## *Chapter 12*

# SCHISTOSOMIASIS

KE MOTT

## INTRODUCTION

Schistosomiasis, sometimes called bilharziasis (after the German physician Theodor Bilharz), ranks high among parasitic diseases in terms of socioeconomic and public health importance in tropical and subtropical areas. One measure of that importance within the development strategies of endemic countries is reflected in executive level planning and inclusion of schistosomiasis control activities in national budgets in Brazil, China, Egypt, the Philippines, and Morocco (World Health Organization 1993).

At least one form of schistosomiasis is now endemic in 74 tropical developing countries. It is estimated that over 200 million people residing in rural agricultural and periurban areas are infected, while 500–600 million more run the risk of becoming infected as a result of poverty, substandard hygiene in poor housing, and inadequate public infrastructure. Schistosomiasis is a major health risk in the rural areas of central China and Egypt and ranks high in other developing countries.

Schistosomiasis is mainly a rural occupational disease that affects people engaged in agriculture or fishing. In many areas, a large proportion of children are infected by the age of 14 years; in other areas, women face the highest risk because of domestic and occupational contact with fresh water. Increased population movements help to propagate schistosomiasis, as evidenced by its introduction into an increasing number of periurban areas of north-eastern Brazil and Africa and among refugees in Somalia, Zimbabwe and Cambodia. Controlling the disease is of strategic relevance for development of tourism in endemic countries; it is recognized that tourists now increasingly acquire schistosomiasis as “off-track” tourism increases, sometimes with severe acute infection and unusual sequelae, including paralysis of the legs.

## Schistosome parasitic worms

The major forms of human schistosomiasis are caused by 5 species of flatworm, or blood flukes, called schistosomes. Urinary schistosomiasis, caused by *Schistosoma haematobium*, is endemic in 54 countries in Africa and the Eastern Mediterranean. Intestinal schistosomiasis, caused by the *mansoni* worm, occurs in 53 countries in Africa, the Eastern Mediterranean, the Caribbean and South America. In 41 countries, both parasites are endemic. Another form of intestinal schistosomiasis caused by *S. intercalatum* has been reported in 7 central African countries. Oriental or Asiatic intestinal schistosomiasis, caused by the *S. japonicum* group of parasites (including *S. mekongi* in the Mekong river basin), is endemic in 7 countries in the South-East Asia and the Western Pacific region.

## Simple and inexpensive diagnosis

Diagnostic methods for schistosomiasis have recently been reviewed (Feldmeier & Poggensee 1993) (Table 12.1). For diagnosis of urinary schistosomiasis, a simple sedimentation can be used efficiently. A syringe filtration technique using filter paper, or polycarbonate or nylon filters, makes it possible for a team of 5 to examine up to 200 children in an hour and a half. Children infected with *S. haematobium* nearly always have microscopic or visual blood in their urine (haematuria). Children needing treatment can be identified by merely looking at the urine specimen or checking for microscopic blood using chemical reagent strips. The eggs of intestinal schistosomiasis can be detected in faecal specimens by a technique using cellophane soaked in glycerine, the Kato or Kato-Katz technique, or

**Table 12.1** Diagnostic methods for schistosomiasis

| *Technique* | *Parasite* | *Sensitivity of single examination* | *Reference* |
|---|---|---|---|
| Kato-Katz | *S. mansoni* | 100% - infections > 100 epg<br>45% - infection 1–50 epg | Sleigh et al. (1982) |
| Glass slide | *S. mansoni* | 100% - infections > 50 epg | Teesdale, Fahringer & Chitsulo (1985) |
| Questionnaire | *S. haematobium* | 77–92% - infections > 64 eggs/10 ml of urine over age 15 years<br>Specificity = 62–82% | Mott et al. (1985a) |
| Reagent strips | *S. haematobium* (haematuria) | Sensitivity = 97 per cent > 64 eggs/10ml of urine<br>Specificity = 85% | Mott et al. (1985b) |
| Reagent strips | *S. haematobium* (haematuria) | Sensitivity = 88%<br>Specificity = 97% | Stephenson et al. (1984) |
| Reagent strips | *S. haematobium* (haematuria) | Sensitivity = 67% overall = 83% = >50 eggs/10ml of urine<br>Specificity = 80% | Savioli et al. (1990) |
| Filtration | *S. haematobium* | 100% - infections > 20 eggs/10ml | Warren et al. (1978) |

epg = eggs per gram

between glass slides. The cost of the tests requiring a microscope is now US$0.01 or less. The chemical reagent strips cost about US$0.05.

## SAFE AND EFFECTIVE TREATMENT

Three safe, effective drugs are now available for schistosomiasis and all can be taken by mouth. Praziquantel, oxamniquine and metrifonate are all included in the WHO's model list of essential drugs (World Health Organization 1990). Their discovery has revolutionized treatment of this disease.

Praziquantel, effective against all forms of schistosomiasis, has proven to have few and only transient side-effects and millions of people have benefited from treatment. Oxamniquine is used exclusively to treat intestinal schistosomiasis in Africa and South America. Metrifonate has now proved to be safe and effective for the treatment of urinary schistosomiasis.

### **Box 12.1** Schistosomiasis: life history of a worm

- People contaminate the environment by their unsanitary habits. They acquire schistosomiasis infection through repeated daily contact with fresh water during fishing, farming, swimming, bathing, washing and recreational activities.
- Eggs, excreted in urine or faeces from an infected person, break open on reaching water, releasing a tiny parasite (a miracidium) which swims frantically through the water by means of the fine hairs (cilia) covering its body, in search of a freshwater snail in which it can develop further. The parasite must find a snail host within 8–12 hours before it perishes.
- Once it has penetrated the snail, the parasite divides many times until thousands of new forms (cercariae) break out of the snail into the water. This phase of development takes 4–7 weeks or longer, depending on the type of parasite.
- Outside the snail, the cercariae, which have long forked tails, can live for 48 hours at the longest. They can penetrate a person's skin within a few seconds in order to continue their growth cycle.
- As the cercaria penetrates the skin using secretions from its special glands, its tail falls off. Within 48 hours it has passed completely through the skin into the blood vessels. Sometimes this process causes itching (swimmer's itch or cercerial dermatitis), but otherwise most people never notice it.
- Within weeks, the young parasite transforms itself into a long worm—either a male or a female. The female can produce eggs only when a male worm is present. Male and female adult worms remain joined together for life (less than 5 years on average, though they can live for up to 40 years); the more slender female is held permanently in a groove in the front of the male's body. Once eggs are produced—over 200 per day depending on the species—the cycle starts again.
- In intestinal schistosomiasis, the worms attach themselves to the walls of the blood vessels lining the intestines. In urinary schistosomiasis, they live in blood vessels of the bladder. Only about half of the eggs leave the body in the faeces (intestinal schistosomiasis) or in the urine (urinary schistosomiasis); the rest remain embedded in the body, damaging organs, including liver, spleen, heart and esophagus.
- Heavy infections with schistosome parasites, occurring mainly in children, cause the actual disease. The eggs laid by the female worm—not the worms themselves—damage the bladder, intestines or other organs.

Even though re-infection may occur after treatment, the risk of developing severely diseased organs is diminished and even reversed in young children in the short term for all forms of schistosomiasis (Jordan, Webbe & Sturrock 1993). In most areas, a reduction in the overall number of cases has been obtained by mass treatment and maintained for 1.5–2 years and in other areas up to 5 years without further intervention.

No long-term follow-up studies after treatment of *S. haematobium* or *S. japonicum* are available.

## DEFINITION AND MEASUREMENT

Historically, the term "bilharziasis" was commonly used for schistosomiasis. Following an expert group report on the epidemiology and control of schistosomiasis (World Health Organization 1967), "schistosomiasis" became the official terminology. Schistosomiasis is a group of diseases, rather than a single entity. The standard criteria on diagnosis of each type of schistosomiasis is the presence of eggs in excreta. The use of the term "schistosomiasis," however, reduces the possibility of discriminating between the different diseases associated with each type of infection.

Efforts were undertaken to refine the reporting of morbidity attributable to schistosomiasis in the development of the Ninth Revision of the International Classification of Diseases (ICD-9), whose preparation began in 1969 and concluded in 1975. The ICD-9 categories are as follows:

120.0 *Schistosoma haematobium*

120.1 *Schistosoma mansoni*

120.2 *Schistosoma japonicum*

120.3 Cutaneous

120.8 Other
Infection by *Schistosoma:*
- *Bovis*
- *Intercalatum*
- *Mattheei*
- *Spindale*
- *cherstermani*

120.9 Schistosomiasis, unspecified

In ICD-9, the adaptation of ICD-O for oncology permitted the designation of the cell type of carcinoma. This was relevant to bladder cancer associated with schistosomiasis (*S. haematobium* only) in which squamous cell types predominate (M805–M808) as compared to transitional cell types (M812–M813).

Deficiencies in the description of morbidity attributable to schistosomiasis (in contrast to the detail of morbidity regarding amoebiasis and Chagas disease) were addressed in preparations for the tenth revision of the International Classification of Diseases (ICD-10). The First Expert Committee on Control of Schistosomiasis proposed eight new definitions to be integrated into ICD-10 either under B65 or as a qualifier under the disease state (World Health Organization 1985). These were provided to the secretariat of ICD-10 as part of the background preparatory documents. The suggestions were partially accepted. In ICD-10, schistosomiasis is classified as B65 and each of the three main species infecting humans are classified separately. *S. intercalatum* and *S. mekongi* come under B65.8—other schistosomiases. The ICD-10 categories are as follows:

B65.0 Schistosomiasis due to *Schistosoma haematobium* [urinary schistosomiasis]

B65.1 Schistosomiasis due to *Schistosoma mansoni* [intestinal schistosomiasis]

B65.2 Schistosomiasis due to *Schistosoma japonicum* [Asiatic schistosomiasis]

B65.3 Cercarial dermatitis

B65.8 Other schistosomiases
Infections due to *Schistosoma:*

- *Intercalatum*
- *Mattheei*
- *Mekongi*

B65.9 Schistosomiasis, unspecified

The category for infection attributable to *S. chestermani* (the same as *S. intercalatum*) in ICD-9 was removed in ICD-10. A number of codes were introduced to classify clinical sequelae of schistosomiasis infection in ICD-10 (see Box 12.2). In ICD-10, haematuria attributable to schistosomiasis is classified under R31 rather than N02 since the origin is the bladder rather than the glomeruli.

Unlike most other infectious diseases, infection with *Schistosoma* is epidemiologically and clinically different from disease attributable to *Schistosoma*. Schistosomiasis is a chronic infection whose onset is difficult to detect. Thus, incidence is generally not measured; if it is, it reflects only the specific geographical area studied and cannot be extrapolated. Prevalence of schistosomiasis is the usual epidemiological variable measured.

Morbidity (disease or impairment of the normal state that affects performance of vital functions) is a result of prior or concurrent infection with *Schistosoma*. With currently available techniques it is, however, difficult to measure clinical impairment in the early stages of disease or in light infections.

