Medical Memoranda

Is the Rat (*Rattus rattus*) ever a Permanent Reservoir of Plague?

Black rats (*Rattus rattus*) are well known as propagators of plague, and Roberts (1950) believes they are the only rodents concerned in outbreaks in Kenya. However, recent work (Heisch, 1952) indicates that wild rodents are the permanent reservoir in Kenya, and that rats are infected secondarily and are incapable of harbouring plague for long periods. Many workers think that rats are never permanent reservoirs of plague; this may be true, but it has not yet been proved.

One of the main features of wild-rodent plague is a tendency for the infection to persist indefinitely in areas once invaded. Rodents often show a marked resistance to the infection, and harbour *Pasteurella pestis* in their tissues and fleas without showing signs of disease. Such invisible or inapparent plague is usually demonstrated by pooling the tissues and/or the fleas of groups of rodents and inoculating guinea-pigs. Such techniques, known as "pooling tests," are of great value in revealing foci of sylvatic plague which might otherwise remain undetected. If *P. pestis* can be isolated from apparently healthy rodents and/or their fleas during silent or interepizootic periods, it strongly suggests that the rodents concerned are permanent reservoirs of plague.

In Kenya, rats (*R. rattus*) are unlikely to be permanent reservoirs. Not only are they highly susceptible, but the infection soon dies out, and *P. pestis* has never been isolated from their tissues or fleas during interepizootic periods. Though far fewer rats than wild rodents have been subjected to pooling tests in Kenya, it seems significant that 827 *R. rattus* and 1,564 *Xenopsylla brasiliensis*, from the old silent focus of Keruguya on the slopes of Mount Kenya, were negative for *P. pestis* (Heisch *et al.*, 1953). Different results have been obtained in other endemic areas. Thus, in India, rats often show a marked resistance and even develop inapparent infections. At the Calcutta Conference of Medical Research Workers in 1934, it was reported that 13 out of 141 tissue pools, derived from 2,107 apparently healthy rats, were positive when inoculated into guinea-pigs. Inapparent infections in commensal rodents have also been demonstrated by Peryassú (1934) in Brazil and Devignat (1940, 1949) in the Belgian Congo; but according to Pollitzer (1954) it was never claimed that such animals played a part in the perpetuation of plague. It seems strange that inapparent infections in wild rodents are considered important while similar infections in commensal rats are disregarded.

It is not clear whether the inapparent infections in commensal rats were found during interepizootic periods or during epizootics when rats may have been incubating plague or recovering from it. It is the interval between epizootics which is the critical period, and determined attempts should be made to isolate *P. pestis* from rats and their fleas at such times. Though pooling techniques have proved invaluable in sylvatic plague studies, they appear not to have been so widely used for detecting *P. pestis* in commensal rodents. This may partly explain why it is still uncertain whether

rats are a permanent reservoir in India, Madagascar, Egypt, Senegal, Peru, Brazil, and elsewhere. The brilliant work of Baltazard (in press) has shown that the gerbil—*Tatera indica*—is an important though perhaps rather unstable reservoir in India, but the possibility that rats may also maintain the infection does not yet seem to have been excluded.

Meyer (1957) has stated that "it is becoming increasingly apparent that the highly susceptible rodents, such as the marmot, the squirrel, and the rat, are not the permanent reservoir of the plague bacillus." This overlooks the fact that a highly susceptible species like *R. rattus* may sometimes develop a marked resistance to the plague bacillus. The terms "susceptible" and "resistant" as applied to rodent species have a relative rather than absolute connotation. The persistence of plague in focal areas is a complex matter, and apparently depends on the presence of susceptible as well as resistant rodent species. It can of course be argued that fleas and not rodents are the chief reservoir of plague. Wild rodents and their fleas are of primary importance in the perpetuation of plague, but it seems doubtful whether the commensal aspect of the reservoir problem is quite in perspective.

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Familial Incidence of the Henoch-Schönlein Syndrome

The Henoch-Schönlein syndrome is now regarded as an allergic or hypersensitivity reaction related to conditions such as erythema nodosum (Wintrobe, 1956) or polyarteritis nodosa (Lecutier, 1952). Visceral forms were first described by Henoch (1874) and the joint symptoms by Schönlein (1837), but these two manifestations are now considered to form part of one syndrome produced by capillary fragility without thrombocytopenia (Ackroyd, 1953). Osler (1904) described a relationship between the skin manifestations of the syndrome and erythema multiforme. The syndrome often occurs after the consumption of certain foods and after upper respiratory infections, frequently due to haemolytic streptococci (Gairdner, 1948). It is not usually regarded as a familial complaint (Davis, 1948), although a family history of allergy is frequently obtained (Alexander and Eyermann, 1929). Wedgwood and Klaus (1955) recorded a series of cases in which no familial incidence of the disease was found. Reports of cases occurring in siblings are uncommon, and the histories of the two children reported here are thought interesting enough to be recorded. The second child, aged 14 months, is much younger than the usual patient with the syndrome; it is believed to be rare under the

age of 2 years, although the youngest authenticated case was aged only 8 months (Philpott, 1952). The two cases are otherwise typical of the syndrome.

