The Susceptibility of Rodents to Schistosome Infection, with Special Reference to *Schistosoma haematobium*

J. H. S. GEAR, D. H. S. DAVIS & R. J. PITCHFORD

In this investigation the susceptibility of several species of rodents—Praomys (Mastomys) natalensis, Saccostomus campestris, Arvicantus niloticus, Aethomys chrysophilus, Tatera brantsi and the white mouse (*SAIMR 200 strain*)—to *Schistosoma haematobium* was determined and the pathology studied. From the results it is clear that these rodents are susceptible to infection with *Schistosoma haematobium.* For various reasons, notably adaptability to laboratory conditions, the most suitable as laboratory animals for the study of bilharziasts are Saccostomus campestris, Arvicantus niloticus and Praomys (Mastomys) natalensis. These three species breed readily in the laboratory and show a high susceptibility to *S. haematobium,* with characteristic lesions involving several organs, including the lungs, liver, spleen, pancreas and intestine.

The pathogenesis and pathology of *Schistosoma mansoni* infections in laboratory animals are well known. Most animals are susceptible and have been extensively used in studies of various aspects of the disease. In contrast, there is little information on *Schistosoma haematobium* infections in common laboratory animals. One of the reasons for this is the difficulty encountered in raising the snails that act as intermediate hosts. However, many species of baboons and monkeys have been infected experimentally; often the infections were patent and eggs were found in the faeces and sometimes in both urine and faeces. Mice and cotton rats have been found to be susceptible and to excrete eggs in the faeces, but usually these are not viable. Hamsters are also susceptible and eggs are excreted in both urine and faeces.

Kuntz & Malakatis (1955), in a systematic study, found that all the common rodents of Lower Egypt could be infected and noted that the Nile rat, *Arvicantus niloticus,* harboured numerous worms and excreted viable eggs for at least three years. With this exception, infections of both primates and rodents were usually light with worms and eggs and tended to self-cure.

In view of the need for suitable small experimental animals for the study of *S. haematobium* infections in the laboratory, it seemed worth while to undertake a systematic study of the susceptibility of the common South African veld rodents. Several of these have been adapted to laboratory conditions by the staff of the Medical Ecology Centre at this Institute and thrive under these conditions.

**MATERIAL AND METHODS**

The six species of rodent chosen for this study were

- *Praomys (Mastomys) natalensis,* the multimammate mouse
- *Saccostomus campestris,* the pouched rat
- *Arvicantus niloticus,* the Nile rat
- *Aethomys chrysophilus,* the African bush rat
- *Tatera brantsi,* the highveld gerbil and the white mouse.

Groups of animals of each species were exposed once to the same number of F₂ cercariae of a Transvaal strain of *S. haematobium,* maintained in the

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*When wild rodents are being used as laboratory animals, the utmost care should be taken to prevent their escape in areas where they are not indigenous. Our practice has been to issue only males of such species for experimental purposes to other laboratories in South Africa.*

*The rodent terminology used in this paper is that proposed by Davis (1962).*
laboratory. The animals were immersed, except for their heads, in water containing the cercariae in narrow bottles for one hour. They were then kept under daily observation in their cages for one year.

Some characteristics of the rodents used in this study

The multimammate mouse, *Praomys (Mastomys) natalensis*, is one of the commonest and most widespread rodents in Africa (see map in Davis, 1962). It has been bred under laboratory conditions since 1939 (Davis & Oetttlé, 1958); it thrives and breeds prolifically and lives for 2½ to 3 years. This small rodent, which weighs up to 100 g, is nocturnal and needs no special care or diet; it is liable to bite when handled. It is highly susceptible to *S. haematobium*, *S. mansoni*, *S. mattheei* and *S. rodhaini* in the laboratory. In the natural state it is known to enter water and has been found to be infected with three schistosome species—*S. bovis*, *S. mansoni* and *S. mattheei* (Pitchford & Visser, 1962; Nelson, Teesdale & Highton, 1962).

