# Observations on Asian Influenza on Two Alaskan Islands

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In OCTOBER 1957, the variant of type A influenza virus commonly referred to as Asian virus was widely prevalent in larger urban centers and many native villages of Alaska. A second wave of influenza was noted early in 1958 in a few villages in southern Alaska which had previously escaped the disease. The overall influenza morbidity in the State is not precisely known but undoubtedly was high. The general mildness of the disease was reflected in influenza and pneumonia mortality rates. They were only slightly higher during October than normally expected for that month, according to the Alaska Department of Health.

Influenza likewise occurred in several remote island communities of Alaska, two of which were St. Paul, Pribilof Islands, and Gambell, St. Lawrence Island, where opportunity was afforded for rather complete investigation. Results of the investigation reported here are of interest because the communities were more isolated than those populations from which most information on Asian influenza has been derived. This isolation facilitated tracing the

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mode of introduction of disease into each village. Attack rates were extremely high, and the frequency of pulmonary complications in St. Paul was higher than observed generally.

## **Background**

St. Paul Island is in the Bering Sea approximately 250 miles northwest of the Aleutian chain (fig. 1). The chief mode of travel to St. Paul is by weekly commercial air service from Cold Bay on the Alaska Peninsula. Travel by boat is limited to infrequent visits by U.S. Fish and Wildlife Service and Coast Guard vessels. The neighboring island community of St. George is only 50 miles from St. Paul, but there was no traffic between them during the outbreak in St. Paul. Consequently, influenza did not occur in St. George.

St. Paul village is comprised of some 350 residents most of whom are predominantly of Aleut descent. The chief industry is fur sealing under U.S. Fish and Wildlife Service jurisdiction, and the majority of the few non-native residents are employees of this agency. The village is compact. The dwellings are generally multiroom and clean, have most of the modern conveniences, and reflect the prosperity of the community. Village ties within racial groups are close, and there is much social interchange among households (defined as one or more persons living as a family group under a single roof). Community life centers around the school, church, movie theater, store, and commissary. The residents are well nourished and warmly clothed. Their health needs

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Figure 1. The State of Alaska, with St. Lawrence, St. Paul, and St. George Islands.

are served by a Public Health Service physician in a small clinic-hospital.

St. Lawrence Island is in the northern part of the Bering Sea approximately 120 miles from Alaska and 40 miles from Siberia (fig. 1). It is inhabited by some 550 Eskimos residing in two communities of similar size, Gambell and Savoonga. At the time of the outbreak, all transportation to the island was by biweekly commercial air service from Nome, Alaska, to Gambell, and all persons traveling to Savoonga pass through Gambell. The Eskimo residents are gregarious, and their villages are usually composed of single-room frame houses which are crowded and poorly constructed in comparison with housing in St. Paul. Community life centers around the school, mission, native store, and National Guard armory. Health services for the island are administered directly by an Alaska Department of Health nurse who

resides in Gambell. Illnesses requiring hospitalization are referred to the Public Health Service's Alaska Native Hospital, Kotzebue.

Influenza occurred in Savoonga also shortly after it was introduced into Gambell. However, details of the outbreak in Savoonga were not sufficiently complete to merit inclusion in this report.

#### Methods

Clinical observations were based on 192 patients (65 percent of the total 294 cases) who were seen at the clinic during the outbreak in St. Paul. Detailed clinical observations were not obtained in Gambell. Epidemiological observations were obtained in St. Paul by a survey of all households immediately after the epidemic, and in Gambell, primarily from health department nursing records and a can-

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vass of all households 1 and 3 months after the outbreak. The majority of illnesses were attended by the nurse who recorded dates and major symptoms. Dates of occurrence of unattended illnesses were obtained in retrospect by relating them to onsets of attended illnesses among household associates.

