EXPERIENCE WITH BUBONIC PLAGUE (HUMAN AND RODENT) IN GALVESTON, 1920.

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The very nearly simultaneous recognition of human bubonic plague in four previously uninfected Gulf ports early in 1920, three of which (Galveston, Beaumont, and Pensacola) are American, created a situation of great anxiety in the minds of all persons interested in public health. The recognition of rodent plague in Port Arthur was a later event. This simultaneous outbreak in four widely separated areas along the Gulf coast is strongly suggestive of some common source, although corroborative evidence was never secured. Since plague is a disease which but few American sanitarians have had opportunity to personally combat, it appeared that an account of some of the experience with the disease in this focus might prove of general interest.

I. EPIDEMIOLOGY OF HUMAN PLAGUE IN GALVESTON, 1920.

In the period between June 16, when the first case of plague was diagnosed, and November 13, when the last case of the 1920 outbreak was recognized, there occurred a total of 18 cases of human bubonic plague attributable to infection acquired in Galveston. None occurred that could be attributed to infection acquired outside the city. Two of these cases left Galveston either in the incubation period or in early stages of the disease, and were diagnosed as plague at Port Arthur and Houston, respectively.

Some essential epidemiological data concerning these cases is presented in Table I.

TABLE I.—*Incidence of human plague in Galveston, 1920.*

No.	Age.	Sex.	Race.	Occupation.	Date of onset.	Duration of illness of fatal cases. (Days.)
1	17	M	W	Collector, feed store	June 8	8
2	25	F	N	Bag sewer, feed store	June 16	.....
3	19	M	N	Longshoreman	July 2	6
4	30 (?)	F	Mex	Housewife	.....	(1)
5	13	F	Mex	.....	.....	(2)
6	35	F	W	Housewife	Aug. 3	.....
7	41	M	N	Waiter on steamship	Aug. 2	2
8	16	M	W	Machinist's helper	Aug. 7	4
9	27	F	N	Houseworker	Aug. 16	2
10	29	F	W	Pathologist	Aug. 23	.....
11	42	M	N	Longshoreman	Aug. 29	4
12	79	F	W	Houseworker	Sept. 2	5
13	17	F	N	....do	Sept. 22	5
14	17	F	W	School girl	Oct. 2	.....
15	3	M	W	.....	Sept. 26	.....
16	35	M	N	Truck driver	Oct. 4	5
17	58	M	W	Night watchman	Oct. 17	.....
18	10	M	N	School boy	Nov. 10	3

[1] Died July 23. [2] Died July 29.

The age, sex, and race of these patients is summarized in the following table:

TABLE II.—*Age, sex, and race of plague patients.*

Race and sex.	Age.							Total.
	1-10.	11-20.	21-30.	31-40.	41-50.	51-60.	61 and over.	
White:								
Male	1	2				1		4
Female		1	1	1			1	4
Total	1	3	1	1		1	1	8
Negro:								
Male	1	1		1	2			5
Female		1	2					3
Total	1	2	2	1	2			8
Mexican:								
Male								
Female		1	1					2
Total		1	1					2
Total	2	6	4	2	2	1	1	18

The greatest number of cases occurred among adolescents and young adults, although no age period was exempt. Among whites the incidence in the sexes was the same; among Negroes, nearly equal; whereas both Mexican cases were females. An equal number of cases occurred among whites and Negroes, which, since the Negro population here is about 20 per cent of the total, indicates a disproportionately high attack rate among Negroes.

Two cases occurred in June, three in July, six in August, three in September, three in October, and one in November, the peak thus being reached in August. The decline, however, was more gradual than the rise.

The occupations of the patients were diverse, and in most instances they do not reveal any special opportunities favoring the contraction of the infection.

A study of the relationship of the human cases to the rodent epizootic reveals a very close correlation between the two. The geographical distribution of both human and rodent cases of plague, several of the former cases being spotted both according to residence and place of employment, is shown in Fig. 1. The details of the evidence indicative of this correlation are presented in Table III.

