Analyses of rubella epidemics and a measles epidemic in isolated Alaskan communities where these diseases had not been present for a long time are reported. It appears that rubella was at least as infectious as measles. Other aspects are discussed, including second rubella infections.

THE INFECTIOUSNESS OF RUBELLA AND THE POSSIBILITY OF REINFECTION

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A SERIES of rubella epidemics occurred in Alaska in 1963-1964 in isolated Eskimo and Aleut communities in which the disease had been absent for at least 20 years. Details of one of these epidemics appear elsewhere.[1,2] In this communication, an attempt will be made to analyze the epidemiological implications of these outbreaks. Several controversial concepts will be expressed concerning the infectiousness of rubella as opposed to measles and the problem of subclinical and second infections with rubella.

Infectiousness of Rubella and Measles

It is well established that rubella, unlike measles, occurs in epidemic waves at seven- to twelve-year intervals.[3] Age-specific attack rates for the two diseases indicate that rubella appears in older children than does measles.[4] Studies among military recruits and pregnant women revealed that about 20 per cent of these individuals were serologically susceptible to rubella, while almost none were serologically susceptible to measles, and that the clinical attack rate in these groups was much higher for rubella than for measles.[5,6] It may be inferred, therefore, that measles is more infectious than rubella.

Our data on rubella in isolated Alaskan populations, however, strongly suggest that this illness in any given outbreak is about as infectious as measles. In 1963, an epidemic of measles occurred on St. George Island.[7] St. George and St. Paul, the two largest of the Pribilof Islands, are located in the Bering Sea off the coast of Alaska. Figure 1 shows the epidemic curve of the measles outbreak on St. George. The disease had not occurred on the island since 1942, and a serological study of 35 per cent of the island's population prior to the epidemic revealed that no one born since 1942 had measles antibody. The illness was introduced on this occasion by an 11-year-old girl returning from Anchorage. From this primary infection, six contacts developed the disease. It then spread through the community exhausting susceptibles in two waves.

Figure 2 presents the epidemic curve for a rubella outbreak on St. Paul Island.[1] The two epidemics occurred almost simultaneously among identical populations. Rubella had not appeared in St. Paul since 1941 and there were

only three serologically immune individuals born since 1941 in a 90 per cent sampling. In contrast to the St. George measles epidemic, there were six primary cases of rubella among children returning home from boarding school. Following the appearance of these six cases, rubella spread rapidly through the island exhausting susceptibles in two epidemic waves.

The curves on Figure 3 represent the cumulative per cent of cases in individuals born since the previous epidemics. The rubella epidemic spread through the community approximately two weeks more rapidly (or one incubation period) than the measles epidemic on the sister island. Since the rubella epidemic was initiated by six index cases while the measles epidemic was initiated by one individual who spread the infection to six contacts, it is perhaps valid to shift the measles curve one incubation period to the left and, in this instance, the two curves virtually coincide. There is certainly no evidence that measles spread more rapidly than rubella.

A virgin population epidemic of rubella occurred in Anaktuvuk Pass, a northern Eskimo village on the mainland of Alaska (Table 1). In this village, a remarkably rapid spread was noted. From one index case, the entire population was infected within two incubation periods. Individuals listed as having date of onset unknown were unable to determine during which of the two waves they had been ill. They did not become ill after the second wave.

Further evidence concerning the high degree of infectiousness of rubella in our populations was the timing of secondary household cases. Of approximately 100 households in which more than one susceptible individual resided, evidence of failure of rubella to exhaust serological susceptibles after initial contact was present in only two instances. In these households, rubella spread from one sibling to another after two weeks, and then to another two weeks later. In our much smaller series of measles households, all susceptibles were exhausted within one exposure.

Second Infections with Rubella Virus

Although second attacks of rubella have been reported,[8] there is no conclusive proof of their occurrence.[4] Recent reports concerning the ratio of inapparent to apparent attack rates have

