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Yersinia pseudotuberculosis Infection: Study of an Epizootic in Squirrel Monkeys

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An epizootic of an acutely fatal enteric disease in a colony of squirrel monkeys (*Saimiri sciureus*) was attributed to infection by *Yersinia pseudotuberculosis* serotype III. Of a total adult population of 96 animals at risk, there were six fatal cases of yersiniosis. Serological evaluation of the colony just after the outbreak ended revealed that 22 of 60 monkeys tested (37%) had significant antibody to *Y. pseudotuberculosis* (microagglutination titer of ≥1:80) but did not have clinical disease. The outstanding pathological lesions noted in dying monkeys were acute, purulent, necrotic and focal enteritis primarily affecting the jejunum and ileum and focal hepatic necrosis and abscessation. *Y. pseudotuberculosis* was isolated from the organs of two of the dying monkeys. Using cold enrichment techniques, *Yersinia* was also isolated from the feces of two apparently healthy monkeys (both seropositive), from the spleen of a monkey dying of other causes, and from the colon contents of a stillborn squirrel monkey baby. All isolates had the same biotype and serotype. An episode of abortions was associated both temporally and spatially with the fatal cases of yersiniosis, and *Y. pseudotuberculosis* was cultured from the uterus of two of the dying monkeys, suggesting that yersinia infection may be associated with abortion, as well as with enteric infection, in these animals.

Yersinia pseudotuberculosis is one member of a generic triad of pathogenic bacteria within the family *Enterobacteriaceae*, which also includes *Yersinia enterocolitica* and *Yersinia pestis*. Infection with *Y. pseudotuberculosis* is zoonotic, as the disease affects wild and domestic animals, nonhuman primates, and humans (16, 22). In fact, infection has been described in more than 75 species of birds and mammals (8, 9, 14). The geographical distribution of the organism includes northern Asia, Europe, North and South America, and Africa. Many infected animals develop abdominal disease which includes enteritis, typhlitis, mesenteric lymphadenitis, and abscessation of liver and spleen. However, birds, rats, mice, and other small rodents may become infected and not develop disease, but instead become chronic carriers of *Y. pseudotuberculosis* with fecal shedding of the organism. These animals are believed to serve as the primary reservoir of infection (14, 16).

Outbreaks of disease due to *Y. pseudotuberculosis* in captive nonhuman primates have been reported in both the United States and Europe (5, 16). Two separate epizootics of yersiniosis, both due to *Y. enterocolitica*, have also been reported in colonies of nonhuman primates in the United States, with lesions similar or identical to those caused by *Y. pseudotuberculosis* (1, 15). These outbreaks involved considerable mortality and in several cases affected a large number of monkeys.

In the spring of 1977, an outbreak of yersiniosis occurred in a colony of squirrel monkeys (*Saimiri sciureus*) housed at the University of California at San Diego. This report describes the epidemiological investigation of that outbreak, which allowed sequential study of both the serological and bacteriological status of the affected animals.

MATERIALS AND METHODS

Description of the colony. In December 1975, the School of Medicine, University of California at San Diego, established a colony of squirrel monkeys at its Elliott field station facility located on 30 acres of brush land 15 miles northeast of downtown San Diego. The colony was initiated with 39 adult males and 68 adult females. Approximately 18 of the monkeys (six males and twelve females) were of the Roman type (Iquitos, Peru) and were originally obtained from Primate Imports, Inc. (Port Washington, N.Y.). The remainder were of the Guyanese type (Georgetown, Guyana) and were obtained from South American Primates, Inc. (Miami, Fla.). All adult monkeys had been trapped as

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adults in the wild. The animals were housed since the inception of the colony in six rectangular wire cages (6 by 20 by 8 ft in height [ca. 1.83 by 6.10 by 2.44 m]) with concrete slab floors. Each cage had approximately 40% of its total area out of doors, and the remaining 60% was inside of a large building with permanent open ventilation to the outside. Cages were adjacent to one another with no more than 18 in. (ca. 46 cm) between them and were numbered one through six from east to west. All monkeys received Purina high protein monkey chow and water ad libitum, fresh fruit twice weekly, and a fruit drink supplemented with vitamins daily.