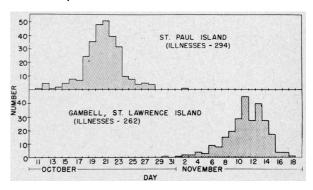
Serologic observations in St. Paul were based on blood serums obtained from 152 persons by Dr. C. M. Eklund, Rocky Mountain Laboratory, Hamilton, Mont., during his investigation of an outbreak of poliomyelitis in 1954 (1), and serums obtained by Weeks from 30 persons 1 to 2 months after the outbreak. Although postepidemic serums in St. Paul were obtained from few persons, most of whom were adults, and donors were not selected on the basis of occurrence of illness during the outbreak, it is felt that the test results are probably representative of adult age groups.

Gambell observations were based on serums obtained from 85 persons during investigation of a mumps epidemic 3 months prior to the influenza outbreak (2) and from 125 persons 3 months after the outbreak. Persons contributing blood specimens prior to the outbreaks were selected only on the basis of availability, cooperativeness, and age. Proportionately fewer serums were obtained from children under 6 years of age than other age groups. Postepidemic serums in Gambell were obtained from all available persons, approximately 6 years or older, who were willing to contribute blood specimens.

Testing was carried out by the hemagglutination inhibition (HI) technique in the manner recommended by Jensen (3) with his suggested modifications (4). Potassium periodate was used to inactivate nonspecific inhibitors to influenza viruses in the serums. Antibody titers are recorded as the final serum dilutions giving inhibitions. Although serums obtained prior to and after the outbreak in each village were not tested simultaneously, human serums of known titers to each test antigen were included as controls in each test.

The influenza virus strains used as HI test antigens included swine (Shope), A/PR8, A/1520 (a Denver-like A' strain isolated in Hamilton, Mont., March 1956), and A/Jap/305/57 (Asian). Antigens were prepared by

Figure 2. Number of illnesses occurring daily during the St. Paul and Gambell influenza outbreaks, 1957.



allantoic inoculation of virus seed into 10-day embryonated chicken eggs. After 4 days' incubation, allantoic fluids were harvested and titrated by hemagglutination against known antiserums. Eight hemagglutinating units of antigen were used in the test.

Pharyngeal swabs were obtained for culture from 38 ill persons during the latter stages of the outbreak in St. Paul. These were immediately planted in stock culture agar (Difco) slants and held at approximately 4° C. for 3 to 6 days until they could be transported to the Infectious Disease Laboratory, Arctic Health Research Center, Anchorage, for processing and bacteriological identification.

Figure 2 shows the chronological occurrence of illness during each outbreak. Two hundred and ninety-four (82 percent) of 358 St. Paul residents were ill between October 11 and November 2. Two hundred and sixty-two (87 percent) of 300 Gambell residents were ill between October 30 and November 18. Each outbreak was sharp, carried high risk of attack, and lasted approximately 3 weeks. The epidemic curves are similar and are characteristic of influenza.

#### Source of Outbreaks

Logs of all incoming traffic are maintained in each community. Examination of the St. Paul log and careful questioning of each passenger indicated that influenza was introduced into the village October 10 when four persons arrived by plane from Cold Bay, Alaska Peninsula. Since two of the passengers had influ-

enza-like illnesses October 20 and 22, they were not considered to be sources of the outbreak. A third passenger (A. M.) had departed from the Navy base, Adak, Aleutian Islands, October 9, and had stayed overnight at Cold Bay while awaiting transportation to St. Paul. In Cold Bay, at noon time on October 10, he met the fourth passenger (S. M.) en route from Anchorage, where he had just been discharged from the Army. The two men were closely associated during the subsequent 3-hour delay at Cold Bay and the 2-hour flight to St. Paul.

Approximately 8 hours after arrival, A. M. developed an influenza-like illness and all members of his family subsequently had similar illnesses, four beginning October 12 and one October 15. Meanwhile, S. M. celebrated his discharge and was jailed at noon, October 11, for disorderly conduct. He remained in jail until October 29, except for visits to the clinic for treatment of an influenza-like illness which began October 12. He could not have been an effective source of illness to the village.

