Epidemiologic observations of recurrent epidemic influenza A2 in rural areas and in selected urban groups in Louisiana in the winter of 1959-1960 are presented. Comparisons are made with the 1957 epidemic, and certain conspicuous differences are pointed out.

EPIDEMIC RECURRENCE OF ASIAN INFLUENZA IN LOUISIANA, 1959-1960

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DURING the past two centuries there have been divergent opinions on the role of immunity in recurrent influenza. While the lessons from the past are clouded for the lack of virus identification, nevertheless, they serve to illustrate the scope of the problem. Edward Gray[1] stated "that many persons who escaped the epidemic of 1775 were affected by that of 1782, and that many who escaped the latter were affected by the former." Noah Webster[2] who was ill during both waves of influenza in the autumn of 1789 and the spring of 1790, alluded to the lack of protection of the first epidemic when he wrote "that in the northern states—the disease was more violent than the preceding autumn." Streeten,[3] reporting on the first influenza survey in England in 1837, noted that most physicians answering the survey believed that "having undergone an attack of the previous epidemic (1833-34) afforded no protection." However, some described a milder episode in 1837 in those who were previously ill. Parsons[4] in his report on influenza during 1889-92 noted the relationship between the severity of an initial attack and a lower incidence during a succeeding epidemic in English towns. Regarding individual immunity, some English physicians believed that those who were ill once were especially susceptible during a recurrence.

The pandemic of 1918 produced more questions than it solved. Numerous authors found that individuals present, but not necessarily ill, in the mild summer epidemic of 1918, were spared during the virulent winter episode six months later.[5-8] However, recurrences at the longer interval of 1920, Vaughan[6] and Jordon and Sharp,[9] demonstrated little differences between the attack rates of those who were ill and those who had escaped in 1918. Pickles and Burnet,[10] describing what were probably three episodes of influenza A in the Wensleydale villages in England, demonstrated an inverse relationship between the severity of an initial attack in 1933 and the succeeding epidemic in 1937. However, this was not true for the 1943-44 epidemic in these villages.

Asian influenza swept across the world in 1957, and there were indications that older age groups that had been attacked lightly during the fall were contributing heavily to the second excess influenza-pneumonia death wave in the winter of 1957-58.[11] In 1958 and in early 1959, there were only sporadic recurrences, but a marked recrudescence

of influenza occurred in the winter of 1959-60.

This report concerns the recurrence of Asian influenza in Louisiana during the winter of 1959-60. The presence of a second major epidemic in this area in a two-year period raised questions that were the basis for the following epidemiologic study. These questions were as follows: Had the 1957 epidemic conveyed any immunity to the community as evidenced by a lower incidence in 1959-60? Were those individuals who were previously ill in 1957 protected during the recurrence? What factors, if any, governed the degree of individual susceptibility?

Methods

On March 15, 1960, as the epidemic was subsiding, two distinct population groups, one unvaccinated, the other partially vaccinated, were chosen for epidemiological surveys. The first group, located in Tangipahoa Parish, a farming community in southeastern Louisiana, was comprised of unvaccinated individuals. It has a population of about 60,000, one-third of which is Negro. The socioeconomic status of this parish is low, but the white population is above that of the Negro. However, Ponchatoula, a more prosperous farming community, has a white population in the middle to upper socioeconomic range. Epidemiological surveys had been made in August and September of 1957 following the severe summer epidemic by Dunn, et al.[12] In these surveys, students from five high schools filled out epidemiologic data for their family members. These schools consisted of three with Negro students (Dillon, West Side, and Greenville Park) and two with white pupils (Ponchatoula and Independence). These data were made available by Dr. Alexander D. Langmuir at the Communicable Disease Center, USPHS. In the present study only the last two classes of high school students were asked to give information on the 1959-60 epidemic because they had been present during the 1957 surveys. Family questionnaire forms were distributed and a verbal description of Asian influenza was presented. It was described to them as an illness characterized by the sudden onset of chills and fever, sore throat, headache, cough, muscular aches and pains, associated with a general "washed-out" feeling. The students gave data on their entire household regarding the presence or absence of "flu" since November, 1959, with approximate dates of onset and the age of each individual. The 1959-60 data on each family member were matched with the data obtained in the 1957 surveys and placed on a single IBM card for analysis. Data were available for both the 1957 and the 1959-60 surveys from 2,696 individuals.