TABLE III.—*Relation of human to rodent plague.*

No.	Dead rats found on premise prior to onset.		Plague rats found within one block of—		Plague rats found within two blocks of—	
	Home.	Place of employment.	Home.	Place of employment.	Home.	Place of employment.
1		Yes		Yes, later		
2		Yes		Yes, later	Yes, later	
3				Yes, later		
4			Yes, later		Yes	
5			Yes, later		Yes	
6	Yes				Yes, later	
7				Yes, later		
8	Yes				Yes, later	
9	Yes		Yes, later			
10[1]						
11			Yes			
12					Yes	
13			Yes			
14					Yes	
15					Yes	
16					Yes	Yes.
17	Yes		Yes		Yes	Yes.
18					Yes	

[1] Pricked finger while doing autopsy on case 9.

When the incidence of human and rodent plague over the entire city is plotted by weeks, the correlation is still observable, as shown in Fig. 2. We did not possess any knowledge of the extent of rodent plague prior to June 20, on which date the examination of rats was begun, although two human cases had occurred before that time. The week of June 6, in which the onset of the first human case occurred, has been arbitrarily taken as the first week of the epidemic, although from the curve of the epizootic it is evident that rodent plague had existed locally for at least a month previous to the onset of the first known human case of plague. The peak of the epizootic was apparently reached early in July, in the fifth week of the rodent outbreak, and thereafter gradually declined, with more or less fluctuation, until the 25th week, in the latter part of November, when a sharp localized epizootic occurred. After its prompt subsidence, no further cases of rodent plague were recognized in 1920, and up to the date of this writing (Mar. 16, 1921) none has been encountered in 1921. To a certain extent it is noticeable that the peaks of human incidence follow the various major and minor peaks of the epizootic by a lag of from one to four weeks, although the last peak of the epizootic was not followed by any human cases. In most instances, the respective human and rodent cases accumulated in these peaks do not represent the same localized geographic areas. The probable explanation lies in the fact that the plague rats secured by trapping operations represented a somewhat random selection of those rats actually infected with plague at any one time, so that while the curves give us a good general idea of the progress of the epizootic, they should not necessarily be expected to coincide, unless

the trapping operations (and hence the sampling of rats) were more intensive. Further corroboration of this view is given by the following circumstances: In 10 instances the existence of rodent plague was known within a two-block radius of the home or the place of

