Epidemiol. Infect. (1997), **118**, 243–252. Printed in the United Kingdom © 1997 Cambridge University Press

The role of young children in a community-wide outbreak of hepatitis A

P. F. SMITH[1, 2]*, J. C. GRABAU[1], A. WERZBERGER[3], R. A. GUNN[4], H. R. ROLKA[4], S. F. KONDRACKI[1], R. J. GALLO[1] AND D. L. MORSE[1, 2]

[1] *Division of Epidemiology, New York State Department of Health, Albany, NY*
[2] *Department of Epidemiology, School of Public Health, State University of New York at Albany, NY*
[3] *Department of Pediatrics, New York Medical College, Valhalla, NY*
[4] *Epidemiology Program Office, Centers for Disease Control and Prevention, Atlanta, GA*

(*Accepted 11 December 1996*)

SUMMARY

An Hasidic Jewish community has experienced recurrent hepatitis A outbreaks since 1980. To assess risk factors for illness during a 1985–6 outbreak, the authors reviewed case records and randomly selected 93 households for an interview and serologic survey. In the outbreak, 117 cases of hepatitis A were identified, with the highest attack rate (4·2%) among 3–5 year olds. Among the survey households, the presence of 3–5 year olds was the only risk factor that increased a household's risk of hepatitis A (indeterminant relative risk, $P = 0{\cdot}02$). Furthermore, case households from the outbreak were more likely to have 3–5 year olds than were control households from the survey (odds ratio = 16·4, $P < 0{\cdot}001$). Children 3–5 years old were more likely to have hepatitis A and may have been the most frequent transmitters of hepatitis A in this community. Hepatitis A vaccination of 3–5 year olds can protect this age group and might prevent future outbreaks in this community.

INTRODUCTION

Hepatitis A continues to cause significant morbidity throughout the world, including developed countries. In the United States, although the incidence has decreased over the past several decades, over 26000 cases were reported in 1994 [1], but because of incomplete reporting, the actual incidence is probably 4–5 times higher than this number [2]. The national cost of the disease has been estimated to exceed $200 million per year [3]. Whereas children, especially under the age of 2 years, are often asymptomatic when infected with hepatitis A virus (HAV), adults with HAV infection usually develop overt symptoms of hepatitis [4]. Approximately 50–60% of cases are associated with recognized risk factors, including contact with a known case, employment or attendance at day-care centres, injection drug use, recent international travel, or association with a food or waterborne outbreak [5]. Large community-wide outbreaks of hepatitis A continue to occur in developed countries [6–9], largely through person-to-person transmission. Such outbreaks are difficult to control, often continuing for many months despite the use of immunoglobulin (IG) for close contacts of known cases [6, 8].

The recent development of inactivated and live-attenuated hepatitis A vaccines has raised the hope of reducing hepatitis A incidence. With hepatitis A vaccine already licensed in several countries, including the United States, much consideration is being given to which groups to vaccinate. In addition to universal childhood vaccination, proposals have included targeting vaccine to travellers to hepatitis A endemic regions, military personnel, children and staff in day

* Address all correspondence and requests for reprints to: Division of Epidemiology; Room 503, Corning Tower, ESP; Albany, New York 12237-0608.

