Plague Epidemic in New Mexico, 1965

Introduction and Description of Cases

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THE SPORADIC occurrence of cases of bubonic plague in man which are acquired from sylvatic sources in the Western United States is a well-recognized contemporary phenomenon. An average of one or two cases has been reported each year for the past 30 years. Generally, these cases were widely separated in both time and location, and epidemiologic investigation usually revealed a history of contact with wild rodents or wild rodent fleas before the onset of illness (*1*).

During the summer of 1965, six cases of bubonic plague occurred among Navajo Indian children in the vicinity of Gallup, N. Mex. This was the largest number of plague cases reported in the United States in a single year since 1924, when an outbreak of urban plague occurred in Los Angeles. The Navajo cases were associated with concurrent epizootics in prairie dog populations. The diagnosis was confirmed bacteriologically in four cases and serologically in two. One child died with evidence of secondary plague pneumonia.

Plague in New Mexico

New Mexico has been prominent in the recent history of sylvatic (or wild rodent) plague in the United States. The first evidence of the existence of plague in the State was seen in 1938 when *Pasteurella pestis* was recovered from tissues of prairie dogs and pools of fleas from prairie dogs and field mice obtained by State and Federal survey teams in Catron County (*2*). Since that time, additional studies have established the presence of plague in a wide variety of animal species and their ectoparasites in New Mexico (*3–5*). *P. pestis* has been found in 22 of the State's 32 counties, and more than 15 different animal hosts, including cottontail rabbits, prairie dogs, ground squirrels, and wood rats, have been incriminated.

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Dr. Collins and Dr. Martin, epidemic intelligence service officers, were assigned to the Epidemiology Branch, National Communicable Disease Center, Public Health Service, Atlanta, at the time of this study. Dr. Kartman is chief, Dr. Hudson is a biochemist, and Dr. Goldenberg is a microbiologist, Zoonoses Section, Ecological Investigations Program, NCDC, San Francisco. Dr. Brutsché was medical officer in charge, Public Health Service Indian Hospital, Gallup, N. Mex. Dr. Doran was head of the Division of Comparative Medicine, New Mexico Department of Public Health. Dr. Hubbert was assigned to the NCDC San Francisco Field Station. Dr. Tirador was chief and Mr. Stacy was chief sanitarian, Community Health Services, Division of Indian Health, Public Health Service, Window Rock, Ariz. Mr. Miller is chief, Vector Control Section, New Mexico State Department of Public Health, Santa Fe. Portions of these papers were presented at the meeting of the American Public Health Association, Epidemiology Section, in San Francisco, November 1, 1966.

The first recognized case of human plague in New Mexico occurred in July 1949 when a 9-year-old boy became severely ill, with a temperature of 106° F., a few days after he killed a prairie dog. He recovered after he was treated with sulfonamide and streptomycin. Of the 26 cases of plague reported in the United States during 1949–65, 18 (69 percent) occurred in New Mexico. Of these 18 cases, seven were fatal—a case fatality rate of 38 percent. Table 1 summarizes New Mexico's plague experience from 1949 through 1965.

Serologic Methods

The diagnosis of human plague often depends almost solely on the specificity and sensitivity of the tests employed. In two of the plague cases in 1965 no isolations were made of the etiological agent, and the diagnosis thus depended on the detection of specific antibodies.

Serums were tested by the passive hemagglutination test for *P. pestis* Fraction I-specific antibodies. Tanned washed sheep erythrocytes and tanned formalinized erythrocytes were used according to the method of Chen and Meyer (*6*). The use of formalinized erythrocytes yielded somewhat better results with regard to both sensitivity and the absence of nonspecific agglutination. Serums to be tested were adsorbed using 1 volume of packed washed sheep erythrocytes to 4 volumes of test serum. Initial dilutions tested were 1 : 4. Negative results therefore represent passive hemagglutination titers of less than 1:4.