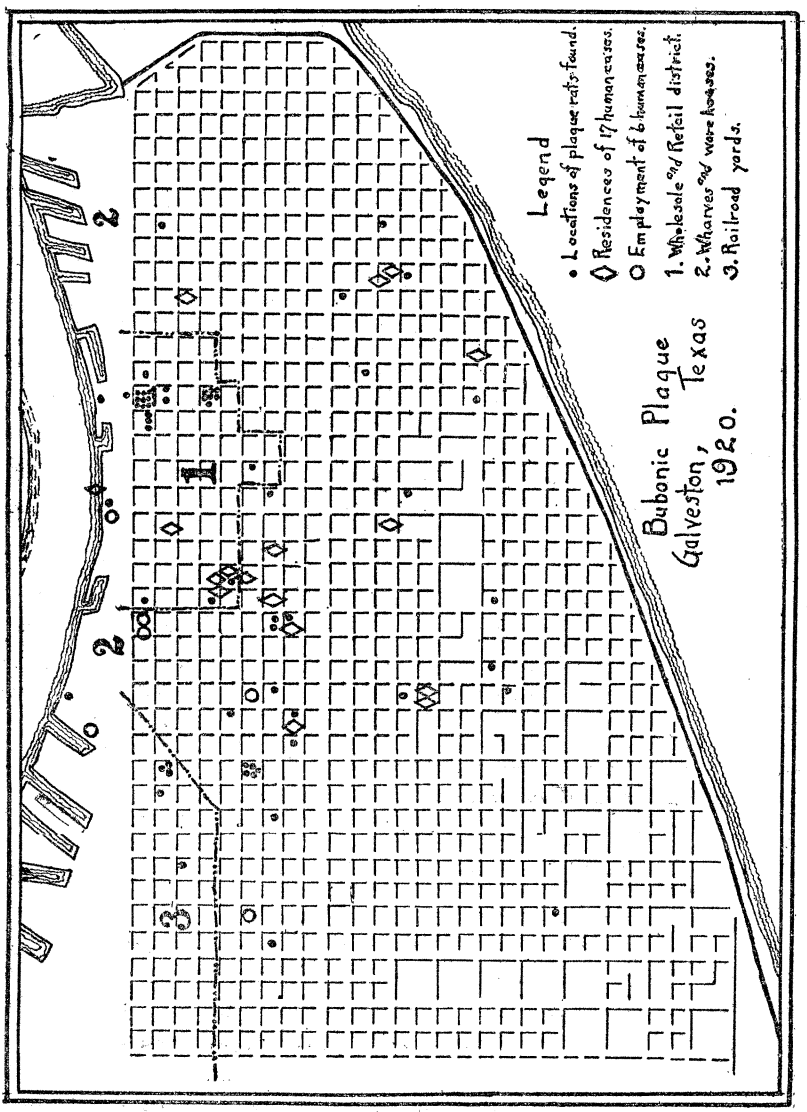

FIG. 1.

employment of a human case before the development of the latter, whereas in six instances the existence of such foci was revealed by the occurrence of human plague before the detection of rodent plague, within a zone of the same radius. No human cases could be

credited to 15 such two-block zones around sites where plague rats were caught.

Most of the infected rats were widely scattered, not more than one known plague rat being found to a focus. In three instances, however, over five positive rats were secured from the same block within a short time, indicating the existence of a very brisk epizootic among the rodent population. Two of these occurred in warehouses in the business district. No human cases arose as a consequence, probably for two reasons: (1) The epizootics were recognized in their incipiency,

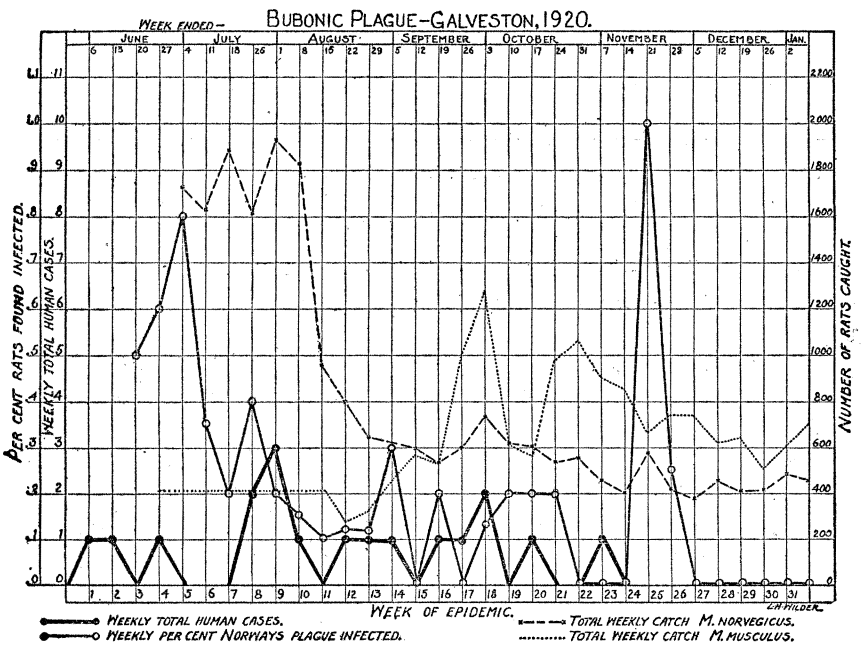

FIG. 2.

before the period when, owing to the ravages of plague, the rat population would have been reduced to such a point that fleas had to seek other hosts for feeding; and (2) the early recognition of these foci permitted the infected buildings to be promptly fumigated with cyanide, so that most, if not all, of the infected rats and fleas were promptly killed.

II. PROGRESS OF THE HUMAN DISEASE.

All cases recognized as plague before death were isolated in the isolation pavilion of Sealy hospital, though in one or two instances the hospitalization was not accomplished as rapidly as was desirable. Suspected cases were removed to isolation pending diagnosis.

Certain particulars pertaining to the administration of these cases are presented in Table IV.