Tissue collection and processing. Animals were necropsied, and tissues were fixed in 10% buffered Formalin. Tissues were embedded in paraffin by using standard techniques, sectioned at 4 to 6 μm, and stained with hemotoxylin and eosin and in some cases with Giemsa or Brown-Brenn stain.

Microbiology. Initially, specimens collected at necropsy were cultured on blood agar plates, on MacConkey agar, and in thioglycolate broth. To identify organisms we utilized standard tubed media and the API 20E identification system (Analytab Products, Plainview, N.Y.). Cold enrichment for *Y. pseudotuberculosis* (6, 7, 18) employed Mg^{2+}- and Ca^{2+}-free phosphate-buffered saline (PBS) (GIBCO Laboratories, Grand Island, N.Y.). Pieces of tissue and rectal swabs or cecum contents obtained with sterile cotton swabs were placed into 2 to 5 ml of PBS and held at 4°C. At approximately 14 and 28 days, a sterile swab was introduced into the PBS and plated onto MacConkey agar, which was incubated at 37°C for 48 h. Pinpoint, translucent colonies with *Yersinia* morphology were tested for the oxidase reaction and for urease production, and oxidase-negative, urease-positive organisms were identified by using the API 20E System. Isolates were sent to the Microbial Diseases Laboratory, California State Department of Public Health, Berkeley, for confirmation of identity, determination of biochemical reactions by using techniques previously described (3), and serotyping by tube agglutination (12).

Serology. Serum antibodies to *Y. pseudotuberculosis* were determined by using a microagglutination test. One of the original isolates from the monkey colony was maintained on blood agar plates subcultured every 1 to 2 months and held at room temperature. The organism was grown on blood agar for 48 h at 20 or 37°C, and a live antigen suspension was made in PBS which was adjusted to a constant cell density of 80% transmission at 640 nm, using a Coleman Junior spectrophotometer. Serum which had been stored at −20°C was thawed and diluted to 1:5 with PBS, and serial twofold dilutions were made to 1:1,280 in PBS. One drop (0.025 ml) of serum dilution was mixed with one drop of antigen suspension on a glass microscope slide and mixed with a wooden applicator stick. The mixture was incubated at room temperature for 30 min; a glass cover slip (18 by 18 mm) was placed over the mixture to reduce drying, and the mixture was incubated for an additional 30 min. Slides were read at ×100 and ×400, and microagglutination was scored as 1+ to 4+. The titer was the highest dilution giving 1+ agglutination. This degree of agglutination was compared with the reaction of negative and positive controls run simultaneously with each test. Control sera consisted of a known positive rabbit antiserum to our isolate of *Y. pseudotuberculosis*, serum from a healthy squirrel monkey with a titer of 1:320, and serum from a squirrel monkey with a titer of <1:10. Rabbit antiserum to *Y. pseudotuberculosis* was prepared by injecting an adult New Zealand rabbit intravenously with 0.5 ml of a Formalin-inactivated preparation of the organism. Injections were made on days 0, 3, 5, 7, 9, and 12, and on day 19 the rabbit was exsanguinated under pentobarbital anesthesia, and the serum was stored at −20°C. This serum had a microagglutination titer of 1:1,280.

Trapping and examination of wild mice. Between September 1977 and June 1978 (5 to 14 months after the start of the epizootic), we trapped and examined 83 wild mice near the monkey colony. A total of 56 *Mus musculus* and 27 *Peromyscus maniculatus* were trapped live, inside or within 50 m of the colony. The mice were euthanized, and the spleen and cecum contents were cultured by cold enrichment. Blood was collected from some mice for serological evaluation.