The second population surveyed included young adults in New Orleans, some of whom had been previously vaccinated The 1,182 individuals in this group consisted of 525 Charity Hospital physicians and student nurses, 364 Tulane medical students and 293 students from the New Orleans Baptist Theological Seminary. They checked "yes" or "no" or "uncertain" regarding the presence or absence of influenza during the winter of 1959-60, winter of 1958-59, or during the 1957 epidemic. If they had received influenza vaccinations they gave the year, but not the number of inoculations, nor the route, nor the source of the vaccine. Answers marked "uncertain" were considered as "no" in analyzing the results.

The presence of infection by influenza virus was investigated by collection of garglings and/or paired blood specimens from suspected cases. Virus isolations were done in ten-day embryonated eggs by two or more passages in the amniotic sac. Serological evidence of influenza was obtained by hemaggluti-

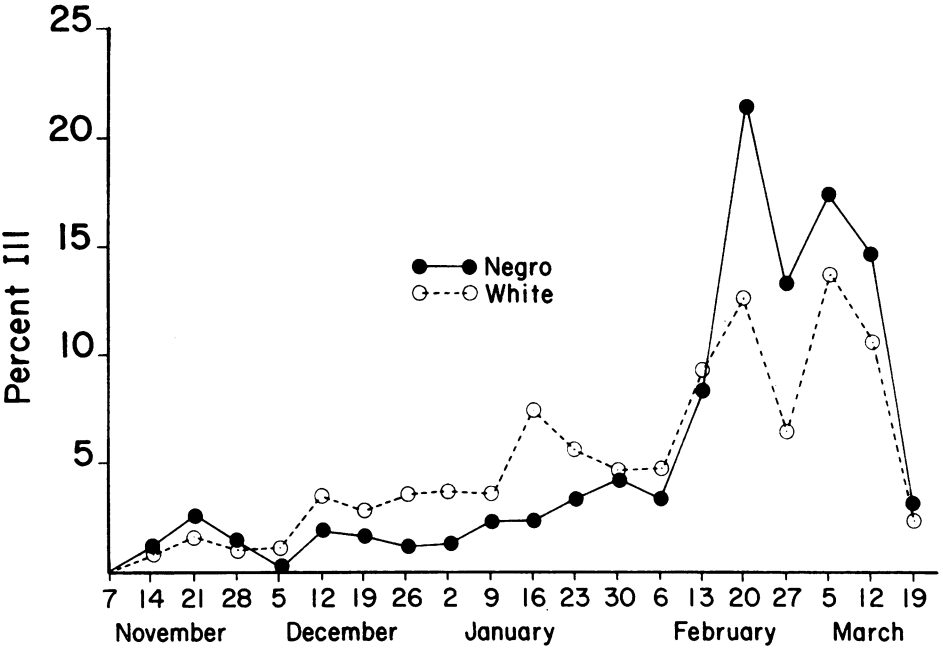

Figure 1—Epidemic Curve, Influenza A2, 1959-60, in Negro and White Populations of Tangipahoa Parish, La. These data were derived from totals of 312 Negro and 347 white pupils who gave reports on date of onset of illness.

nation-inhibition with serums treated with receptor destroying enzyme of Vibrio cholerae. Determinations were made with an egg or monkey kidney tissue culture line of Asian virus isolated in New Orleans in 1957.[13] In those instances in which antibody was not demonstrable with the egg line the procedure was repeated with the tissue culture propagated virus.

Results

Tangipahoa Parish Epidemic

Clinical Features—The clinical findings did not differ from those in the cases of Asian influenza in 1957. Complaints included chills, fever, sore throat, cough, malaise, headache, and myalgia. The physical findings in many were compatible with acute bronchitis. As in 1957, the severity of the clinical illness varied widely. Some had fever up to five days with severe bronchitis, while others had a short one- or two-day febrile course, with a minimum of upper respiratory symptoms. These general observations held true for students and medical personnel observed in New Orleans as well as patients in Tangipahoa Parish.