TABLE IV.—*Administration of patients.*

	Days elapsing between onset and diagnosis.								Days elapsing between diagnosis and hospitalization.[1]								Duration of illness in 10 fatal cases. (Days.)							
	Same day.	2	3	4	5	6	7	8	Same day.	2	3	4	5	6	7	8	1	2	3	4	5	6	7	8
Number of cases	2	5	3	3	3	1	0	1	8	3	2	0	0	0	0	0	0	2	1	2	3	1	0	1

[1] Died on way to hospital, 1; not hospitalized, 4.

The great length of time elapsing between diagnosis and onset in many instances was due to two factors, chiefly, (1) delay on the part of a patient's family in calling for a physician, and (2) in some instances to the attending physician's not considering plague as a possibility. As the presence of the disease became more widely known, this last factor of delay disappeared. All diagnoses were confirmed microscopically, and, in some instances, including the first case, by cultural study of the organism isolated and by animal inoculation.

Seventeen of these cases were bubonic in type and one was apparently a case of primary pneumonic plague. In 12 instances the primary bubo was in the femoral region, on one side; in two additional instances femoral buboes were present on both sides, and it was impossible to distinguish between the primary and secondary buboes; one patient had a primary inguinal bubo; and in two cases the primary bubo was axillary in situation. One patient had a definite phlyctenule on the leg.

Immediately following the recognition of the first case, a small supply of Mulford's antiplague serum was made available by the State board of health. Later, an abundance of the serum prepared by the Pasteur Institute, Paris, was jointly supplied by the State board of health and the United States Public Health Service. The serum, previously diluted with an equal volume of physiological saline, was administered intravenously. The average dose employed for individuals of 150 pounds weight was 120 c. c., which was repeated every 24 hours if the patient's condition had not improved. One case (10) received a total of 600 c. c. The results secured from its administration are shown in Table V.

TABLE V.—*Cases treated and untreated with plague serum.*

Day of illness on which serum was given.	Number of fatal cases to whom serum was administered.	Number of cases that recovered, to whom serum was administered.
First	0	1
Second	0	1
Third	3	3
Fourth	3	0
Fifth	0	0
Sixth	0	0
Seventh	0	0
Eighth	0	0
None given	6	1
Total	12	6

Thus, of the 18 cases, 11 received treatment with antiplague serum. Of these, six died and five recovered. One of the deaths in this series was due to anaphylactic shock. The recovered cases all received their initial dose of serum not later than the third day of their illness. Seven cases did not receive treatment with antiplague serum. Of these, one recovered. Although these observations are limited, they suggest that the antiplague serum is of distinct therapeutic value. No limit should be placed upon the amount employed.

Five white cases recovered and three died, whereas one Negro recovered and seven died.

All the fatal cases whose death took place in Galveston came to autopsy. A study of the post-mortem findings will be published later by Prof. H. O. Hartman, of the University of Texas, and will not receive consideration here.

Case 10 of this series presents some features of unusual interest. This patient, a young physician, pricked her gloved finger (left forefinger) near the nail root, with the needle with which she was sewing up the scalp of case 9, after having performed the autopsy. Two hours later the area was cauterized. On the day following a lymphangitis of the finger and forearm was observed. She was given a prophylactic dose of 50 c. c. of antiplague serum and one dose of Haffkine's vaccine. On the fifth day the onset of plague developed, with general muscular pains, headache, fever, and a chill. Very shortly thereafter there were evident enlarged and painful occipital, submaxillary, cervical, axillary, and femoral glands, of which those in the left axilla were largest. Smears made from gland juice secured by puncture showed organisms morphologically resembling *B. pestis*. Large doses of serum were given until she had received a total of 600 c. c. After a week's illness convalescence was rapid and the patient recovered.

III. OBSERVATIONS ON PATHOLOGY OF RODENT PLAGUE.

The examination of rats for evidence of plague was initiated under the direction of Passed Asst. Surg. C. L. Williams, of the United States Public Health Service.

The technique of the examinations followed closely the directions given by Surg. Geo. W. McCoy, Director of the Hygienic Laboratory, Washington, D. C.[1]

From the beginning of antiplague operations until the 31st of December, 1920, 46,623 rats were examined in the laboratory. Sixty-seven of these, or 0.14 per cent, were found to be plague infected. Of these, 66 were *Mus norvegicus* and one was a *Mus rattus*.