RESULTS

Clinical findings. A diagnosis of yersiniosis was made postmortem in six adult squirrel monkeys. The diagnosis was made on the basis of pathological lesions (monkeys 612, 58, and 33), pathological lesions and culture of *Y. pseudotuberculosis* (monkeys 47 and 53), and pathological lesions and a high serum antibody titer (1:1,280; monkey 24). Clinical signs associated with disease are shown in Table 1. The duration of illness varied from 2 to 7 days, but in most cases lasted only 2 to 3 days. Three of the five female monkeys were pregnant at the time of death or had evidence of a recently terminated pregnancy, and one of these females died during dystocia. Several of the monkeys were treated with fluids and antibiotics. Three of the six were euthanized while severely ill.

Pathological findings. Distribution of lesions is shown in Table 1. The outstanding lesions in all six monkeys were intestinal, characterized grossly as raised, reddened, elliptical nodules, or raised, annular, tan nodules on the serosal, antimesenteric surface of the jejunum and ileum. On the mucosal surface, there were elliptical foci characterized by a red-brown perimeter and a grey center. In one monkey the cecum was involved, with a red-black discoloration and a thickened wall. Microscopically, there was acute to chronic-active, multiple, focal, ulcerative enteritis. Lesions often eroded into and through the submucosa, and several were transmural. Lesions were often associated with a Peyer's patch. One monkey had acute, hemorrhagic, chronic-active typhlitis with ulceration. In some

TABLE 1. *Summary of clinical and pathological findings in squirrel monkeys dying of Y. pseudotuberculosis infection*

Monkey no.[a]	Cage no.	Clinical signs	Histological lesions[b]			
			Small intestine	Liver	Spleen	Uterus
612 (M)	1	Unknown (euthanized)	+	+	+	
58 (F)	6	Dystocia, found dead	+	−	−	−
47 (F)	6	Pregnant, comatose	+	−	−	+[c]
33 (F)	5	Weakness, depression, diarrhea	+	+	−	NE[d]
53 (F)	6	Vaginal hemorrhage, weakness, abortion?	+	+[c]	−	+[c]
24 (F)	4	Depression, dehydration, diarrhea	+	+	−	NE

[a] Sex of monkey is given within parentheses.
[b] Significant microscopic lesions noted to be present (+) or absent (−).
[c] Indicates that *Y. pseudotuberculosis* was cultured from this organ.
[d] NE, Not examined microscopically.

monkeys, mesenteric lymph nodes were grossly reddened and microscopically showed hypermia and reactive hyperplasia but no lymphadenitis.

Liver lesions, when macroscopic, consisted of multiple, punctate, 0.5- to 2-mm-diameter foci of white-grey discoloration. Microscopically, liver lesions varied from small foci of acute hepatic necrosis to larger microabscesses. Rod-shaped bacteria were seen in some liver lesions and were gram negative.

Splenitis in one monkey was characterized by foci of abscessation. Uterine lesions were characterized by acute, erosive endometritis, with areas of focal necrosis, hemorrhage, and variable myometritis.

Microbiological findings. *Y. pseudotuberculosis* was recovered from monkeys 47 and 53, using routine microbiological techniques (Table 1). Monkeys 612, 33, and 58 were not cultured at necropsy. Cultures from monkey 24 yielded *Klebsiella pneumoniae*, *Enterobacter cloacae*, and a *Streptobacillus* from the liver, mesenteric lymph nodes, and spleen, but no *Yersinia* was isolated even by cold enrichment of a number of tissues. In addition to *Yersinia*, *Escherichia coli* was isolated from the kidney, mesenteric nodes, intestines, liver, and peritoneum, and a group D streptococcal organism was isolated from the uterus, intestines, and liver of monkey 47.