Controls for nonspecific agglutination consisted of parallel series of dilutions of test serums which were tested using washed and tanned erythrocytes to which no antigen was added. All reagents and diluents were identical to those used for the test itself except for the absence of *P. pestis* Fraction I antigen. Controls for serologic specificity consisted of a battery of normal, *P. pestis* positive, and *P. pseudotuberculosis* positive serums. The latter were included because of the close antigenic relationship between the plague and pseudotuberculosis organisms. Antiserums to *P. pseudotuberculosis* types 1B, 2A, 2B, 3, 4, and 5 were tested. A minor amount of cross-reactivity was noted with regard to *P. pseudotuberculosis* type 4 (table 2). Eight *P. pestis* antiserums yielded initially identical results with both tests, although the formalinized erythrocytes yielded somewhat higher titers. The ranges and geometric mean titers are shown in table 2.

Description of Cases

The six cases of plague occurred between June 21 and August 26, 1965. Four of the six stricken Navajo children were admitted to the Public Health Service Indian Hospital in Gallup and two were admitted to the Service's Indian Hospital in Crown Point, N. Mex. A

Table 1. Cases of human plague in New Mexico, 1949–65

Case No.	Month and year	County	Sex	Age	Animal implicated	Outcome
1	July 1949	Taos	Male	9	Prairie dog	Recovered.
2	July 1949	Sandoval	Male	34	Gopher	Recovered.
3	November 1949	Lincoln	Male	7	Ground squirrel	Fatal.
4	January 1950	Lea	Male	27	Cottontail rabbit	Recovered.
5	July 1950	Santa Fe	Male	15	Prairie dog	Fatal.
6	January 1951	Lea	Male	?	Cottontail rabbit	Fatal.
7	July 1959	Bernalillo	Female	12	Cottontail rabbit	Fatal.
8	February 1960	Chaves	Male	23	Cottontail rabbit	Recovered.
9	February 1960	Chaves	Male	24	Cottontail rabbit	Recovered.
10	June 1961	San Miguel	Male	38	Uncertain	Fatal.
11	July 1961	Santa Fe	Male	38	Uncertain	Fatal.
12	August 1961	San Miguel	Male	23	Uncertain	Recovered.
13	July 1965	McKinley	Female	3	Prairie dog	Recovered.
14	July 1965	McKinley	Male	2	Uncertain	Recovered.
15	August 1965	McKinley	Male	9	Prairie dog	Recovered.
16	August 1965	McKinley	Female	4	Prairie dog	Recovered.
17	August 1965	McKinley	Female	3	Prairie dog	Recovered.
18	August 1965	McKinley	Male	14	Prairie dog	Fatal.

SOURCE: Reference 5.

Table 2. Comparison of sensitivity and specificity of the two passive hemagglutination techniques used to test serums for *Pasteurella pestis* Fraction I-specific antibodies

Number and types of serum	Washed unfixed erythrocytes			Formalinized erythrocytes		
	Number positive	Range of titers	GMT [1]	Number positive	Range of titers	GMT [1]
15 human plague convalescent-stage serums	14	0 to 1:64	1:29	15	1:8 to 1:1,024	1:110
8 *P. pestis* antiserums [2]	7	0 to 1:4,096	1:560	8	1:8 to 1:1,024	1:570
13 *P. pseudotuberculosis* antiserums (rabbit)	[3] 2	1:8 to 1:32	--------	[3] 2	1:8 to 1:32	---------
19 normal serums (rabbit)	0	----------	--------	0	----------	---------

[1] Geometric mean titer.
[2] 1 burro, 7 rabbit serums.
[3] Type 4.

summary of the clinical data on these cases appears in table 3.