After the examinations were under way, the rats reported as positive or plague infected were divided into two classes; namely, (1) those reported as positive without animal inoculation, and (2) those reported positive by animal inoculation. The first of these groups comprises for the most part those rodents presenting the five cardinal lesions of rodent plague; namely, injection, bubo, granular liver, congested spleen, and pleural effusion, always with positive microscopical findings, on which basis the diagnosis of rodent plague in a known plague focus is justified without necessarily requiring cultural or virulence confirmation. The second group comprises those rodents in which the manifestations of plague may be considered to be slightly atypical, though highly suspicious, less than five of the cardinal lesions being noted. The suspicions raised by these findings were proved or disproved by animal inoculation.

Thirty-four of these rats were reported positive without animal inoculation. Of these, 21 showed all five lesions, 9 showed four, 2 showed two lesions, and the lesions in 2 were not recorded. The frequency with which these were found is shown in Table VI.

TABLE VI.—*Frequency of cardinal lesions in class 1 rats.*

Rats having—	Particular lesions.				
	Injection.	Buboes.	Liver lesions.	Spleen lesions.	Pleural effusion.
5 lesions	21	21	21	21	21
4 lesions	8	9	8	8	3
2 lesions	1	2	2	1	0
Total	30	32	31	30	24

Thirty-three rats were reported positive after animal inoculation. Five showed all five lesions; 11, four lesions; 10, three lesions; 4,

[1] The Technique of the Laboratory Examination of Rats for Plague. Reprint No. 89, from the Public Health Reports, Vol. XXVII, No. 30, July 26, 1912. The reader is referred to this article for an excellent description of the lesions of rodent plague.

two lesions; 2, one lesion; and, presumably, 1, no lesion The frequency with which these lesions were found is shown in Table VII.

TABLE VII.—*Frequency of cardinal lesions in class 2 rats.*

Rats having—	Particular lesions.				
	Injection.	Injected glands (bubo).	Liver lesions.	Spleen lesions.	Pleural effusion.
5 lesions	5	5	5	5	5
4 lesions	11	11	5	10	6
3 lesions	10	10	5	7	3
2 lesions	4	3	0	1	0
1 lesion	1	0	0	1	0
Total	31	28	15	24	14

In one instance a combination inoculation was made with five rats, all apparently negative, secured at the fumigation of a known plague focus. The guinea pig died on the sixth day and presented typical lesions of plague, indicating that at least one of the five rats was infected.

Thus, of 65 positive rats, subcutaneous and general injection was present in 61, buboes were present in 60, liver lesions were present in 46, splenic lesions were present in 54, and pleural effusion was present in 38.

In the rats of class 1 (positive without animal inoculation) the injection was marked in 25 instances, moderate in 5, and in no instance slight in degree. In the other class (positive by animal inoculation) it was marked in 21 instances, moderate in 9, and slight in 1 instance.

The definite buboes were usually multiple. The application of this term is properly limited to those glands which are enlarged, injected, softened, and microscopically positive. They were found as follows:

	Rats of class 1 (pos. without A. I.).	Rats of class 1 (pos. by A. I.).
Cervical bubo alone	13	13
Cervical and axillary buboes	2	1
Cervical and inguinal buboes	2	1
Axillary bubo alone	7	3
Axillary and inguinal buboes	4	0
Axillary, inguinal, and pelvic buboes	1	2
Inguinal bubo	2	5
Inguinal and pelvic buboes	1	3

The buboes are thus seen to be chiefly in the anterior half of the body, in the cervical and axillary glands, either or both, in 60 per cent of the rats, and the posterior half alone in 16 per cent.

Twenty-four of the rats of class 1 presented the small foci of necrosis in the liver, known as "granular" liver, which was also seen

in seven of class 2. Three livers, in addition to the "granules," appeared of the so-called "fatty" type. In 20 instances, all told, the liver was distinctly congested; but congestion is not necessarily associated with the presence of focal necrosis. It is probably seen where the rat is examined during the acute stage of infection, but as the convalescence progresses the congestion subsides, whereas the necrosis remains visible indefinitely.