Y. pseudotuberculosis isolates were initially identified in our laboratory as gram-negative rods which were oxidase negative, growing as pinpoint-size to 1-mm translucent colonies on MacConkey agar. They were urease-, *o*-nitrophenyl-β-D-galactopyranoside-, nitrate-, and esculin-positive and fermented glucose, mannitol, rhamnose, and arabinose. Isolates were sent to the Microbial Diseases Laboratory and confirmed to be *Y. pseudotuberculosis*, with biochemical reactions as listed in Table 2. All isolates had the same biotype, all were serotype III, and none of the isolates fermented melibiose (an unusual characteristic for *Y. pseudotuberculosis*).

Epizootiology of the outbreak. The temporal characteristics of the *Y. pseudotuberculosis* epizootic in the colony are shown in Fig. 1. Between December 1975 when the colony was started and March 1977, there had been three deaths in adult monkeys (monthly death rate, 2.1 per 1,000). From April to July 1977, there were eight adult deaths, six due to *Y. pseudotuberculosis* (monthly death rate, 22 per 1,000). Coinciding with the adult deaths was an unusual occurrence of abortions (seven in 1 month). Between July 1977 and July 1978, the adult death rate was 4.9 per 1,000 per month, and the breeding season of 1978 was not characterized by a high incidence of abortions (three in 1978 versus eight in 1977).

The spatial characteristics of the epizootic are shown in Table 3. The adult deaths attributed to *Yersinia*, as well as the abortions and infant deaths for the period from April to December 1977, were clustered at the west end of the facility, and the highest incidence of deaths due to all causes occurred in cage 6. In cage 6 during the 1977 breeding season, eight of the nine pregnancies ended in abortion or infant deaths.

In August 1977, we initiated a survey of cages 1, 4, 5, and 6 to determine the serological status of squirrel monkeys to *Y. pseudotuberculosis* and to detect latent carriers of the organism by fecal culture. Between August and October 1977, 4 to 6 months after the start of the epizootic, monkeys in these cages were bled for serum, and a fecal culture for *Y. pseudotuberculosis* was performed by using cold enrichment (Table 3). Of the monkeys sampled, 37% (22 of 60) had serum antibody titers ≥1:80, with titers ranging to 1:1,280. Geometric mean titers were similar in all four cages. Only 2 of 59 monkeys (3%) cul-

TABLE 2. *Biochemical characteristics of Y. pseudotuberculosis isolates*

Test	Result[a]	Test	Result[a]
Oxidase	0	Glucose	A
Catalase	+	Lactose	–
Motility	+(25°C)	Maltose	A
Gelatin	0	Sucrose	–
Citrate	0	Mannitol	A
Urease	+	Raffinose	–
Indole	0	Adonitol	–
Nitrate	+	Xylose	A
Voges-Proskauer	0	Dulcitol	–
Malonate	0	Inositol	–
Triple sugar iron	Alkaline/acid/–	Sorbitol	–
Esculin	+	Salicin	A
Sodium acetate	v[b]	Trehalose	A
Phenolpyruvate	0	Cellibiose	–
β-galactosidase	+	Rhamnose	A
Lysine decarboxylase	0	Melibiose	–
Arginine dihydrolase	0	Arabinose	A
Ornithine decarboxylase	0		

[a] (+) Positive reaction; (0) negative reaction; (A) fermentation; (–) no fermentation. All incubation was done at 35°C unless otherwise noted.
[b] Variable reaction noted.

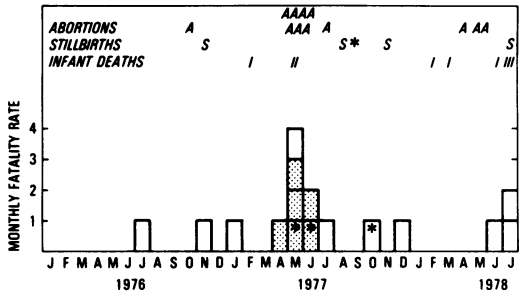

FIG. 1. *Temporal distribution of adult and infant deaths and reproductive failures in the squirrel monkey colony during a Y. pseudotuberculosis outbreak. Each rectangle represents one adult death; shaded rectangles are deaths attributed to Y. pseudotuberculosis; and open rectangles are deaths due to other causes. Symbols: (A) abortion; (S) stillbirth; (I) infant death; (*) Y. pseudotuberculosis was isolated from monkey at necropsy.*