Case No. 1, a 3-year-old girl from Red Rock, was admitted to the hospital outpatient clinic on June 21, 1965, with a temperature of 103° F. and what appeared to be an upper respiratory infection. After treatment with penicillin for 1 week her condition improved, but on June 30 it deteriorated; she became very irritable and cried when moved. On July 1, when admitted to the hospital, the child had a temperature of 104° F. She was lethargic and irritable, and she had signs of meningismus. No enlarged lymph nodes were noted on admission. A lumbar puncture revealed cloudy cerebrospinal fluid containing 196 white blood cells per cubic millimeter, and gram stain showed large gram-negative rods. The child was then started on penicillin, sulfadiazine, and chloramphenicol; she improved steadily.

On July 14, a 3- by 3-cm. node was seen in the left groin. When cultured, material aspirated from the node yielded large gram-negative rods which appeared similar to those observed in the cerebrospinal fluid. The child completed a course of antibiotic therapy, was discharged from the hospital on July 24, and subsequently remained well. Subcultures from this patient were sent to the New Mexico Department of Public Health Laboratory and the National Communicable Disease Center for identification but, unfortunately, viable organisms could not be recoverd from the plates. This child's illness was not recognized initially as plague, and she was discharged with a diagnosis of gram-negative meningitis. With the subsequent occurrence of confirmed cases, the patient's history was reviewed and retrospectively considered to be compatible with plague. The diagnosis of plague was then confirmed by demonstration of a significant rise in passive hemagglutination titer for *P. pestis* (*7*).

Case No. 2, a 2-year-old boy living in Gamerco, had a sudden onset of vomiting and fever on July 8. The child was admitted to the hospital July 9 with a temperature of 102.5° F. Subsequently a 3- by 4-cm. right axillary lymph node was noted and found to be exquisitely tender. There were no lesions on the extremities. Because of meningismus, a lumbar puncture was performed; the cerebrospinal fluid was normal. Therapy consisting of penicillin and heat applied to the area of the lymph node was started, but during the next 5 days the child continued to have a temperature of 102° to 104° F. A needle aspiration of the node was performed on July 14, and because of developing ataxia and meningismus, another lumbar puncture was performed on July 15. The cerebrospinal fluid contained 2,400 white blood cells per cubic millimeter, and gram-negative organisms were seen on microscopic examination. Large doses of aqueous penicillin, sulfadiazine, and chloramphenicol were administered, and the child gradually recovered during the next 3 weeks.

Cultures of cerebrospinal fluid revealed gram-negative bipolar rods, and subcultures of these were sent to the NCDC's Special Projects Laboratory in Atlanta and the San Francisco Field

Station. The subcultures were positively identified as *P. pestis* by colonial morphology, fluorescent antibody staining, phage typing, and animal inoculation. The child continued to do well, but in mid-August he still had a prominent 2- by 2-cm. non-tender node in the right axilla.

Case No. 3, a 9-year-old boy from Prewitt, was admitted on August 1. His chief complaints were a severe headache and an infected finger. The boy had been well until 3 days before admission, when a bluish sore on the tip of his left index finger was noted. He subsequently suffered high fever, headache, and vomiting. When brought to the hospital, the child also complained of pain under his left arm. His temperature was 104° F. on admission and soon rose to 105° F. A pustule on the left index finger was noted, and a tender, enlarged lymph node was found in the left axilla. Scrapings were taken from the index finger for culture, and the node in the left axilla was aspirated. Material from both these sources was subsequently shown to contain organisms that were identified and confirmed as *P. pestis* at the San Francisco Field Station. After the boy was treated with streptomycin and chloramphenicol, his temperature returned to normal. However, 2 days after admission he became confused and appeared to be having hallucinations. A lumbar puncture was performed, and the fluid was found to be normal. The remainder of the child's convalescence was uneventful, and he was discharged after approximately 10 days in the hospital.