The pouched mouse, *Saccostomus campestris*, occurs from the Cape to north of the equator in East Africa (see map in Davis, 1962). It is not a common species but is widely distributed, inhabiting sub-tropical savanna bush country. It is nocturnal. Pouched mice are small rodents weighing up to 90 g and need no special diet. They are easily handled, except during the mating season, when they become vicious and readily bite; the pregnant female is liable to kill the male if the two are kept together. Pouched mice are relatively easy to breed, having about four litters in a year during the restricted breeding season between September and March (southern summer). They live in underground burrows, often in association with gerbils (*Tatera*) and tend to avoid water. They have not been found naturally infected with any schistosome, but are highly susceptible to *S. haematobium*, *S. mansoni*, *S. mattheei* and *S. rodhaini* in the laboratory.

The African bush rat, *Aethomys chrysophilus*, has a sub-tropical distribution, extending northwards through the savanna to the southern Congo and north of the equator (see map in Davis, 1962) and is one of the commonest species of bush rat. It is nocturnal, usually nests underground among the roots of trees and makes well-marked surface runs. In the laboratory it breeds poorly and has to be handled with care. It is a medium-sized rodent, weighing up to about 120 g, and needs no special diet. It is susceptible to *S. haematobium*, *S. mansoni* and *S. mattheei*. It has not been found naturally infected and tends to avoid water. In view of its poor breeding habits it is not recommended as a laboratory animal.

The highveld gerbil, *Tatera brantsi*, is widely distributed through the Eastern Cape, the Orange Free State, Natal and the Kalahari region to Barotseland and Zambia (see map in Davis, 1962). It is nocturnal and does not breed easily in the laboratory; specimens caught wild are used for experimental purposes. It is advisable to quarantine these animals for at least two weeks to prevent the introduction of plague and other natural diseases. Gerbils are easy to handle, weigh up to 130 g, and need no special diet. They are susceptible to *S. haematobium*, *S. mansoni* and *S. mattheei*, but tend to avoid water and have not been found naturally infected.

The Nile rat, *Arvicanius niloticus*, occurs north of the Zambesi and is widely distributed in central, west, north and east Africa; it is particularly numerous in cultivated areas. It is mainly nocturnal and breeds freely in the laboratory provided that it is fed potatoes. It is relatively easy to handle and weighs up to about 150 g. The laboratory-bred stock used in these laboratories have been bred from a nucleus established at the Virus Research Institute, Entebbe, Uganda. The species is highly susceptible in the laboratory to *S. haematobium*, *S. mansoni*, *S. mattheei* and *S. rodhaini*, but has not been found naturally infected, although it does not avoid water (Kuntz & Malakats, 1955).

RESULTS

The number of animals exposed and the mortality during the year after exposure are shown in Table 1. The heaviest mortality occurred three to six months after exposure, probably as a result of damage caused by egg-laying.

None of the rodents that died was examined histologically. The surviving animals were sacrificed approximately one year after exposure, and detailed histological studies were made. Findings in regard to the presence of eggs and worms are set out in Table 2. A detailed description of the pathological changes seen in various organs of the infected rodents follows; Table 3 indicates the organs affected in each species of rodent.

*Praomys (Mastomys) natalensis*

Sixteen animals were exposed to infection in February 1962: nine died and two were sacrificed for
transmission experiments before the end of the period of observation. The five survivors were sacrificed in late February or early March 1963; all showed active schistosomai infection.

**Brain.** In none of the animals were bilharzial lesions found in the brain.

**Heart.** No worms or eggs were observed in the heart muscle or in the heart cavities.

**Lungs.** In four of the five animals, bilharzial lesions affected the lungs (see Fig. 1). Each of these had remnants of dead worms in an advanced stage of dissolution encapsulated in thick fibrous tissue surrounded by an area showing marked inflammatory reaction. The walls of the vessels containing worms were greatly thickened and the surrounding cellular infiltration included large numbers of polymorph and eosinophil leucocytes and round cells, including plasma cells. Three of the animals showed bilharzial tubercles developed in relation to ova.

**Liver.** All five animals showed liver infection (see Fig. 2-6). The capsule was not thickened, but the portal tracts showed marked cellular infiltration. In four animals there were dead worms in the portal veins in an advanced stage of dissolution, encapsulated in fibrous tissue which obliterated the lumen of the vein. There was a surrounding cellular reaction of polymorph leucocytes and round cells, including plasma cells. Numerous bilharzial tubercles, developed in relation to ova, were present in the portal tracts. A typical bilharzial tubercle consisted of an egg or the remnants of eggs, often engulfed in multinucleated giant cells surrounded by endothelial cells, surrounded in turn by polymorph and eosinophil leucocytes, with a peripheral zone of round cells, including plasma cells. There was some distortion and compression of parenchymal cells in its immediate neighbourhood. The sinusoids showed a slightly increased number of inflammatory cells, including polymorph and eosinophil leucocytes. The Kupffer cells contained yellowish brown or black pigment and were sometimes heavily loaded. One of the animals showed marked proliferation of the bile ducts in relation to the inflammatory reactions to eggs.