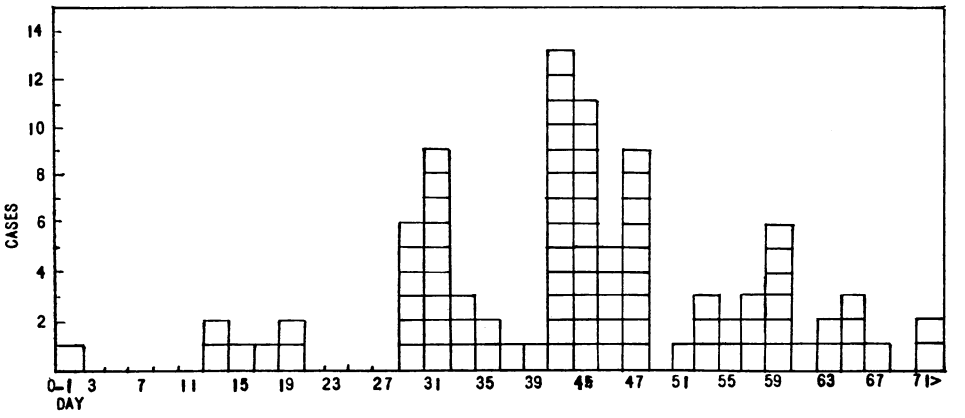

Figure 1—St. George measles epidemic, 1963, epidemic curve

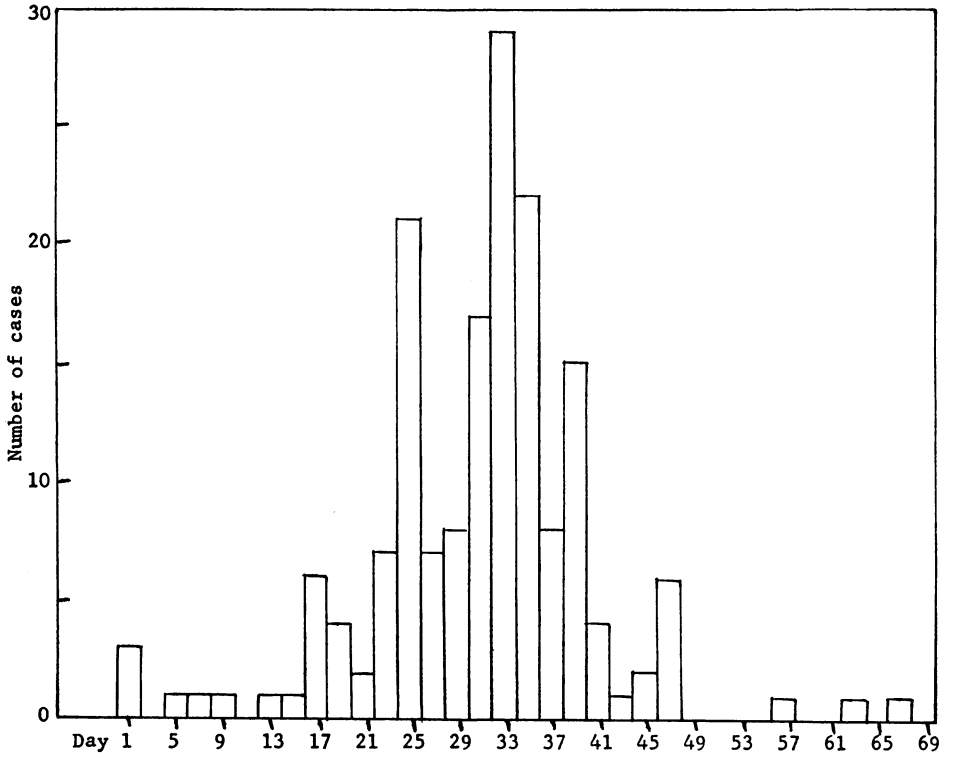

Figure 2—St. Paul rubella epidemic, 1963, epidemic curve

varied from nine apparent cases per inapparent case to one apparent case per seven inapparent cases.[1,9-12] These wide discrepancies suggest the possibility that we are seeing two distinct phenomena, one an inapparent infection in a susceptible individual, and the other a second infection in a person with previous contact with rubella virus. For the purpose of this discussion, the term "true susceptibles" is used to refer to individuals who by history were unlikely to have been exposed to rubella, while "serological susceptibles" refers to individuals who have no demonstrable antibody, but were alive and present during a previous rubella epidemic.

In Krugman's studies at Willowbrook[12] and Horstmann's studies at Yale,[11] the ratio of apparent to inapparent cases was approximately 1:1. These studies were conducted on the East Coast of the United States among young children who were "true susceptibles," since rubella had been absent from this area for seven or eight years prior to the investigations.[12]

In the rubella epidemic previously referred to on St. Paul Island, the disease had been absent since 1941. Virtually all residents under age 21 developed antibody to rubella following the epidemic. Among children under 15 years of age, the apparent to inapparent attack rate was between 1:2 to 1:1. However, among the 57 individuals age 15 to 21, 90 per cent experienced clinical rubella with rash. In addition, these people were sicker than the younger children, the majority having rather severe cases of rubella similar to those which are frequently reported among

pregnant females. It seemed probable that on St. Paul the ratio of apparent to inapparent infections among susceptible adults would be similar to the 9:1 rate we found among those aged 15 to 21, and that symptoms would be relatively severe.

We were surprised, therefore, that in a group of 18 individuals born before 1942 who were serologically negative at the beginning of the rubella epidemic, there were no cases with rash, no recorded illness, and only an occasional finding of posterior auricular lymphadenopathy. All 18 developed antibody during the epidemic.

It is unlikely that rubella on St. Paul Island was increasingly severe in older age groups up to age 21, but suddenly changed in character to become an almost totally inapparent infection in those 22 and over. Significantly, those over age 22 had been present during the 1941 rubella outbreak and fall into the suggested classification of "serological susceptibles."