Epidemic Sequence—In Figure 1, separate epidemic curves are shown for the families of the white schools (Ponchatoula and Independence) and the Negro schools (Dillon, West Side, and Greenville Park). The epidemic assumed definite proportions during the second week of February, reaching a peak one week later. Both races had a similar timing of the epidemic peaks. This differed from the 1957 survey which showed one white school (Ponchatoula) to have an epidemic peak four weeks later than the Negro schools.[12] Further breakdown of these data from

Table 1—Influenza Attack Rates 1957 and 1959-60, Tangipahoa Parish Survey by School Family Groups

Name of School	Total Persons	Per cent Ill	
		1957	1959-60
Ponchatoula	827	20.7	42.2
Independence	475	29.7	27.2
Dillon	245	51.0	42.4
West Side	623	58.7	27.3
Greenville Park	526	60.3	21.3
Totals	2,696	41.5	32.0

the 1959-60 survey did not reveal any significant differences in the two white schools to warrant separate curves for each school.

Analysis of the epidemic timing by age revealed no marked differences in the onset or epidemic peaking in the different age groups. However, the 1957 survey had revealed an earlier epidemic peak for high school students in the school-families from West Side, Independence, and Greenville Park.[12]

Comparison of the 1957 and 1959-60 Attack Rates—In Table 1 the attack rates for both epidemics by each school-family group are shown. Here the 1957 attack rates are compared with the incidence of illness in 1959-60. Those ill in 1959-60 were also analyzed with respect to the presence or absence of influenza in 1957 (Table 2). In regard to the parish as a whole, it can be seen in the summary that the 1959-60 attack rate was lower than 1957 with a reduction from 41.5 to 32.0 cases per hundred. The inverse relationships between the attack rates of the two epidemics that are noted at Ponchatoula, West Side, and Greenville Park may reflect the effect of the previous epidemic, in that they were at the extremes of influenza incidence in 1957. This type of reciprocal relationship is also noted by combining all schools and comparing them as to race. These data revealed a change in the 1957 attack rates, in opposite directions, for the white and Negro races. White families had a

Table 2—Influenza Attack Rates 1959-60, Tangipahoa Parish Survey, According to History of Being Ill or Not Ill in 1957, and by School-Family Groups

Name of School	Total Persons Surveyed		Per cent Ill 1959-60	
	Ill 1957	Not Ill 1957	Of 1957 Ill	Of 1957 Not Ill
Ponchatoula	171	656	51.5	39.8
Independence	141	334	33.3	24.6
Dillon	125	120	50.4	34.2
West Side	366	257	26.2	28.8
Greenville Park	317	209	19.9	23.4
Totals	1,120	1,576	31.9	32.2

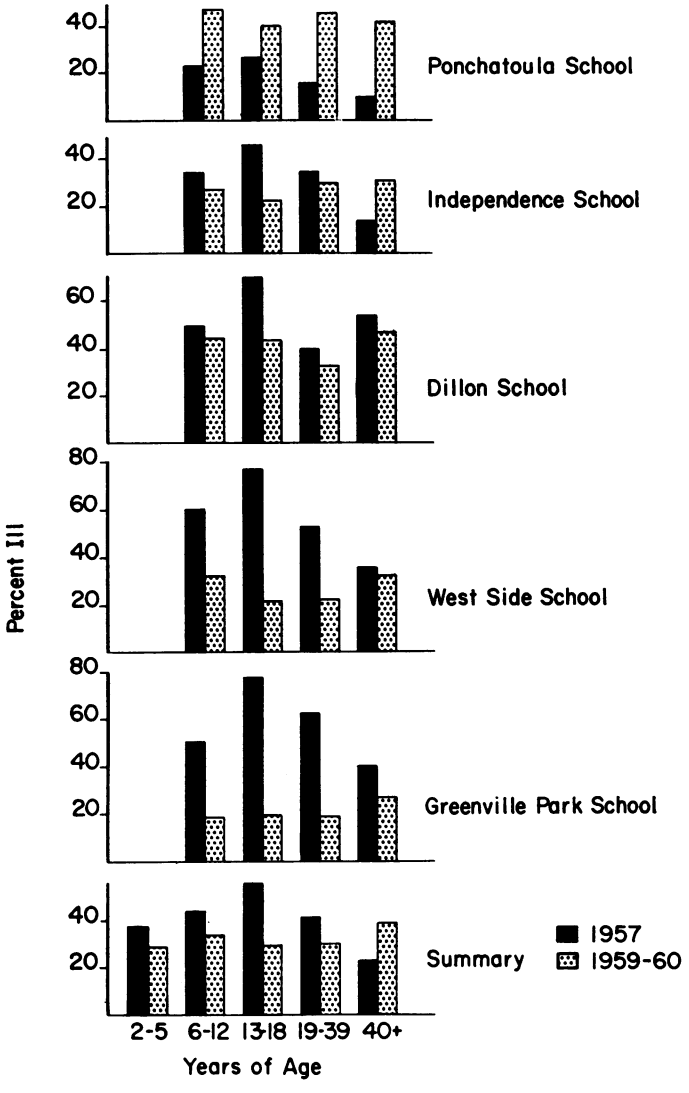

Figure 2—Comparison of the 1957 and the 1959-60 Influenza Attack Rates by Age in Each of the School-Family Survey Groups.

