SOCIOLOGIC FACTORS IN THE SPREAD OF EPIDEMIC HEPATITIS IN A RURAL SCHOOL DISTRICT

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(With 4 Figures in the Text)

The virus of epidemic hepatitis is transmitted in a variety of ways which contribute to the complexity of the epidemiology of the disease. Contact or air-borne infection is presumably the most frequent mode of transmission with both droplet and finger transmission as likely mechanisms. Contact accounts for most sporadic and endemic cases of hepatitis, although undoubtedly also for the pandemic waves which were seen two or three times in the past half century, involving most parts of the world. During the pandemic of the early 1920's, well over two hundred localized outbreaks were noted in New York State (Williams, 1923).

Isolated major outbreaks of epidemic hepatitis in interpandemic periods are often due to a common source involving transmission through water, food or other vehicles. However, contact infection is always a possibility for the epidemiologist to consider when called upon to investigate conspicuous outbreaks. The distinction between common source and contact infection obviously is of practical importance, since the former would call for enforcement of environmental sanitation.

The epidemic which is described in this paper occurred in a rural area of Central New York in 1950. It is interpreted as a typical contact epidemic. Extent, attack rate, incidence curve and seasonal appearance are almost identical to those of a hepatitis epidemic which occurred a few years ago in a small urban community in Iowa (Davis & Hanlon, 1946). Water-borne transmission which was a matter of some discussion in the latter epidemic was highly unlikely in the rural area.

The epidemic offered an opportunity to study relationships of attack rates to certain basic social structures of the community, primarily family propinquity. This interest has often been pursued in the study of secondary attack rates in communicable diseases of childhood (Chapin, 1925; Greenwood, 1931; Pope, 1926; Stocks & Karn, 1928; Top, 1935; Wilson, Bennett, Allen & Worcester, 1939), but the analysis is simplified in this instance because susceptibility was almost identical in all age groups, presumably due to lack of recent experience with the disease in the community. Hence, separation of immune family members, which is necessary in studies of measles in families, was of no concern in this study.

THE EPIDEMIC

In the early spring of 1950, the District Health Officer of Geneva, New York, was alerted by an unusual number of reported cases of epidemic hepatitis from Yates County. Cases were mostly from three villages, Rushville, Middlesex and Potter, east of Lake Canandaigua. A preliminary investigation by the Health Officer in March, pointed out that school children were most frequently attacked, although a number of adults and pre-school children had also contracted the disease. There were reasons to believe that the area of the epidemic extended over the district which furnished pupils to the Middlesex Valley Central School situated near Rushville, with an enrolment of well over 500 children.

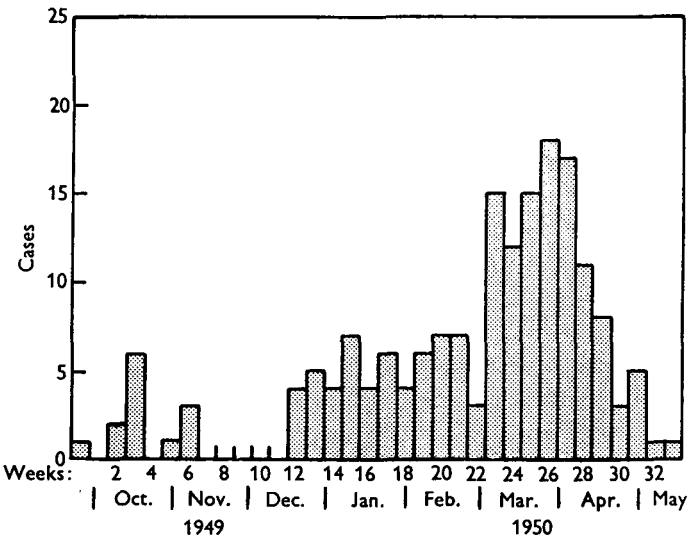

Fig. 1. Distribution by week of 176 cases of epidemic hepatitis in Middlesex Valley Central School District, New York.

Two subsequent field investigations disclosed that 176 cases of epidemic hepatitis had occurred from the last week of September 1949, to the middle of May 1950, when the epidemic subsided. During that period only forty cases had been reported from Yates and Ontario Counties. The weekly number of onsets are presented in Fig. 1.