Schistosomiasis infection is thought to be associated with increased risk of other parasitic infections. Aside from the theoretical assumptions on

**Box 12.2** Tenth revision of the International Classification of diseases (ICD 10) codes for schistosomiasis sequelae

| *ICD 10 code* | *Sequela* |
|---|---|
| K77.0 Liver disorders in infectious and parasitic diseases classified elsewhere | Hepatosplenic schistosomiasis<br>Portal hypertension in schistosomiasis |
| J70.8 | Cor pulmonale attributable to schistosomiasis would probably be designated under J70.8 |
| N29.1 | Disorders of the kidney and ureter in schistosomiasis (bilharziasis) |
| N33.8 Bladder disorder in schistosomiasis (bilharziasis) | |
| R31 | Haematuria due to schistosomiasis is to be classified under R31 rather than N02 since the origin is the bladder rather than the glomeruli |

Source: World Health Organization (1987)

the frequency of the association of schistosomiasis with other diseases at the population level (Bundy et al 1991), published reports on community studies in Brazil (Lehman et al. 1976), Chad (Buck et al. 1970), Egypt (El Malatawy et al. 1992), Kenya (Thiongo and Ouma 1987), Sierra Leone (White et al. 1982), and Zambia (Wenlock 1979), have shown that *Schistosoma* infection is not consistently associated with other parasitic infections. When present, the consequences of multiple infections on morbidity, disability and mortality have not been adequately studied.

In summary, a “gold standard” for the measurement of schistosomiasis morbidity has not been developed. This chapter will attempt to clarify this matter so that, within national development priorities, the impact of schistosomiasis can be accurately identified and the ensuing changes induced by control and development can be reliably assessed.

## POTENTIAL CONFOUNDING DISEASES

Of all the types of schistosomiasis, disease attributable to *Schistosoma haematobium* is the most likely to be confounded with other diseases. When haematuria is present, the disease may be misdiagnosed as glomerulonephritis (ICD-10 code N02.9) or haematuria of unknown etiology (ICD-10 code R31).

In a known endemic area, the direct correlation between gross and microscopic haematuria and infection attributable to *S. haematobium* permits the valid use of haematuria as a proxy for infection even after large scale treatment. The picture may be further complicated if renal biopsy is performed, since both *S. mansoni* and *S. haematobium* infection have been associated with morphological changes with clinical manifestations ranging from nephrotic syndrome (ICD-10 code subdivisions .5 and .6, i.e. membranoproliferative glomerulonephritis types 1–3).

In most endemic areas of schistosomiasis attributable to *S. haematobium*, the incidence of squamous cell bladder cancer is high. Epidemiological data do not usually cite the association with schistosomiasis as an etiological agent. Such detail is subject to the thoroughness of the surgical pathological studies (incomplete in endemic areas) or the training and personal interest of the surgeon (as in Egypt).

Although most people in the endemic areas have light infections with no symptoms, the economic and health effects of schistosomiasis should not be underestimated. In the north-east of Brazil, in Egypt, and in Sudan, the work capacity of rural inhabitants is severely reduced because of the weakness and lethargy caused by the disease. The school performance and growth patterns of infected children are also retarded; after treatment, remarkable improvement occurs.

A major constraint in the determination of the burden of disease of schistosomiasis is that it does not occur in isolation. It is one of the diseases of poverty and is linked to all other infectious and parasitic diseases of poverty. Within any community, it most affects the poorest segment of the population.

## DATA SOURCES AVAILABLE AND THEIR BIASES

The standard references for the current distribution and prevalence of schistosomiasis are two WHO documents. In the *CEGET/WHO Atlas of the global distribution of schistosomiasis* (Doumenge et al. 1987), the geographical distribution of all forms of schistosomiasis is identified on the basis on an exhaustive compilation of published epidemiological studies as well as surveys carried out by ministries of health. The prevalence was estimated (extrapolated) using this geographical representation of data and the actual surveys or epidemiological data (Utroska et al. 1989).

The heterogeneity of the spatial distribution of the prevalence of parasitic diseases is widely recognized (Ashford, Craig & Oppenheimer 1992, 1993). The clustering of heavy infections attributable to *S. mansoni* and *S. japonicum* is also heterogeneous (Mott 1982). With the exceptions of Algeria, Brazil, Botswana, China, Egypt, Mali, Malawi, Morocco, and Zimbabwe, however, the data available to Ministries of Health are not disaggregated to the village level; thus most national statistics are intrinsically inadequate.

## REVIEW OF THE EMPIRICAL DATABASES BY WORLD BANK REGIONS

There is no single standard database for schistosomiasis. Schistosomiasis is not usually reported as an infection nor are any of its disease manifestations reported separately (Iarotski & Davis 1981). The *CEGET/WHO Atlas of the global distribution of schistosomiasis* (Doumenge et al. 1987) comprises the most complete database to the village level of endemic countries. Systematic extrapolation of these data to obtain accurate national estimates of the prevalence or morbidity is not possible because of the heterogeneity of the data and the distribution of the infection and disease within any particular country. Despite these limitations, an attempt has been made to estimate prevalence in order to assess the potential need for antischistosomal drugs (Utroska et al. 1989).

Unlike the analysis of information on acute infectious diseases, the interpretation of incidence data has many limitations in schistosomiasis (Jordan, Webbe & Sturrock 1993). To calculate incidence accurately requires greater sensitivity of the diagnostic techniques than those currently available or in use by health services (Sleigh & Mott 1986).

Any age-specific data on prevalence of schistosomiasis attributable to *S. mansoni* and *S. haematobium* should be interpreted with caution and compared with the age distributions shown in Tables 12.2–12.8. There seems to be no standard age distribution for *S. japonicum*. This is probably because no endemic area has not been exposed to intervention since 1950 and in some areas, transmission has been eliminated.

Extensive analytical reviews of the published literature on the morbidity of schistosomiasis have been conducted by Gryseels (1989) and Sleigh & Mott (1986). There is general agreement that heavy infections, as measured by faecal or urinary egg counts, are associated with disease. Most of the relevant studies are referred to in Tables 12.2–12.8 or discussed in the text of this review. The major information gap is the lack of statistics on schistosomiasis as a cause of outpatient visits, hospitalization and even as a cause of death.

### Established Market Economies

Only Japan is endemic in this region. No new cases have been reported since 1978. Thus no mortality could be attributed below age 15 years.

### Formerly Socialist Economies

Schistosomiasis is not endemic in these countries.

### India

*S. haematobium* has been confirmed near Hyderabad. The extent of the problem requires further investigation and any estimates cannot be confirmed.

### China

The current prevalence estimate of 1.5 million people infected, proposed by the Ministry of Public Health of the People's Republic of China, is based on a national sample survey in 1989 (China Ministry of Public Health 1992). The sex ratio in the sample survey was 14.2:8.3 (male:female). Prevalence studies for China are summarized in Table 12.2.

**Table 12.2** *S. Japonicum* empirical databases, China

| *Date* | *Location* | *Prevalence (%)* | *Morbidity* | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | *Mortality Rate per million* | | | |
| | | | | | *Age group (years)* | *M* | *F* | | |
| 1990 | China<br>65 counties | Not stated | Not stated | Surveys on nutrition and health in 49 counties | 1–4 | 0 | 0 | Junshi et al. (1990) | Data from 49 counties used in calculations. Only 20 are endemic. Mortality estimates are probably low.<br>Note: rectal cancer and colon cancer (registered separately) had lower cumulative mortality rates for 0–64 years than schistosomiasis. All these were significantly associated with schistosomiasis japonica. |
| | | | | | 5–9 | 0 | 1 | | |
| | | | | | 10–14 | 1 | 0 | | |
| | | | | | 15–19 | 2 | 1 | | |
| | | | | | 20–24 | 5 | 1 | | |
| | | | | | 25–29 | 8 | 2 | | |
| | | | | | 30–34 | 17 | 6 | | |
| | | | | | 35–39 | 30 | 11 | | |
| | | | | | 40-44 | 42 | 16 | | |
| | | | | | 45–49 | 70 | 34 | | |
| | | | | | 50–54 | 95 | 58 | | |
| | | | | | 55–59 | 122 | 84 | | |
| | | | | | 60–64 | 197 | 108 | | |
| | | | | | 65–69 | 181 | 135 | | |
| | | | | | 70–74 | 247 | 183 | | |
| | | | | | 75-79 | 209 | 177 | | |
| | | | | | 80+ | 231 | 221 | | |
| | | | | | All | 29 | 21 | | |

*continued*

**Table 12.2** *S. Japonicum* empirical databases, China *(continued)*

| *Date* | *Location* | *Prevalence (%)* | *Morbidity* | *Source* | *Results* | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|
| 1992 | China | 11.8 | 57 600 advanced cases in the entire country | Stratified group random sample = 1 per cent of the population of the endemic area: Hubei, Hunnan, Anhui, Jiangxi | *Age group (years)* | *Prevalence (%)* | China Ministry of Public Health | |
| | | | | | 0–4 | 2.8 | | |
| | | | | | 5–9 | 6.9 | | |
| | | | | | 10–14 | 10.0 | | |
| | | | | | 15–19 | 13.6 | | |
| | | | | | 20–29 | 12.3 | | |
| | | | | | 30–39 | 8.4 | | |
| | | | | | 40–49 | 13.4 | | |
| | | | | | 50–59 | 13.5 | | |
| | | | | | 60+ | 12.8 | | |
| | | | | | M | 14.2 | | |
| | | | | | F | 8.3 | | |

M = males, F = females

### OTHER ASIA AND ISLANDS

Schistosomiasis attributable to *S. japonicum* and *S. mekongi* are present in five countries of this region. Available prevalence data are summarized in Table 12.3. The prevalence of infection resulting from *S. japonicum* in Indonesia is less than 1 per cent among a population at risk of 10 000 persons. *S. malayensis* has only been reported in 10 persons of a single ethnic group in Malaysia. In Thailand, less than 5 cases have been reported. Thus schistosomiasis is prevalent only in Cambodia and the Lao People's Democratic Republic (*S. mekongi*) and the Philippines (*S. japonicum*). There are no studies on comparative morbidity between *S. japonicum* and *S. mekongi*. The distribution in the Lao People's Democratic Republic overlaps with a higher prevalence of opisthorchiasis, and increased morbidity may be present in these areas.

In the Philippines, Tanaka et al. have attempted to measure incidence among schoolchildren (Tanaka et al. 1984) and to assess the impact of treatment on incidence (Tanaka et al 1985). Incidence at age 7 years was 18.6 per cent and at age 12 years was 33.9 per cent; no difference between sexes was observed.

### SUB-SAHARAN AFRICA

Schistosomiasis attributable to *S. mansoni* or *S. haematobium* is endemic in 41 countries of this region. It is not endemic in Cape Verde, Comoros, Djibouti, and Lesotho. Representative population-based studies of the prevalence and morbidity of schistosomiasis are shown in Table 12.4. Brinkmann et al. (1988) have published data on age and sex distribution in 13 village clusters in Mali of infection with *S. mansoni* or *S. haematobium*.

Schistosomiasis resulting from *S. intercalatum* has received more attention recently and is now reported from eight West and Central African states. The geographical extent of risk is unknown, but the morbidity seems low (see Table 12.5).

One example of the limitations of estimating national prevalence of infection has been published by Ratard et al. (1992) concerning Cameroon. The data derived from a national school survey of 5th grade primary school students (average age 13 years: range 10–19 years) were extrapolated to show a maximum number of 719 000 cases (range 392 900–1 027 800) in contrast to an estimate of 2 239 591 cases (range 1 261 355–3 267 963), which was based on an assessment of published literature (Utroska et al. 1989). In general, national surveys have not been done in sub-Saharan African; where available, as in Zimbabwe (Taylor & Makura 1985), the limitations of these data are recognized and no attempt is made to estimate national prevalence.