shift in attack rate from 24.0 to 36.6 while the Negro incidence of illness dropped from 57.9 to 27.2. It should be noted that the survey group was evenly divided by race, 48.4 per cent white and 51.6 per cent Negro. Independence and Dillon, unlike the other schools, did not show the inverse relationship of influenza incidence in the two epidemics, but both had a slightly lower attack rate in 1959-60. Furthermore, Dillon differed in that its 1959-60 attack rate was double that of the other Negro schools. The data regarding the Negro individuals is of additional interest inasmuch as only 422 of a total of 1,394 persons surveyed (30.3 per cent) escaped clinical influenza after

Table 3—Influenza Attack Rates in Individuals Over 40 Years of Age, Tangipahoa Parish Survey

Age	Total	1957 Cases/100	1959-60 Cases/100
40-49	411	24.1	35.0
50-59	214	22.0	34.6
60-up	84	25.0	38.1
Totals	709	23.6	35.3

Table 4—Influenza Attack Rates in 1959-60 According to Family Size, Tangipahoa Parish Survey

No. of Members in Family	No. of Families	Total No. of Individuals	Per cent Ill
2	16	32	43.7
3	70	210	33.3
4	106	424	32.1
5	92	460	30.2
6	81	486	32.5
7	58	406	24.9
8	43	344	36.9
9	26	234	32.5
10	21	210	33.3
11	11	121	25.6
12	2	24	16.7
13	2	26	26.9
14	2	28	14.3
18	1	18	22.2
Totals	531	3,023	31.1

two epidemics. This could represent the expected incidence of subclinical Asian influenza for a population that probably had nearly all its members in contact with the virus after two epidemics.

In evaluating the effect of the 1957 epidemic on individuals (Table 2) it was noted that the previous illness did not protect against a recurrence during the 1959-60 epidemic. These data suggest that, although there might have been factors reducing attack rates on a community-wide basis, there appeared to be little effect at the individual level.

Comparison of the 1957 and 1959-60 Attack Rates by Age and School Group —Incidence of influenza in the families of the five school populations was analyzed according to age groups and comparisons were made between the 1957 and 1959-60 epidemics as shown in Figure 2. The role of socioeconomic status and of race in affecting the susceptibility of each age group can also be evaluated in these data. The 2-5-year-old group was not shown on the graph except in the summary because of its small size. In 1957, the highest incidence of illness occurred in the 13-18-year-old group, followed by the younger school children (6-12 years). The young adults (19-39 years) and the preschool children (2-5 years) had less, while the 40 years and older group

Table 5—Influenza Attack Rates, 1957 and 1959-60, New Orleans Survey by Selected Population

Name of Group	Number of Persons Surveyed	Per cent Ill 1957	Per cent Ill 1959-60
Charity Hospital resident physicians, and student nurses	525	26.5	17.7
Tulane University School of Medicine students	364	36.0	18.1
New Orleans Baptist Theological Seminary students	293	24.6	19.1
Totals	1,182	28.9	18.2