Clinically, the disease was typical of epidemic hepatitis, and there were no unusual complications. Forty-three of the 176 cases were not jaundiced, and there was no particular sex or age predominance of jaundiced cases. Few other infectious diseases occurred in the area at the time of the epidemic. A few cases of 'flu' presented some problems of differential diagnosis. Prolonged fatigue and malaise were regarded as pathognomonic of non-icteric hepatitis. Cases lasting 1 or 2 days and having no time connexion with cases of hepatitis were not regarded as non-icteric hepatitis. None of the patients died.

METHODS OF INVESTIGATION

The Bureau of Epidemiology and Communicable Disease Control, Albany, New York, assisted the District Health Officer in the detailed investigations. The first survey, on 1 and 2 May 1950, was made to determine the extent of the epidemic over the area. Five public health physicians visited about 175 households along the main roads through the three villages. In each house in which people were at home, household rosters were made listing age and sex of members in the house. Cases of jaundice were noted together with illnesses characterized by nausea, prolonged fatigue, dark urine, and malaise of more than transient nature, with dates of onset and duration. Past histories of jaundice were noted. For each household, information on milk and water supply was collected, and a general impression of sanitary conditions recorded.

This extensive inquiry revealed that most cases occurred in households with school children, and that the geographic extent of the epidemic was roughly that of the Middlesex Central School District. A second survey on 26 and 27 July, aimed at intensive study of all families with children in that school who were known to the school authorities as having had jaundice or illnesses suggesting hepatitis. The point of departure was in this case the school list of absenteeism. Seven investigators (five junior public health interns and two visiting public health physicians) followed the routes of the school buses and made the same inquiries as in the first survey in all households with suspected cases of hepatitis among school children. Several households were visited twice, and some households not on the school's sick list were also visited when neighbours knew of purported cases. A few cases with onset after 1 May were found, but the second survey was strictly retrospective. The number of households visited in the two surveys totalled 226.

A DEMONSTRATION OF CONTAGION

No samples of blood or faeces were taken for laboratory study, but a clear demonstration of the nature of the disease occurred fortuitously as a consequence of the first field study. Two of the physician investigators from Albany developed typical epidemic hepatitis 4–5 weeks after their participation in house-to-house visits on 1 and 2 May. They had no known contact with material or patients of this disease, except their relatively short stay in the homes in Yates County where only conversation with convalescents or their relatives had taken place. Neither water nor food were taken in any of these homes. The second survey reported that a visitor to one of the homes containing cases of hepatitis had stayed for 10 min., only sitting on a chair at the door. Three weeks later he came down with jaundice in his home town, 30 miles away.

TOPOGRAPHY OF THE AREA

The area of the Middlesex Valley Central School district extends over some 30 square miles. The region is typical of the 'Finger Lake' part of Central New York State. Middlesex and Rushville villages are 8 miles apart in a valley parallel to Lake Canandaigua, separated from the lake by a 200–300 ft. high crest, and

a similar crest separates the Middlesex village from Potter village situated 3–4 miles east of Middlesex. East of the road connecting Potter and Rushville, the terrain flattens out in swampy fields. Most houses are farms, except in a few areas in the centres of the villages. The families involved in the epidemic were scattered evenly over most of the area from the slopes down to Lake Canandaigua and to the fields east and south of Potter. No particular focus of high incidence could be distinguished. The water supply was separate for each house, mostly dug wells, and the geography of the region would suggest that at least three different watersheds existed. Investigation of the water supply of the school revealed nothing of epidemiological importance. Routine *coli* tests of the school water five times during the epidemic period were all negative. The milk supply was equally scattered, mainly indigenous to the farms.

THE POPULATION AND GENERAL ATTACK RATE

A delineation of the exposed population is difficult, because cases occurred in parts of three rural townships of which those of Middlesex and Potter are in Yates County, and the third in the adjacent part of Ontario County. The 1940 census has 764 inhabitants in Middlesex and 1109 in Potter township. Children aged 5–14 were 15·4 % of the 1940 census in the two townships. With 466 children aged 5–14 years enrolled in the Central School in 1949, the population concerned would be 3026, which is the best estimate to be had. The overall morbidity based on the 176 cases of hepatitis was then at least 5·7 %.