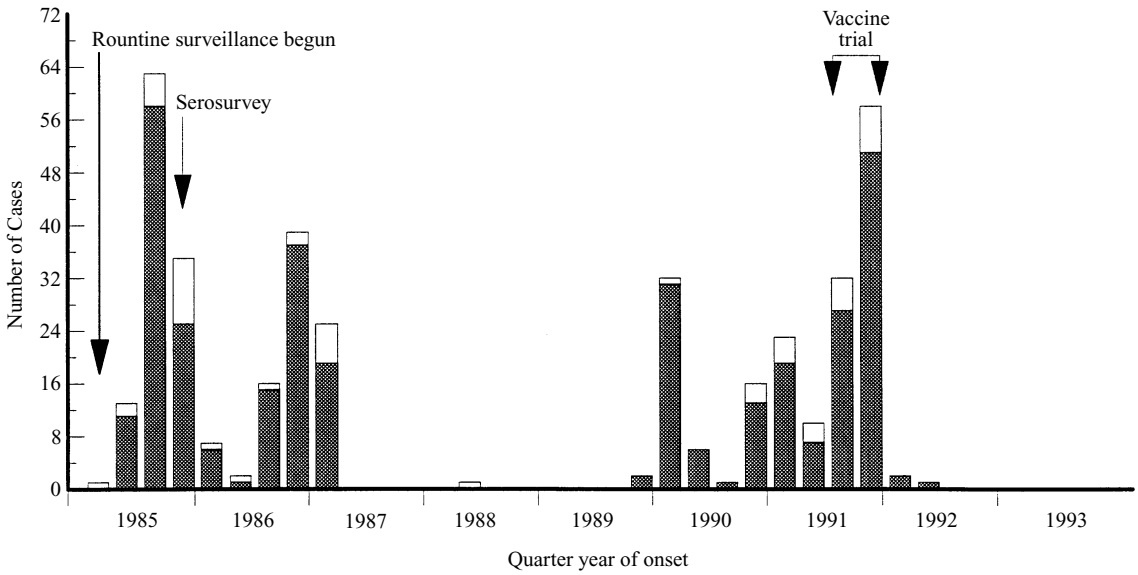


Fig. 1. Clinical cases of hepatitis A by onset date and age, Kiryas Joel, New York, 1985–93 (Excludes 39 cases with unknown dates of onset and two cases occurring during the 1985–6 study period with unknown ages). Cases: ▩, ⩽ 16 years old; □, > 16 years old.

care, persons in selected occupations (e.g. food handlers, sewage workers), and members of frequently affected communities [10, 11].

The village of Kiryas Joel, New York, has been affected by recurrent community-wide outbreaks of hepatitis A and was the site of a successful 1991 vaccine trial [10]. This report describes one of these community outbreaks and the results of a serologic survey that was performed during the outbreak. The objectives of the survey were to determine the susceptibility of the population to hepatitis A, to identify subclinical cases of hepatitis A, and to assess risk factors for infection, all with the goal of better understanding the transmission pattern of HAV in this community.

Background

Kiryas Joel is located 40 miles north of New York City in a residential section of Orange County. Composed entirely of Satmar Hasidic Jewish residents, whose beliefs do not permit contraception, the village is characterized by very rapid growth resulting from a high birth rate, with an average family size of more than six members. Adding to the rapid population growth are the many families that have moved to Kiryas Joel from older Hasidic communities in Brooklyn and Rockland County, New York, and from Europe, Canada, and the Middle East. From the founding of the village in 1972, the population of Kiryas Joel grew to approximately 2000 in 1980, 5200 in 1985, and 9600 in 1992. Close social and economic ties have been maintained with the Brooklyn Hasidim. The economy of Kiryas Joel is supported by the working adult males, most of whom commute daily to jobs in New York City. The village, which remains culturally isolated from the surrounding communities, strictly adheres to religious and social traditions, which include one unified but sexually-segregated village school system.

Kiryas Joel has had two large hepatitis A outbreaks since 1985 when routine surveillance for hepatitis was instituted (Fig. 1). One outbreak occurred in 1985–7 and the second in 1989–92. Anecdotal reports also suggest the occurrence of an earlier outbreak in 1980. Most cases since 1985 occurred in children 16 years of age or younger. Each outbreak lasted about 2 years, involving well over 100 persons in the community, and each was followed by a period when virtually no cases occurred. There has been no evidence to suggest that these outbreaks resulted from a common-source exposure. For example, the community water for Kiryas Joel is supplied by three deep village wells, is chlorinated, and has always met drinking standards. Food is prepared at individual homes except for school lunches which are prepared at one location and distributed to the village schools. There have been no cases of hepatitis A identified among food workers. There is no organized infant day-care system; children

remain at home until age $2\frac{1}{2}$ years when they start school.

The first large outbreak of hepatitis A was recognized in mid-1985 by the only paediatrician (author AW) with an office in Kiryas Joel. He provides most of the paediatric medical care to the community and maintains close rapport with the residents, many of whom do not speak English. During the early months of this outbreak, public health workers and local physicians aggressively promoted control measures, including educating residents regarding proper hygiene to decrease the transmission of HAV, as well as administering IG to family and neighbourhood contacts of cases. By September 1985, it was apparent that the outbreak was continuing despite these measures, and the authors undertook the serologic survey that is the subject of this report.