**Spleen.** Two of the animals had eggs occurring singly and one had a small egg-nest with surrounding fibrosis (see Fig. 7).

**Pancreas.** The pancreas was involved in only one of the animals (Fig. 8); large egg-nests with considerable distortion and replacement destruction of the parenchymal lobules were seen.

**Kidneys and adrenals.** No bilharzial lesions were seen in these organs.

**Bladder.** In one animal there were numerous eggs and an associated inflammatory reaction in the submucous coat (Fig. 9).
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</table>

*a* D = dead; L = living; O = ova; S = schistosomes.

*In 7 Tatara no eggs or schistosomes were seen.*
FIG. 1
MASTOMYS LUNG (× 37.5)

S. haematobium egg-nest and inflammatory reaction

FIG. 2
MASTOMYS LIVER (× 37.5)

S. haematobium adults and inflammatory reaction

FIG. 3
MASTOMYS LIVER (× 37.5)

Dead S. haematobium worm

FIG. 4
MASTOMYS LIVER (× 37.5)

Fibrosis due to S. haematobium infection

FIG. 5
MASTOMYS LIVER

S. haematobium tubercle (× 240)

FIG. 6
MASTOMYS LIVER

Early S. haematobium infection (×240)
FIG. 7

MASTOMYS SPLEEN (× 37.5)

S. haematobium eggs and inflammatory reaction

FIG. 8

MASTOMYS PANCREAS (× 37.5)

S. haematobium egg-nests

FIG. 9

MASTOMYS BLADDER (× 37.5)

S. haematobium eggs and inflammatory reaction

FIG. 10

MASTOMYS GUT (× 37.5)

S. haematobium egg-nest and inflammatory reaction
FIG. 11
SACCOSTOMUS LUNG (× 37.5)

Dead S. haematobium and inflammatory reaction

FIG. 12
SACCOSTOMUS LUNG (× 60)

S. haematobium lesions

FIG. 13
SACCOSTOMUS LIVER PORTAL TRACT (× 37.5)

S. haematobium infection

FIG. 14
SACCOSTOMUS LIVER PORTAL TRACT

Giant cells (× 240); S. haematobium infection

FIG. 15
SACCOSTOMUS SPLEEN (× 37.5)

S. haematobium eggs in pulp
FIG. 16
SACCOSTOMUS PANCREAS (×37.5)

S. haematobium egg-nests

FIG. 17
SACCOSTOMUS BLADDER (×37.5)

S. haematobium egg and inflammatory reaction

FIG. 18
SACCOSTOMUS GUT (×37.5)

S. haematobium eggs and inflammatory reaction

FIG. 19
SACCOSTOMUS MESENTERY (×37.5)

S. haematobium egg-nest

FIG. 20
TATERA BLADDER (×37.5)

S. haematobium egg-nests and inflammatory reaction
TABLE 3
ORGANS AFFECTED BY S. HAEMATOBium INFECTION

<table>
<thead>
<tr>
<th>Species</th>
<th>Number exposed</th>
<th>Number studied</th>
<th>Number with lesions of</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Brain</td>
</tr>
<tr>
<td>Praomys (Mastomys) natalensis</td>
<td>16</td>
<td>5</td>
<td>0</td>
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<td>16</td>
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<td>0</td>
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<tr>
<td>Arvicantbus niloticus</td>
<td>17</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Aethomys chrysophilus</td>
<td>13</td>
<td>3</td>
<td>1  b</td>
</tr>
<tr>
<td>White mouse</td>
<td>11</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Talara brantsi</td>
<td>17</td>
<td>13</td>
<td>0</td>
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</table>

a Animals showed area of eosinophil degeneration of heart muscle of unknown etiology and significance.
b Animal showed area of eosinophil degeneration of the brain.