### LATIN AMERICA AND THE CARIBBEAN

Only schistosomiasis attributable to *S. mansoni* is present in this region. Several prevalence distributions are presented in Table 12.6. Mortality due to schistosomiasis in Brazil and Surinam is discussed below.

**Table 12.3** *S. japonicum* empirical databases by region, Other Asia and Islands

| *Date* | *Location* | *Prevalence (%)* | *Morbidity* | *Source* | *Results* | | | *References* |
|---|---|---|---|---|---|---|---|---|
| 1979 | Philippines | 40.1 | 25% of those infected had hepatomegaly or splenomegaly | Village<br>N = 851 (45% of the residents) | *Age group (years)* | *Prevalence (%)* | | Lewert, Yogore & Blas (1979) |
| | | | | | | *M* | *F* | |
| | | | | | 1–4 | 0 | 2 | |
| | | | | | 5–9 | 14 | 10 | |
| | | | | | 10–14 | 57 | 37 | |
| | | | | | 15–19 | 63 | 47 | |
| | | | | | 20–29 | 73 | 44 | |
| | | | | | 30–39 | 62 | 65 | |
| | | | | | 40–49 | 71 | 60 | |
| | | | | | 50+ | 57 | 50 | |
| 1980 | Philippines | 43 | Hepatomegaly and splenomegaly assoication with infection | Village<br>N = 755 (90% of the residents) | *Age group (years)* | *Prevalence (%)* | | WHO workshop (1980) |
| | | | | | | *M* | *F* | |
| | | | | | <1 | 0 | 0 | |
| | | | | | 1–4 | 12 | 0 | |
| | | | | | 5–9 | 40 | 21 | |
| | | | | | 10–14 | 68 | 48 | |
| | | | | | 15–19 | 81 | 52 | |
| | | | | | 20–24 | 81 | 25 | |
| | | | | | 25–34 | 74 | 46 | |
| | | | | | 35–44 | 77 | 59 | |
| | | | | | 45–54 | 65 | 58 | |
| | | | | | 55–64 | 63 | 23 | |
| | | | | | 65+ | 63 | 17 | |

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<tr>
<td>1980</td>
<td>Philippines</td>
<td>35</td>
<td>Hepatomegaly associated with infection</td>
<td>Village<br>N = 1010</td>
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<tr><th rowspan="2">Age group (years)</th><th colspan="2">Prevalence (%)</th></tr>
<tr><th>M</th><th>F</th></tr>
<tr><td>1–4</td><td>4</td><td>8</td></tr>
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<tr><td>60+</td><td>42</td><td>11</td></tr>
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<td>Domingo et al. (1980)</td>
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<td>1983</td>
<td>Philippines</td>
<td>Village A: 26<br>Village B: 39<br>Village C: 44</td>
<td>Hepatomegaly and splenomegaly associated with infection</td>
<td>Village A<br>N = 289<br>Village B<br>N = 824<br>Village C<br>N = 1113</td>
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<tr><th colspan="6">Village</th></tr>
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</table>

M = males F = females

**Table 12.4** *S. mansoni* empirical databases, sub-Saharan Africa

| *Date* | *Location* | *Prevalence* | *Morbidity* | | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|---|
| 1972 | Uganda | 89% | *Age group (years)* | *Haematemesis (ever) (%)* | Village N = 231 | *Age group (years)* | *Prevalence (%) (estimated from graph)* | | Ongom & Bradley (1972) | Raw data not presented |
| | | | 0–4 | 0 | | < 1 | 30 | | | |
| | | | 5–9 | 6 | | 1–4 | 60 | | | |
| | | | 10–19 | 3 | | 5–9 | 80 | | | |
| | | | 20–39 | 9 | | 10–14 | 95 | | | |
| | | | 40+ | 9 | | 15–19 | 100 | | | |
| | | | | | | 20–29 | 100 | | | |
| | | | | | | 30–39 | 100 | | | |
| | | | | | | 40–49 | 100 | | | |
| | | | | | | 50–59 | 100 | | | |
| | | | | | | 60+ | 100 | | | |
| 1976 | Kenya | 82% | Splenomegaly 3% vs 0 in uninfected | | Village N=416 | *Age group (years)* | *Prevalence (%)* | | Siongok et al. (1976) | Kato technique used |
| | | | | | | | *M* | *F* | | |
| | | | | | | 1–4 | 71 | 47 | | |
| | | | | | | 5–9 | 91 | 95 | | |
| | | | | | | 10–19 | 100 | 97 | | |
| | | | | | | 20–29 | 97 | 80 | | |
| | | | | | | 30–39 | 80 | 67 | | |
| | | | | | | 40–49 | 73 | 70 | | |
| | | | | | | 50–59 | 67 | 58 | | |
| | | | | | | 60–69 | 100 | 67 | | |
| | | | | | | 70+ | 20 | 86 | | |

<table>
<tr><td>1976</td><td>Ethiopia</td><td>Village A = 43.3%<br>Village B = 10.7%</td><td>Low</td><td>50% household sample:<br>Village A N = 352<br>Village B N = 155</td><td>
<table>
<tr><th rowspan="3"><i>Age group (years)</i></th><th colspan="4"><i>Prevalence (%)</i></th></tr>
<tr><th colspan="2"><i>Village A</i></th><th colspan="2"><i>Village B</i></th></tr>
<tr><th><i>M</i></th><th><i>F</i></th><th><i>M</i></th><th><i>F</i></th></tr>
<tr><td>1–4</td><td>12</td><td>8</td><td>0</td><td>0</td></tr>
<tr><td>5–9</td><td>52</td><td>49</td><td>6</td><td>7</td></tr>
<tr><td>10–14</td><td>93</td><td>60</td><td>18</td><td></td></tr>
<tr><td>15–19</td><td>85</td><td>25</td><td>5</td><td>20</td></tr>
<tr><td>20–29</td><td>81</td><td>27</td><td>8</td><td>29</td></tr>
<tr><td>30–39</td><td>48</td><td>25</td><td>0</td><td>0</td></tr>
<tr><td>40–49</td><td>36</td><td>9</td><td>0</td><td>0</td></tr>
<tr><td>50+</td><td>22</td><td>36</td><td>0</td><td>0</td></tr>
</table>
</td><td>Hiatt (1976)</td><td>Kato technique used</td></tr>
<tr><td>1979</td><td>Kenya</td><td>47%</td><td>Abdominal pain</td><td>30% household sample</td><td>
<table>
<tr><th rowspan="2"><i>Age group (years)</i></th><th colspan="2"><i>Prevalence (%)</i></th></tr>
<tr><th><i>M</i></th><th><i>F</i></th></tr>
<tr><td>0–4</td><td>15</td><td>17</td></tr>
<tr><td>5–9</td><td>23</td><td>32</td></tr>
<tr><td>10–14</td><td>57</td><td>55</td></tr>
<tr><td>15–19</td><td>73</td><td>50</td></tr>
<tr><td>20–24</td><td>85</td><td>38</td></tr>
<tr><td>25–29</td><td>78</td><td>67</td></tr>
<tr><td>30–39</td><td>58</td><td>57</td></tr>
<tr><td>40–49</td><td>100</td><td>47</td></tr>
<tr><td>50–59</td><td>71</td><td>67</td></tr>
<tr><td>60+</td><td>90</td><td>50</td></tr>
</table>
</td><td>Smith, Warren & Mahmoud (1979)</td><td>Kato technique used</td></tr>
</table>

*continued*

**Table 12.4** *S. mansoni* empirical databases, sub-Saharan Africa *(continued)*

| *Date* | *Location* | *Prevalence* | *Morbidity* | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|
| 1988 | Burundi | 33% | Hepatomegaly and splenomegaly associated with infection | 5% population sample<br>n = 6 203 | *Age group (years)* | *Prevalence (%)* *M* | *F* | Gryseels (1988) | Kato technique used |
| | | | | | 0–4 | 11 | | | |
| | | | | | 5–9 | 27 | | | |
| | | | | | 10–14 | 43 | | | |
| | | | | | 15–19 | 46 | | | |
| | | | | | 20–29 | 44 | | | |
| | | | | | 30–39 | 37 | | | |
| | | | | | 40–49 | 34 | | | |
| | | | | | 50–59 | 35 | | | |
| | | | | | 60+ | 36 | | | |
| 1992 | Uganda | M = 82%<br>F = 81% | High intensity of infection | 20% population sample | *Age group (years)* | *Prevalence (%) (estimated from graph)* *M* | *F* | Kabaterine et al. (1992) | |
| | | | | | 0–4 | 51 | 73 | | |
| | | | | | 5–9 | 83 | 77 | | |
| | | | | | 10–14 | 93 | 94 | | |
| | | | | | 15–19 | 90 | 91 | | |
| | | | | | 20–29 | 90 | 81 | | |
| | | | | | 30–39 | 91 | 76 | | |
| | | | | | 40+ | 75 | 84 | | |

M = males F = females

**Table 12.5** *S. intercalatum* empirical databases by region, sub-Saharan Africa

| *Date* | *Location* | *Prevalence (%)* | *Morbidity* | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|
| 1990 | Equatorial Guinea | 21 | Diarrhoea and blood in the stool associated with infection | Periurban<br>N = 1221 | *Age group (years)* | *Prevalence (%)* *M* | *Prevalence (%)* *F* | Simarro, Sima & Mir (1990) | Note infection almost absent after age 45 years.<br>Kato technique used. |
| | | | | | 0–4 | 12 | 10 | | |
| | | | | | 5–9 | 38 | 35 | | |
| | | | | | 10–14 | 32 | 48 | | |
| | | | | | 15–24 | 18 | 26 | | |
| | | | | | 25–44 | 7 | 9 | | |
| | | | | | 45+ | 3 | 9 | | |
| 1992 | Gabon | 29 | Splenomegaly caused by infection | Village<br>N = 354 | *Age group (years)* | *Prevalence (%)* *M* | *Prevalence (%)* *F* | Martin-Prevel et al. (1992) | Sedimentation used first, Kato for quantification. |
| | | | | | 0–4 | 14 | 8 | | |
| | | | | | 5–9 | 48 | 46 | | |
| | | | | | 10–14 | 52 | 65 | | |
| | | | | | 15–19 | 60 | 53 | | |
| | | | | | 20–29 | 50 | 35 | | |
| | | | | | 30–39 | 25 | 22 | | |
| | | | | | 40–49 | 0 | 18 | | |
| | | | | | 50–59 | 0 | 0 | | |
| | | | | | 60+ | 7 | 4 | | |

M = males, F = females

**Table 12.6** *S. mansoni* empirical databases by region, Latin America and the Caribbean

| *Date* | *Location* | *Prevalence (%)* | *Morbidity* | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|
| 1962 | Brazil | 83 | 52% spleen | Town | Age 10–14 years = 50% of spleen and heaviest infections | | | Kloetzel (1962) | First study of its kind Limited to children |
| 1976 | Brazil | 80 | | Groups N = 363 | *Age group (years)* | *Prevalence (%)* *M* | *Prevalence (%)* *F* | Lehman et al. (1976) | Kato technique used |
| | | | | | < 1 | 10 | 50 | | |
| | | | | | 1–4 | 39 | 33 | | |
| | | | | | 5–9 | 65 | 77 | | |
| | | | | | 10–14 | 69 | 86 | | |
| | | | | | 15–19 | 89 | 86 | | |
| | | | | | 20–24 | 100 | 100 | | |
| | | | | | 25–34 | 85 | 96 | | |
| | | | | | 35–64 | 80 | 90 | | |
| | | | | | 65+ | 73 | 79 | | |

M = males, F = females

### MIDDLE EASTERN CRESCENT

*S. haematobium* is endemic in 15 countries in this region and *S. mansoni* is present in 7 of these countries. Some distributions of prevalence by age and sex are shown in Tables 12.7 and 12.8.