The evidence, therefore, indicates that A. M. was probably the source of the outbreak. In

Table 1. Occurrence of illness during St. Paul and Gambell influenza outbreaks, by age and sex, 1957

Age and sex	St. Pa	ul	Gambell		
J	Number <sup>1</sup>	Per- cent	Number <sup>1</sup>	Per- cent	
Age (years)					
Under 1  1-4  5-9  10-14  15-19  20-29  30-39  40-49  50-59  60-69  70-79  80 and over  Sex	7/11 39/44 42/45 45/48 21/27 47/59 41/50 31/39 12/17 6/15 3/3	64 89 93 94 78 80 82 79 71 40 100	12/13 41/42 53/56 33/36 13/14 30/39 22/32 19/24 15/18 14/15 7/8 3/3	92 98 95 92 93 77 69 79 83 93 88	
MaleFemale	160/196 134/162	82 83	138/156 124/144	88 86	
Total	294/358	82	262/300	87	

<sup>&</sup>lt;sup>1</sup> Numerator represents persons with illness; denominator represents persons at risk.

Table 2. Occurrence of illness during the St. Paul influenza outbreak, by race according to age and sex, 1957

	Whi	te	Aleut			
Age and sex	Num- ber <sup>1</sup>	Per- cent	Num- ber <sup>1</sup>	Per- cent		
Age (years)						
Under 5 5-19 20-39 40 and over	$0/2 \\ 3/4 \\ 3/10 \\ 1/14$	0 75 30 8	46/53 105/116 85/99 51/60	87 91 86 85		
Sex						
MaleFemale	$\begin{array}{c} 6/17 \\ 1/13 \end{array}$	35 8	$154/179 \\ 133/149$	86 89		
Total	7/30	23	287/328	88		

<sup>&</sup>lt;sup>1</sup>Numerator represents persons with illness; denominator represents persons at risk.

view of the fact that the two servicemen were not closely associated after arrival in the village, it is possible that A. M. was infectious to S. M. during the incubation period (8-12 hours prior to onset of symptoms).

The airline schedule showed that influenza was probably introduced into Gambell October 31 when a flight arrived from Nome carrying four passengers, all residents of Gambell and members of the National Guard who were returning from a course of training at Fort Ord, Calif. Three of the guardsmen had influenzalike illnesses in California. Upon arrival in Anchorage October 30, the fourth passenger developed influenza-like symptoms which continued after his arrival in Gambell. members of his household and three neighbors became ill November 1, 2, 4, 6, and 7. The evidence, therefore, suggests that the fourth passenger acquired influenza from exposure before arrival in Alaska and was the direct source of the outbreak in Gambell.

### **Patterns of Morbidity**

Five hundred and fifty-six (84 percent) of 658 residents in both villages were ill during the outbreaks. Attack rates were slightly lower in St. Paul than in Gambell. However, there were similar patterns of attack by age

and sex, that is, risk of illness was only slightly greater for children than adults, and there was no difference between males and females (table 1).

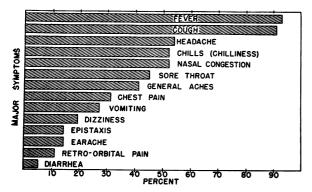
Table 2 compares the proportion of white and Aleut residents of St. Paul who were ill. Attack rates were similar among the 328 St. Paul Aleuts and 300 Gambell Eskimos (table 1) but distinctly lower among the 30 St. Paul white residents.

It is believed that the lower attack rate in white persons was at least partially due to their less frequent and intense exposure to the dis-There was little intermingling of white with other St. Paul residents except in the school and places of occupation. Nearly onehalf of initial household cases occurred among school children, suggesting that the school played an important role in dissemination of the disease in the community. Of 14 white households (comprising an average of 2.1 persons), only 5 had illnesses. Two of the five had school children, whereas all nine households without illnesses consisted wholly of adults. On the other hand, all but 1 of 61 native households (comprising an average of 5.4 persons) had illnesses, and most of these households had school children.