The 226 households covered by the two surveys comprise 861 persons of whom children aged 5–14 are 196 or 22·8 % (Table 1). This is expectedly somewhat more

Table 1. *Total population in survey, Yates County,* 1949–50
by age, by sex and by households

	Households (226)									
	With cases (85)				Without cases (141)					
	With school children (75)		Without school children (10)		With school children (33)		Without school children (108)		Total	
Age	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
0–4	27	24	2	2	5	8	10	13	44	47
5–9	42	28	—	—	16	11	—	2	58	41
10–14	42	41	—	—	8	6	—	—	50	47
15–24	28	34	1	3	12	7	8	11	49	55
25–34	9	18	3	1	6	11	19	17	37	47
35–44	38	42	—	1	17	14	9	11	64	68
45–54	22	9	3	5	8	7	15	19	48	40
55–64	9	3	2	2	2	1	17	25	30	31
65–74	2	4	—	—	—	4	23	28	25	36
75+	2	4	1	2	3	2	10	14	16	22
Unknown (adults)	—	—	—	—	—	—	3	3	3	3
	221	207	12	16	77	71	114	143	424	437
Average family size	5·7		2·8		4·4		2·4		3·8	

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than in the general population since one of the surveys concentrated on school-children's families. Table 1 shows that households with cases contained a larger proportion of school children. A total of 300 families had children in the school, and 70 of these families had known cases of hepatitis. Of 118 families without school children which were visited at random during the first survey, only 10 had cases. One may, therefore, assume that most existing cases in the area became known through the second household survey, which took issue from the cases among school children reported to the school.

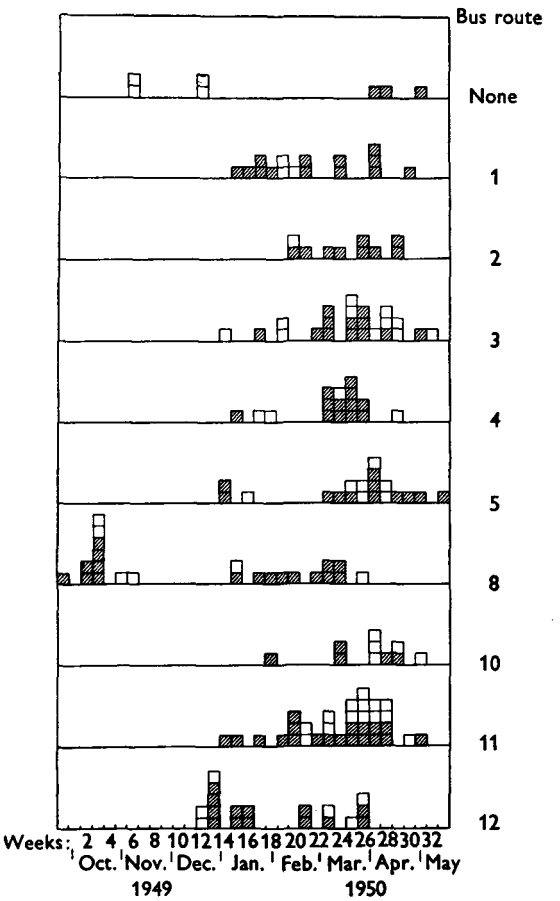

Fig. 2. Weekly cases by neighbourhoods, indicated by school bus route. ('None' are residents near school.) School children ▨; pre-school children and adults, □.

The time pattern of the epidemic suggests certain aggregations of cases in neighbourhoods. The routes of the school buses were chosen as indications of neighbourhoods and areas of closer family contact. Fig. 2 shows that the first cases appeared on bus-route 8 which extended north into Ontario County. The first case (20 September 1949) was a single child in one family. For the next 4 weeks, eight cases appeared in a neighbouring family of 11. There were no cases from the middle of October till the middle of December, when six cases occurred

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in a family in route 5, the south-west part of the region. This family lived on an isolated farm 15 miles south of the school on the top of the crest east of Middlesex Valley. After Christmas 1949, cases appeared all over the region with some clustering around bus-route neighbourhoods.