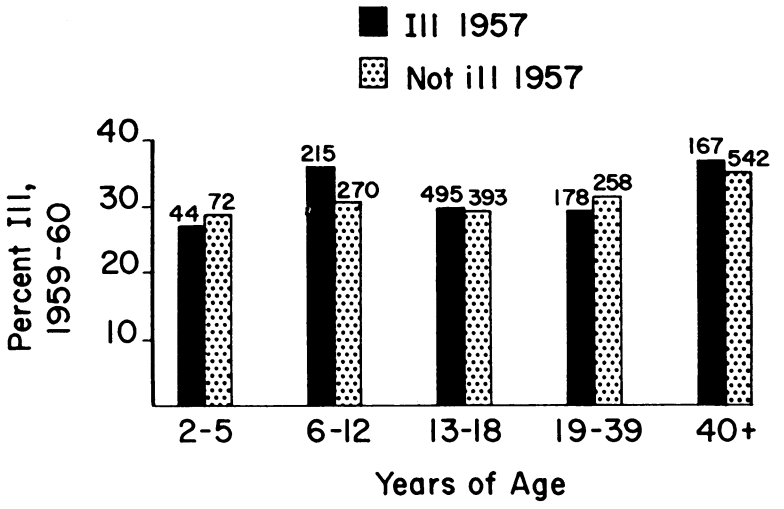

Figure 3—Comparison of the 1959-60 Influenza Attack Rates of Those Ill and Not Ill in 1957 by Age Groups. Total numbers of individuals surveyed in each group are shown at the top of their respective columns.

experienced the lowest attack rate. In 1957 the Negro families of all age groups had a higher incidence of influenza than those of the white race and, in 1959-60, all Negro groups had a lower attack rate than in 1957. The white families from Ponchatoula and Independence showed an interesting difference. In the age groups 6-12, 13-18, and 19-39, Ponchatoula had a low 1957 attack rate and then a subsequent higher incidence in 1959-60. The reverse was true of Independence. In the 40 years and older group both Ponchatoula and Independence showed a low 1957 occurrence of illness, but then a large increase in 1959-60. These observations suggested that immunity might be playing a role at the community level especially in areas where an inverse relationship existed between the attack rates of the two epidemics. These were most outstanding in the 13-18 age group at West Side and Greenville Park; in the 19-39 age group at Ponchatoula and Greenville Park, and in both white schools in the 40 years and older group.

In the 19-39 age group it is of interest to note that, as the 1957 attack rate increased in the Negro schools, there was a corresponding decrease in the 1959-60 incidence of illness. The 40 and older individuals were unique in that they were the only age group that had an attack rate in 1959-60 that was greater than 1957. The older segments of the population (above 60 years) did not show a greater resistance to influenza than the rest of the 40 years and older individuals. This is shown in Table 3 where the 40 and older age range is subdivided into decades.

Further analysis of the 1959-60 attack rates according to age groups as related to those ill and not ill in 1957 is shown in Figure 3. These data again show the lack of effect in all age groups of clinical influenza in 1957 on the incidence of illness in the second epidemic.

Recurrent Influenza — The incidence of recurrent illness in each age and school group is shown in Figure 4. Children of school age (6-12 and 13-18 years) had the greatest per cent of re-

current illness (16 per cent). The young adults and preschool children had a lower recurrence rate, while the older adults had the lowest of all. These results are related to the 1957 incidence of illness in these age groups, which would govern the number of individuals that would be available for recurrence. It is likely that the increased number of social contacts of the school-age children could account for their greater rate of recurrent illness.

In the school-family groups, the Negroes had a higher per cent of recurrent illness than did the white families. The high recurrence rate at Dillon as compared to the other Negro schools reflects its high 1959-60 attack rate, which was twice that of West Side and Greenville Park. However, there were no obvious reasons for these differences.

Attack Rates in 1959-60 by Family Size—In the 1957 epidemic Dunn, et al.,[12] showed a gradual increase in incidence of influenza as the family size increased. However, in Table 4 it is seen that this relationship was not present in the 1959-60 survey. While the extremely large families had a low attack rate, the numbers were too small to be very significant. Further analysis of family size by school groups yielded similar results with the exception of Independence. In the latter case there were 55 cases per 100 for those families with eight or more members versus 23 cases per 100 for those with less than eight members.

New Orleans Survey

Comparison of the 1957-58 and 1959-60 Attack Rates—The results of surveys made shortly after recession of outbreaks in selected medical and university populations are shown in Table 5. Except for lower attack rates in both epidemic periods, the findings were quite similar to those derived from Tangipahoa Parish. The incidence of clinical illness was lower in 1959-60 than in 1957-58; however, it appears that an infection two years previously had no protective effect for the individual against the current influenza A2 virus (Table 6).

