Letters

Morbidity in Schistosomiasis mansoni I

In their article on the control of morbidity due to schistosomiasis mansoni in subsaharan Africa[1], Gryseels and Polderman make several interesting and valid points. However, some of their arguments and statements, although emphatically presented, are more controversial. From the perspective of schistosomiasis mansoni as it presents as a public health problem in Kenya, we would like to draw attention to several issues on which we would strongly disagree.

Gryseels and Polderman develop the implicit argument that 'decompensated portal hypertension' (DPH) is not merely the most but rather the *only* important clinical manifestation of schistosomiasis mansoni that needs to be prevented. While we are unclear as to the authors' definition of DPH, we would argue that this is a very extreme stance. In Kenya, apart from those areas of low prevalence and intensity of infection, in which schistosomiasis mansoni is of little public health significance, we would identify two distinct scenarios:

(1) Areas of high prevalence and intensity of infection but with little evidence of severe morbidity. Hepatomegaly can be detected in 15% of primary schoolchildren but hepatosplenomegaly (and portal hypertension) are rare[2]. In such areas, chemotherapy of infected schoolchildren once every three years is sufficient to reduce to low levels the prevalence of both high intensity infections and hepatomegaly[2]. In addition, although difficult to quantify, we have consistent reports, from schoolteachers and parents, of general improvement in health (particularly of abdominal complaints), school attendance and physical and academic performance.

(2) Areas of comparably high prevalence and intensity of infection but with evidence of a high prevalence of more severe morbidity[3]. Hepatosplenomegaly is present in up to 20% of primary schoolchildren and, in many cases, is massive and associated with demonstrable oesophageal varices, which may progress to haematemesis. In such areas, apart from the common complaint of bloody diarrhoea, the organomegaly *itself* is (not surprisingly) perceived as a serious clinical problem. It is associated with poor nutritional status[4], reduced physical activity and the search for and substantial payment for treatment, both from conventional sources and from traditional healers (as evidenced both by direct interview and by the presence of abdominal scars in children with organomegaly). In these areas, annual chemotherapy of infected primary schoolchildren leads to a progressive but very slow reduction in the prevalence of gross hepatosplenomegaly (G.G. Mbugua *et al.*, unpublished). The reasons for such focal differences in morbidity, in spite of similar intensities on infection, remain uncertain[3] and are currently a subject of study in Kenya. Factors contributing to a difference in morbidity between different areas could include poor nutrition, interactions with other infections and immunological phenomena associated with recent immigration to an endemic area. Such factors might be associated with new land settlement, as increasing population pressure drives subsistence farmers from long-settled and fertile areas into more arid and less productive regions. If this is the case, foci of severe morbidity associated with schistosomiasis mansoni may be expected to increase.

Although the impact of chemotherapy of schoolchildren on transmission may be slight, we have found that both intensities of infection and morbidity (as reflected by organomegaly) *can* be controlled by repeated treatment of primary schoolchildren who are most at risk of developing high-intensity infections and severe morbidity[2]. Likewise, in Brazil, the chemotherapy of highly infected children successfully prevents the development of severe schistosomiasis[5]. In areas of intense transmission, it may be justifiable (as suggested by Gryseels and Polderman) to treat all children of primary school age without individual diagnosis of infection. Whether such an approach is appropriate in all endemic areas will depend on local (national) circumstances, which will include the relative availability of foreign currency (for drugs and fuel) and of local currency (for salaries of staff involved in diagnosis and delivery of treatment). For such programmes to be sustainable, it is not *a priori* essential that they be based entirely on primary health care structures; an essential element of vertical, centrally organized control may be desirable as well as feasible.

In Kenya, a chemotherapy-based control programme for schistosomiasis and other intestinal helminths is currently being launched and is considered to be both necessary and feasible in the short and middle term. In the longer term, other measures would be desirable. These may include not only social and economic measures (including health education and the provision of water supplies) but also the application of vaccines as these become available. Current vaccine coverage in Kenya, with 71% of children being fully vaccinated within an EPI programme (A.B. Mutie, pers. commun.), supports the feasibility of such an approach.

References

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J.H. Ouma
Division of Vector-Borne Diseases
Ministry of Health
PO Box 20750, Nairobi, Kenya

G.G. Mbugua
Centre for Microbiology Research
Kenya Medical Research Institute
PO Box 54840, Nairobi, Kenya

A.E. Butterworth
Department of Pathology
Tennis Court Road
Cambridge CB2 1QP, UK

Reply I

Indeed, in our paper we raised some controversial issues, but with the intention of triggering a clarifying discussion about the objectives of morbidity control in schistosomiasis mansoni. We are pleased that Ouma, Mbugua and Butterworth have taken up the challenge.

We used the term 'decompensated portal hypertension' (DPH) for cases in which periportal liver fibrosis due to schistosomiasis leads to clinical consequences, particularly haematemesis. This may not be the most accurate or appropriate term but it is at least more tangible than 'hepatosplenic disease', which has been used for anything from mild liver enlargement to what we know as DPH. This confusion, in itself, indicates the need for a more precise definition of 'morbidity' and 'morbidity control' in schistosomiasis mansoni.