METHODS

Surveillance

From April 1985 through January 1986, the authors conducted intensive surveillance (in addition to routine surveillance) for cases of hepatitis A by reviewing county health department surveillance reports and contacting the four physicians who provided the majority of medical care to the residents of Kiryas Joel. For each reported case, information was obtained regarding the date of onset of symptoms, age, sex, and address. A clinical case of hepatitis A during this period was defined as physician-diagnosed hepatitis A in a resident of Kiryas Joel. In addition, cases had to have either immunoglobulin M antibody to HAV (IgM anti-HAV) or close contact with an IgM anti-HAV positive case. Age-specific attack rates were calculated by using census data from a comprehensive, computerized village directory that was maintained by the community's school system. Residents were grouped into the following age categories: 0–2 years (children not yet in school), 3–5 years (young school children), 6–9 years (older school children), 10–16 years (pre-adolescents/adolescents), and $>$ 16 years (older adolescents and adults, grouped together since age-specific population estimates for this group were not available).

Serologic survey

For the serologic survey, 93 households (11·5% sample) were randomly selected from the 809 households listed in the village directory. The sample size was determined primarily by feasibility (i.e. the number of participants that could be interviewed and bled with available resources). Members of the selected households were encouraged to participate in the study both by a personal letter from the paediatrician in the community and by a telephone call from his nurse. Following a protocol approved by the state Department of Health review board and after written informed consent, the authors asked participants to complete a questionnaire which included the following information: age, sex, length of residence in Kiryas Joel, former place of residence, type of dwelling (single *vs.* multi-family), history of hepatitis A or any contact with someone with hepatitis A other than a household member, and presence of diapered children in the home. During scheduled appointments between 17 November and 6 December 1985, the authors reviewed the questionnaire information with an adult household member, and a blood sample was drawn from household members who were over the age of 1 year. The blood samples were tested by the New York State Health Department's Wadsworth Center for Laboratories and Research for total antibody to HAV (anti-HAV) and IgM anti-HAV using commercially available radioimmunoassay test kits (HAVAB and HAVAB-M kits, Abbott Laboratories, North Chicago, IL).

To assess risk factors for hepatitis A infection in survey households, the authors compared households with at least one member with IgM anti-HAV (serologic case households) with the remaining survey households without IgM-positive members. For analysis purposes household members were considered to have been susceptible to hepatitis A during the outbreak if they lacked anti-HAV or had IgM anti-HAV, indicating recent HAV infection.

It was not possible to identify the index (first) case of hepatitis A within each household because the timing of infection among household members with IgM anti-HAV could not be determined from the serologic survey. Therefore, patterns of person-to-person transmission could not be precisely studied. However, as an indirect measure of intrahousehold transmission, an analysis using individuals as the unit of observation was performed, by assessing the association between serologic cases, defined as survey participants with IgM anti-HAV, and household exposure to other children with IgM anti-HAV. Three separate analyses were performed to assess exposure to children with IgM anti-HAV in three age groups: 1–2 years, 3–5 years, and 6–9 years.

Case-control study

Because information on the age and number of household members was available for clinical cases reported through surveillance during the outbreak, a case-control household study was also performed to determine if the presence of different age groups of children was associated with hepatitis A in households, after controlling for household size. Households in which clinical cases had occurred between April 1985 and January 1986 and for which information was available regarding age of children and family size were defined as surveillance case households. Control households were those households from the serologic survey that had at least one member susceptible to hepatitis A (i.e., negative anti-HAV) but none with IgM anti-HAV.

Statistical methods

Significance testing for the crude estimation of risk factors for hepatitis A and for subgroup differences was performed using either the χ^2 2-tailed Fisher's exact, or student's t test with Epi Info software [12]. For multivariable analysis of risk factors, interactions and confounding, logistic regression was performed using the Statistical Package for Interactive Data Analysis [13]. Final preferred models were derived by sequentially eliminating non-significant variables as shown by the P-values for Wald's criterion and the likelihood ratio test statistic. Potential confounding was assessed during the model fitting process by observing changes of magnitude in effect measures for variables remaining in the model when other variables were removed.