Case No. 4, a 4-year-old girl residing in Tinian, a remote area of the reservation 125 miles northeast of Gallup, suffered painful swelling in the right supraclavicular area, fever, and vomiting on August 7. When she was seen in the outpatient clinic the following day, she had a temperature of 104° F. She was given penicillin parenterally. The girl failed to improve, and she was admitted to the hospital in Crown Point on August 11. On admission she was lethargic and had a temperature of 100° F. Physical examination revealed diffuse tender swelling in the right parotid area and a prominent walnut-size tender right supraclavicular lymph node. Aspirated material from the supraclavicular node showed no growth on culture. Penicillin therapy was maintained, and the patient's temperature returned to normal in 3 days.

The child was discharged on August 18 with a tentative diagnosis of mumps. When an illness of the patient's cousin (case 5) was confirmed as plague, she was readmitted to Crown Point where re-aspiration of the supraclavicular node yielded only nonhemolytic staphylococci. A residual 3- by 3-cm. swelling over the right cheek was thought to represent pre-auricular lymphadenopathy. The patient was then treated with streptomycin, and the size of the enlarged nodes rapidly decreased. The diagnosis of plague was confirmed serologically by the passive hemagglutination test, as shown in table 4. This child's illness, which was mild and resolved without specific plague chemotherapy, fits the description of pestis minor or ambulant plague as described by Pollitzer (*8*).

Case No. 5, a 3-year-old girl residing in Tinian, was admitted to the Crown Point hospital on August 16. Three days before admission, the child complained to her parents of a vague pain across her upper anterior chest. This pain continued and was accompanied by fever. The parents noted what appeared to be an insect bite on the right anterior chest. On the day before admission, the child complained of

Table 3. Clinical data on 6 children with plague, 1965

Case No.	Age	Sex	Date of admission	Clinical description	Outcome
1	3	Female	July 1	Meningitis, inguinal bubo	Recovered.
2	2	Male	July 9	Axillary bubo, meningitis	Recovered with mild ataxia.
3	9	Male	Aug. 1	Pustule on finger, axillary bubo	Recovered.
4	4	Female	Aug. 11	Pestis minor, supraclavicular and pre-auricular bubo.	Recovered.
5	3	Female	Aug. 16	Ulcerating lesion on chest wall, axillary bubo.	Recovered.
6	14	Male	Aug. 26	Septicemia with secondary pneumonia	Fatal.

Table 4. Laboratory data on cases of plague in 6 children

Case No.	Bacteriology	Date	Serology—hemagglutination test results	
			Washed sheep erythrocytes	Formalin fixed erythrocytes
1	Negative	July 15, 1965	1:32	1:256
		Sept. 9, 1965	1:64	1:128
		Dec. 10, 1965	1:32	1:1,024
		July 1966	1:16	1:64
2	Positive culture from spinal fluid and bubo	Aug. 1, 1965	0	----------
		Aug. 17, 1965	1:16	----------
		Sept. 9, 1965	1:64	1:256
		Nov. 3, 1965	1:32	1:64
		Mar. 7, 1966	1:4	1:16
3	Positive culture from spinal fluid and bubo	Aug. 2, 1965	0	----------
		Sept. 9, 1965	1:16	1:256
		Nov. 3, 1965	1:16	1:32
4	Negative	Aug. 22, 1965	0	----------
		Sept. 3, 1965	1:32	1:1,024
		Nov. 3, 1965	0	1:64
		July 1966	1:32	1:64
5	Positive culture from skin lesion	Aug. 22, 1965	0	----------
		Sept. 3, 1965	1:64	1:512
		Nov. 3, 1965	1:16	1:64
		March 1966	1:4	1:8
6	Positive culture from postmortem lung and lymphatic tissue.	----------	None	None

severe chest pain and appeared seriously ill. When examined on admission the patient was afebrile, but soon her temperature rose to 104° F. The lesion on her right anterior chest was a 3- by 3-cm., well-demarcated, erythematous area with many small vesicles on the surface. These vesicles had begun to coalesce, but there was no loss of skin continuity. No lymphadenopathy was found.