## MORTALITY FROM SCHISTOSOMIASIS

Mortality from schistosomiasis has been poorly documented in most endemic countries. Death certificates and patients' records rarely identify schistosomiasis as the underlying cause of death. Well-designed prospective autopsy studies have nevertheless been published (Cheever et al. 1978). In that study, schistosomiasis was the direct cause of death in 9.2 per cent of the infected individuals and was the attributable cause of death in 6.2 per cent of all deaths. The breakdown of these causes among those infected is shown in Table 12.9. Table 12.10 summarizes the results of available mortality studies by World Bank region; these results are discussed below.

### SUB-SAHARAN AFRICA

Annual mortality attributable to *S. haematobium* infection in East Africa has been estimated at 1–2 per 1000 infected adults (Forsyth 1969). In Chad, in 1966, the mortality rate of schistosomiasis attributable to *S. haematobium* infection was 1 per 1000 infected persons (Buck et al. 1970).

### LATIN AMERICAN AND THE CARIBBEAN

In 1984, the annual mortality attributable to schistosomiasis caused by *S. mansoni* in Brazil was estimated at 0.5 per 100 000 total population; at the same time in Suriname the figure was estimated to be 2.4 per 100 000 inhabitants. The control of schistosomiasis through large-scale chemotherapy in Brazil was associated with a decline in annual mortality between 1977 and 1988, from 0.67 to 0.44 deaths per 100 000 inhabitants. *S. intercalatum* infection has never been reported as a cause of death in the region.

### CHINA

Before the introduction of praziquantel in China, severe acute schistosomiasis attributable to *S. japonicum* had a 2.5–20.7 per cent mortality rate, and in Leyte, the Philippines, the annual mortality among 135 untreated patients with severe schistosomiasis was 1.8 per cent (Blas et al. 1986). It is expected that the more widespread use of current antischistosomal drugs for morbidity control in highly endemic areas will also reduce mortality.

The national survey by the Ministry of Health of China appears to confirm the total annual mortality rate. A total of 982 severe cases were detected among 165 384 people examined; (16 953 people in the sample cohort were infected). If it is assumed that only infected people had severe disease, the rate of severe cases was 5.79 per cent. This is probably not reasonable since many advanced cases do not excrete eggs. The peak mor-

**Table 12.7** *S. haematobium* empirical databases by region, Middle Eastern Crescent

| *Date* | *Location* | *Prevalence (%)* | *Morbidity* | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|
| 1981 | Egypt | 29 | | 2 villages<br>N = 2403 | *Age group (years)* | *Prevalence (%) (estimated)* *M* | *F* | Mansour et al. (1981) | |
| | | | | | <1 | 18 | 20 | | |
| | | | | | 1–4 | 19 | 18 | | |
| | | | | | 5–9 | 55 | 36 | | |
| | | | | | 10–14 | 75 | 43 | | |
| | | | | | 15–19 | 54 | 18 | | |
| | | | | | 20–24 | 30 | 15 | | |
| | | | | | 25–29 | 35 | 9 | | |
| | | | | | 30–39 | 27 | 5 | | |
| | | | | | 40–49 | 30 | 7 | | |
| | | | | | 50–59 | 26 | 5 | | |
| | | | | | 60+ | 24 | 12 | | |
| 1982 | Egypt | 37 | | 6 villages<br>N = 5 998 | *Age group (years)* | *Prevalence (%)* *M* | *F* | King et al. (1982) | |
| | | | | | 1–4 | 20 | 21 | | |
| | | | | | 5–9 | 57 | 41 | | |
| | | | | | 10–14 | 75 | 49 | | |
| | | | | | 15–19 | 66 | 35 | | |
| | | | | | 20–24 | 41 | 21 | | |
| | | | | | 25–29 | 42 | 17 | | |
| | | | | | 30–39 | 43 | 11 | | |
| | | | | | 40–49 | 42 | 12 | | |
| | | | | | 50–59 | 40 | 13 | | |
| | | | | | 60+ | 32 | 13 | | |

M = males, F = females

**Table 12.8** *S.mansoni* empirical databases by region, Middle Eastern Crescent

| *Date* | *Location* | *Prevalence* | *Morbidity* | | | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1976 | Sudan | 48 | Spleen | | | Village<br>N = 1747 | *Age group (years)* | *Prevalence (%)* | | Omer et al. (1976) | |
| | | | *Age group (years)* | *Infected (%)* | *Not infected (%)* | | | *M* | *F* | | |
| | | | 1–4 | 9 | 4 | | 5–9 | 24 | 24 | | |
| | | | 5–9 | 22 | 10 | | 10–19 | 80 | 80 | | |
| | | | 10–14 | 29 | 23 | | 20–29 | 60 | 50 | | |
| | | | 15–19 | 20 | 6 | | 30–39 | 55 | 45 | | |
| | | | 20–39 | 17 | 17 | | 40–49 | 40 | 22 | | |
| | | | 40+ | 8 | 6 | | 50+ | 50 | 15 | | |
| 1977 | Egypt | *S. mansoni:* 40.5 | | | | 25% sample in 8 villages<br>N = 8712 | *Age group (years)* | *Prevalence (%) (estimated from graph)* | | El-Alamy & Cline (1977) | |
| | | | | | | | 2–5 | 15 | | | |
| | | | | | | | 6–10 | 33 | | | |
| | | | | | | | 11–15 | 50 | | | |
| | | | | | | | 16–20 | 40 | | | |
| | | | | | | | 21–30 | 25 | | | |
| | | | | | | | 31+ | 18 | | | |

*continued*

**Table 12.8** *S.mansoni* empirical databases by region, Middle Eastern Crescent *(continued)*

| *Date* | *Location* | *Prevalence* | *Morbidity* | | | *Source* | *Results* | | | *Reference* | *Comments* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1980 | Egypt | 74 | Intensity (geometric mean no. eggs/gram) | | | Village<br>N = 537 | | *Prevalence (%)* | | Abdel-Wahab et al. (1980) | |
| | | | *Age group (years)* | *M* | *F* | | *Age group (years)* | *M* | *F* | | |
| | | | 1–4 | 134 | 95 | | 1–4 | 25 | 33 | | |
| | | | 5–9 | 204 | 67 | | 5–9 | 71 | 57 | | |
| | | | 10–19 | 331 | 213 | | 10–19 | 89 | 81 | | |
| | | | 20–29 | 165 | 199 | | 20–29 | 80 | 71 | | |
| | | | 30–39 | 233 | 151 | | 30–39 | 86 | 78 | | |
| | | | 40–49 | 194 | 109 | | 40–49 | 80 | 56 | | |
| | | | 50–59 | 194 | 112 | | 50–59 | 75 | 55 | | |
| | | | 60+ | 83 | 100 | | 60+ | 50 | 50 | | |

M = males, F = females

**Table 12.9** Causes of death related to schistosomiasis

| *Attributed cause of death* | *Percentage of deaths* | *Mean age (years)* |
|---|---|---|
| Obstructive uropathy | 2.5 | 42 |
| Bladder cancer | 2.4 | 38 |
| Symmer's fibrosis | 3.1 | 30 |
| Colonic polyposis | 0.8 | 35 |
| Salmonellosis | 0.4 | — |
| Total deaths in infected persons | 9.2 | |

Source: Cheever et al. (1978).

tality was in the 45–59 year age group. Overall mortality rates were 29 per million population for males and 21 per million population for females.

The most recent data on mortality from to schistosomiasis have been produced by a study of 65 Chinese counties (Junshi et al. 1990). Schistosomiasis ranked as the most significant cause of death resulting from colorectal, rectal and colon cancer. Only 20 of these counties were currently endemic for schistosomiasis. The cumulative mortality rate attributable to schistosomiasis for both sexes was as high as 38.99 per 1000 deaths.

*S. japonicum* is possibly carcinogenic in humans, causing colorectal carcinoma. The cumulative data from China support this conclusion; see also the comprehensive review by the International Agency for Research on Cancer (1994).

### OTHER ASIA AND ISLANDS

One in 20 deaths in the Khong district of the Lao People's Democratic Republic were attributable to schistosomiasis (*S. mekongi*) according to survey data obtained in preparation for the national control programme on opisthorchiasis and schistosomiasis (A. Sleigh, personal communication 1992).

The case fatality rate of schistosomiasis attributable to *Schistosoma japonicum* infection in the Philippines is estimated to be 1.78 per cent per year (Blas. et al 1986). The recognized number of cases in Thailand and Malaysia is so low that neither the populations nor the populations at risk should be included in the totals.

### MIDDLE EASTERN CRESCENT

Mortality from schistosomiasis is the cause of 6 per cent of all deaths in Egypt. The annual crude mortality rate is 1 per 1000 infected individuals, and is higher in men than in women. The male-to-female sex ratio is 47.2:23.0, based on the combined raw data of King et al. (1982) and Mansour et al. (1981).

**Table 12.10** Studies of mortality rates associated with schistosomiasis, World Bank regions

| *Region and type* | *Reference* | *Sex* | *Age group (years)* | *Population (number)* | *Number of people infected* | *Number of deaths per year* |
|---|---|---|---|---|---|---|
| China — *S. japonicum* only | China Ministry of Public Health (1992) | Males | 0–14 | 157 240 000 | 205 427 | 50 |
| | | | 15–65+ | 427 959 000 | 741 240 | 530 |
| | | Females | 0–14 | 148 347 000 | 120 073 | 40 |
| | | | 15–65+ | 400 147 000 | 433 260 | 380 |
| Middle East Crescent — *S. mansoni* | El Malatawy et al. (1992) | Males | 0–14 | 106 506 000 | 1 550 687 | 1.5 |
| | | | 15–65+ | 149 883 000 | 3 419 463 | 34 |
| | | Females | 0–14 | 101 733 000 | 1 347 793 | 3 |
| | | | 15–65+ | 144 953 000 | 2 972 057 | 30 |
| Middle East Crescent — *S. haematobium* | Utroska et al. 1989. | Males | 0–14 | 106 506 000 | 2 127 152 | 21 |
| | | | 15–65+ | 149 883 000 | 1 719 416 | 1 719 |
| | | Females | 0–14 | 101 733 000 | 1 036 544 | 10 |
| | | | 15–65+ | 144 953 000 | 837 858 | 600 |
| Latin America & Caribbean — *S. mansoni* only | Lopes (1984) | Males | 0–14 | 80 845 000 | 1 755 725 | 3 |
| | | | 15–65+ | 140 766 000 | 3 244 275 | 64 |
| | | Females | 0–14 | 78 420 000 | 1 986 486 | 3 |
| | | | 15–65+ | 144 264 000 | 2 913 514 | 62 |
| Sub-Saharan Africa — *S. haematobium* | Buck et al. (1970) | Males | 0–14 | 117 742 000 | 27 318 640 | 27 |
| | | | 15–65+ | 134 580 000 | 22 082 160 | 2 208 |
| | | Females | 0–14 | 116 848 000 | 21 607 502 | 21 |
| | | | 15–65+ | 141 104 000 | 13 872 797 | 1 000 |
| Other Asian Countries & Islands — *S. japonicum* (Philippines & Indonesia) | Blas et al. 1986 | Males | 0–14 | | 151 252 | 3 |
| | | | 15–65+ | | 208 748 | 55 |
| | | Females | 0–14 | | 99 248 | 2 |
| | | | 15–65+ | | 140 752 | 39 |
| *S. mekongi* (Lao PDR & Cambodia) | A. Sleigh (personal communication 1992) | Males | 0-14 | | 50 000 | 5 |
| | | | 15–65+ | | 75 000 | 25 |
| | | Females | 0–14 | | 50 000 | 5 |
| | | | 15–65+ | | 75 000 | 25 |

## DISABILITY

In a critical review, Prescott (1979) pointed out that, at that time, no clear empirical basis had been found for the assumption that schistosomiasis seriously impairs labour productivity. He attributed this to possible sampling bias resulting in the exclusion of workers with severe disease. Subsequent studies have lent more support to the thesis that not only does severe infection cause disability but infection alone limits productivity and well-being.