RESULTS

Results of surveillance

From surveillance investigations, 117 (2·3%) persons were identified who met the clinical case definition among the 5200 residents of Kiryas Joel. Eighty-two (10·1%) of the community's 809 households had at least one member with hepatitis A. The male-to-female ratio for cases was nearly equal (1:1·2), although 11 of the first 13 cases in the outbreak were male. For the 115 cases with known age, ages ranged from 16 months to 36 years, with a mean of 10·4 years and a median of 8 years. The attack rate was highest in the 3–5 year age group (4·2%) and declined to 1·0% among those greater than 16 years old (Table 1). There was no apparent geographic clustering within the community overall or when cases were examined by one-month time periods, or within classrooms in the school system.

Results of serologic survey

For the serologic survey, 53 of the 93 selected households participated (57·0% response rate). Participating households were larger on average (mean, 7·3 members) than non-participating households (mean, 5·3 members), ($P = 0{\cdot}005$). This difference was due to the lack of participation of 17 of the 23 households without children. If only the households with children (less than 16 years old) are considered, the mean number of members in participating and non-participating households was nearly equal at 8·0 and 7·8, respectively. The male-to-female ratios within participating (1·3:1) and non-participating (1·1:1) households were similar ($P = 0{\cdot}22$). The mean age of children 16 years or younger was 7·1 years for participating households and 7·5 years for non-participating households ($P = 0{\cdot}40$).

Within the 53 participating households, there were 375 members over the age of 1 year and thus eligible for serologic testing for anti-HAV. Of these, 341 (90·9%) provided blood samples for testing. The mean age of participating members was 15·3 years, compared with 18·6 years for non-participating members ($P = 0{\cdot}19$). The male-to-female ratio was 1·1:1 for participating members and 1·8:1 for non-participating members ($P = 0{\cdot}22$).

Overall, 147 (43·1%) of the participants had antibody to HAV. The seroprevalence among males and females was similar (41% *vs.* 45%, respectively). The prevalence of total antibody increased with age, from 10% in 1–2 year olds to 100% in those 40 years and older ($P < 0{\cdot}001$, χ^2 test for linear trend (Fig. 2). Seventeen persons from 10 households had IgM anti-HAV: only 3 had symptoms consistent with hepatitis A during the previous 6 months. Sixteen of the 17 IgM anti-HAV positive persons were under the age of 10 years, and 9 were in the 3–5 year age group. Seven were male.

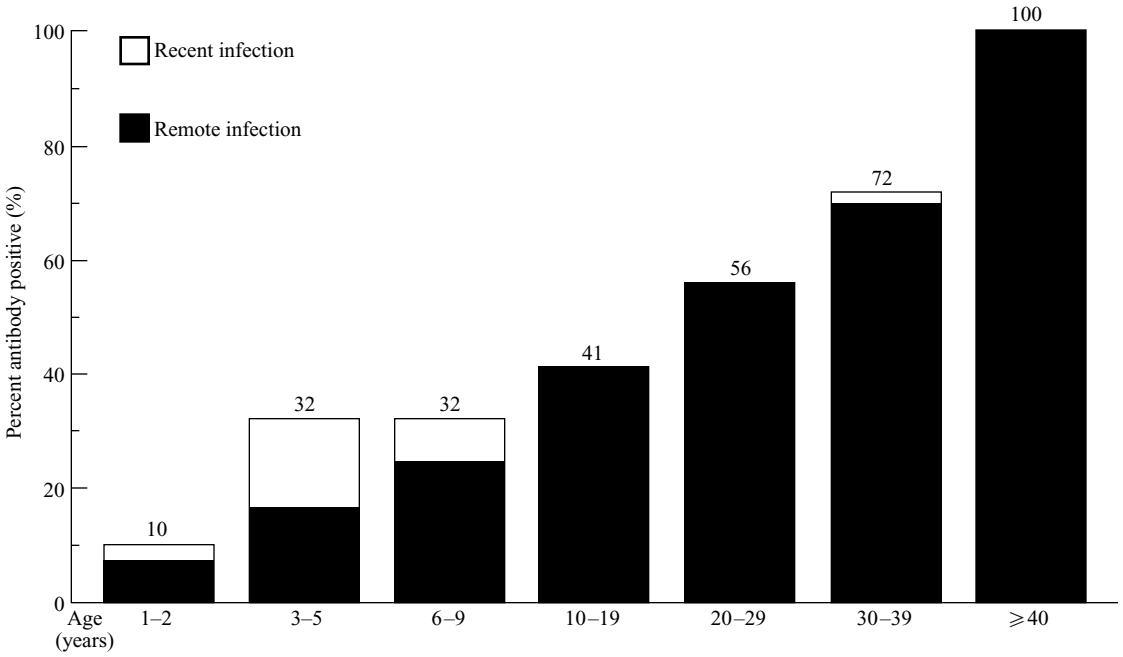
The age-specific attack rates for HAV infection among recently susceptible children are shown in Table 2. Three-to-five year old children had the highest estimated attack rate (19·1%), which was five times greater than the rate among children 1–2 years old.