Intestine. In the sections examined only one of the animals showed bilharzial tubercles in association with eggs in the intestine (Fig. 10). Three of the other four showed subacute inflammatory reaction with infiltration of polymorph and eosinophil leucocytes and round cells, including plasma cells, in the submucous coats, presumably in response to eggs or adults not cut in the sections examined.

In summary, Praomys (Mastomys) natalensis is highly susceptible to infection with S. haematobium and many of the worms survive for at least one year, but the number of dead worms found suggests that the infection was being overcome and possibly in time would have been eliminated.

Saccostomus campestris

Sixteen animals were exposed to S. haematobium infection in February 1962. Nine died, one was sacrificed for transmission and the surviving animals were sacrificed during January and February 1963. All showed evidence of schistosomal infection.

Brain. No bilharzial lesions of the brain were found.

Heart. No worms or eggs were found in the heart or its cavities.

Lungs. Bilharzial lesions were found in five of the six animals (see Fig. 11, 12). In two, dead worms were encapsulated and surrounded by thickened intima and fibrous tissue obliterating the lumen of the blood vessels. The surrounding inflammatory reaction consisted of polymorph and eosinophil leucocytes and round cells. Numerous eggs occurring singly and in nests, with surrounding cellular inflammatory reaction, were scattered throughout the lungs. Many of the large parabronchial blood vessels showed great thickening, especially of the intima, presumably a reaction to schistosomal infection.

Liver. The liver was infected in all the animals (see Fig. 13, 14). One contained only dead worms, which were encapsulated in thick fibrous tissue. The absence of schistosome eggs suggested that this animal had a unisexual infection, an opinion supported by the finding at autopsy of 60 male worms in the liver and four in the mesenteric veins, but no females. In all the other animals the portal tracts were heavily infiltrated with inflammatory cells and showed numerous characteristic bilharzial tubercles. The involvement of the portal tracts, with associated cellular infiltration and fibrosis, resulted in considerable distortion, with compression, of the adjacent parenchymal tissue. The sinusoids contained an increased number of inflammatory cells, including polymorph and eosinophil leucocytes. The Kupffer cells were heavily loaded with yellowish brown or black pigment.

Spleen. In two of the animals the spleen was affected, showing scattered eggs and the remnants of shells in the pulp with surrounding cellular reaction (Fig. 15); one also showed acute peri-splenitis. All six showed congestion and it was noted
that the Malpighian corpuscles were prominent. Large numbers of cells, heavily loaded with pigment, were scattered in the pulp.

Pancreas. The pancreas was affected in four of the animals. In these there were numerous eggs occurring in large egg-nests that displaced and destroyed the parenchymal tissue (Fig. 16). In one animal a live worm was found in a vein with no surrounding cellular reaction.

Kidneys and adrenals. In two animals there was congestion of the kidneys, one of which had a presumed bilharzial tubercle in the renal cortex, but no eggs or remnants of eggs could be identified. No bilharzial lesions were detected in the adrenals, but in one there was an inflammatory infiltration of round cells, including plasma cells, in the medulla.

Bladder. In one of the animals there was great thickening of the submucous coat, which contained a few bilharzial tubercles developed in relation to eggs (Fig. 17).

Intestine. The intestines were infected in all the animals except the one with a presumed unisexual male infection. Numerous egg-nests were present in the submucous coat, with marked cellular and fibrous reaction. Some animals had eggs in the mucous coat (Fig. 18, 19).

In summary, all the animals showed signs of heavy or very heavy infections, with indications of continuing progression. It is clear from these findings that Saccostomus campestris is highly susceptible to infection with S. haematobium and is an animal eminently suitable for laboratory studies of this infection.

Arvicathanus niloticus

Seventeen Arvicathanus were infected in February 1962. Of these, eight survived for one year and were sacrificed in February and March 1963. All were found to be infected.

Brain. No lesions of the brain were found.

Heart. There was eosinophil degeneration and inflammatory reaction of the heart muscle in two animals. The nature of these lesions was not identified, but possibly could be related to schistosomal infection. However, neither eggs nor worms were found in the muscle or cavities.

Lungs. The lungs were infected in six of the eight animals and showed eggs associated with an inflammatory reaction consisting of polymorph and eosino-phil leucocytes and round cells, including plasma cells. In one animal a live worm was found.