In the workplace, decreased productivity has infrequently been shown to be the result of to schistosomiasis. In contrast, increased absenteeism has consistently been shown to be related to schistosomiasis. In irrigation workers, who are at greatest risk, schistosomiasis infection was associated with a 3.67 per cent greater absenteeism rate than among those not infected; in cane-cutters, schistosomiasis infection was associated with 1.64 per cent greater absenteeism (Foster 1967). These figures are based on observation of over 28 000 consecutive work shifts and the difference related to the irrigation workers was considered statistically significant. As Prescott points out, the determinants of absenteeism were not analysed.

Since Prescott's review, a substantial amount of data has been published supporting the hypothesis that infection without clinical manifestations causes work days lost: 26.2 to 41.6 work days lost per year as a result of *S. japonicum* (Blas 1989); 4.4 work days per year as a result of *S. haematobium* (Ghana Health Assessment Project Team 1981, Morrow et al. 1984). Absenteeism was noted to be twice as high among labourers infected with *S. mansoni* as among those uninfected (Ndamba et al. 1991); 6 working days per year were estimated to be lost due to *S. mansoni* infection (Chowdhry & Levy 1988); in Madagascar 50 per cent of people reported unable to work had evidence of urinary schistosomiasis and remained out of work for at least 10 days (Breuil, Moyroud & Coulanges 1983). There is a latency period of 5–15 years between infection and development of severe disease due to *S. mansoni*, thus hepatosplenomegaly with portal hypertension attributable to *S. mansoni* occurs after age 20 years. Approximately 10 per cent of those infected will develop severe disease.

Since 1980 a number of studies have shown that schistosomiasis resulting from *S. mansoni* has a negative effect on productivity. A reduction of 18 per cent in the work output of those individuals with heavy infection or hepatosplenomegaly can be expected (Awad El Karim et al. 1980). According to a recent study, physical performance increased 4.3 per cent after treatment, and work output (as measured by the amount of sugar cane cut in a given time) increased 16.6 per cent (Ndamba et al. 1993). Infected individuals without organomegaly were observed to have no reduction of work output (Van Ee & Polderman 1984).

A minimum of 12 per cent lower exercise capacity was found in children with *S. haematobium* infection in Zimbabwe (Ndamba 1986). This deficit was recovered within one month after treatment. Similarly, in Kenya, a

7–10 per cent improvement in exercise capacity was found one month after treatment in children with *S. haematobium* infection (Latham et al. 1990).

Of those with hepatosplenomegaly attributable to *S. mansoni*, 33 to 67 per cent have oesophageal varices (De Cock et al. 1982, Saad et al. 1991). Of those with oesophageal varices, 3 to 4 per cent have recurrent upper gastrointestinal bleeding. After oesophageal varices have bled once, there is a tendency for the bleeding to recur, and 3–4 months per year will be lost either in or out of hospital.

Disease of the central nervous system, affecting the spinal cord, is more frequent and causes more disability than is currently recognized, especially among migrants into endemic areas of *S. mansoni* transmission (World Health Organization 1989, Joubert et al. 1990). In the Philippines, the minimum contribution of schistosomiasis attributable to *S. japonicum* to the etiology of epilepsy is 6 per 1000 of the total rate of 11 per 1000 (Hayashi 1979). Spinal cord involvement attributable to schistosomiasis (*S. mansoni* mostly) is the cause of 1 in 4 of all hospital admissions to the neurological nervice of a general hospital in Durban, South Africa (Haribhai et al. 1991).

Average disability weights for cases of schistosomiasis infection were estimated using expert panels, person-trade off methods for 22 indicator conditions, and rankings of other conditions (including schistosomiasis) against the indicator conditions as described by Murray & Lopez (1996a). The resulting average disability weights were 0.005 for children aged 0–14 years and 0.006 for adults aged 15 years and over.

## ESTIMATION OF DISABILITY-ADJUSTED LIFE YEARS

General methods used for the calculation of disability-adjusted life years (DALYs) lost due to a disease are given by Murray & Lopez (1996a). DALYs are the sum of years of life lost due to mortality (YLLs) and years lived with disability (YLDs). The prevalence of schistosomiasis and deaths attributable to schistosomiasis were calculated for the eight World Bank regions, based on the data reviewed above. The resulting estimates are shown in Tables 12.11 and 12.12 and have also been published by Murray & Lopez (1996b).

YLLs are calculated directly from the deaths shown in Tables 12.11 and 12.12 Total global deaths in 1990 resulting from schistosomiasis were estimated to be around 8000. It must be emphasised that these are direct schistosomiasis deaths and do not include the cancer and other disease deaths attributable to schistosomiasis (as discussed below).

YLDs are usually calculated from regional estimates of incident cases and average durations. However, because of the very limited information available to estimate incidence rates for schistosomiasis from prevalence and case fatality data, YLDs for schistosomiasis were calculated directly from the prevalence estimates (in effect assuming a one year duration and prevalence equal to incidence).

Tables 12.11 and 12.12 also show the estimated DALYs for the eight World Bank regions and the regional variation in DALYs per 100 000 population resulting from schistosomiasis.

## SCHISTOSOMIASIS AS A RISK FACTOR FOR OTHER DISEASES

### CANCER

Squamous cell bladder carcinoma is the leading cause of cancer among 20–44 year old men in Egypt and is a leading cause of death in this age group (Koroltchouk et al. 1987). El-Bolkainy & Chu (1981) estimated that the rate of squamous cell carcinoma among people with *S. haematobium* infection in Egypt is 2 per 1000 and 4 per 1000 among rural residents. In an older study, squamous cell bladder cancer was estimated to occur in 10 of every 1000 people infected with *S. haematobium* (Halawani & Tamami 1955); 26 per cent of deaths attributable to schistosomiasis were associated with bladder cancer in Egypt. The latency between initial infection and onset was estimated to be 20–30 years, and the male-to-female ratio was 9.5:1.

In Angola the peak age of incidence of squamous cell carcinoma of the bladder is 44 years of age. An incidence rate of 8 per 10 000 rural inhabitants is suggested on the basis of epidemiological and clinical data (Lopes 1984). In Malawi, Mozambique and Zambia, the incidence rates of squamous cell bladder cancer are eight times as high as in the United States of America or the United Kingdom (Lucas 1982).

It has been estimated that primary prevention to control urinary schistosomiasis would reduce the global rate of carcinoma of the bladder by 5000–10 000 cases per year (Koroltchouk et al. 1987).

### OTHER DISEASES

Intestinal schistosomiasis caused by *S. mansoni* and *S. japonicum* may complicate a variety of other diseases that increase disability.

*Typhoid fever.* If a person with schistosomiasis acquires typhoid fever, the duration of the typhoid fever will increase 2–3 times the average duration and may lead to total incapacity or death in 30 per cent of untreated persons (Salih et al. 1977).

*Hepatitis B.* The effect of schistosomiasis infection on concurrent hepatitis B has been recently reviewed, and the limitations of the interpretation of clinical and epidemiological data have been analysed (Chen et al. 1993). There is a consensus among clinicians in the highly endemic areas of China and Egypt that in patients with schistosomiasis the duration of hepatitis increases up to 5 times the average, and the risk of chronic liver disease is greater and may result in total incapacity (Ghaffar et al. 1990).

**Table 12.11** Epidemiological estimates for schistosomiasis, 1990, males

| | Prevalence | | Deaths | | YLLs | | YLDs | | DALYs | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Age group (years)* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* |
| *Established Market Economies (EME)* | | | | | | | | | | |
| 0–4 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 15–44 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 45–59 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 60+ | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| All Ages | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| *Formerly Socialist Economies (FSE)* | | | | | | | | | | |
| 0–4 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 15–44 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 45–59 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 60+ | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| All Ages | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| *India* | | | | | | | | | | |
| 0–4 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 15–44 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 45–59 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 60+ | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| All Ages | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| *China* | | | | | | | | | | |
| 0–4 | 35 | 59 | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 170 | 175 | 0 | — | — | 0 | 1 | 1 | 1 | 1 |
| 15–44 | 501 | 164 | 0 | 0 | 4 | 1 | 4 | 1 | 9 | 3 |
| 45–59 | 145 | 199 | 0 | 0 | 4 | 6 | 1 | 1 | 5 | 7 |
| 60+ | 96 | 196 | 0 | 1 | 2 | 4 | — | 0 | 3 | 6 |
| All Ages | 947 | 162 | 1 | 0 | 11 | 2 | 7 | 1 | 17 | 3 |
| *Other Asia and Islands* | | | | | | | | | | |
| 0–4 | 35 | 79 | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 167 | 198 | 0 | — | — | 0 | 1 | 1 | 1 | 1 |
| 15–44 | 192 | 119 | 0 | — | 1 | 1 | 2 | 1 | 3 | 2 |
| 45–59 | 55 | 162 | 0 | 0 | 2 | 6 | — | 0 | 2 | 6 |
| 60+ | 37 | 182 | 0 | 1 | 1 | 5 | — | 0 | 1 | 5 |
| *All Ages* | 485 | 141 | 0 | 0 | 4 | 1 | 3 | 1 | 7 | 2 |
| *Sub–Saharan Africa* | | | | | | | | | | |
| 0–4 | 11 148 | 23 478 | 0 | — | 1 | 2 | 24 | 51 | 25 | 53 |
| 5–14 | 45 013 | 64 068 | 0 | 0 | 12 | 17 | 261 | 371 | 273 | 389 |
| 15–44 | 39 991 | 38 540 | 1 | 1 | 22 | 21 | 348 | 335 | 371 | 358 |
| 45–59 | 7652 | 37 682 | 1 | 3 | 8 | 39 | 47 | 231 | 56 | 276 |
| 60+ | 3442 | 32 758 | 0 | 5 | 3 | 29 | 14 | 133 | 18 | 171 |
| *All Ages* | 107 246 | 42 504 | 2 | 1 | 47 | 19 | 695 | 275 | 742 | 294 |
| *Latin America and the Carribean* | | | | | | | | | | |
| 0–4 | 301 | 1 047 | | | — | | 1 | 3 | 1 | 3 |
| 5–14 | 1 455 | 2 791 | | | — | | 8 | 15 | 9 | 17 |
| 15–44 | 2 549 | 2 444 | | | 3 | 3 | 22 | 21 | 25 | 24 |
| 45–59 | 496 | 2 230 | | | 1 | 4 | 3 | 13 | 4 | 18 |
| 60+ | 199 | 1 398 | | | — | | 1 | 7 | 1 | 7 |
| *All Ages* | 5 000 | 2 256 | | | 5 | 2 | 35 | 16 | 40 | 18 |