PREVIOUS HEPATITIS IN THE POPULATION

A physician who had practised in the area for several decades reported that jaundice had last occurred in epidemic proportions in the nineteen-twenties. Inquiry as to previous attacks of jaundice (except those due to gall-bladder disease) during the survey elicited affirmative answers from 35 persons out of 861 (4·1 %). Table 2 shows that these persons were all adults and that the times

Table 2. *Previous cases of jaundice (except due to gall-stones) in the surveyed population, by age, and by 10-year period*

Years of occurrence	Age of persons in 1950								Total	Percentage of persons alive from period
	0–14	15–24	25–34	35–44	45–54	55–64	65–74	75+		
1880–89	—	—	—	—	—	—	0	1	1	1·5
1890–99	—	—	—	—	—	1	1	1	3	2·3
1900–09	—	—	—	—	0	3	1	1	5	2·5
1910–19	—	—	—	1	1	0	3	0	5	1·6
1920–29	—	—	1	2	1	2	1	0	7	1·7
1930–39	—	1	1	0	3	2	0	1	8	1·6
1940–49	0	5	0	1	0	0	0	0	6	0·8
Total	0	6	2	4	5	8	6	4	35	—
Persons	287	104	84	132	88	61	61	38	—	—
Percentage	0	5·8	2·4	3·1	5·8	13·1	9·8	10·5	—	—

of disease were evenly distributed over the past 70 years. In all preceding decades 1–2 % of the population had jaundice, and the accumulation of experience in the high age group is too low to influence attack rates during the recent epidemic. Although the result of this subjective interrogation is of doubtful reliability because it is entirely based on memory, the general result confirms the impression that the epidemic in question can be regarded as an unusual event for the community, and that susceptibility is almost uniform in all ages due to low acquired herd immunity.

AGE SPECIFIC ATTACK RATES

The percentage by age and sex of people attacked is presented in the last column of Table 3 where the rates are presented both for the population of households with one or more cases and for the whole population surveyed. Sex differences in attack rate cannot be demonstrated. The slight predominance of males is hardly significant. The higher attack rates in ages 5–14 are partly explained by the bias of sampling households being chosen because they contained school children with jaundice. One hundred and seventeen cases occurred among the 532 school children, an attack rate (22·0 %) which is similar to that of all other ages in the infected families (23·4 %). In the adult population, the attack rate is about the

same in all ages, and the rate in pre-school children is not significantly different from that in adults.

Table 3. 176 *cases of epidemic hepatitis, Yates County*, 1949–50, *by age, sex and clinical form*

	Males (97)		Females (79)		Attack rate (%)			
					In families with cases		In all families surveyed	
Age	Jaundiced	Not jaundiced	Jaundiced	Not jaundiced	M.	F.	M.	F.
0–4	5	4	5	2	31·1	26·9	20·5	14·9
5–9*	25	7	15	6	76·2	75·1	55·2	52·5
10–14*	27	2	20	3	69·1	56·2	58·0	49·0
15–24	7	2	7	1	31·1	21·6	18·3	14·5
25–34	3	1	5	1	33·3	31·5	10·8	13·1
35–44	6	—	5	5	15·8	23·2	9·5	14·7
45–54	1	1	—	2	8·0	14·3	4·3	5·1
55–64	3	1	—	1	36·4	20·0	13·3	3·2
65–74	—	—	—	—	—	—	—	—
75+	1	1	—	1	66·7	16·7	12·5	4·5
Total	78	19	57	22	41·7	35·4	23·0	18·2

* The apparent high attack rates for ages 5–14 are due to the manner of sampling (see text).

ATTACK RATE IN THE SCHOOL POPULATION

Attention is now given to the conditions in the two major places of contact, the school and the home. The total roster of 532 pupils in the school was studied with respect to age, family grouping and school grades. The attack rate by grades is given in Table 4, and presented graphically in the lower part of Fig. 3. The scatter of these rates, from 6 to 48 %, is somewhat more than expected random variation ($P = 3$ %). The highest attack rate was found in grade 7 with ages 11–12 years.