tured for fecal shedding of *Y. pseudotuberculosis* were found to be positive. Both of these monkeys (36 and 43) had serum antibody titers of 1:160 and were clinically healthy. When sampled 45 days later, both monkeys were still clinically healthy, and *Y. pseudotuberculosis* was not recovered by fecal culture at this time. Serum antibody titers in both monkeys declined thereafter.

In December and January and again in April 1978, 1 year after the start of the epizootic, all seropositive monkeys in cages 4, 5, and 6 again were sampled. None were found to be shedding *Yersinia*. Changes in serum antibody titers with time are shown in Fig. 2. Of the 17 monkeys followed sequentially, 10 showed a twofold or less change in titer between August and September 1977 and April 1978, and 7 showed a greater than twofold decline in titer. Geometric mean antibody titers for the 17 monkeys were 1:181 in the period from August to September, 1:83 in the period from December to January, and 1:77 in April.

Between August and October 1977, *Y. pseudotuberculosis* was cultured two more times from monkeys in the colony. A stillborn monkey delivered in August was found to have a pure culture of *Y. pseudotuberculosis* in the colon but no lesions associated with this infection. In October, a male monkey from cage 1 died of pneumonia, pleuritis, and a lung abscess due to *Streptococcus pneumoniae*. *Y. pseudotuberculosis* was isolated from the spleen of this monkey by cold enrichment, but again no lesions were associated with this infection, and no antibody was detected by microagglutination. All monkeys, including neonates, infants, and some aborted fetuses, which died between July 1977 and January 1979 were cultured for *Yersinia* by using cold enrichment. Other than the instances described above, no *Yersinia* was isolated. There have been no additional deaths with lesions resembling those caused by *Y. pseudotuberculosis*.

Findings in wild mice. Of the 83 wild mice trapped in or near the monkey colony, 3 were found to be infected with *Y. pseudotuberculosis*. *Yersinia* was isolated from the cecum contents but not the spleens of the three mice. All three

TABLE 3. *Mortality, serological, and culture data (from April to December 1977) by cage for the squirrel monkey colony during a Y. pseudotuberculosis outbreak*

Cage no.	Total adults at risk	No. of deaths by:		No. of abortions	No. of stillbirths	No. of infant deaths	No. seropositive (%)[a]	Geometric mean titer[b]	Fecal shedding[c]
		Yersiniosis	Other						
1 (East)	22	1	1	NA[d]	NA	NA	4/19 (21)	190	0/16
2	11	0	0	0	0	0	NE[e]	NE	NE
3	15	0	1	1	1	0	NE	NE	0/3
4	16	1	0	0	0	0	4/16 (25)	190	0/15
5	16	1	1	1	1	0	9/14 (64)	201	2/14
6 (West)	16	3	1	6	0	2	5/11 (45)	183	0/11

[a] Number seropositive per number tested (sampled from August to October 1977).
[b] Reciprocal geometric mean titer of seropositive monkeys.
[c] Number of monkeys shedding *Y. pseudotuberculosis* in the feces per number cultured.
[d] NA, Not applicable since cage 1 contained all male animals.
[e] NE, Not examined.