Penicillin therapy was initiated, but the patient remained toxic. By August 18 her temperature had risen to 105° F. At this time, the original vesicular lesion had become a deep subcutaneous ulcer; red lymphangitic streaks extended from the ulcerative lesion to the right axilla, where palpable lymph nodes were noted. Sulfonamides and streptomycin were added to the regimen after material from the ulcerative lesion was cultured. The child showed immediate and sustained improvement and subsequently remained well. Culture of the skin lesion yielded colonies of pleomorphic gram-negative bipolar rods, subsequently confirmed as *P. pestis*.

Case No. 6, a 14-year-old boy residing in Red Rock near the first stricken child, was admitted to the Gallup hospital on August 26 with headache, fever, and vomiting of 1 day's duration. On arrival at the hospital, he was semistuporous and had a temperature of 104° F. No buboes were noted. Chest X-ray on admission was normal. Lumbar puncture was performed, and the spinal fluid was normal. High fever and stupor continued despite symptomatic therapy with salicylates and intravenous fluids. On the evening of the second hospital day, the boy's temperature rose to 104.4° F., and he became restless, his breathing was rapid and labored, and he coughed bloody sputum. Another chest X-ray revealed widespread pulmonary infiltration. Cardiorespiratory arrest ensued, and the patient was pronounced dead at 10:20 p.m., August 27.

Autopsy performed on August 30 revealed hemorrhagic lymphadenitis of the left axilla, bilateral pulmonary edema, and congestion of liver and spleen. Postmortem specimens of lung and lymphatic tissue yielded *P. pestis*. All the persons who lived with the child (three adults, four children) were hospitalized and treated with prophylactic antibiotics. They were subsequently released because they showed no evidence of having plague infection. The nurses and the physicians who had cared for the patient received antibiotics, and they also showed no subsequent evidence of infection.

The laboratory findings for the six cases are summarized in table 4, and the geographic relationships are shown in figure 1. The importance of the serologic tests in diagnosis is emphasized by the fact that an additional presumptive case in a 3-year-old boy had been originally considered solely on the basis of a rise in passive hemagglutination titer from negative on August 22 to 1:16 on September 2. A critical re-examination of the serum showed this to be a nonspecific agglutination.

Epidemiologic Investigations

Approximately 100,000 Navajo Indians reside on or near the reservation, which encompasses 25,000 square miles of western New Mexico and eastern Arizona and Utah. The Indians in this area live on a high desert plateau, generally in small shacks, tents, or traditional "hogans" made of wood and mud. They tend to cluster in family camps of two or three hogans rather than in large communities. Families frequently abandon their homesites and move considerable distances. Those who care for livestock remain in remote grazing areas for months at a time.

Except for several missions, the Gallup hospital and the other Service units provide all the medical care on the reservation. Despite this network of health facilities, the Indians often do not seek medical care, and occasionally serious illnesses and even deaths are not reported. For example, in early August a report was received of the death of an 18-year-old shepherd following a 4-day febrile illness. He lived near Tinian, close to the victims in cases

Figure 1. Sites where six cases of human plague occurred, McKinley County, N. Mex., 1965