Buescher's data in military recruits[9] essentially are in agreement with our data for those over age 22. Among military recruits, the majority of whom have lived through a rubella epidemic, 10 to 30 per cent were serologically susceptible to rubella, and among these there appeared only one apparent case to seven inapparent infections. Buescher's evidence for infection among these inapparent cases was indisputable since, in addition to serological conversion, the rubella virus was isolated on several occasions.[10]

While it is possible that Buescher's ratios of apparent to inapparent cases were biased by the fact that rash was not as aggressively searched for as in the other studies and mild cases may have been missed, this could not be the complete explanation for the vast discrepancies of apparent to inapparent infections of 9:1 as opposed to 1:7. Further, it was our impression that the overwhelming majority of St. Paul teenagers, 90 per cent of whom developed clinical illness, were sufficiently sick for at least one day to have made it impossible for them to participate in the rigors of recruit training.

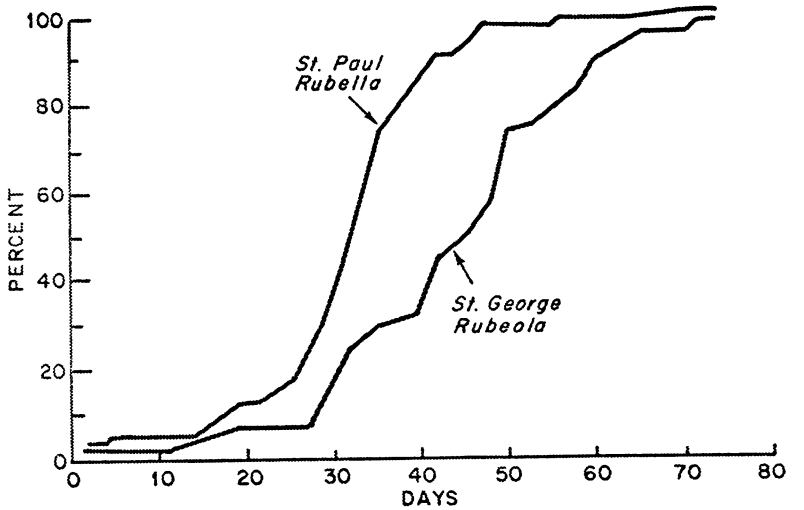

Figure 3—Pribilof Islands—1963, comparison of rubella and rubeola epidemics (cumulative per cent)

Table 1—Anaktuvuk Pass rubella, 1964

	Timing of Clinical Cases					
Week of	Jan. 12	Jan. 26	Feb. 9	Feb. 23	?	Total
Male cases	1	16	14	2	6	39
Female cases	0	7	15	0	14	36
Total cases	1	23	29	2	20	75

Discussion

There can be little doubt from the accumulated world experience that more adults are susceptible to rubella than to measles. This has led to the widespread belief that measles is more infectious than rubella. Our data suggest that this is not the case, since following known exposure rubella spread as rapidly as measles through the community and susceptibles were exhausted at approximately equal rates. It is possible that the discrepancy in age-specific attack rates is more closely related to the peculiarities of the rubella virus which cause it to appear at seven- to twelve-year intervals rather than at the much shorter intervals associated with measles. The causes for cyclic variations in epidemic waves of diseases are poorly understood. To claim that this phenomenon is related only to infectiousness, however, would be hazardous in view of the body of information on periodicity of other respiratory spread viruses such as influenza, smallpox, mumps, and, on occasion, measles.

In populations where possible exposure to rubella occurred some time in the past, about one in five individuals have no antibody by present serological methods of testing.[13] Our data indicate that these serologically susceptible individuals become infected and develop detectable rubella antibody, but rubella symptoms are very mild or totally inapparent. On the other hand, when prior exposure to rubella has not occurred, 90 per cent of older teenagers develop full-blown rubella with rash. It is suggested, therefore, that the majority of those seronegative individuals who have lived through a rubella epidemic may actually possess antibody at a low and undetectable level as a result of previous infection, and upon re-exposure may have a second infection which produces a highly modified form of illness.

The problem arises, does sufficient amount of virus circulate in pregnant females who suffer a highly modified infection to cause fetal damage? Existing data suggest that fetal damage is not caused under these circumstances. Extensive studies by Lundstrom in Sweden[13] and McDonald in England[14] showed that there was no increased risk of fetal damage among pregnant women in households including a child with rubella than the rest of the population unless the woman herself suffered a full-blown case of rubella.

This issue may be resolved during the major epidemic of rubella in the United States in 1964. If one in five pregnant women are without antibody,[6] the risk of fetal damage should theoretically be calculated on the basis of one in five mothers at risk. If, however, the great majority of serologically susceptible adults who have lived through a previous rubella epidemic have partial immunity and do not develop full-blown disease, the rate of fetal damage should be much lower.

Summary

Analysis of rubella epidemics and a measles epidemic in isolated Alaskan communities where these diseases had not occurred for many years indicated that rubella was at least as infectious as measles. Further, inapparent rubella with seroconversion was much more common among those who were alive during the previous epidemic; while among those who were born subsequent to the last epidemic, the rate of apparent illness was much higher. It is possible that these older individuals were partially immune as a result of the prior exposure and the recent episode was a second rubella infection.

ACKNOWLEDGMENT—I wish to thank Dr. John Sever, head, Section on Infectious Diseases, NINDB, who conducted the serologic tests for these studies.

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(This paper was submitted for publication in August, 1965.)