Splenic congestion was noted in 27 of the first group and in 20 of the second group of rats, the organ appearing quite tense. Past acute congestion, as revealed by transverse linear scars of the capsule, was seen in 7 instances. Four rats showed large, solitary abscesses in the spleen. Transverse linear scars of the capsule can not be taken unreservedly as evidence of convalescence from plague or "resolving" plague. We are inclined to believe that any acute infection may produce acute splenic congestion with transverse rupture of the capsule. We noted three nonplague rats, in which *Trypanosoma lewisii* was found, that showed such splenic scars.

Of the first group of rats, pleural effusion was marked in 12, moderate in amount in 5, and scanty in volume in 7. Of the second group, 4 showed a marked effusion, 8 a moderate volume, and 2 only a slight amount of fluid. In most cases the effusion was clear and limpid, though not infrequently it was noted to be blood stained. The possibility of a severe contusion of the thorax by the blow of a spring trap, as a factor in the production of the latter type of effusion, must be borne in mind.

IV. RAT FLEAS.

Owing to inability to secure cage traps by which live rats could be secured for flea observations, very few live rats were brought to the laboratory; consequently the flea observations leave much to be desired. The few that were made suggest a very high number of fleas per rat, the bulk of which were *Leomopsylla cheopis*. The only other flea observed was *Ceratophyllus fasciatus*, which was seen only on a few occasions, and then associated with *L. cheopis*.

V. ANTIPLAGUE OPERATIONS.

In the foregoing account of Galveston's experience with bubonic plague no reference has been made to the energetic efforts directed toward its control and eradication, for the reason that neither of the writers had personally participated in them, apart from the work done in the laboratory. For the sake of completeness, however, the following should be mentioned:

Early in July, 1920, through the cooperation of the city of Galveston, the Texas State Board of Health, and the United States Public Health Service, active operations against rodents were begun.

These activities comprised (1) the trapping of rodents, (2) the removal of rat harborages, (3) rat-proofing, and (4) the fumigation of buildings and the shipping on the water front included in plague areas. The success of these measures may be gauged by three results:

1. A steady diminution of the number of rats caught in traps, as indicated in Figure 2.

2. An increase in the number of mice caught in traps set in positions likely to be frequented by rats, also shown in Figure 2.

3. The cessation of plague, both human and rodent.

PREVALENCE OF POLIOMYELITIS.

The following table gives the number of cases of poliomyelitis (infantile paralysis) reported to the Public Health Service by State health officers from May 29 to July 23, 1921, inclusive. These reports are preliminary and necessarily incomplete. The cases are widely scattered. For instance, the 15 cases reported from Illinois for the week ended July 15 were from 13 different places, no more than 2 cases being reported from any one locality.

Poliomyelitis (infantile paralysis)—Number of cases of poliomyelitis occurring in various States, as reported to the Public Health Service by the State health officers in weekly telegraphic or mail reports.

[States omitted are those from which no reports have been received or which have reported no poliomyelitis during the period covered. Leaders indicate that reports were received but no cases of poliomyelitis were reported.]

State.	Week ended (1921)—							
	June.				July.			
	4	11	18	25	2	9	16	23
Arkansas				1				
California	1	1	2	5		3	3	6
Connecticut				6	2	3	2	4
District of Columbia						3	4	3
Georgia		1	1		1			
Illinois		2	4	5	10	12	15	24
Indiana			2	1	1	1		6
Iowa					1	1	3	1
Kansas		1				2	2	
Kentucky		1			1	2	2	1
Maine		3					1	
Maryland		1	2	3	4	1	4	8
Massachusetts	1	2		1	4	3	6	4
Minnesota		1	1	2	10	1	3	5
Mississippi		1						
Missouri			(1)	6		(1)	8	3
Montana	1							
Nebraska		1			3		1	2
New Jersey		2	1	1	2	(1)	3	1
New York [2]				1		2	4	10
North Carolina	1		3		4	3	1	
South Dakota							3	
Vermont						3	1	
Virginia		(1)	(1)	1		(1)	2	1
Wisconsin	2				1	4	9	14

[1] No report received.

[2] Exclusive of New York City.