Liver. The liver was infected in all the animals, but in none was there thickening of the capsule. Worms were found in four of the eight livers and dead worms were associated with marked inflammatory reaction. The portal tracts were thickened and there was considerable inflammatory infiltration, often associated with fibrosis. Numerous bilharzial tubercles had developed in relation to ova, some viable, others dead, and others with remnants of the shells only. Several showed large egg-nests with surrounding fibrosis and cellular infiltration of polymorph leucocytes and round cells, including monocytes and plasma cells. Many cells were heavily loaded with brownish black pigment. One liver showed an acute submiliary abscess, the nature of which was not determined.

The parenchyma adjacent to the portal tracts was compressed, but otherwise showed no marked change. The sinusoids had an increased number of cells, including polymorph and eosinophil leucocytes; the Kupffer cells contained brownish black pigment and were often heavily loaded.

Spleen. Sections of the spleen of three animals showed bilharzial lesions, with scattered eggs and egg-nests associated with cellular reaction. The Malpighian corpuscles were prominent, with active germinal centres, some showing a few with pyknotic nuclei cells. Large numbers of cells heavily loaded with brownish black pigment were scattered in the pulp.

Pancreas. The pancreas was infected in six animals, with scattered eggs and egg-nests displacing and destroying the parenchymal tissue and associated with marked inflammatory cellular and fibrous reaction.

Kidneys and adrenals. No bilharzial lesions were detected in the kidneys but it was noted that all showed dilatation of the tubules of the cortex and scattered collections of round cells associated with the veins. The adrenals showed congestion but no bilharzial lesions.

Bladder. No bilharzial lesions were found in the bladder.

Intestine. The intestines were infected in all the animals and there were large numbers of eggs occurring singly or in nests in the submucous coat, with marked subacute inflammation and fibrosis.

In summary, Arvicathanus niloticus is highly susceptible to S. haematobium and the infection con-
tinues to progress for at least one year. This animal therefore appears to be suitable for experimental studies of *S. haematobium* infections.

*Aethomys chrysophilus*

Of the 13 animals exposed in February 1962, only three survived to be sacrificed in February 1963; all three showed signs of heavy infection.

*Brain.* No bilharzial lesions were found in the brain.

*Heart.* The heart was not infected.

*Lungs.* In one of the three animals eggs were found in the lung, with surrounding cellular reaction. The lymphoid nodules were prominent. Great thickening and tortuosity of the blood vessels were noted at one apex, possibly a reaction to a dead worm, although this could not be identified.

*Liver.* The liver was infected in all three animals and live worms were found in one. The capsule was not thickened. The portal tracts showed marked involvement, with infiltration of inflammatory cells, including polymorph leucocytes, numerous eosinophil leucocytes and round cells, including monocytes and plasma cells. Large numbers of pigment cells loaded with yellowish brown or black pigment were seen. There were numerous bilharzial tubercles in the portal tracts, with central giant cells, surrounding endothelial cells and polymorph and eosinophil leucocytes and round cells, including plasma cells. There were areas in which the parenchymal cells were less granular than normal, associated with an infiltration of polymorph and eosinophil leucocytes and other inflammatory cells. There was an increase in the number of inflammatory cells, including polymorph and eosinophil leucocytes in the sinusoids, and the Kupffer cells contained masses of brownish black pigment.

*Spleen.* No bilharzial lesions were detected in the spleen, but there was some congestion and in one of the animals fibrous nodules were present.

*Pancreas.* No lesions were found in two animals, but the other showed egg-nests associated with inflammatory cellular reaction replacing the parenchymal tissue.

*Kidneys and adrenals.* No bilharzial lesions were found in the kidneys or adrenals.

*Bladder.* None of the animals showed bladder infection.

*Intestine.* In two of the three animals there was infection of the intestine, with thickening and fibrosis of the submucous coat in relation to bilharzial tubercles.

In summary, *Aethomys chrysophilus* is highly susceptible to *S. haematobium* and from this point of view is suitable for use as a laboratory animal. However, a high mortality rate was noted in this study and possibly would be a disadvantage. Moreover, this rodent breeds poorly in the laboratory.

*White mouse*

Eleven white mice were exposed to infection in February 1962; only two survived for one year, one of which was sacrificed in January and the other in February 1963. Both showed infection.

*Brain.* The brains showed no lesions.

*Heart.* No bilharzial lesions were found in the heart.