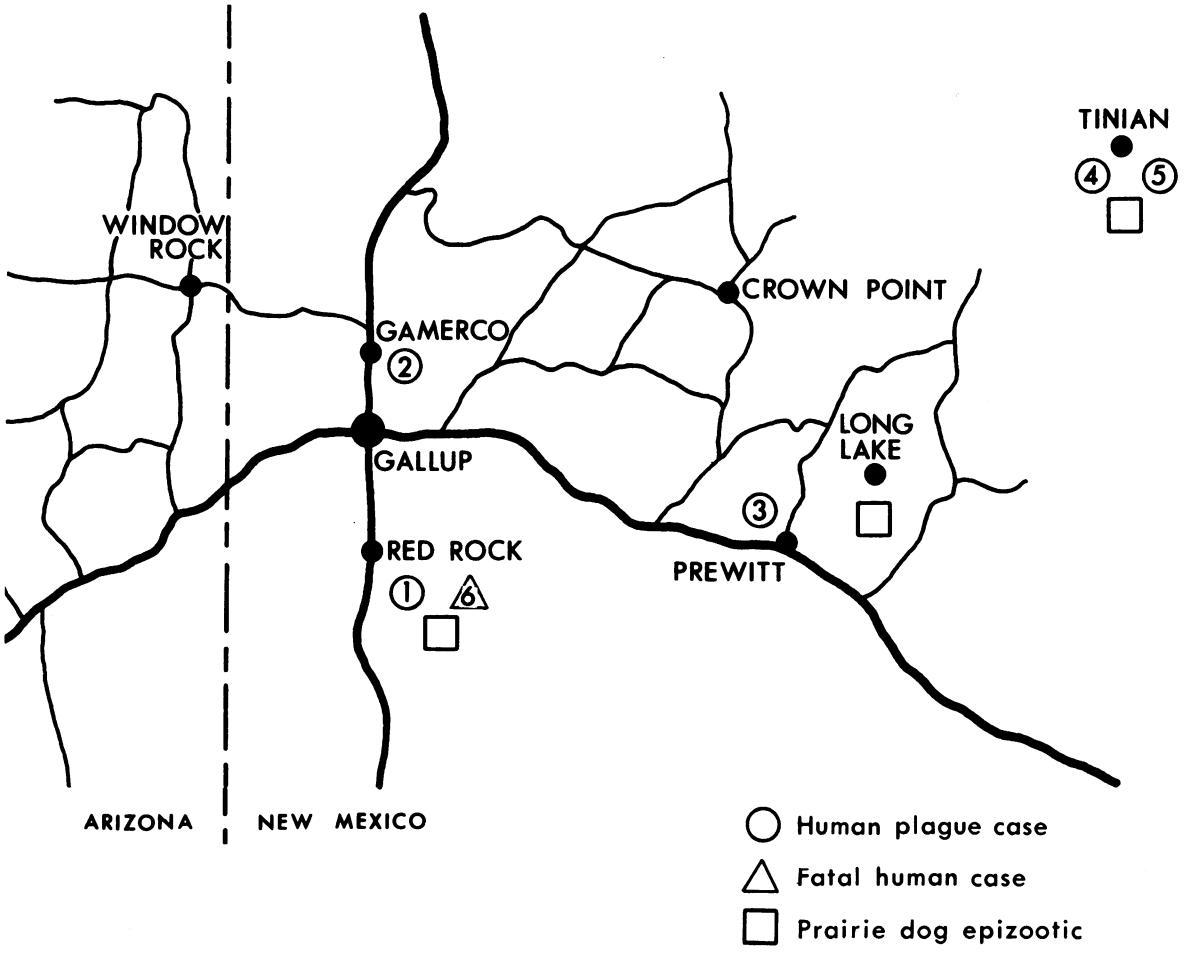

Figure 2. Tents used by a family of a plague victim. These dwellings, in a remote area, were located on the site of a prairie dog colony undergoing a plague epizootic.

4 and 5. The circumstances of his illness were not clear, and they varied among different informants. One unconfirmed report described the presence of buboes and subcutaneous hemorrhage before death. He was not seen by medical personnel before he died and no autopsy was performed. The question of whether or not this illness was another fatal case of plague is unresolved, but the temporal and geographic relationship to other confirmed cases is highly suggestive of plague.

A visit was made to the home of each patient to inspect the environment and interview family members. Epidemiologic histories varied considerably in detail, depending in part on the amount of elapsed time between the illness and the interview and the willingness of the families to discuss events concerning each illness. Certain features were common to all six cases: each patient lived or had visited and played in areas with known prairie dog die-offs and each household contained domestic animals, such as dogs and cats, which frequently brought animal carcasses into the home. A specific history of contact with prairie dogs before illness was elicited for all but the patient in case 2. Epidemiologic histories for the patients in cases 3, 5, and 6 were particularly illustrative of the frequent contact between Navajo children and wild rodents.