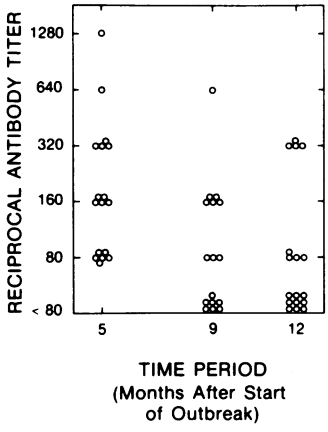

FIG. 2. *Reciprocal antibody titers to Y. pseudotuberculosis as determined by microagglutination in 17 squirrel monkeys sampled at 5, 9, and 12 months after the start of the outbreak. Each circle represents the titer of a single animal. Titers of less than 80 were not considered significant.*

mice were *Mus musculus* and were trapped within the first 2 months of the survey (September and October 1977). The biotype and serotype of all three isolates were identical to those isolates recovered from infected monkeys. The only lesion noted at necropsy of these infected wild mice was enlargement of the mesenteric lymph nodes. These three mice and 14 culture-negative mice trapped during the same period had no detectable antibody to *Y. pseudotuberculosis* (titer <1:40).

DISCUSSION

Infections caused by *Y. pseudotuberculosis* or *Y. enterocolitica* in nonhuman primates have been reported as sporadic, isolated cases as well as full-blown epizootics. Poelma et al. (19) have reviewed and described some of the isolated cases reported in the literature occurring primarily in Europe, and there have been three case reports of multiple animal involvement from regional primate centers in the United States (1, 5, 15). The clinical disease and pathological lesions in monkeys are similar for both of these *Yersinia* species. Poelma et al. (19) reported on 10 fatal cases of *Y. enterocolitica* infection in nine species of monkeys, the primary pathological findings being abscesses in liver and spleen, ulceration and necrosis of the jejunum, ileum, and colon, and hyperplasia of mesenteric lymph nodes. McClure et al. (15) reported three cases of *Y. enterocolitica* in two vervet monkeys (*Cercopithecus aethiops*) and one mangabey (*Cercocebus torquatus*), with lesions including small to large caseous abscesses of the liver and multiple, focal areas of enteritis with necrosis and ulceration, involving in one case Peyer's patches of the ileum. Baggs et al. (1) reported fatal *Y. enterocolitica* infection in 29 owl monkeys (*Aotus trivirgatus*). Animals had purulent and necrotizing enteritis, hepatitis, and splenitis. These authors succeeded in reproducing the disease experimentally in owl monkeys by oral or intravenous inoculation. Bronson et al. (5) reported an epizootic of *Y. pseudotuberculosis* in 29 monkeys of four different species. The characteristic lesions were again multiple, focal hepatitis with necrosis, multiple, focal splenitis, and areas of mucosal necrosis with inflammation in the intestine, often underlying lymphoid follicles. Lesions described in these four previous reports are similar or identical to those observed by us. Of the five outbreaks, the organism was identified serologically to species level in three, and in the other two instances, the organisms appeared to have definitive biochemical characteristics. Thus, yersiniosis in nonhuman primates, whether caused by *Y. enterocolitica* or *Y. pseudotuberculosis*, appears to be a single

pathological disease entity affecting a number of primate species and which can result in significant mortality.

Our finding of a common biotype and serotype among isolates from the dying monkeys, fecal carriers, the latently infected monkeys, and the wild mice suggests that there was a common source of contamination. The fact that these isolates did not ferment melibiose (an unusual characteristic for *Y. pseudotuberculosis*) strengthens this suggestion. We commonly observed feral mice and ground squirrels running within or near the squirrel monkey cages and occasionally found evidence that the monkeys sometimes caught and ate wild mice within their cages. Also, monkeys commonly ate biscuits that had fallen to the cage floor and become moistened. We postulate that the epizootic began by contamination of food by feces of feral mice or by the eating of an infected mouse and then spread through the colony by fecal-oral transmission (14, 16). Since disease, as well as the number of seroconversions, appeared to be concentrated at one end of the colony (cages 4 to 6), the original episode of infection may have occurred there, although the finding of the first adult case in the opposite end of the colony suggests that there may have been several point-sources of infection.