*Lungs.* In one of the animals, dead dissolving worms were associated with great thickening of the intima and walls of the blood vessels, with surrounding inflammatory infiltration. The other showed a few tubercles related to schistosome ova.

*Liver.* The capsules were not thickened, but the portal tracts showed considerable thickening, with fibrosis and subacute inflammatory infiltration. The veins were dilated and there were numerous eggs with surrounding fibrous reaction and cellular infiltration, including polymorph and eosinophil leucocytes, round cells and plasma cells. There were many pigment cells and several bilharzial tubercles were present in various stages of formation, from recently developed to old fibrous nodules. The Kupffer cells contained brownish black pigment.

*Spleen.* The spleen showed no bilharzial lesions but the pulp contained numerous pigment cells.

*Pancreas.* In one of the animals the pancreas had a few egg-nests, but the other showed no bilharzial lesions.

*Kidneys and adrenals.* Neither kidneys nor adrenals showed any lesions.

*Bladder.* No bilharzial lesions were seen.

*Intestine.* In one of the animals no ova or worms were found; the other contained a few scattered eggs associated with inflammatory reaction in the submucosa.

In summary, the white mouse is susceptible to infection by *S. haematobium*, but a high mortality rate was noted in this study.
**Tatera brantsi**

Seventeen animals were infected in February 1962; 13 survived for one year and were sacrificed in late January and February 1963. Of the 13 animals examined, seven contained no eggs or schistosomes but lesions, presumably those of bilharziasis, were detected in all.

**Brain.** There was eosinophil degeneration in the olfactory lobes of one brain but presumably this was not related to bilharziasis. The other brains showed no lesions.

**Heart.** No eggs or worms were found in the heart of any of the animals.

**Lungs.** The lungs were infected in two animals. They showed nodules of inflammatory cells associated with the remnants of ova. In one, one dead schistosome, associated with marked inflammatory reaction, was found.

**Liver.** Bilharzial lesions were found in the livers of all the animals, but in most they were relatively slight. One liver had an infection with considerable cellular infiltration of the portal tracts and several bilharzial tubercles, some with viable ova and others with remnants of ova and shells with surrounding multinucleate giant cells, endothelial cells and inflammatory reaction, including polymorph and eosinophil leucocytes and round cells, including numerous plasma cells. It was noted that in some animals the parenchymal cells were pale. The Kupffer cells contained brownish black pigment.

**Spleen, pancreas, kidneys and adrenals.** No bilharzial lesions were found in any of these organs.

**Bladder.** In one, the heavily infected animal, many eggs, occurring singly and in nests, were found in the submucous coat (Fig. 20).

**Intestine.** Large egg-nests were found in the intestine of one animal. In the others no ova or worms were detected, although two of them showed an inflammatory reaction in the submucous coat, possibly related to bilharziasis.

From these findings it appears that *Tatera brantsi*, except for isolated individuals, is not as susceptible to *S. haematobium* as the other rodents studied in this investigation.

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**Susceptibility of Other Wild Rodents to Schistosomes**

The following species of wild rodents have also been found in this laboratory to be susceptible to schistosomes—

<table>
<thead>
<tr>
<th>Rodent</th>
<th>Schistosome</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Lemniscomys griselda</em></td>
<td><em>S. haematobium</em> (poor)</td>
</tr>
<tr>
<td>(diurnal)</td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td><em>Rattus rattus</em></td>
<td><em>S. mattheei</em></td>
</tr>
<tr>
<td>(nocturnal)</td>
<td><em>S. mansoni</em> (poor)</td>
</tr>
<tr>
<td><em>Steatomys pratensis</em></td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td>(nocturnal)</td>
<td><em>S. mattheei</em></td>
</tr>
<tr>
<td><em>Rhabdomys pumilio</em></td>
<td><em>S. haematobium</em> (poor)</td>
</tr>
<tr>
<td>(diurnal)</td>
<td><em>S. mansoni</em> (poor)</td>
</tr>
<tr>
<td><em>Tatera leucogaster</em></td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td>(nocturnal)</td>
<td><em>S. mattheei</em></td>
</tr>
<tr>
<td><em>Mystromys albicaudatus</em></td>
<td><em>S. haematobium</em></td>
</tr>
<tr>
<td>(nocturnal)</td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td></td>
<td><em>S. mattheei</em></td>
</tr>
</tbody>
</table>

None of these species, with the exception of *Rattus* and *Mystromys*, has yet been bred in the laboratory.