CASE 1

This girl, aged 5 years, was admitted to the children's ward of Dorking General Hospital on March 6, 1958. She had been unwell and listless since an attack of influenza at the beginning of January. On March 1 she had her second injection of anti-diphtheria and pertussis vaccine (Glaxo). She had vomited occasionally since that evening until her admission: the vomitus had once contained flecks of fresh blood. Since March 4 she had complained of lower abdominal pain and intermittent pain in both calves and the metacarpo-phalangeal joints of both thumbs. There was no family history of allergy.

On admission she was flushed and febrile, and had numerous petechial haemorrhages on the hard and soft palates. There were palpable soft lymph nodes on both sides of the neck and in the axillae and groins. She had diffuse abdominal tenderness, but no abnormal masses or enlarged viscera were felt.

Investigations gave the following results. Haemoglobin, 88%; total white-cell count, 14,000/c.mm. (P. 77%, E. 1%, L. 22%); platelet count, 495,000/c.mm.; bleeding-time, 2 minutes; clotting-time, 1 minute (Dale and Laidlaw); throat-swab culture yielded a heavy mixed growth of *Neisseria catarrhalis* and *Streptococcus viridans*; urine contained no albumin; chest radiograph, clear.

The day after admission she developed small haematomata on both upper eyelids. Her lower abdominal pain persisted, and on the fourth day after admission she passed a stool containing a little altered blood. She had a brief epistaxis on the same evening, but then had no further overt bleeding. Intermittent colic continued for another week, and she then became symptom-free. She was discharged home on March 23. During the next six weeks she had several attacks of abdominal pain and some transient swelling of the left ankle. She was last seen three months after her discharge, when she had been quite well for six weeks.

CASE 2

This boy of 14 months was the only sibling of Case 1. He was admitted to the same hospital (after a domiciliary consultation) on March 22, with six days' history of a purulent discharge from the left ear. Several crops of red spots had occurred on his legs during the same period. His left knee had been swollen for four days, and for three days he had been passing fresh blood per rectum: his urine was said to be red. On the morning of admission he had developed spontaneous swelling and discoloration around his left eye. He had been treated with oral penicillin, some of which he vomited.

He had a left orbital haematoma and an ecchymosis on the right upper eyelid. There was a purulent otorrhoea from a perforation of the left eardrum. There was oedema of both hands and feet, with a reddish maculo-papular rash over both legs and forearms. Both knee-joints were swollen.

Investigations resulted as follows. Haemoglobin, 72%; total white-cell count, 13,000/c.mm. (P. 57%, E. 3%, L. 33%, M. 7%); platelets, 225,000/c.mm.; bleeding-time 2½ minutes; clotting-time, 3½ minutes (Dale and Laidlaw); ear-swab culture yielded a light growth of coagulase-negative staphylococci. Urine (non-catheter) contained a small amount of amorphous urates and occasional calcium oxalate crystals and yielded a mixed growth of *Proteus vulgaris* and *Streptococcus faecalis*.

He was pyrexial for a fortnight, and was given a full course of crystalline penicillin and ephedrine nose-drops. His otitis media cleared and the orbital haematoma quickly resolved. The rash and joint swellings gradually subsided,

and he was discharged on May 4. When seen again six weeks later he had had no recurrence of symptoms.

COMMENT

The only previous case of a familial incidence of the Henoch-Schönlein syndrome is reported by Gibson and Hobson (1932) in two siblings who died after developing purpura without thrombocytopenia while recovering from mild attacks of scarlet fever. These children were thought to have a constitutional susceptibility to the scarlatinal toxin. Such a definite common predisposing cause was not present in the two cases reported here, but it seems unlikely that the almost concurrent development of the syndrome was coincidental. It is improbable that an article of diet was responsible in both cases, because, although both children received a similar diet before admission, only one of them had further symptoms after discharge. In Case 1 the syndrome appeared soon after immunization against diphtheria and pertussis, and in Case 2 it was associated with an upper respiratory infection. It seems that these two different factors provoked the same hypersensitivity reaction—that is, the Henoch-Schönlein syndrome. The type of hypersensitivity reaction, rather than the nature of its provocation, appears to be the common familial factor in these two cases.

I am grateful to Dr. Philip Ellman for his help in the preparation of this paper and for permission to report these two cases under his care, and also to Dr. D. M. Kellett Carding, who referred both children.

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"While many factors affect the development of children into vigorous adults, it is certain that abundant physical exercise (followed by long sleep at night) is of primary importance. . . . When children attend day school it is not easy to ensure that they take daily exercise, and an important proportion of them dislike organized games. School buses provide a passive means of reaching school for many of the county's scholars, and parents who have lost the habit—or never had it—of taking exercise themselves do not encourage their children to walk, run, or cycle. So the development of less formal types of exercise, such as the experimental ten-day adventure courses and canoe courses, are particularly welcome as a means of arousing an interest and desire for physical exertion which can more easily continue into adult life. When the time comes that every normal adult retires to bed at night tired from bodily movement during the day which has been sufficient to make him or her out of breath for a short while, we shall see less ill-health, particularly heart trouble, and a lowering of the number of cases of neurosis." (*Annual Report of the School Medical Officer for the Year 1958*, Staffordshire County Council.)