practice committee should become publicly accountable. Fewer than half its members should come from the industry: the majority should represent the health professions and the public. (The United Kingdom Advertising Standards Authority, which has analogous functions, has only a minority of members from the advertising industry.) Complainants should have the same right to be heard as defendants; hearings should be open to the public, reports published, and a mechanism for appeals introduced. Adequate self regulation needs teeth: it needs sanctions that work. If publicity and mandatory corrections are ineffective then fines may be needed. Changes along these lines should be made as soon as possible, and their efficacy monitored by an impartial body. Editors of journals carrying advertisements could play a special part by performing their own critical scrutiny and taking a more robustly independent line.

We thank Mr A D W Massam, secretary of the ABPI code of practice committee, for patiently answering our questions about the administration of the code.

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Letter from Brasilia

Fertile flukes and mediating molluscs

P D Marsden

We are at a school deep in the Brazilian interior on the borders of the state of Bahia with that of Goiás. A rural community of less than 200 people, the school is small and there is a single teacher. We have got permission from the mayor to examine the children today for schistosomiasis. As we enter the children stand, but I note that a little boy at the back has difficulty in standing up and getting out of his form—his belly is unusually large. The teacher has arranged a sort of examination couch made of desk tops, and I ask if I can examine that boy first. He comes forward slowly, kicks off his sandals, and lies on the desk top. His name is José, and he is 12 years old. Lifting up his shirt I can see that his abdomen is distended, though there are no visible dilated veins. On palpation I can show an enlarged spleen by ballottement but no liver. Percussion confirms the presence of a considerable ascites. He probably has advanced hepatosplenic schistosomiasis due to *Schistosoma mansoni*. The distribution of faecal pots is supervised by the teacher, and José goes to the bush (there is no lavatory) to return with a faecal specimen.

Outside, in the back of our transport, two technicians are processing stool specimens with the Kato quantitative technique. José's specimen shows a heavy infection with the laterally spined eggs of *S. mansoni*. Meanwhile inside, the routine clinical examination for hepatosplenomegaly continues. Eventually we have the preliminary results of our little survey. Over half the children have palpable livers or spleens, but the parasitology is even more impressive. The technicians examining stools find that only one child does not have the characteristic eggs.

The students are finishing up the protocols in the school and the technicians still documenting, preserv-

ing, and storing the specimens for future reference. Accompanying my chief, Professor Aluizio Prata, we stroll down to the river. Like most of the areas where we work on schistosomiasis it is the only source of water for bathing, washing, and drinking. Later, in the heat of the day, we will see these same children bathing from the little beach. Looking under the vegetation hanging over the water we quickly find the vector snail *Biomphalaria glabrata*, and, putting two or three in a glass of water in the sunlight, we can just see the cercarias being shed by the snails into the water. This is the minute, tadpole like infective form for humans, which enters the skin within minutes to produce eventually the adult worm in the portal circulation. Fertile pairs of worms give rise to the characteristic eggs in the stool, and such eggs induce the severe portal fibrosis, producing presinusoidal portal hypertension as seen in José.

Theoretically you could break this parasitic life cycle at least at three points: the shedding of eggs into the environment, the snail, and humans. Rural Brazilians are often broadcast defecators, and latrine building has not been a great success. They get turned into storage rooms or chicken houses. Educational programmes are hard to implement. Like all mammals we live in an environment contaminated by our own faeces. It is a question of degree. Witness the famous unpublished experiment in an American middle class home where after the family had ingested fluorescein ultraviolet light picked up the dye on the mantelpiece, the cutlery, etc. The experiment was not published because the Americans are already obsessed about washing their hands and what else can you do?

So the eggs get into the water and the infective form for the snail, the miracidium, produces innumerable

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Oedema associated with *Schistosoma mansoni* infection in young Brazilian girl

multiplying forms in the digestive gland of the snail, which may die of the infection. Snails are tough and difficult to control with molluscicides. In St Lucia, Caribbean, a good degree of control was achieved but with a lot of Rockefeller money and in isolated island valleys. Of the many studies of snail control in Brazil, few have had more than transitory success in controlling the three species of snail responsible for *S. mansoni* transmission.