What is the rationale for intervention in the first-described scenario, of high prevalence and little severe morbidity, described in the preceding letter? In schistosomiasis, prevalence and intensity of infection, and even hepatomegaly, are not public health problems in their own right but, at most, possible indicators of morbidity. The implementation of a programme of population-based chemotherapy requires considerable means and national long-term commitment. Policy makers, who have to confront a panoply of health problems with scarce resources, need objective and controlled evidence of a general improvement in health before starting such a programme.

The second scenario, of high prevalence and significant severe morbidity, represents a situation comparable to the one we described in Maniema. The problem is evidently recognized by the health services and the suffering community, and control measures are clearly justified. How chemotherapy will be delivered depends indeed on national circumstances and resources. A 'vertical' component (eg. a specialized central team of the Ministry of

Health) may be needed to develop, initiate and then further coordinate control measures. However, the problem will not go away with one or a few treatments. A programme based on chemotherapy has to be maintained in the long term, and therefore has to be integrated into existing, sustainable health structures. Large vertical programmes generally depend too crucially on foreign assistance to fit this description. The development of primary health care may be, in our view and experience, of greater importance for sustained schistosomiasis control than the quick and easy successes of specialized mobile teams.

As long as the detection of *Schistosoma mansoni* infection is based on microscopic screening, individually selective strategies remain too labour intensive to be integrated into the primary health care activities of most subsaharan countries. Mass treatment of selected target communities or groups, based on, and monitored by, reliable health statistics and/or epidemiological intelligence, would appear a more, if not the only, feasible approach. In this respect, the development of cheap and easy tools for community diagnosis, such as those available for urinary schistosomiasis, would be a major advance for the control of schistosomiasis mansoni. Reagent strips for antigen detection may answer this need in the near future. It would also be worthwhile to investigate how far the control of intestinal and hepatosplenic morbidity can be achieved by a 'passive' case detection approach based on adequate curative health care and health education.

The provision of domestic water supply and health education remain the only ways to achieve durable control of schistosomiasis. Again, these are essential components of primary health care, and require intersectoral cooperation at all levels. Preferably, they should not be implemented through a vertical, single-disease programme.

However exciting recent scientific developments may be, it would appear premature to base the development of a control strategy on the prospect of a vaccine becoming available soon.

B. Gryseels and A.M. Polderman
Department of Parasitology and Tropical Medicine
University of Leiden
PO Box 9605 2300 RC Leiden
The Netherlands

Morbidity in Schistosomiasis mansoni II

Gryseels and Polderman[1] raise some interesting points, but comment on two misleading statements would not be out of place.

Their criteria for decompensated portal hypertension are unclear but one factor seems to be the development of oesophageal varices, which may be fatal. This is far from the recognized difference between compensated and decompensated portal hypertension, namely, in the latter the development of abnormal ammonia tolerance and liver function tests[2], with patients dying in hepatic coma[3].

It is a pity that they did not look critically at the report from Brazil[4], to which they refer, before claiming that hepatosplenic disease is disappearing in areas *not* subject to control. In fact, severe disease was reduced (in the absence of *specific* schistosomiasis control) but only in the part of Minas Gerais State where piped water availability in dwellings increased from 17% to 44%. In areas with no improved water, and no control, there was no change in the rate of splenomegaly[5]. In a second area with no control, which they claimed to show a spontaneous reduction in disease, two-thirds of those who improved had in fact received specific treatment[6].

More generally, studies showing less disease in black people compared with caucasians in Brazil[7,8] and with asians in St Lucia[9] suggest that morbidity may be genetically influenced. These findings are in line with the views of early workers that severe morbidity from *Schistosoma mansoni* in Africans in sub-Saharan Africa is rare[10–12].

However, the finding of a focus of severe morbidity in children in Kenya[13] reminds us that in Africa, with massive human movement owing to population growth, famine, war and possible climatic changes, the epidemiology of schistosomiasis can change and new problems may arise at any time.

References

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P. Jordan
21 Linwood Road
Ware SG12 7JQ, UK

Reply II

We are very pleased that Peter Jordan has brought his vast experience into this discussion.

We recognize that our definition of decompensated portal hypertension is different from that of other authors. However, as already stated in our reply to Ouma, Mbugua and Butterworth, there is a lack of standardized criteria and terminology for hepatosplenic disease and morbidity in schistosomiasis mansoni, and thus also of clearly defined objectives for 'morbidity control'.

The variation of morbidity patterns between and within countries (and ethnic groups) was one of the basic observations of our paper and has further been dealt with in the above-cited letter and reply.

We certainly did not wish to imply that, in some areas in Brazil, hepatosplenic disease (whatever it means) is disappearing spontaneously, but rather that this is happening without systematic population-based treatment. We are well aware that water supply, sanitation and the availability of specific drugs in the curative sector have been forwarded as possible explanations. These observations support, rather than refute, our view that the development of primary health care is essential and perhaps even sufficient for the control of severe schistosomiasis morbidity.

B. Gryseels and A.M. Polderman

Erratum

The text of the letter from A. Suhrbier in the December issue of *PT* (pp 340–341) contains an error: the reference to 'Ungureanu *et al.*' should read 'Weiss'.

We apologize to all concerned.