Other investigators have suggested, or presented evidence, that *Yersinia* outbreaks may be associated with feral rodents. Mair (14) reported that after an outbreak of *Y. pseudotuberculosis* in birds at the Bristol Zoo, 6 of 14 mice trapped near the bird house were found to be carrying the organism. During an outbreak of *Y. pseudotuberculosis* at the National Zoological Park in Washington, D.C., Baskin et al. (2) isolated the organism from wild rats and pigeons trapped in the vicinity of the affected zoological species. Kapperud (10) reported that 10% of small wild rodents trapped in Denmark were carriers of *Y. enterocolitica* and isolated *Y. pseudotuberculosis* from small rodents as well, suggesting a significant potential reservoir of yersinia infection in this population. As laboratory mice have been shown to excrete *Y. enterocolitica* in the feces for up to 135 days after experimental infection without showing signs of illness (20), it is not difficult to accept the probable role of wild mice in outbreaks of yersiniosis in animals.

The findings of this study, as well as those of others on yersinia outbreaks, suggest that *Y. pseudotuberculosis* and *Y. enterocolitica* both can be considered primarily as enteric pathogens in nonhuman primates. Certainly in the squirrel monkey cases described here, enteritis was the outstanding and most severe lesion, severe enough to have caused the death of the animal. Obwolo (17) found that in guinea pigs experimentally infected with *Y. pseudotuberculosis* by the oral route that animals dying within the first 10 days had predominantly gastrointestinal lesions, primarily multiple foci of necrosis in the ileum and cecum. Guinea pigs which died between 10 and 20 days had fewer but larger intestinal lesions and more visceral involvement, with abscesses in the liver and spleen. The early intestinal lesions seen in the experimentally infected guinea pigs were histologically similar to those found in naturally infected monkeys. This suggests that most monkeys infected with *Yersinia* probably died acutely and thus presented primarily intestinal lesions rather than the large, sometimes caseous, visceral abscesses often associated with pseudotuberculosis.

Our serological data strongly suggest that during the epizootic described herein the colony attack rate exceeded 40%. Monkeys were observed carefully several times daily by experienced workers during the period of the epizootic, and clinical illness (with the exception of abortion) was limited to those animals which ultimately died, suggesting that primates may become infected with *Yersinia* without developing disease. Our finding of only two fecal carriers of the organism among more than 20 monkeys with significant titers also suggests that some squirrel monkeys may serve as healthy carriers and shed *Y. pseudotuberculosis*, albeit for an unknown duration, and that most monkeys overcome or do not develop the intestinal carrier state while remaining seropositive. It is also possible that the number of organisms shed from some carriers may be so low as to preclude detection. Further, serum antibody responses in the colony suggest that whereas in some monkeys titers drop below significant levels fairly rapidly, some animals maintain titers for at least 1 year after infection. It has been previously assumed that antibody levels to *Y. pseudotuberculosis*, at least in humans, are short lived (4).

The association of *Y. pseudotuberculosis* infection with abortion is based on the following observations made by us in squirrel monkeys: (i) the occurrence of abortions coincided both temporally and spatially with disease due to *Yersinia*, (ii) *Y. pseudotuberculosis* was isolated from the uterus of two of the monkeys, (iii) there was endometritis associated with this infection, and (iv) *Yersinia* was isolated in pure culture from the colon contents of a stillborn squirrel monkey, indicating possible infection of the amniotic fluid. Bronson et al. (5) isolated *Y. pseudotuberculosis* from the amniotic fluid of a cy-

nomolgus monkey dying of yersiniosis, but did not report any uterine lesions associated with this infection or the relation to any abortions. *Yersinia* has been associated previously with abortion or fetal infection in bovines (11, 13) and ovines (21). The importance of *Yersinia* as a possible abortifacient in animals and humans is unknown, but considering the epidemiology of the organism and its worldwide distribution, this aspect of yersiniosis deserves investigation. Certainly, *Yersinia* may pose a potential threat to colonies established for the captive breeding of nonhuman primates.

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