Table 4. *Cases of infective hepatitis in school children and mean number of siblings in school, by grade, Middlesex Valley Central School, New York*, 1949–50

Grade	No. of pupils	No. of siblings in school*	Cases of hepatitis	Attack rate per 100	No. of siblings in school per child
K	38	57	7	18·4	1·50
1	60	64	13	21·7	1·07
2	57	78	8	14·0	1·37
3	43	62	11	25·6	1·44
4	52	79	12	23·1	1·52
5	38	53	9	23·7	1·39
6	47	72	13	27·7	1·53
7	31	63	15	48·4	2·03
8	51	71	15	29·4	1·39
9	31	47	5	16·1	1·52
10	29	40	2	6·9	1·38
11	35	40	4	11·4	1·14
12	20	26	3	15·0	1·30
	532	752	117	22·0	1·41

* The total number of children in the school who are siblings of the pupils in a given grade.

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The physical environment of class-rooms did not suggest higher degrees of exposure in some rooms, nor is a specific susceptibility in that age group a reasonable assumption. A satisfactory explanation was found by counting the number of siblings which each child had in school. While children in the lowest and highest grades have less siblings in the school, those of the middle grades had the highest number of siblings. The more siblings, the higher is the probability of a communicable disease being introduced into the family. Once the disease is introduced, the family propinquity offers higher exposure than any other social contact. The upper part of Fig. 3 shows the average number of siblings per child of each grade.

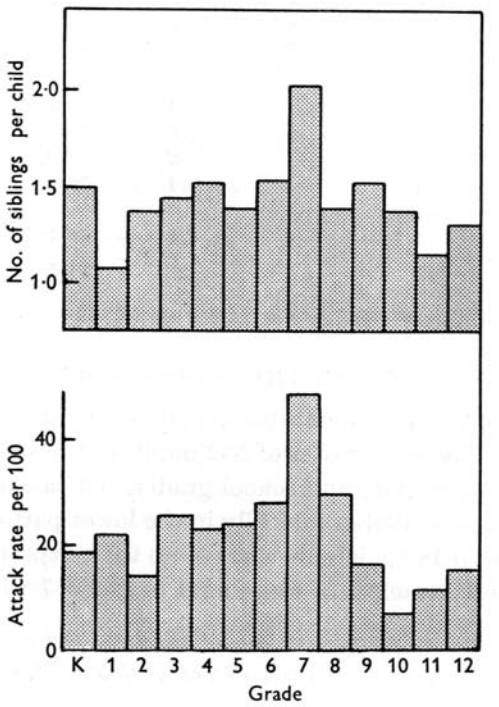

Fig. 3. Attack rates per 100 children in school grade (lower graph), and average number of siblings in school per each child of a given school grade (upper graph).

The distribution conforms with that of the attack rate by grade. Correction for 'school family size' brings the variation of attack rate within the order of expectancy for random variation ($P = 10$ %).

The dependency of attack rate and family size is demonstrated in Table 5 where the 300 families of the 532 school children are distributed according to 'school family size'. Families with one or more cases are in the third column. If the overall chance for one child of contracting hepatitis in the school be p, and the chance of escape be $1-p=q$, then the probability of a family of s children having one or more cases is $1-q^s$. Each of the seven sizes of families observed (Table 5) provides an independent estimate of q and consequently of p. From the one-child families we have, directly, $q = 142/165 = 0{\cdot}861$; $p = 0{\cdot}139$. For the 2-child families, we have $q^2 = 53/69 = 0{\cdot}768$, $q = 0{\cdot}876$ and $p = 0{\cdot}124$. The p's for 3-, 4-, 5-, 6-, and

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7-child families are respectively 0·133, 0·307, 0·242, 0·109 and 1·00. The mean of these seven estimates of p, weighted by the number of families of each size is 0·146. The observed proportion of attacked families is listed in the fourth column of Table 5 with expected frequencies in the sixth column, computed for $p = 0{\cdot}146$. The distribution of expected number of attacked families agrees satisfactorily with that of the observed. Thus, the primary attack rate in families is considered to be 14·6 %, while the higher secondary attack rate within families increases the total school attack rate to 22·0 %.