### **Clinical Observations**

The illnesses were generally mild and clinically characteristic of influenza in less remote populations. Figure 3 shows the frequency of various symptoms among 192 ill persons who were medically attended in St. Paul. Fever

Figure 3. Frequency of major symptoms among 194 influenza patients in St. Paul, 1957.



and cough were almost universal. The maximum recorded temperatures of febrile illnesses ranged from 99° F. to 106° F., with a mean maximum of 101.5° F. Thirty-seven percent of ill persons had productive cough, and it is noteworthy that 7 percent had hemoptysis. Painful deglutition was not observed, but many patients complained of "scratchy" throats. Nearly one-third complained of chest pain, usually substernal. Epistaxis was infrequent, but several individuals had severe nosebleeds which stubbornly resisted control. Earaches, predominately in children, complicated 14 percent of illnesses. Few of these progressed to drainage.

Of particular importance was the high frequency of lower respiratory complications (table 3). Since nearly all severely ill persons very likely sought medical care, frequencies were based on total illnesses by age and sex. One-third of illnesses had pulmonary or bronchial complications. Frequency of such complications, particularly those which were most severe, correlated directly with increasing age. Complications were equally frequent among males and females. Ten persons were hospitalized for pneumonia which was confirmed by chest X-ray. Five other persons had X-ray evidence and eight had clinical findings suggestive of pneumonia. Thus, 23 persons had pulmonary complications. There was no correlation between occurrence of lower respiratory complications and the presence of potentially pathogenic bacteria in the throats of 38 patients from whom cultures were obtained.

Two deaths were attributed to complications of influenza during the St. Paul outbreak. A 78-year-old man developed pneumonia complicated by congestive failure 9 days after onset of influenza. He died on the 11th day of illness despite intensive antibiotic therapy. A 27-year-old man developed right, middle lobe pneumonia 3 days after onset of influenza. Although he was hospitalized and given massive doses of antibiotics, including penicillin, tetracycline, streptomycin, and chloramphenicol, he steadily became worse and died on the third hospital day. A pure culture of Staphylococcus aureus, which was resistant in vitro to the antibiotics administered, was obtained from

Table 3. Frequency of lower respiratory tract complications among Aleuts with illness during the St. Paul influenza outbreak, by age and sex, 1957

		Lower respiratory tract complications					
Age and sex	Total ill	Severe 1		Mild <sup>2</sup>		Total	
		Number	Percent	Number	Percent	Number	Percent
Age (years) Under 10	30	1 2 4 7 3 2 4	1 3 9 18 10 17 44	15 18 13 7 13 5	17 28 29 18 43 42	16 20 17 14 16 7	18 31 38 35 53 58 56
Sex           Male	154 133	14 9	9 7	39 33	25 25	53 42	34 32
Total	287	23	8	72	25	95	33

<sup>&</sup>lt;sup>1</sup> Patients who had either X-ray evidence of pneumonia or cough productive of "rusty" or "bloody" sputum, or both.

bronchial aspirations. It is presumed that death was due to an overwhelming secondary infection by this agent.

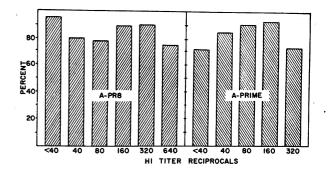
The proportion of St. Lawrence Eskimos who had pulmonary complications is not known. There were, however, no deaths, and the health department nurse gained the impression from clinical observation that complications were infrequent.

### Prior Experience With Influenza

In view of the high morbidity during the two outbreaks, it was of interest to determine prior experience of these population groups with influenza A. There is, to our knowledge, no historical evidence that influenza occurred on St. Lawrence Island in 1918-19. Examination of medical logbooks, kept daily on all persons receiving clinical care on St. Paul Island during the years 1918-20, showed no evidence of an outbreak which could have been influenza. Figure 4 shows the proportion of persons in each village who had specific preepidemic HI antibody titers to preexisting strains of influenza A (including swine, A/PR8, and A') according to three broad age groups. The majority of persons tested in each village, including those 37 years of age or older, had no demonstrable swine antibodies. These results may be contrasted with the serologic findings by Lackman that persons from Montana and Idaho who were born prior to 1920 had a high prevalence of swine antibodies (5).

Few persons in either village born after 1944 had demonstrable HI titers to A/PR8, whereas most persons born prior to 1944 had such antibodies. Several residents stated that an outbreak similar to that in 1957 occurred in St. Paul during the early 1940's. No information

Figure 4. Percentage of tested persons with preepidemic HI antibody titers equal to or greater than specified against influenza A strains of past years.