https://doi.org/10.1017/S0950268897007462 Published online by Cambridge University Press

Table 1. *Estimated attack rates for clinical cases of hepatitis A by age group, Kiryas Joel, New York, 1985–6*

Age (years)	Number of clinical cases	Estimated population*	Attack rate (%)
0–2	9	692	1·3
3–5	29	692	4·2
6–9	32	960	3·3
10–16	28	1118	2·5
> 16	17	1738	1·0
All ages	117†	5200	2·3

* Community-based census data.
† Total includes two cases of unknown age.



Number of persons with recent and remote infection

Recent	1	9	6	0	0	1	0
Remote	2	9	18	35	15	37	14
Total tested	29	56	76	86	27	53	14

Fig. 2. Prevalence of class-specific antibody to hepatitis A virus by age group during an outbreak of hepatitis A, Kiryas Joel, New York, 1985. Recent infection [positive immunoglobulin M antibody to hepatitis A virus (IgM anti-HAV)]; Remote infection (positive antibody to hepatitis A virus but negative IgM anti-HAV).

Several potential risk factors for HAV infection were assessed among the 53 survey households. The crude summary statistics appear in Table 3. Households with children 3–5 years of age were at significant risk of having an IgM anti-HAV positive person. None of the other variables in Table 3 significantly increased a household's risk. In addition, the impact of place of prior residence appeared to be minimal: of the 47 households that had moved from Brooklyn or Rockland County, New York, 8 (17·0%) were serologic case households, compared with 1 (20·0%) of 5 households that had moved from Belgium, Canada, or Israel. One household was of unknown previous residence.

Multivariable analysis of risk factors for hepatitis A among the 53 survey households resulted in a regression model that included regression terms for the presence of 3–5 year old boys, 3–5 year old girls, and diapered children. Since there were no serologic case households that did not have at least one 3–5 year old, the analytic problem of 'quasicomplete separation' (limited overlap in the distribution of the

https://doi.org/10.1017/S0950268897007462 Published online by Cambridge University Press

Table 2. *Estimated attack rates for hepatitis A virus infection among recently susceptible children by age group, Kiryas Joel, New York, 1985*

Age (years)	Number tested	Number susceptible*	Number IgM positive	Attack rate (%) among susceptibles	RR† (95% confidence interval)	*P* value
1–2	29	27	1	3·7	1·0 (reference)	—
3–5	56	47	9	19·1	5·2 (0·7–38·6)	0·08
6–9	76	58	6	10·3	2·8 (0·4–22·1)	0·42

* A child was considered recently susceptible if the child lacked antibody to hepatitis A virus (HAV) or tested positive for immunoglobulin M antibody to HAV.
† Relative risk.

Table 3. *Crude analysis of potential risk factors for the presence of immunoglobulin M antibody to hepatitis A virus (IgM anti-HAV) in one or more household members, among 53 households in the serologic survey, Kiryas Joel, New York, 1985*

	Households with risk factor			Households without risk factor				
Potential risk factor	Serologic case households*	Total	AR† (%)	Serologic case households	Total	AR† (%)	RR‡ (95% confidence interval)	*P* value
> 5 Household members	9	38	23·7	1	15	6·7	3·6 (0·5–25·7)	0·25
Household member(s)								
Aged < 3 years	7	34	20·6	3	19	15·8	1·3 (0·4–4·5)	1·00
Aged 3–5 years	10	37	27·0	0	16	0·0	Indeterminant	0·02
Aged 6–9 years	9	39	23·1	1	14	7·1	3·2 (0·5–23·3)	0·26
Diapered child(ren) in household	6	29	20·7	4	24	16·7	1·2 (0·4–3·9)	1·00
> 50% of household members susceptible to hepatitis A§	8	32	25·0	2	21	9·5	2·6 (0·6–11·2)	0·28
Residence in community less than 10 years	9	42	21·4	1	11	9·1	2·4 (0·3–16·7)	0·67
Household member(s) with contact with hepatitis A case	5	24	20·8	5	29	17·2	1·2 (0·4–3·7)	1·00
Residence in multifamily dwelling	8	46	17·4	2	7	28·6	0·6 (0·2–2·3)	0·60