Table 5. *Number of families of school children with one or more cases*

(Comparison with expectancy according to probability hypothesis where $1-q=p$ is chance of one child being infected in school.)

Size of school family (S)	No. of school families	Families with cases	Proportion of families attacked	Expectancy $1-q^s$ ($p=0{\cdot}146$)	Expected no. of attacked families
1	165	23	0·139	0·146	24·0
2	69	16	0·231	0·270	18·6
3	46	16	0·347	0·376	17·3
4	13	10	0·769	0·467	6·1
5	4	3	0·750	0·545	2·2
6	2	1	0·500	0·611	1·2
7	1	1	1·000	0·668	0·7
Total	300	70	—	—	70·1

ATTACK RATE IN HOUSEHOLDS

With the central school as the major focus of contact, the infection of adults and pre-school children would depend largely on exposure to infected school children. The attack rates in these two categories are presented in Table 6, from which it is evident that attack rates of the family members not school children depend on whether or not there are diseased school children in the family. Attack rates in families without school children do not differ significantly from those in families with school children without known hepatitis. The attack rate in these two categories of families indicates the risk of infection in the community outside the school, which is about 5 %, contrasted to the risk in the school which was estimated to be 14·6 %. However, four of the 17 cases in families with no sick school children were school teachers who presumably were as much exposed as the pupils. Four of twenty-five teachers were attacked, a rate of 16 % which is identical with the primary rate of the pupils. The rate for all of the forty-four adult school employees was 5/44 or 11·6 %.

Table 6. *Attack rate among pre-school children and adults by family exposure to school children*

	Families without school children	Families with school children	
		No sick children	Sick children
Pre-school	2/29 (6·9 %)	1/22 (4·5 %)	12/52 (23·1 %)
Adults	9/256 (3·5 %)*	5/93 (5·4 %)*	30/176 (17·0 %)†
	11/285 (3·9 %)	6/115 (5·2 %)	42/228 (18·4 %)

* Two cases were school teachers.

† One case was a school secretary.

MULTIPLE FACTORS IN HOUSEHOLD INFECTIVITY

The classical technique for study of the infectivity of a contagious disease is the evaluation of secondary attack rates. The method relies on accuracy in observation of dates of onset, so that primary cases can be distinguished from secondary cases on the basis of the recognized incubation period of the disease. In this study, the majority of the case data were collected retrospectively and mainly based on lay peoples' memory of events 2–9 months back. Although it was fairly well established who had had jaundice in a family, it happened that there was difficulty in recalling the sequence of family cases.

The selection of primary cases could not with any degree of accuracy be based on time data. Working from the hypothesis that the school was the principal area of infection, school children with the disease would essentially be considered as primary or introductory cases, and adults and pre-school children of these homes as secondary cases. Thus, the criterion for a primary case is changed from a time observation to a sociological attribute. Secondary cases are persons without direct contact with the infected area.

Pre-school children and adults are equally susceptible, since attack rates of the two groups, as shown in Table 6, are practically identical.

The 'secondary' attack rate, defined as the attack rate among pre-school children and adults, is determined by several factors which are more or less mutually interdependent. Three such factors were selected and the secondary attack rate related in a multiple regression analysis based on the date of Table 7. One causative factor is the number of sick school children, x, in the family, the second is the total family size, s, and thirdly, the hygienic standard in the household was based on a statement of the overall impression of cleanliness, waste disposal, appearance of the family, etc. The hygienic condition, z, was classed in three categories 'good', 'fair' and 'poor', which were assigned numerical values of 0, 1 and 2 respectively.