*continued*

**Table 12.11** Epidemiological estimates for schistosomiasis, 1990, males *(continued)*

| | *Prevalence* | | *Deaths* | | *YLLs* | | *YLDs* | | *DALYs* | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Age group (years)* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* |
| *Middle Eastern Crescent* | | | | | | | | | | |
| 0–4 | 630 | 1 531 | | | — | | 1 | 2 | 2 | 5 |
| 5–14 | 3 048 | 4 664 | | | 7 | 11 | 18 | 28 | 25 | 38 |
| 15–44 | 4 038 | 3 545 | 1 | 1 | 15 | 13 | 35 | 31 | 50 | 44 |
| 45–59 | 786 | 3 519 | | 2 | 6 | 27 | 5 | 22 | 10 | 45 |
| 60+ | 315 | 2 308 | | 2 | 2 | 15 | 1 | 7 | 4 | 29 |
| *All Ages* | 8 817 | 3 439 | 1 | 1 | 30 | 12 | 60 | 23 | 91 | 35 |
| *World* | | | | | | | | | | |
| 0–4 | 12 149 | 3 781 | | | 1 | | 26 | 8 | 28 | 9 |
| 5–14 | 49 852 | 9 044 | 1 | | 19 | 3 | 289 | 52 | 308 | 56 |
| 15–44 | 47 271 | 3 782 | 2 | | 46 | 4 | 412 | 33 | 458 | 37 |
| 45–59 | 9 135 | 2 924 | 1 | | 21 | 7 | 56 | 18 | 77 | 25 |
| 60+ | 4 089 | 1 868 | 1 | | 10 | 5 | 17 | 8 | 27 | 12 |
| *All Ages* | 122 495 | 4 616 | 5 | | 97 | 4 | 800 | 30 | 898 | 34 |

YLLs, years of life lost; YLDs, years lived with disability; DALYs, disability-adjusted life years.

Furthermore, there is a decreased response to hepatitis B vaccine (Bassily et al. 1987, Ghaffar et al. 1990).

*Rift Valley Fever.* During epidemics of Rift Valley Fever in Egypt and East Africa, it has been hypothesized that persons with intestinal schistosomiasis had a high mortality rate due to liver failure (WHO Regional Office for the Eastern Mediterranean 1983). The haemorrhagic form may occur in up to 6 per cent of patients and the mortality rate of this form of RVF is about 30 per cent.

## ECONOMIC BURDEN AND INTERVENTION

The economic constraints in developing countries where schistosomiasis is endemic require careful analysis and review to assess the feasibility of control with limited resources. The cost of disease and the price of health have been a focus of attention of the World Health Organization since its inception (Winslow 1951).

### ECONOMIC COSTS

The economic costs of schistosomiasis are substantial. In 1953 schistosomiasis was estimated to cost Egypt about U$57 million dollars and to decrease productivity by 33 per cent (Wright & Dobrovolny 1953). The projected savings to be made by eradicating schistosomiasis from Japan was estimated in 1952 to be US$3 million per year (Hunter et al. 1952). In Fukuoka and Saga prefectures alone there were 28 617 infected people representing 24 213 520 man-hours lost per year and wages lost per year equivalent to US$2 522 200, while the estimated cost of treatment of infected invividuals per year was U$177 938 (U$6.22 per person).

Audibert (1986) estimated that, in the rice irrigation projects of north Cameroon, a 10 per cent increase in the prevalence of schistosomiasis resulted in a 4.9 per cent decrease in rice output.

### ESTIMATION OF COSTS OF INTERVENTIONS

An estimation of the cost of different interventions is the first step in a cost-effectiveness analysis of control strategy options. In a strategy aimed primarily at morbidity control, the main elements are chemotherapy and health education. The simplest item to estimate is the actual cost of the antischistosomal drugs, but the cost of central and peripheral storage, transport to distribution centres, and administrative support must also be considered.

After choosing the most appropriate drug and establishing a dosage schedule, the next cost is that of delivery. This will reflect the choice of drug and the dosage schedule. Cost calculations must also include the requirements for diagnosis. Selective diagnosis of *S. haematobium* infection using questionnaires or chemical-reagent strips costs relatively little compared with the high cost of stool examination to detect *S. mansoni*

**Table 12.12** Epidemiological estimates for schistosomiasis, 1990, females

| | *Prevalence* | | *Deaths* | | *YLLs* | | *YLDs* | | *DALYs* | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Age group (years)* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* |
| *Established Market Economies (EME)* | | | | | | | | | | |
| 0–4 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 15–44 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 45–59 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 60+ | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| All ages | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| *Formerly Socialist Economies* | | | | | | | | | | |
| 0–4 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 15–44 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 45–59 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 60+ | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| All Aaes | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| *India* | | | | | | | | | | |
| 0–4 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 15–44 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 45–59 | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 60+ | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |
| All ages | 0 | — | 0 | — | — | 0 | — | 0 | 0 | 0 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| *China* | | | | | | | | | | |
| 0–4 | 21 | 36 | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 99 | 110 | 0 | — | — | 0 | 1 | 1 | 1 | 1 |
| 15–44 | 293 | 103 | 0 | — | 1 | 0 | 3 | 1 | 4 | 1 |
| 45–59 | 84 | 131 | 0 | 0 | 2 | 3 | 1 | 2 | 3 | 5 |
| 60+ | 56 | 108 | 0 | 1 | 2 | 4 | — | 0 | 2 | 4 |
| All ages | 553 | 101 | 1 | 0 | 5 | 1 | 4 | 1 | 9 | 2 |
| *Other Asia and Islands* | | | | | | | | | | |
| 0–4 | 26 | 61 | 0 | — | — | 0 | — | 0 | 0 | 0 |
| 5–14 | 124 | 154 | 0 | — | — | 0 | 1 | 1 | 1 | 1 |
| 15–44 | 146 | 91 | 0 | — | — | 0 | 1 | 1 | 2 | 1 |
| 45–59 | 42 | 120 | 0 | 0 | 1 | 3 | — | 0 | 1 | 3 |
| 60+ | 28 | 12 | 0 | 1 | — | 0 | — | 0 | 1 | 4 |
| All ages | 365 | 107 | 1 | 0 | 1 | 0 | 2 | 1 | 5 | 1 |
| *Sub–Saharan Africa* | | | | | | | | | | |
| 0–4 | 7 512 | 15 972 | 0 | — | — | 0 | 16 | 34 | 17 | 36 |
| 5–14 | 30 330 | 43 442 | 0 | 0 | 8 | 12 | 176 | 252 | 184 | 264 |
| 15–44 | 28 124 | 26 468 | 1 | 1 | 19 | 18 | 245 | 231 | 264 | 248 |
| 45–59 | 5 382 | 24 333 | 0 | 1 | 4 | 18 | 33 | 149 | 37 | 167 |
| 60+ | 2 421 | 19 017 | 0 | 2 | 1 | 8 | 10 | 79 | 11 | 86 |
| All ages | 73 769 | 28 598 | 1 | 1 | 33 | 13 | 480 | 186 | 513 | 199 |
| *Latin America and the Caribbean* | | | | | | | | | | |
| 0–4 | 340 | 1 230 | 0 | — | — | 0 | 1 | 4 | 1 | 4 |
| 5–14 | 1 646 | 3 244 | 0 | — | — | 0 | 10 | 20 | 10 | 20 |
| 15–44 | 2 289 | 2 199 | 0 | 0 | 3 | 3 | 20 | 19 | 22 | 21 |
| 45–59 | 446 | 1 908 | 0 | 0 | 1 | 4 | 3 | 13 | 4 | 17 |
| 60+ | 179 | 1 062 | 0 | 0 | — | 0 | 1 | 6 | 1 | 6 |
| All ages | 4 900 | 2 200 | 1 | 0 | 5 | 2 | 34 | 15 | 38 | 17 |

*continued*

**Table 12.12** Epidemiological estimates for schistosomiasis, 1990, females *(continued)*

| | *Prevalence* | | *Deaths* | | *YLLs* | | *YLDs* | | *DALYs* | |
|---|---|---|---|---|---|---|---|---|---|---|
| *Age group (years)* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* | *Number (thousands)* | *Rate per 100 000* |
| *Middle Eastern Crescent* | | | | | | | | | | |
| 0–4 | 408 | 1 028 | 0 | — | — | 0 | 1 | 3 | 1 | 3 |
| 5–14 | 1 976 | 3 187 | 0 | — | — | 0 | 11 | 18 | 11 | 18 |
| 15–44 | 2 994 | 2 792 | 0 | 0 | 10 | 9 | 26 | 24 | 36 | 34 |
| 45–59 | 584 | 2 614 | 0 | 1 | 2 | 9 | 4 | 18 | 6 | 27 |
| 60+ | 234 | 1 512 | 0 | 1 | 1 | 7 | 1 | 6 | 2 | 13 |
| All ages | 6 194 | 2 511 | 1 | 0 | 13 | 5 | 43 | 17 | 56 | 23 |
| *World* | | | | | | | | | | |
| 0–4 | 8 307 | 2 686 | 0 | 0 | 0 | 0 | 18 | 6 | 19 | 6 |
| 5–14 | 34 175 | 6 503 | 0 | 0 | 9 | 2 | 198 | 38 | 207 | 39 |
| 15–44 | 33 846 | 2 824 | 1 | 0 | 33 | 3 | 295 | 25 | 328 | 27 |
| 45–59 | 6 537 | 2 101 | 1 | 0 | 11 | 4 | 40 | 13 | 51 | 16 |
| 60+ | 2 917 | 1 084 | 1 | 0 | 6 | 2 | 12 | 4 | 17 | 6 |
| All ages | 85 781 | 3 282 | 3 | 0 | *58* | 2 | *563* | 22 | 622 | 24 |

YLLs, years of life lost; YLDs, years lived with disability; DALYs, disability-adjusted life years.

infection. The cost of large-scale chemotherapy will be affected by the method of identifying communities for treatment.

Cost calculations frequently omit the cost of employing the necessary personnel. The cost of equipment, particularly vehicles, usually appears in the accounting, but the cost of offices and laboratories is rarely considered, nor is the depreciation of these capital assets. In a strategy that aims at morbidity control and the reduction of transmission, the additional costs of, for example, mollusciciding and environmental management must be considered.

The cost of mollusciciding includes the purchase of molluscicides and the cost of their application (e.g. transportation, storage, administration, equipment and personnel). The cost of environmental management includes capital investments in engineering and equipment and recurrent operational costs (e.g. personnel and materials). Since the cost estimates above are intended to allow comparison, a uniform method of cost calculation must be used so that costs can be adjusted to reflect local variation.

### THE COSTS OF CONTROL

Studies on the costs of control programmes based on chemotherapy were evaluated by the World Health Organization Expert Committee (WHO 1993), which noted the inadequacy of the available data. Costs estimated in studies ranged from US$0.70 to US$3.10 per person, but development costs, salaries, and the cost of failure were often excluded.

The government health allocations for schistosomiasis control were last reviewed in 1976 (Iarotski & Davis 1981). Nineteen endemic countries responded to a World Health Organization questionnaire regarding: (1) the total health budget in US dollars and its proportion of the total national budget and (2) the allocation for schistosomiasis control in US dollars and as a proportion of the health budget. In those countries responding, between 2 per cent (Saudi Arabia) and 18 per cent (Dominican Republic) of the national budget was allocated to health and between 0.002 per cent (Indonesia) to 2.8 per cent (Saint Lucia) of the health budget was directly allocated to schistosomiasis control. Current expenditures for schistosomiasis control are not available.