A few weeks after infection the snail begins to shed the infective cercarias. One answer for control has come from the drug companies. Oxamniquine, the drug most used in Brazil, was the result of a happy accident of contamination of a manufacturing vat at Pfizer by a fungus that metabolised a standard antischistosomal drug into a much more active agent. Praziquantel had been used for years in veterinary medicine but Bayer was persuaded to put it on the market for human use, and it is the only drug active against all three common species causing human

schistosomiasis—namely, *S. mansoni*, *S. haematobium*, and *S. japonicum*. Both these drugs are given in a single oral dose that kills most of the worms. Oxamniquine costs about a dollar a dose, and side effects are usually slight. So this is one answer—targeted mass treatment. As we walk back from the river to the school we see the box of drugs being lifted out of the back of the jeep. We watch our staff and the teacher organise the administration, checking that each dose is ingested. But why the children? What about the adults? and the comment is valid. There is, however, a question of cost, and children are most at risk of developing severe forms of the disease. These forms are usually related to worm load. Portal hypertension in children is more common with *S. japonicum* because each female produces 10 times the number of eggs each day that *S. mansoni* produces.

Many diseases came to Brazil from Africa with the slave trade, and schistosomiasis *mansoni* is a good example. The slaves were carrying much more *S. haematobium* but there was no suitable snail vector in the New World so fortunately it never established itself. The sugar cane industry on the Brazilian coast created a favourable environment for transmission and still today the states involved in that first agricultural economy of Brazil have the greatest problem with schistosomiasis. It continues to spread inland, and there is a vigilance programme in the federal district where Brasilia stands to monitor transmission. Fortunately to date there have been only minor instances. Certainly the fact that Brasilia's lake is so grossly polluted with insecticides and sewage may help to avoid colonisation by vector snails.

Brazilian *S. mansoni* is much more destructive in humans than is the case in Africa. Of my three lectures on schistosomiasis in the University of East Africa at Makerere, two were devoted to haematobium and only one to mansoni. Severe portal hypertension was virtually restricted to fishermen in Lake Victoria, but in Brazil you see cases like José. The answer may lie not in the parasite but in the host for there is good evidence from our group that white caucasian stock develops portal hypertension more often than negroid stock. Obviously we want to know why.

ANY QUESTIONS

Do bodybuilding mixtures have any effect?

The development of muscular tissues increases the body's demand for protein, but the protein content of people's diets in the United Kingdom would normally be adequate to support this additional requirement, especially if they are physically active with a moderate to high intake as is likely to be the case among bodybuilding enthusiasts. A widespread belief exists among athletes, particularly those who need to maintain a high mass, that a high—in some instances very high—protein intake is required, though there is little scientific evidence to support this view.¹ A strong psychological element is evident in achieving consistently high performances, and special dietary regimens may play a part in building confidence even if only through a placebo effect. Although physical exercise is associated with increased urinary excretion of nitrogen, there is little evidence that this carries the implication that a higher protein intake is required for exercise.

Bodybuilding mixtures provide supplementary amino acids that would contribute to protein intake; they include the major essential amino acids and presumably are formulated in the proportions found in muscle. They may have a slight stimulatory effect on growth, but if they are eaten with a normal consumption of protein the effects would be small. There is much evidence that increased intakes of amino acids may stimulate amino acid oxidation during the fasting periods between meals so the overall effects would probably be negligible. The amounts consumed are relatively small and would almost certainly have minimal risks, and if the practice encourages performance it probably does little harm.²—D A T SOUTHGATE, head, nutrition and food quality department, Agricultural and Food Research Council's Institute of Food Research, Norwich

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Are erythrocytes commonly found in the urine after exercise, and if so, what, if any, advice should be given?

An increase in urinary erythrocytes of glomerular origin is commonly found after exercise.¹ Occasionally microscopic haematuria has been reported immediately after strenuous exercise such as a 10 000 metre run and is thought to be due to contusion of an empty bladder.² Nevertheless, microscopic haematuria after exercise rarely causes any clinical difficulty because urine is rarely tested at this time. If it was chanced on a doctor would need to satisfy himself that no urinary tract disease was overlooked. This could be achieved by repeating microscopy of fresh urine when it should be free of erythrocytes before exercise and after exercise when erythrocytes would be present. More than two observations would be necessary because microscopic haematuria is intermittent.³ The age of the patient is relevant: in a middle aged or elderly person a pathological cause of haematuria is more likely to be present than in a younger subject. The haematuria of exercise requires no further investigation or action.—ROGER GABRIEL, renal physician, London

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