The Effect of Vaccination and Previous Influenza—In Figure 5 the attack rates of the New Orleans groups were analyzed in terms of antigenic experi-

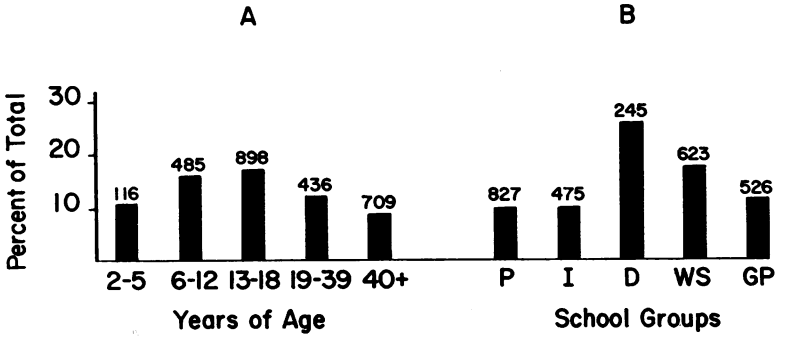

Figure 4—Incidence of Recurrent Influenza Analyzed According to (A) Age and (B) School-Family Survey Groups. Total numbers of individuals surveyed in each group are shown at the top of their respective columns.

Table 6—Influenza Attack Rates, 1959-60, New Orleans Survey, According to History of Being Ill or Not Ill in 1957 and by Selected Population

Name of Group	Total Persons Surveyed		Per cent Ill 1959-60	
	Ill 1957	Not Ill 1957	Of 1957 Ill	Of 1957 Not Ill
Charity Hospital resident physicians and student nurses	139	386	18.0	17.6
Tulane University School of Medicine students	131	233	16.8	18.9
New Orleans Baptist Theological Seminary students	72	221	18.1	19.5
Totals	342	840	17.6	18.5

ences by means of vaccination or disease. The results show little difference in attack rates except for those individuals that had been immunized shortly before the epidemic. (p of 1959 vaccine vs. 1957 vaccine = 0.05 and 0.16 vs. 1958 vaccine.)

Laboratory Observations

Virus Isolations — Viruses isolated during the epidemic period, January-March, 1960, were identified as influenza A2 by hemagglutination-inhibition. Characteristics were like the 1957 strains and virus was recovered by one or two passages in the amniotic sac of the chick embryo.[13] Hemagglutination titers with human Type O and fowl erythrocytes were the same as had been observed with the 1957 viruses. The 1960 strains were compared and all were found similar as determined by hemagglutination-inhibition with fowl and rabbit antiserums prepared against a 1957 New Orleans isolate. The 1960 strains also were antigenically like the latter virus. During the epidemic period a total of 55 cases of influenza A2 were documented by virus isolation and/or hemagglutination-inhibition. Twenty of these cases were from areas near the study populations. The remainder were cases from the study groups that had been selected to document the presence of influenza A2 infection. A total of 15 isolations was made during the epidemic.

Serologic Determinations — In 40 cases there was a fourfold or greater increment in hemagglutination-inhibition antibody titers as determined with an egg or tissue culture line of influenza A2/NO 1/57. In four instances it was necessary to use the monkey kidney cul-

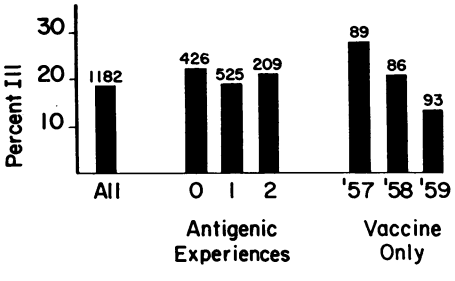

Figure 5—The Combined Effect of Previous Influenza and Vaccination and of Vaccine Alone on the 1959-60 Attack Rates in the New Orleans Groups. An antigenic experience was either an influenza episode or an Asian influenza vaccination. Total numbers of individuals surveyed in each group are shown at the top of their respective columns.

ture line to demonstrate antibody titer rises. Acute serum inhibition titers were less than 1:4 in 30 of these, and 1:4 in the remainder.

Clinical Correlation with Laboratory Diagnoses—During the epidemic period cases of clinical influenza were selected for laboratory diagnosis. These served to document the presence of the virus and to determine the validity of the clinical diagnoses. In practically all cases selected for study it was possible to obtain laboratory confirmation of the clinical impression during the period of observation.