* Defined as those households with at least one member with IgM anti-HAV.
† Attack rate.
‡ Relative risk for households with risk factor compared to those without risk factor.
§ Household members were considered susceptible to hepatitis A during this outbreak if they lacked antibody to hepatitis A or had IgM anti-HAV.

covariates in the model) had to be addressed in order to perform the logistic regression [14]. Sex categories for the 3–5 year age group were used so that adjusted odds ratios could be obtained since some serologic case households did have only 3–5 year old girls or boys, respectively.

Only the presence of 3–5 year old boys [odds ratio (OR) 9·9, 95% confidence interval (CI) 1·3–76·9)] and of 3–5 year old girls (OR 8·1, 95% CI 1·3–52·5) was strongly associated with hepatitis A infection. The odds ratio for the presence of diapered children was 0·2 (95% CI 0·03–1·3). Effect estimates (odds ratios) for 3–5 year old boys and 3–5 year old girls increased by about 45% when analyzed with the diapered-children variable in the model compared to when they were fit to a model without controlling for the

https://doi.org/10.1017/S0950268897007462 Published online by Cambridge University Press

Table 4. *Crude analysis from the case-control study of the presence of household members by age group as a risk factor for hepatitis A, among 63 surveillance case households* and 40 control households,† Kiryas Joel, New York, 1985*

Potential risk factor	Surveillance case households with risk factor		Control households with risk factor		OR‡ (95% confidence interval)	*P* value
	Number	Percent	Number	Percent		
Household member(s) aged < 3 years	48	76·2	26	65·0	1·7 (0·7–4·5)	0·31
Household member(s) aged 3–5 years	61	96·8	26	65·0	16·4 (3·3–154·6)	< 0·001
Household member(s) aged 6–9 years	55	87·3	29	72·5	2·6 (0·9–8·1)	0·10

* Surveillance case households were those households in which clinical cases had been reported through surveillance and for whom information about the ages of children and household size was available.
† Control households were those households in the serologic survey with at least one member susceptible to hepatitis A but none with immunoglobulin M antibody to hepatitis A virus.
‡ Odds ratio.

presence of diapered children. This is suggestive of confounding since young children before toilet training are known to be associated with HAV transmission within households [4] and diapered children were associated with the household presence of 3–5 year old children in this study. The proportion of households that had a 3–5 year old among those with diapered children was 0·86, compared with 0·54 for households without diapered children. Therefore, the presence of diapered children was controlled for as a confounder in the regression model. None of the following factors was predictive of HAV infection or found to contribute to the model: number of household members (measured as actual count), presence of boys and presence of girls in each of two age groups (0–2 years and 6–9 years), history of a household member's contact with a case of hepatitis A, and the percentage of household members susceptible to hepatitis A.

In the analysis that used individuals as the unit of observation, the risk of having IgM anti-HAV was highest among those with household exposure to 3–5 year olds with IgM anti-HAV [relative risk (RR) 13·6, 95% CI 3·5–53·2]. In comparison, the relative risk from exposure to 1–2 year olds with IgM anti-HAV was 6·9 (95% CI 1·6–30·1), and to 6–9 year olds with IgM anti-HAV was 5·0 (95% CI 1·6–15·7).