<sup>&</sup>lt;sup>2</sup> Patients other than those classified "severe" who had chest pain, abnormal pulmonary signs, or productive cough, or both.

is available on such an outbreak on St. Lawrence.

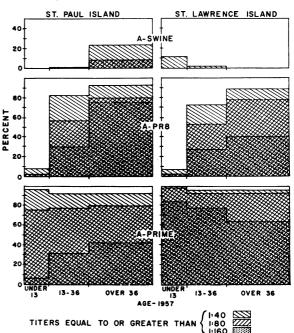
Nearly all persons tested in both villages had demonstrable antibodies to influenza A' indicating recent occurrence of the disease. Since serums were not obtained from very young children, the precise date of the last occurrence of influenza A' could not be determined.

#### Other Serologic Observations

Asian influenza infection rates could not be obtained by serologic methods because monovalent vaccines containing 200 chicken cell agglutinating (CCA) units of Asian virus per milliliter were administered during each outbreak. In St. Paul, two weekly 0.2-ml. doses of vaccine were given intracutaneously to each of 68 well persons beginning October 21, 12 days prior to onset of the last case. Likewise, single 0.1-ml. doses were given intracutaneously to 47 well Gambell residents, November 14, 4 days prior to onset of the last case. Obviously, there was insufficient time for vaccines to affect the course of the outbreaks. However, it was impossible to differentiate naturally acquired from artificially induced Asian strain antibodies among vaccinated persons.

Table 4 shows the distribution of HI titers to Asian influenza among vaccinated and nonvaccinated persons before and after the out-

Figure 5. Percentage of persons with stated preepidemic hemagglutination inhibition antibody levels to influenza A strains who were ill during St. Paul and Gambell influenza outbreaks, 1957.



breaks in each village. It is evident that demonstrable titers were infrequent and when present were low prior to the outbreak. In contrast, most nonvaccinated as well as vaccinated persons had demonstrable antibodies, generally of high titer, after the outbreaks.

Table 4. Distribution of hemagglutination inhibition titers against Asian (A/Jap/305) strain among
St. Paul and Gambell residents before and after the 1957 influenza outbreaks

Place and time of titration	Num- ber of							Percent 40 or
	serums tested	Under 40	40	80	160	320	Over 320	higher of total tested
St. Paul 1954:1 Unvaccinated	152	129	7	12	4	0	0	15
Unvaccinated Unvaccinated Vaccinated	21 9	0	0 0	$\begin{bmatrix} 2\\3 \end{bmatrix}$	12 3	$\begin{bmatrix} 6 \\ 2 \end{bmatrix}$	1 0	100 89
Gambell July 1957:1 Unvaccinated	85	80	3	2	0	0	0	6
February 1958: 2 Unvaccinated Vaccinated	100 25	24 6	10 2	16 4	$\begin{bmatrix} 32 \\ 7 \end{bmatrix}$	14 3	$\frac{4}{3}$	76 76

<sup>&</sup>lt;sup>1</sup> Before the outbreak.

<sup>&</sup>lt;sup>2</sup> After the outbreak.

Serums were obtained during the outbreak in St. Paul from 16 persons with illness. Only three had demonstrable A/Jap/305/57 antibodies. Data not shown have established that there were no essential differences in prevalence of HI antibodies to the other influenza strains before and after the outbreaks.

The magnitude of the outbreaks in each village and the evidence that residents had had extensive experience with type A influenza in the past strongly indicate that prior infection conferred no immunity to influenza in 1957. Figure 5 shows the proportion of persons who were ill grouped according to their preepidemic HI antibody levels to A/PR8 and A' strains.

#### Discussion

Influenza morbidity rates were generally higher in 1957 than during any influenza epidemic in recent years. An estimated 25 percent of the population of the United States was attacked (6). Rates ranging from 10 to 70 percent were reported in population groups of varying composition and size elsewhere in the world (7-14). However, to our knowledge, illness rates during the St. Paul and Gambell outbreaks were as high as any thus far reported.