Some hypothetical projections have been made based on pilot projects. The cheapest regimen using praziquantel involved existing health services in areas of low endemicity. When specialized programmes were required, costs varied from US$1.50 to US$6.53 per person per year. Costs per infected person treated were inevitably higher, with a minimum cost of US$3.00 for the specialized programmes.

When the annual per capita expenditure on all forms of primary health care is only between US$1 and US$4 in many endemic countries, most cannot afford vertical programmes, except perhaps in small areas. Drugs account for a relatively low proportion of total delivery costs in vertical programmes. On the other hand, their cost is more prominent and may be the major expenditure in programmes integrated into primary health care. A reduction in the price of praziquantel could significantly reduce the cost of such programmes.

The cost of identifying communities with a high prevalence of *S. haematobium* infection can be substantially reduced by using questionnaires and reagent strips. Chemotherapy targeted at schoolchildren will also reduce the costs of control, and in most communities will reach the groups with the highest prevalence and severity of infection. Increasing the interval between treatments will also reduce costs, but this has to be balanced against a reduced impact on morbidity. Further studies are needed on ways of reducing the cost of identifying communities for treatment (especially for *S. mansoni* infection) and the cost of drug delivery.

### Estimation of effectiveness

Effectiveness has been estimated in terms of coverage (number of people receiving treatment per number of people infected or at risk) or cure (coverage per drug efficacy). This allows estimation of the cheapest approach to delivering treatment, which is not necessarily the same as the most cost-effective approach to reducing disease.

For a better assessment of effectiveness, more information is required on the morbidity attributable to schistosomiasis at the community level, the relationship between this morbidity and the prevalence and intensity of infection, and the relationship between morbidity and disease predisposition. This approach may require modelling the dynamics of transmission and morbidity, and the impact of control, in similar ways to methods developed for other helminthic infections.

### Indirect costs

In assessing indirect costs of the disease, a distinction is traditionally made between the cost of health care for infected people and the cost of reduced economic productivity as a result of the disease. Little is known about the health-seeking behaviour of people suffering from schistosomiasis and the direct cost incurred by the health system; more research is needed in this area.

Given levels of prevalence and intensity of infection can be associated with very different levels of clinical disease both between and within countries. Economic estimates show similar variations in the indirect cost of the disease. These inconsistencies do not imply that schistosomiasis has no demonstrable economic impact, but rather that the indirect costs might be considerable in certain circumstances, as in areas where the prevalence of clinical disease is high or in communities that have recognized schistosomiasis as a significant health problem. In the assessment of indirect costs, rural household income should be the focus of analysis rather than the wages of individual workers.

The long-term economic consequences of the disease should also be explored. *S. haematobium* infection can be associated with malnutrition and stunting of growth in children. There is a positive correlation between education and productivity, and if schistosomiasis inhibits educational achievement it could also eventually affect production.

## DISCUSSION AND CONCLUSIONS

This chapter has documented the data and methods used to estimate the global burden of schistosomiasis in 1990. The paucity of epidemiological data, particularly on the sequelae and consequences of schistosomiasis infection, have inevitably required that a very simplified disease model be used for the estimations. Ideally, a number of other factors should be considered in the calculation of the burden of disease attributable to schistosomiasis.

### MULTIPLE SCHISTOSOMA INFECTIONS

In 35 sub-Saharan countries and in 6 countries of the Middle Eastern Crescent, both *S. mansoni* and *S. haematobium* are present and overlap in some areas. The age and sex distribution is similar to that of each infection alone (Saladin et al. 1983, Granier et al. 1985, Kazura et al. 1985, El Malatawy et al. 1992). The only study to analyse the interaction between the two infections in the same individuals nevertheless showed that the intensity of *S. haematobium* infection is greater in those with concomitant *S. mansoni* infection than in those with *S. haematobium* alone; the converse was not observed (Robert, Bouvier & Rougemont 1989).

### MULTIPLE PARASITIC INFECTIONS

Schistosomiasis may be only one of multiple parasitic infections in the same individual. Various intestinal helminthic infections have been associated with *S. mansoni* infection (Lehman et al. 1976)

Polyparasitism is the rule rather than the exception in most developing countries. Its occurrence is, however, focal and wide variations are present within any country (Bundy et al. 1991). Estimating the proportional contribution of each of multiple infections to the burden of disease has not been attempted.

### REINFECTION

In endemic areas re-exposure to infection is constant or seasonal and is proportional to the overall prevalence, i.e. high prevalence is associated with high rates of reinfection. In assessing the burden of schistosomiasis, the risk of reinfection should be considered.

It is hoped that future revised estimates of the global burden of disease for schistosomiasis will be able to take into account improved epidemiological information on the incidence, prevalence, sequelae and case fatality of the various types of schistosomiasis infection, and that control efforts will have resulted in reductions in the prevalence of this disease in most regions.

## References

Abdel-Wahab MF et al. (1980) Schistosomiasis mansoni in an Egyptian village in the Nile Delta. *American Journal of Tropical Medicine and Hygiene*, 29:868–874.

Ashford RW, Craig PS, Oppenheimer SJ (1992) Polyparasitism on the Kenya Coast. 1 – Prevalence and the association between parasitic infections. *Annals of Tropical Medicine and Parasitology*, 86:671–679.

Ashford RW, Craig PS, Oppenheimer SJ (1993) Polyparasitism on the Kenya Coast: 2 – Spatial heterogeneity in parasite distributions. *Annals of Tropical Medicine and Parasitology*, 87:283–293.

Audibert M (1986) Agricultural non-wage production and health status: A case study in a tropical environment. *Journal of Development Economics*, 24:275–291.

Awad El Karim MA et al. (1980) Quantitative egg excretion and work capacity in a Gezira population infected with *S. mansoni*. *American Journal of Tropical Medicine and Hygiene*, 29:54–61.

Bassily S et al. (1987) Immunogenicity of hepatitis B vaccine in patients infected with Schistosoma mansoni. *American Journal of Tropical Medicine and Hygiene*, 36:549–553.

Blas BL et al. (1986) An attempt to study the case fatality rate in *Schistosoma japonicum* infection in the Philippines. *Southeast Asian Journal of Tropical Medicine and Public Health*, 17:67–70.

Blas B (1989) *The Schistosomiasis Control Programme in the Philippines. Meeting on the Strategy of Control of Morbidity due to Schistosomiasis,* Geneva, 10–13 October 1989. World Health Organization, Geneva, (unpublished abstracts).

Breuil J, Moyroud J, Coulanges P (1983) Tentatives d'appreciation du retentissement socio-economique des schistosomiases a Madagascar [Attempts to evaluate the socioeconomic repercussions of schistosomiasis in Madagascar]. *Archives de l'Institut Pasteur de Madagascar*, 50:97–111.

Brinkmann UK et al. (1988) Experiences with mass chemotherapy in the control of schistosomiasis in Mali. *Tropical Medicine and Parasitology*, 39:167–174.

Buck AA et al. (1970) *Health and disease in Chad: epidemiology, culture and environment in five villages*. Baltimore, Johns Hopkins University Press.

Bundy DAP et al. (1990) The epidemiological implications of a multiple-infection approach to the control of human helminth infections. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 85:274–276.

Chen MG et al. (1993) Hepatitis B and schistosomiasis: interaction or no interaction. *Tropical Diseases Bulletin*, 90:R97–R115.

Cheever AW et al. (1978) *Schistosoma mansoni* and *S. hematobium* infections in Egypt: III Extrahepatic pathology. *American Journal of Tropical Medicine and Hygiene*, 27:55–75.

China Ministry of Public Health (1992) *Abstracts. International Symposium on Schistosomiasis. November 24–26 1992, Beijing*. pp. 1–12.

Chowdhury AW, Levy BS (1988) *Morbidity estimates of occupational illnesses and injuries in Kenya: human and economic costs.* Paper presented at the Annual

Medical Scientific Conference, Kenya Medical Research Institute – Kenya Tropical Research Institute, Nairobi, 2 February (unpublished).

De Cock KM et al. (1982) Esophageal varices in Nairobi, Kenya: a study of 68 cases. *American Journal of Tropical Medicine and Hygiene*, 31:579–588.

Domingo EO et al. (1980) Morbidity in schistosomiasis japonica in relation to intensity of infection: study of a community in Leyte, Philippines. *American Journal of Tropical Medicine and Hygiene*, 29:858–867.

Doumenge J-P et al. (1987) *CEGET/WHO atlas of the global distribution of schistosomiasis*. Presses Universitaires de Bordeaux.

El-Alamy MA, Cline BL (1977) Prevalence and intensity of *Schistosoma haematobium* and *S. mansoni* infection in Qalyub, Egypt. *American Journal of Tropical Medicine and Hygiene*, 26:470–472.

El-Bolkainy MN, Chu EW (1981) *Detection of bladder cancer associated with schistosomiasis*. Cairo, National Cancer Institute.

El Malatawy A et al. (1992) Selective population chemotherapy among schoolchildren in Beheira governorate: the UNICEF/Arab Republic of Egypt/WHO Schistosomiasis Control Project. *Bulletin of World Health Organization*, 70: 47–566.

Feldmeier H, Poggensee G (1993) Diagnostic techniques in schistosomiasis control: a review. *Acta Tropica*, 52:205–220.

Forsyth DM (1969) A longitudinal study of endemic urinary schistosomiasis in a small East African Community. *Bulletin of World Health Organization*, 40:771–783.

Foster R (1967) Schistosomiasis on an irrigated estate in East Africa: Ill-Effects of asymptomatic infection on health and industrial efficiency. Journal of Tropical Medicine and Hygiene, 70:185–195.

Ghaffar YA et al. (1990) Hepatitis B vaccination in children infected with Schistosoma mansoni: correlation with ultrasonographic data. *American Journal of Tropical Medicine and Hygiene*, 43:516–519.

Ghana Health Assessment Project Team (1981) A quantitative method of assessing the health impact of different diseases in less developed countries. *International Journal of Epidemiology*, 10:73–80.

Granier H et al. (1985) Etude épidémiologique des bilharzioses intestinale et urinaire dans la région de Tala Mokolo (Monts Mandara-Nord Cameroun): [Epidemiologic study of intestinal and urinary bilharziasis in the Tala Mokolo region (Mandara Mountains, North Cameroon]. *Médecine Tropicale*, 45:27–33.

Gryseels B (1988) The morbidity of schistosomiasis mansoni in the Rusizi Plain (Burundi). *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 82:582–587.

Gryseels B (1989) The relevance of schistosomiasis for public health. *Tropical Medicine and Parasitology*, 40:134–142.

Halawani A, Tamami M (1955) Preliminary report on the cytological diagnosis and incidence of bilharzia cancer of the bladder in Egypt. *Journal of the Egyptian Medical Association*, 38:455–465.

Haribhai HC et al. (1991) Spinal cord schistosomiasis: a clinical, laboratory and radiological study, with a note on therapeutic aspects. *Brain*, 114:709–726.

Hayashi M (1979) Clinical studies on cerebral schistosomiasis japonica in the Philippines. *Bulletin of the Tokyo Medical and Dental University*, 26:287–297.

Hiatt RA (1976) Morbidity from *Schistosoma mansoni* infections: an epidemiologic study based on quantitative analysis of egg excretion in two highland Ethiopian villages. *American Journal of Tropical Medicine and Hygiene*, 25:808–817.