Results of case-control study

To further assess age groups of household members as risk factors, 63 households (298 members) of the 82 households with a clinical case of hepatitis A reported through surveillance (surveillance case households) were compared with all 40 survey households (566 members) that had at least one member susceptible to hepatitis A but none with IgM anti-HAV (control households). Nineteen of the 82 surveillance case households were excluded from this analysis since information on the ages of children and household size could not be obtained. The crude summary statistics appear in Table 4. Of the 63 surveillance case households, 61 (96·8%) had children aged 3–5 years, compared with 26 (65·0%) of the 40 control households (OR 16·4). Surveillance case households were also more likely to have children aged 6–9 years and under three years than were the control households, but the associations were weaker. In a logistic regression model that included the following factors, only the presence of 3–5 year old children was associated with hepatitis A: number of household members (OR 1·0, 95% CI 0·8–1·3), children 0–2 years old (OR 1·0, 95% CI 0·4–2·8), children 3–5 years old (OR 11·5, 95% CI 2·1–64·6), and children 6–9 years old (OR 1·4, 95% CI 0·4–5·4). The odds ratio for the presence of 3–5 year old children in the final preferred model that included only this variable was 14·7 (95% CI 3·1–69·6).

DISCUSSION

This outbreak of hepatitis A is similar in some aspects to numerous non-point-source community-wide outbreaks which have been reported previously [6, 8, 9, 15–20]. In fact, the similarity among these earlier outbreaks is so great that they have been described as belonging to a specific genre of hepatitis A epidemic [6, 19]. Typically they occur in 9–12 year

https://doi.org/10.1017/S0950268897007462 Published online by Cambridge University Press

intervals, last for 6–18 months, and often occur in communities of low to middle socioeconomic status. Age-specific attack rates are usually highest in the 5–9 year age group with a secondary peak incidence among young female adults who are probably exposed during close contact with young infected children in the family. Large family size, crowding within the home, and having children in day care have been reported as risk factors for hepatitis A during such community outbreaks [6, 8, 15, 18–21].

With no common source of exposure or organized day care to explain the epidemic in Kiryas Joel, how was hepatitis A transmitted in this community? This investigation strongly suggested that 3–5 year old children played an important role. This group of children had the highest attack rate among physician-diagnosed cases and had the highest proportion with anti-HAV in the serologic survey. Furthermore, the only significant risk factor among households in both the serologic survey and case-control analyses was the presence of 3–5 year old children in the household, although the results of both analyses also showed non-significantly elevated risk for households with 6–9 year olds.

Children under 3 or over 5 years were probably less important hepatitis transmitters in this outbreak. The results of this investigation suggested that the presence of diapered children in households failed to significantly increase a household's risk of hepatitis A. This finding is similar to the results of an investigation of a large outbreak in Zanesville, Ohio [6], and a study of sporadic hepatitis A cases in England which found that having a household member between 3–10 years old, but not less than 3, was associated with infection [22]. In contrast, several studies of outbreaks in children's day-care centres [23–25] and hospital nurseries [26] have shown an increased risk of hepatitis A associated with contact with very young children, who are often asymptomatic when infected with HAV and present a risk to their caretakers who handle their diapers or help with toilet-training. We believe that the absence of day-care centres or baby-sitting groups in Kiryas Joel resulted in little opportunity for diapered children to be exposed to hepatitis A outside the household, and therefore, they were unlikely to transmit HAV to their households. All young children remain at home until the age of about 3 years when they begin school. Moreover, studies in day-care centres [23] and elementary schools [27] have suggested that children aged 5 years and older are not frequent transmitters of HAV. Thus, although the majority of children of all ages were susceptible to hepatitis A in the survey, it seems likely that 3–5 year olds were the most important transmitters of HAV in this community.

In the serologic survey, only 53 of the 93 selected households participated. However, it is unlikely that the conclusions from this study were seriously biased by the lack of participation of some households since participating and non-participating households were similar in all assessed characteristics except the number of household members. Non-participating households had fewer members because households without children tended not to participate. Since numerous studies have shown that small family size decreases the risk of hepatitis A [6, 15, 18, 21, 28], it is likely that the non-participating households, many of which lacked children, were at decreased risk of hepatitis. Furthermore, the risk associated with 3–5 year old children was also found in the case-control study in which case households were drawn from surveillance reports, independent of participation in the survey.

Prevalence rates for anti-HAV vary greatly for different populations and are determined by multiple factors, including environmental and socioeconomic conditions [29–31], as well as the occurrence of epidemics. The overall prevalence of anti-HAV in our sample of Kiryas Joel residents was 43% and increased with age to 100% for residents 40 years and older (Fig. 2). In comparison, it is estimated that only 50% of United States residents have antibody by the age of 50 [4]. The high rate of antibody prevalence among Kiryas Joel residents probably resulted not only from the community outbreaks of hepatitis A but also from the immigration to Kiryas Joel of persons from highly endemic areas, such as the Hasidic communities in Brooklyn, New York, and Israel. Israel has a high rate of hepatitis A with more than 50% of the population estimated to have anti-HAV by age 18 years [28].