The fact that each outbreak arose from a single introduction of virus, the date of which could be identified, afforded unusual opportunity for identifying the precise time limits of virus activity in each community. On the basis of the short duration of 3 weeks and the sharpness of epidemic curves, as well as clinical characteristics of illness, there is no doubt that nearly all illnesses occurring within this time were influenza. This raises the question as to why influenza morbidity was so high in these population groups.

The frequent and intense exposure resulting from unusually close community ties was highly favorable for rapid and wide dispersion of Asian virus. There was daily contact between households either at work, at school, at church, at play, or during social activities.

Lack of experience with preexisting influenza A strains could not have been responsible for the high attack rates. Not only had residents of each village been exposed to type A and A' strains but they actually had a higher preva-

lence of HI antibodies than various population groups of comparable age in other Northwestern States (5).

The differences between attack rates in children and adults were not so striking as those noted by others (7, 10, 14, 15). Such differences are thought to reflect a broadening of experience with increasing age to the various antigenic components of influenza A virus (7). Older children and adults in these two villages had experienced both type A and A' infections, whereas young children had been exposed only to A' virus. However, there was no evidence on the basis of circulating HI antibody levels to indicate that past experience with either type A or A' strains reduced the risk of attack by Asian influenza. The degree of immunity may be related to intensity of exposure. It is possible that exposure was so intense during these outbreaks that immunity acquired from prior influenza A infections was not sufficient in most instances to prevent attack against the radically different Asian strain.

It was observed that swine HI antibodies were infrequent in St. Paul and absent in Gambell adults born prior to 1920. This is in contrast with the high prevalence of swine antibodies in this age group in most areas (5, 16-18). In a negative way this supports the hypothesis that a swine-like strain of influenza A virus was responsible for the 1918-19 pandemic since there was no evidence to indicate that the disease occurred in either community. This question is being investigated further during antibody surveys in other Alaskan native communities. In a small Indian village where approximately 20 percent of the population succumbed to influenza in 1918, all of 10 persons born prior to 1922 had swine HI antibodies in titers ranging from 1:40 to 1:320, whereas only 1 of 22 persons born after 1922 had such antibodies.

The frequency of pulmonary complications during the St. Paul outbreak was somewhat greater than that generally observed with Asian influenza (6, 7). However, severity of illness was not comparable to that during the 1918–19 pandemic or during the outbreak of influenza in 1949 among Eskimos of Victoria Island, Canada (19), where 20 percent died. There

was no clinical evidence to suggest that any of the 23 cases of pneumonia in St. Paul were viral in origin or that the epidemic strain was any more virulent than Asian strains encountered elsewhere in 1957.

## **Summary**

Asian influenza outbreaks occurred during October and November 1957 in the remote communities, St. Paul, Pribilof Islands, and Gambell, St. Lawrence Island, Alaska. Introduction of virus into each village could be dated precisely and in each instance was attributed to a returning serviceman.

Two hundred and ninety-four (82 percent) of 358 St. Paul residents and 262 (87 percent) of 300 Gambell Eskimos were ill during the outbreaks. Attack rates were higher among the Aleut than the white residents of St. Paul. Attack rates were higher among children than adults, but the difference was not so great as generally noted with Asian influenza.

The disease was generally mild and clinically similar to influenza noted elsewhere. There were two deaths in St. Paul. Pulmonary complications were somewhat more frequent than generally noted. There was, however, no evidence that such complications were viral in origin or that the epidemic strain of virus was more virulent than observed elsewhere.

Both villages had high prevalence, prior to the outbreaks, of HI antibodies to type A and A' influenza. Distribution of these antibodies by age indicated that type A influenza last occurred in 1943–44 and A' within recent years. Antibodies to the swine strain were infrequent in all age groups, and there was no historical evidence to indicate that influenza occurred in either community in 1918–19. Persons with type A and A' antibodies were as susceptible to illness during the outbreaks as persons without these antibodies. Prevalence of Asian influenza HI antibodies was low before but high after each outbreak.

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