The attack rates (measured in probits of per cent, y) were significantly correlated to each of these single factors; the correlation coefficients for attack rates and number of sick children, x, being 0·825, attack rates and family size, s, 0·815, and attack rates and hygiene, z, 0·587. There is, however, strong interdependence between the three factors, especially between x and s, since expectedly the larger families will have more school children infected. Also, hygienic standard will to some extent depend upon family size, although there were several exceptions. Clean houses with six children might escape the further spread of infection, even after two children came home with the disease, or one child would infect all adults in a home with low sanitary standards. A relative weighting of the three factors was obtained by multiple regression analysis which resulted in the following equation for expected attack rate

$$Y = 3{\cdot}02 + 0{\cdot}242x + 0{\cdot}071s + 0{\cdot}187z,$$

where Y is probit of expected attack rate, and x, s and z are given above. A similar technique was used in a study of factors influencing attack rates for streptococcal

infections among student nurses (Ipsen, 1950). The agreement between observed and expected attack rates conforms with random variation. The independent action of the three factors is given by the weighting coefficients of this equation. Thus, addition of one infected child (increase of x) will influence the attack rate as much as would increase of family size, s, by three members. Lowering in hygienic standard, z, by one unit has somewhat the same effect as introducing one more primary case.

Table 7. *Attack rate among pre-school children and adults in households by exposure to school children, by family size and by hygienic condition in household*

No. of sick school children (x)	Household hygiene (z) Good (0) Cases Persons	Family size (s)	Fair (1) Cases Persons	Family size (s)	Poor (2) Cases Persons	Family size (s)	Total Cases Persons	Family size (s)
0*	8/182 (4·4 %)	2·4	3/103 (2·9 %)	2·5	—	—	11/285 (3·9 %)	2·4
0†	3/61 (4·9 %)	4·6	3/48 (6·3 %)	4·7	0/6	4·5	6/115 (5·2 %)	4·7
1	2/80 (2·5 %)	4·7	10/50 (20·0 %)	5·6	2/12 (16·7%)	5·3	14/142 (9·9 %)	5·1
2	3/9 (33·3 %)	4·5	0/7	6·5	5/20 (25·0 %)	6·5	8/36 (22·2 %)	5·8
3	1/6 (16·7 %)	6·5	6/18 (33·3 %)	6·8	6/11 (54·5 %)	7·7	13/35 (37·1 %)	7·0
4	—	—	—	—	3/6 (50·0 %)	7·5	3/6 (50·0 %)	7·5
5	—	—	—	—	0/3	10·0	0/3	10·0
6	—	—	4/6 (66·7 %)	12·0	—	—	4/6 (66·7%)	12·0
Total	17/338 (5·0 %)	3·4	26/232 (11·2 %)	4·0	16/58 (27·6 %)	6·5	59/628 (9·4 %)	3·8

* No school children in household. † No sick school children.

CLINICAL FORM AND DOSAGE

Of the 176 cases of hepatitis observed in the survey, 41 were without jaundice. Presumably, a diagnosis in the latter group is less definite than for jaundiced cases, and more cases of hepatitis without jaundice might have existed in the area. Fifteen per cent of the cases among school children were not jaundiced, while 23 of 59 cases among non-school persons (or 39 %) were of that clinical form. There was no significant difference in the distribution of clinical form in pre-school children and in adults. It is suggested that the higher exposure which school children sustained explains the higher proportion of jaundiced cases, but selectivity due to use of school children as index cases cannot be excluded. The hypothesis of exposure as a determinant for clinical form is supported by the distribution of clinical form among pre-school and adult persons, by degree of exposure to school

children (Table 8, Fig. 4). In spite of small numbers within each class of exposure, the increase in relative numbers of jaundiced cases with higher exposure is beyond doubt. This phenomenon suggests the importance of dose, not only in determining probability of infection, but also in determining degree of illness among those infected.

Table 8. *Clinical type of hepatitis in pre-school children and adults, by degree of exposure*

Exposed to	Cases	Jaundiced	Not jaundiced	Percentage jaundiced	Total attack rate
No school children	11	3	8	27	3·9
No sick school children	6	2	4	33	5·2
1 sick school child	14	7	7	50	9·9
2 sick school children	8	5	3	63	22·2
3 sick school children	13	12	1	92	37·1
4–6 sick school children	7	7	0	100	46·7
Total	59	36	23	63·9	9·4