Discussion

Recurrence of Asian influenza during the winter of 1959-60 provided a unique opportunity to make epidemiologic observations on a disease that had been pandemic two years previously and was again epidemic. Among the influenza viruses this had not occurred with an antigenic newcomer in such proportions during the period of modern virology. Accordingly, it was possible to document that similar strains of influenza A2 were responsible for both epidemics. This could not have been done during the 1918-19 pandemic. Moreover, the availability of survey records of the Tangipahoa Parish population allowed a comparison of the features of the 1957 and 1959-60 epidemics in an area that had previously experienced an extremely high incidence of illness.[12]

The recurrence of epidemic Asian influenza after a two-year period was unexpected, at least in the proportions observed in areas which were heavily attacked in 1957. In 1958 there were no isolations of influenza A2 from a total of 1,130 garglings collected from university, industrial, and military groups in areas near the study groups.[14] The absence of endemic foci in this area during the interepidemic period would facilitate the rapid decrease of antibody titers in the population, especially after only one exposure to an antigen as different from the other influenza A strains as the Asian variant.

The most surprising aspect of the epidemiologic surveys reported herein was the inability to show any reduction in the incidence of Asian influenza that could be correlated with an individual's past history of that disease. In addition, this made it difficult to explain why the 1959-60 attack rate was lower than 1957. Certainly the presence of a partially immune community with an apparent absence of individual protection could not be readily rationalized from the data presented. Yet, such a phenomenon was suggested by the inverse relationships between the attack rates of the two epidemics in some of the school, racial, and age classifications used in the analysis of these data. While the studies presented did not include data which would relate the length of illness to the presence or absence of previous influenza, nevertheless, one might speculate that the partially immune individual might interfere with viral dissemination via a shorter illness and possibly a reduction of viral particles produced in the respiratory tract. There seem to be no studies available that have sought to quantitate virus production in partially immune individuals during recurrent influenza. However, Bell, et al.,[15] in 1957 noted that vaccinated volunteers had a milder and shorter illness than unprotected controls when challenged with Asian influenza virus.

As another explanation of the before mentioned paradox, it is reasonable to speculate about the validity of the surveys. One may suggest that the 1959-60 attack rates reflect only the sporadic character of influenza, unrelated to the prior epidemic; that the index individuals have confused influenza with other winter respiratory disease; that the index persons have weighted the

surveys with more accurate information regarding themselves. The fact that the same individual has recorded the events in both epidemics and has indicated many examples of 1959-60 attack rates that were inverse to those in 1957, suggests that the above speculations about the surveys are not valid. In addition, a separate analysis of the 531 index persons (teenagers) revealed a 1957 attack rate of 58.9 cases per hundred which fell to 32.8 in 1959-60. When the ill and not ill of this 1957 group were compared as to incidence of illness in 1959-60 the rates were 34.2 and 30.7 cases per hundred, respectively. These data compare closely with the entire 13-19 age group, and the community as a whole, which also failed to receive individual protection by the 1957 epidemic.

In the analysis of certain factors that might lead to variation in individual susceptibility to influenza, the role of age has been a subject of much interest. The low incidence of illness of the older adult population during the 1957 pandemic[12] has suggested that these individuals may be more immune than the younger segment of the population.[17,18] There are, however, several lines of evidence which suggest that this concept is not valid: (1) The lower attack rate of the older adult population was not correlated with the presence of pre-epidemic antibody to the Asian strain except with a small percentage of individuals who were over 70 years of age.[16,17] (2) The preschool children, with little influenza antigenic experience, also had a low incidence of illness in 1957.[12] (3) If some immunity exists in the older population, then it should have been reinforced by the 1957 epidemic. On the contrary, the incidence of influenza greatly increased in the 40 and older age group with the second epidemic. In addition, the 1959-60 attack rates for the older adults were found to be the same for those individuals who were ill in 1957 and for those who had escaped. (4) Within the 709 individuals who made up the 40 and older age group there was a conspicuous absence of a lower attack rate in the older segment of this group (above 50 years of age). (5) The attack rates of individuals in different age ranges varied widely with the racial and socioeconomic status in Tangipahoa Parish. These lines of evidence, coupled with the knowledge that the children of school age had the highest attack rates in 1957 and the greatest recurrence in 1959-60, suggest that individual social mobility probably plays a major role in varying the susceptibility of different age groups, and that probably all age ranges lack immunity to a new variant of influenza.

The surveys carried out in the New Orleans group supported the findings in Tangipahoa Parish. Thus, it was not surprising that vaccination as well as the natural illness supplied no protective effect unless it was administered shortly before the epidemic. It appears that numerous antigenic experiences in a considerable proportion of the population might be necessary to cause disappearance of certain influenza viruses with distinct immunologic capacities.