The white-tailed rat or South African hamster, *Mystromys albicaudatus*, is the only member of the subfamily Cricetinae in sub-Saharan Africa and is restricted to the south east of southern Africa. It has been bred in the laboratory since 1940 and has been extensively used as an experimental animal. In the laboratory it is long-lived and may reach the age of 6 years. It breeds readily and may produce over 20 litters in its lifetime (Davis, 1962). It is thoroughly adapted to laboratory conditions and is now easy to handle. It weighs up to 180 g, needs no special care or diet and is susceptible to *S. haematobium*, *S. mansoni* and *S. mattheei*. In the field it avoids water but does not occur in the areas where bilharziasis is endemic.

Natural schistosomal infections in wild rodents in Africa have been described and tabulated (Pitchford & Visser, 1962). Since then, Nelson et al. (1962) have incriminated several other species in Kenya. The present position is as follows:

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**Susceptibility of Other Wild Rodents to Schistosomes**

The following species of wild rodents have also been found in this laboratory to be susceptible to schistosomes—

<table>
<thead>
<tr>
<th>Rodent</th>
<th>Schistosome</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Lophuromys flavopunctatus</em></td>
<td><em>S. bovis</em></td>
</tr>
<tr>
<td><em>Praomys (Mastomys)</em></td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td><em>natalensis</em></td>
<td><em>S. mansoni</em> (var. rodentorum)</td>
</tr>
<tr>
<td></td>
<td><em>S. mattheei</em></td>
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<tr>
<td></td>
<td><em>S. rodhaini</em></td>
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<tr>
<td><em>Pelomys</em></td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td></td>
<td><em>S. rodhaini</em></td>
</tr>
<tr>
<td><em>Otomys angoniensis</em></td>
<td><em>S. haematobium</em></td>
</tr>
<tr>
<td></td>
<td><em>S. mansoni</em></td>
</tr>
<tr>
<td></td>
<td><em>S. mattheei</em></td>
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</tbody>
</table>
RÉSUMÉ

Etant donné que de petits animaux de laboratoire sont nécessaires à l’étude expérimentale des infections à *Schistosoma haematobium*, les auteurs ont recherché systématiquement la réceptivité au parasite de plusieurs rongeurs sauvages d’Afrique du Sud: *Praomys (Mastomys) natalensis*, *Saccostomus campestris*, *Arvicanthus niloticus*, *Aethomys chrysophilus*, *Tatera brantsi* et des souris blanches de la souche SAIMR 200. Des groupes d'ani naux de chaque espèce ont été observés quotidiennement pendant un an après avoir été exposés une fois au même nombre de cercaires d’une souche de *S. haematobium* du Transvaal entretenue au laboratoire.

A l’autopsie, ni les parasites ni leurs œufs n’ont été retrouvés au niveau du cœur ou du cerveau des animaux infectés; on a observé, assez rarement, la présence d’œufs au niveau de la vessie et de la rate. En revanche, les poumons, le foie, le pancréas et les intestins présentaient fréquemment, à l’examen nécropsique, des œufs, et, dans le foie et les poumons notamment, des vers adultes vivants ou morts. De nombreux vers ont survécu au moins un an chez *Praomys (Mastomys) natalensis* qui s’est montré très réceptif. Mais la présence de nombreux vers morts donne à penser que le rongeur était en voie de guérison. Tous les *Saccostomus campestris* ont présenté une forte ou une très forte infection; ces rongeurs semblent convenir parfaitement aux études expérimentales sur la bilharziose à *S. haematobium*; il en est de même d’*Arvicanthus niloticus*, également très réceptif à l’infection qui évolue chez cet animal pendant au moins un an. *Aethomys chrysophilus* et les souris blanches ont témoigné d’une forte réceptivité mais chez ces rongeurs, l’infection a entraîné une mortalité élevée. A l’exception de quelques individus, *Tatera brantsi* a été moins sensible que les autres espèces étudiées.

Les auteurs donnent en complément de leur étude, une liste de rongeurs réceptifs à l’infection mais qui n’ont pas encore été élevés en captivité ainsi qu’une liste de rongeurs d’Afrique qui présentent une infection naturelle par divers schistosomes.

REFERENCES