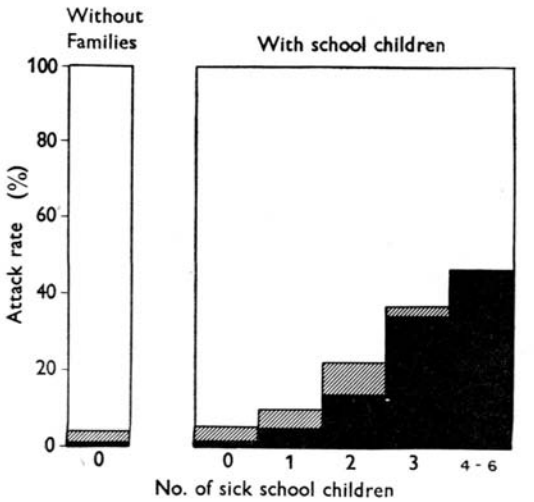

Fig. 4. Attack rates among pre-school children and adults by number of school children in household with hepatitis. Cross hatched: proportion without jaundice, ▨; proportion with jaundice, ■.

DISCUSSION

The argument for disregarding a common source of infection in this epidemic of hepatitis is established indirectly. First, no such source as water or food could be found by routine epidemiologic examination. Secondly, the form of the epidemic curve (Fig. 1) is not one usually associated with a vehicle-borne outbreak, where one would expect an early peak and a prolonged fall. The extent of the epidemic over a period several times the incubation period of epidemic jaundice points to contact transmission.

Direct proof of contact infection is difficult to establish where the infective agent is not readily demonstrable. The hypothesis was substantiated by showing that attack rates were highest where human contacts were maximum. The incidence among school children was highest among those who had the highest number of

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contacts, i.e. the highest number of siblings. It was shown that the general attack rate in the school was higher than expected if all children had been equally exposed. The incidence among family contacts outside the school also conformed with a hypothesis of person-to-person contact, the most important factor being the number of infected school children. Other sociological factors such as crowding and hygiene also had some influence, although each of them was highly dependent on the number of sick school children.

An attempt was made to enter these three factors in an expression for expected attack rates. Such attempts are, assuredly, rather theoretical. The equation expresses expectancies on the assumption that one could arrange epidemiological experiments with humans in which three variables 'dosage', 'group size' and 'transmission favorability' could be changed at will. It is more realistic, however, to recognize that in human communities, large families are less hygienic, and the larger the family the more school children will probably be infected. Nevertheless, an effort to quantitate observations and a study of their relative values is preferred to a vague narrative account of the epidemiologic importance of 'socio-economic factors'.

Although it is believed that sufficient evidence is brought out to explain this epidemic on the basis of contact transmission from house to house, some vehicle transmission could have occurred within the home. Indeed, it is highly probable that the high attack rate in homes of low hygienic standard could have been caused by contamination of food or well water. Probably because of the isolation of the families in this rural community, such vehicles were in no instance shown to transmit infection outside the family.

While the spread and extent of this epidemic were largely due to sociological factors, such as family grouping and size, and housing, the genesis of the epidemic is also explained by sociologic circumstances. The reasons why this rural area had a considerable epidemic of hepatitis are first, that the remoteness of the community over a number of years had favoured accumulation of susceptibles and secondly, that a modern educational development created possibilities of more intense contact. The construction of the consolidated school 10 years before the epidemic led to an aggregation of susceptible persons, which did not exist at the time when the children of the several villages met in one-room school-houses, still to be seen scattered around the area.

SUMMARY

An epidemic of epidemic hepatitis occurred in the district of a rural Central School in Yates and Ontario Counties of Central New York. The epidemic comprised 176 cases of which 135 were jaundiced, and extended from September 1949 to May 1950, with most cases in March 1950.

No common source could be demonstrated and the transmission was believed to be due to human contact alone.

The overall morbidity rate was about 5 %, attack rate among school children 22 %, and among the adults and pre-school family contacts of infected school children the attack rate was 18 %, with no significant age difference in attack rate.

Differences in attack rate in the various school grades were explained by higher number of siblings of children in certain grades.

Household attack rates among non-school persons were related to number of sick school children, family size, and hygienic standard of the home. The first of these factors seemed most important.

More jaundiced cases occurred in families where the exposure was heaviest.

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