or even a reliable figure for the various provinces. In addition, the distribution of the snail hosts and sites of transmission are delimited poorly. An expertise, not presently available, in both epidemiology and malacology will be required to resolve these problems.

Both urbanization and a high rate of population growth are apt to influence the nature of schistosomiasis as it occurs in Zambia. It is

recognized that Zambia is one of the most urbanized countries in all of Africa, with 45% of its population in urban centres and urbanization occurring at an annual rate of 6.5% [8]. The country also has a high annual growth rate, estimated at 3.6%. This shift in population may alter existing patterns of transmission and distribution from predominantly rural to urban. Hira [37–39] has demonstrated the occurrence of urban transmission in Lusaka and its environs. Rampant urbanization may exacerbate marginal housing and poor sanitation, leading to contamination of peridomestic snail habitats and increased transmission. Alternatively, the restrictions of space imposed by a rapid rise in the urban population might usurp existing habitats or modify them so that transmission is reduced or ceases. The need for future studies on the patterns of urban transmission is obvious. Countries with high population growth rates are characterized by population pyramids with broad bases, i.e. a marked increase in age groups represented by children and young adults. In Zambia, as in other endemic areas, these age groups exhibit the highest prevalence rates, have the greatest potential for developing morbidity in the future, and are the major contributors to transmission. Known hyperendemic foci, such as the villages on the shores of Lake Kariba or those in the Gwembe Valley, will probably change very little with respect to prevalence. They will remain a constant reservoir of infection, serve as a threat to irrigated agriculture in the Southern Province and require continual surveillance.

In the past, control of schistosomiasis was relegated to the environmental health staff at the district or provincial level [80]. Control measures suggested by the Ministry of Health [81] included the following: (i) health education, (ii) provision of adequate sanitary facilities, and (iii) provision of safe water supplies for domestic use. Implementation depended on provincial resources and there is no evidence that these measures were carried out to any substantial degree. The ability to carry out extensive control programs and to limit transmission is often beyond the financial and manpower resources of developing countries. It is not surprising, therefore, that Zambia has no national control programme, and the recent decline in the Zambian economy will possible limit the initiation of future health programmes [82]. Zambia was selected by the WHO to participate in a multicentre trial to test the efficacy and safety of praziquantel [83]. Consequently, over the past decade, the TDRC has collaborated with the WHO in testing this drug under field conditions [84,85]. Recently, a Phase III trial was conducted and the results indicated that 67.2% of 551 individuals treated remained free of infection for 1 year [86]. In the short-term, morbidity and serious diseases will probably be controlled by chemotherapy, the extent of the control programme will be related directly to the availability and cost of these drugs. In the long-term, Zambia's socioeconomic climate will determine the extent to which the problem of schistosomiasis can be resolved.

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