Interviews with members of the household of the patient in case No. 3 indicated that the boy cut the index finger of his left hand 4 days before his admission to the hospital. Later the same day the patient and his family traveled approximately 8 miles northeast of their home to an area called Long Lake, where they hunted and shot five prairie dogs. These animals were taken home, gutted, cooked, and eaten the following day. We learned that the boy had assisted his father with the dead animals. The patient subsequently had fever, a pustule on his left index finger, and classic signs of local infection with regional lymphadenitis. We believe that the cut on his left hand was the portal of entry for infection. When his condition deteriorated during the next 2 days, he was brought to the Gallup hospital. No other mem-

ber of the family or persons in the community (composed of 12 dwellings) became ill or noticed enlarged lymph nodes on themselves.

In case No. 5 the patient also had a clear history of contact with wild rodents. The girl and seven other persons in her family lived in a desolate area of the reservation. They had set up their camp, consisting of several tents, in this area only a week before the onset of the patient's illness. A visit to the site revealed that the tents were almost entirely surrounded by an extensive prairie dog colony (fig. 2). The patient's father stated that the family was accustomed to hunting prairie dogs for food, and that several dead animals (including prairie dogs, rabbits, and a squirrel) had recently been found on the ground near the tents.

The family had continued to hunt prairie dogs until shortly before the patient's illness, when they allegedly discontinued this practice because of repeated warnings over the Navajo radio station that these animals might be infected. The child often played in the fields around the house, and her parents noticed that she frequently had insect bites on her body. There were four dogs in the camp; they apparently lived on table scraps. No other members of the family had become ill. A search of the region surrounding the tents quickly revealed many fresh prairie dog carcasses. Carcasses were collected and sent to the San Francisco Field Station for examination.

The child's illness apparently began with an insect bite on the chest wall, and she subsequently had regional lymphadenitis. *P. pestis* was recovered from the ulcerating wound. This patient seemed to have acquired plague by the bite of an infective flea. There was ample evidence of an extensive epizootic in prairie dogs surrounding her home.

Members of the family of the patient in case No. 6 were interviewed while they were being retained for observation at the hospital following his death. The parents were notably reluctant to discuss the situation, but two of his brothers stated that the patient had hunted prairie dogs shortly before onset of his illness. This was further corroborated by a family that lived near the patient; members of the family indicated that hunting prairie dogs was a common practice of the victim and his friends.

Epidemiologic Features and Results of Field Studies

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THE 1965 epidemic of bubonic plague revealed certain unique epidemiologic features of great interest to persons concerned with Indian health and to students of communicable disease.

The epidemic, as is common in human infections associated with sylvatic sources, was related to epizootics among wild rodents, primarily prairie dogs. The history of wild rodent plague in this country is replete with accounts of prairie dog die-offs, and it has become fairly well established that whenever plague occurs among prairie dogs the colony is decimated. In many instances "dog towns" have been wiped out over hundreds of square miles. A recent summary of the literature on prairie dogs and plague appears in an account of an epizootic in Colorado (*9*).

Materials and Methods

Field. During the epidemic, field observations and field collections were made in areas associated with cases in human beings and in other areas located on and adjacent to the Navajo Indian Reservation in New Mexico and Arizona. Briefly, the following methods were used.

1. A search was made for evidence of epizootics and die-offs of rodents. Each dead animal, regardless of its condition, was placed in a plastic bag, labeled, and packed with dry ice for shipment to the San Francisco laboratory.

2. Burrows of prairie dogs in epizootic areas were swabbed for fleas with flannel cloths attached to 6-foot-long wires. Swabs were placed in cloth sacks that were tightly closed and labeled. Subsequently, the fleas were etherized and placed in vials containing 2 percent saline solution.

3. Live-catch traps (Sherman) baited with cereal were set for overnight collection of small rodents in the area where prairie dog mortality had been observed. The trapped animals were