Hunter GW et al. (1952) Studies on schistosomiasis: VI – Control of the snail host of schistosomiasis in Japan with sodium pentachlorophenate (Santobrite). *American Journal of Tropical Medicine and Hygiene*, 1:831–847.

Iarotski, LS, Davis A (1981) The schistosomiasis problem in the world: results of a WHO questionnaire survey. *Bulletin of the World Health Organization*, 59:115–127.

Inaba Y et al. (1984) A case-control study on liver cancer with special emphasis on the possible aetiological role of schistosomiasis. *International Journal of Epidemiology*, 13:408–412.

International Agency for Research on Cancer (1994) *Schistosomes, liver flukes and helicobacter pylori*. Lyon, International Agency for Research on Cancer (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 61).

Jordan P, Webbe G, Sturrock RF, eds. (1993) *Human Schistosomiasis*. Wallingford, CAB International.

Joubert J et al. (1990) Schistosomiasis of the spinal cord: underdiagnosed in South Africa? *South African Medical Journal*, 77:297–299.

Junshi C et al. (1990) *Diet, life-style and mortality in China: a study of the characteristics of 65 Chinese counties*. Oxford, Oxford University Press.

Kabatereine NB, Ariho C, Christensen NO (1992) *Schistosoma mansoni* in Pachweach, Nebbi District, Uganda, 40 years after Nelson. *Tropical Medicine and Parasitology*, 43:162–166.

Kazura JW et al. (1985) Swamp rice farming: possible effects on endemicity of schistosomiasis mansoni and haematobia in a population in Liberia. *American Journal of Tropical Medicine and Hygiene*, 34:107–111.

King CL et al. (1982) Prevalence and intensity of *Schistosoma haematobium* infection in six villages of Upper Egypt. *American Journal of Tropical Medicine and Hygiene*, 31:320–327.

Kloetzel K (1962) Splenomegaly in *schistosomiasis mansoni*. *American Journal of Tropical Medicine and Hygiene*, 11:472–476.

Koroltchouk V et al. (1987) Bladder cancer: approaches to prevention and control. *Bulletin of the World Health Organization*, 65:513–520.

Latham MC et al. (1990) Metrifonate or praziquantel treatment improves physical fitness and appetite of Kenyan schoolboys with *Schistosoma haematobium* and hookworm infections. *American Journal of Tropical Medicine and Hygiene*, 43: 170–179.

Lehman JS et al. (1976) The intensity and effects of infection with *Schistosoma mansoni* in a rural community in northeast Brazil. *American Journal of Tropical Medicine and Hygiene*, 25:285–294.

Lewert RM, Yogore MG Jr., Blas BL (1979). Schistosomiasis japonica in Barrio San Antonio, Basey, Samar, The Philippines. I. Epidemiology and morbidity. *American Journal of Tropical Medicine and Hygiene*, 28:1010–25.

Lopes CAS (1984) *Cancerizaçao vesical e schistosomiase [Bladder cancer and schistosomiasis].* Faculdade de Medicina da Universidade de Porto.

Lucas SB (1982) Squamous cell carcinoma of the bladder and schistosomiasis. *East African Medical Journal*, 59:345–351.

Mansour NS et al. (1981) A longitudinal study of Schistosoma haematobium infection in Qena governorate, Upper Egypt: 1-Initial epidemiological findings. *American Journal of Tropical Medicine and Hygiene*, 30:795–805.

Martin-Prevel Y et al. (1992) An epidemiological study of a Schistosoma intercalatum focus in south-east Gabon. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 86:401–405.

Morrow RH (1984) The application of a quantitative approach to the assessment of the relative importance of vector and soil transmitted diseases in Ghana. *Social Science and Medicine*, 19:1039–1049.

Mott KE (1982) Epidemiological considerations for parasite vaccine development. *Pontificiae Academiae Scientarium Scripta Varia*, 47:5–23.

Mott KE et al. (1985a) Evaluation of reagent strips in urine tests for detection of *Schistosoma haematobium* infection: a comparative study in Ghana and Zambia. *Bulletin of the World Health Organization*, 63:125–133.

Mott KE et al. (1985b) Indirect screening for *Schistosoma haematobium* infection: a comparative study in Ghana and Zambia. *Bulletin of the World Health Organization*, 63:135–142.

Murray CJL, Lopez AD (1996a) The global burden of disease. Global Burden of Disease and Injury Series, Volume I. Cambridge, Harvard University Press.

Murray CJL, Lopez AD (1996b) Global health statistics: a compendium of incidence, prevalence and mortality estimates for over 200 conditions. Global Burden of Disease and Injury Series, Volume II. Cambridge, Harvard University Press.

Ndamba J (1986) Schistosomiasis: its effects on the physical performance of school children in Zimbabwe. *Central African Journal of Medicine*, 32:289–293.

Ndamba J et al. (1991) Morbidity due to *Schistosoma mansoni* among sugar-cane cutters in Zimbabwe. *International Journal of Epidemiology*, 20:787–795.

Ndamba J et al. (1993) The physical fitness and work performance of agricultural workers infected with *Schistosoma mansoni* in Zimbabwe. *Annals of Tropical Medicine and Parasitology*, 87:553–561.

Omer AHS et al. (1976) Infection with *Schistosoma mansoni* in the Gezira area of the Sudan. *Journal of Tropical Medicine and Hygiene*, 79:151–157.

Ongom VL, Bradley DJ (1972) The epidemiology and consequences of *Schistosoma mansoni* infection in West Nile, Uganda: 1–Field studies of a community at Panyagoro. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 66:835–851.

Prescott NM (1979) Schistosomiasis and development. *World Development*, 7:1–14.

Ratard RC et al. (1992) Estimation of the number of cases of schistosomiasis in a country: the example of Cameroon. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 86:274–276.

Robert CF, Bouvier S, Rougemont A (1989) Epidemiology of schistosomiasis in the riverine population of Lagdo Lake, northern Cameroon: mixed infections and ethnic factors. *Tropical Medicine and Parasitiology*, 40:153–158.

Saad AM et al. (1991) Oesophageal varices in a region of the Sudan endemic for Schistosoma mansoni. *British Journal of Surgery*, 78:1252–1253.

Saladin B et al. (1983) A pilot control trial of schistosomiasis in central Liberia by mass chemotherapy of target populations, combined with focal application of molluscicide. *Acta Tropica*, 40:271–295.

Salih SY et al. (1977) Salmonellosis complicating schistosomiasis in the Sudan. *Journal of Tropical Medicine and Hygiene*, 80:14–18.

Savioli L et al. (1990) Control of morbidity due to *Schistosoma haematobium* on Pemba Island: Egg excretion and haematuria as indicators of infection. *American Journal of Tropical Medicine and Hygiene*, 43:289–295.

Simarro P, Sima FO, Mir M (1990) Urban epidemiology of *Schistosoma intercalatum* in the city of Bata, Equitorial Guinea. *Tropical Medicine and Parasitology*, 41:254–256.

Siongok TKA et al. (1976) Morbidity in schistosomiasis mansoni in relation to intensity of infection: study of a community in Machakos, Kenya. *American Journal of Tropical Medicine and Hygiene*, 25:273–284.

Sleigh AC and Mott KE (1986) *Schistosomiasis. Clinics in tropical medicine and communicable diseases*. London, Saunders, 1:643–670.

Sleigh A et al. (1982) Comparison of filtration staining (Bell) and thick smear (Kato) for the detection and quantitation of *Schistosoma mansoni* eggs in faeces. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 76:403–406.

Smith DH, Warren KS, Mahmoud AAF (1979) Morbidity in schistosomiasis mansoni in relation to intensity of infection: study of a community in Kisumu, Kenya. *American Journal of Tropical Medicine and Hygiene*, 28:220–229.

Stephenson LS et al. (1984) Sensitivity and specificity of reagent strips in screening of Kenyan children for *Schistosoma haematobium* infection. *American Journal of Tropical Medicine and Hygiene*, 33:862–871.

Tanaka H et al. (1984) Epidemiology and transmission dynamics: evaluation of control measures by means of annual incidence of infection with *Schistosoma japonicum* among school children at Dagami, Leyte, Philippines. *Southeast Asian Journal of Tropical Medicine and Public Health*, 15:480–481.

Tanaka H et al. (1985) Longitudinal study on *Schistosoma japonicum* infections in the Philippines: 4 – Effect of mass-chemotherapy with praziquantel in incidence at Dagami, Leyte. *Japanese Journal of Experimental Medicine*, 55:161–165.

Taylor P, Makura O (1985) Prevalence and distribution of schistosomiasis in Zimbabwe. *Annals of Tropical Medicine and Parasitology*, 79:287–299.

Teesdale CH, Fahringer K, Chitsulo L (1985) Egg count variability and sensitivity of a thin smear technique for the diagnosis of *Schistosoma mansoni*. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 79:369–373.

Thiongo FW, Ouma J (1987) Prevalence of schistosomes and other parasites in Taito Division of Taita-Taveta District. *East African Medical Journal*, 64:665–671.

Utroska JA et al. (1989) *An estimate of global needs for praziquantel within schistosomiasis control programmes*. Geneva, World Health Organization (WHO/SCHISTO/89.102).

Van Ee JH, Polderman AM (1984) Physiological performance and work capacity of tin mine labourers infested with schistosomiasis in Zaire. *Tropical and Geographical Medicine*, 36:259–266.

Warren KS et al. (1978) Quantification of infection with Schistosoma haematobium in relation to epidemiology and selective population chemotherapy. 1 – Minimal number of daily egg counts in urine necessary to establish intensity of infection. *Journal of Infectious Diseases*, 138:849–855.

Wenlock RW. The epidemiology of tropical parasitic diseases in rural Zambia and the consequences for public health. *Journal of Tropical Medicine & Hygiene*, 82: 90–98.

White PT, Coleman M, Jupp BP (1982) Swamp rice development, schistosomiasis and onchocerciasis in Southeast Sierra Leone. American Journal of Tropical Medicine and Hygiene, 31:490–498.

Winslow, CEA (1951) *The cost of sickness and the price of health*. Geneva, World Health Organization (WHO Monograph Series, N. 7).

World Health Organization (1967) World Health Organization Scientific Group. *Measurement of the public health importance of bilharziasis*. Geneva, World Health Organization (WHO Technical Report Series No. 349).

World Health Organization Regional Office for the Eastern Mediterranean (1983) *Technical guide for the diagnosis, prevention and control of Rift Valley Fever in man and animals*. WHO/EMRO Technical Publication No. 8.

World Health Organization (1985) WHO Expert Committee on Control of Schistosomiasis. *The control of schistosomiasis*. Geneva, World Health Organization (WHO Technical Report Series, No. 728).

World Health Organization (1990) *World Health Organization model prescribing information: drugs used in parasitic diseases*. Geneva, World Health Organization.

World Health Organization (1993) WHO Expert Committee on Control of Schistosomiasis. *The control of schistosomiasis. Second report*. Geneva, World Health Organization, (WHO Technical Report Series, No. 830).

World Health Organization (1989) Progress in assessment of morbidity due to schistosomiasis: reviews of recent literature. Schistosoma haematobium, Schistosoma intercalatum, Schistosoma japonicum, Schistosoma mansoni. Reprints from the *Tropical Diseases Bulletin* 85 Nos. 6 & 10 (1988); 86 Nos. 4 & 8 (1989).

Wright WH, Dobrovolny CG (1953) Experiments in the control of schistosomiasis in Brazil. *Public Health Reports*, 68:1156–1160.