Summary

Epidemiologic observations of recurrent epidemic influenza A2 in rural areas and in selected urban groups in Louisiana in the winter of 1959-60 have been presented. Attack rates of 32.0 per cent in the school-family groups in Tangipahoa Parish, and 18.2 per cent in the university and medical personnel in the city of New Orleans were found. These were considerably less than the 1957 incidence of Asian influenza, but the rates of illness were the same whether or not there was a past history or record of the disease. Vaccine did not appear to reduce the incidence

either, unless it was administered during the year of the epidemic.

Certain differences between the two epidemics were conspicuous. These included an inverse relationship in incidence of influenza in certain school, racial, and age groups. Family size did not affect the numbers who became sick in the second epidemic.

There were 15 isolations of influenza A2 and all strains were antigenically alike as determined by hemagglutination-inhibition and did not differ from a virus isolated in New Orleans in 1957. Hemagglutination - inhibition antibody was not detectable in most of the acute serums of those individuals in whom a serologic diagnosis of influenza was made.

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REFERENCES

1. Gray, E. "An Account of the Epidemic Catarrh of the Year 1782." In Annals of Influenza by T. Thompson. London, England: Sydenham Society, 1852, p. 122.
2. Webster, N. A Brief History of Epidemic and Pestilential Diseases. Hartford, Conn.: Hudson and Goodwin, 1799. Vol. I, p. 290.
3. Streeten, R. J. N. "Report upon the Influenza or Epidemic Catarrh of the Winter of 1836-37." In Annals of Influenza by T. Thompson. London: Sydenham Society, 1852, p. 292.
4. Parsons, H. F. Further Report and Papers on Epidemic Influenza 1889-92. London: Local Government Board, 1893, p. 50.
5. Stanley, L. L. Influenza at San Quentin Prison, California. Pub. Health Rep. 34:996–1008, 1919.
6. Vaughan, W. T. Influenza—An Epidemiologic Study. Am. J. Hyg. Monograph Ser. No. 1, Baltimore, Md., 1921.
7. Gibbon, J. G. Acquired Immunity in Influenza. Lancet 1:583, 1919.
8. Joltrain, E., and Baufle, P. Epidemic of Influenza. Abstr. in J.A.M.A. 1919, p. 527.
9. Jordon, E. O., and Sharp, W. B. Influenza Studies I. Immunity in Influenza. J. Infect. Dis. 26–27:463–468, 1920.
10. Pickles, W. N.; Burnet, F. M.; and McArthur, N. Epidemic Respiratory Infection in a Rural Population with Special Reference to the Influenza Epidemics of 1933, 1936-37, and 1943-44. J. Hyg. 45:469–473, 1947.
11. McDonald, J. C. Asian Influenza in Great Britain, 1957-58. Proc. Roy. Soc. Med. 51:1016–1018, 1958.
12. Dunn, F. L.; Carey, D. E.; Cohen, A.; and Martin, J. D. Epidemiologic Studies of Asian Influenza in a Louisiana Parish. Am. J. Hyg. 70:351–371, 1959.
13. Mogabgab, W. J.; Pelon, W.; Burch, G. E.; and Holmes, B. Characteristics of the Asian Strain of Influenza A. Proc. Soc. Exper. Biol. & Med. 99:116–120, 1958.
14. Mogabgab, W. J. Unpublished observations.
15. Bell, J. A.; Ward, T. G.; Kapikian, A. Z.; Shelkov, A.; Reichelderfer, T. E.; and Huebner, R. J. Artificially Induced Asian Influenza in Vaccinated and Unvaccinated Volunteers. J.A.M.A. 165: 1366–1372, 1957.
16. Mulder, J., and Masurel, N. Pre-epidemic Antibody Against 1957 Strain of Asiatic Influenza. Lancet 1:810–814, 1958.
17. Davenport, F. M., and Hennessy, A. V. The Clinical Epidemiology of Asian Influenza. Ann. Int. Med. 49:493–501, 1958.
18. Jensen, K. E.; Dunn, F. L.; and Robinson, R. Q. "Influenza, 1957, A Variant and the Pandemic." In Progress in Medical Virology. New York, N. Y.: Hafner Publishing Co., 1958, p. 164.

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