Although some features of the outbreaks in Kiryas Joel are similar to those of typical community outbreaks, one important difference is the time interval between epidemics. Kiryas Joel has now experienced hepatitis A outbreaks beginning in 1980, 1985 and 1989, with intervals between epidemics of less than 5 years. The more typical interval in other communities is 9–12 years, probably reflecting the necessary elapsed time to accumulate sufficient numbers of susceptible children to sustain an outbreak. However, the population of Kiryas Joel has

steadily increased as a result of high family growth and influx of new families into the community. Both of these factors likely caused rapid accumulation of newly susceptible residents after each epidemic. With the recurrent importation of HAV into Kiryas Joel from other closely-associated Hasidic communities where hepatitis A is endemic, it is not surprising that Kiryas Joel has experienced frequent epidemics.

IG has been shown to be effective in preventing symptomatic hepatitis A in individuals [4, 21, 32] and in controlling outbreaks in some settings [33–36]. However, as in other community outbreaks [6, 8], the administration of IG to household and close contacts of cases did not control this outbreak, nor prevent the recurrent outbreak in Kiryas Joel in 1990. More aggressive use of IG, with mass administration in a small religious community on the Utah–Arizona border, may have been partially effective in ending one outbreak [9]. However, such use of IG does not provide long-term protection and may serve only to postpone cases of hepatitis A and increase the number of older susceptible persons who are likely to experience more severe illness at time of infection [4].

Clearly a better strategy for preventing community outbreaks of hepatitis A is needed. Active immunization with an effective vaccine offers new hope. Such a vaccine would ideally provide long-lasting protection to vaccinees, as well as prevent them from transmitting HAV. Preliminary evidence from animal studies [37] suggests that active immunization may prevent both clinical disease and HAV infection, thus interrupting transmission. Recently, the mass immunization of susceptible residents in several Alaskan native communities with an inactivated hepatitis A vaccine appeared to be effective in ending community outbreaks of hepatitis A [38]. Adverse reactions from the inactivated vaccine appear to be minimal, consisting primarily of local reactions at the injection site [10]. The need for booster doses to prevent disease later in life, when hepatitis A typically causes more severe morbidity, is not yet clear.

Kiryas Joel has experienced frequent hepatitis A outbreaks at regular intervals and might expect another outbreak soon. During the most recent outbreak, Kiryas Joel was the site for the 1991 controlled vaccine trial [10] that resulted in 519 children, ages 2–16 years, receiving hepatitis A vaccine (Fig. 1). By November 1993, further vaccine studies had resulted in the vaccination of 1187 additional children, providing vaccine coverage for an estimated 45% of children between the ages of 2 and 16 years of age, and 63% of children between 3 and 5 years of age (Alan Werzberger, New York Medical College, Valhalla, New York, personal communication, 1994). With a substantial percentage of children in Kiryas Joel now actively immunized in vaccine trials, it will be important to see if the pattern of hepatitis A changes.

The history of recurrent outbreaks of hepatitis A in Kiryas Joel emphasizes that community outbreaks continue to cause significant morbidity. The results of this investigation show that 3–5 year olds in this community were more likely to be affected. Vaccination can protect this vulnerable group and might help to prevent future outbreaks in communities like Kiryas Joel where children under 3 have little opportunity for exposure outside the home.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the help of Dr Donna Stroup (Centers for Disease Control and Prevention, Atlanta, Georgia) for statistical advice, Shirley VanZetta (Orange County Health Department) for providing surveillance case information, Barbara Mensch (Kiryas Joel Institute of Medicine) for providing vaccine trial information, and the following persons from the New York State Department of Health, Albany, New York: Jaromir Mikl for computer programming, and Jean Hanson, Greer Fitting, and Kelly Cirilli for assistance in the preparation of the manuscript and data entry.

REFERENCES

1. Centers for Disease Control and Prevention. Summary of notifiable diseases, United States, 1994. MMWR 1994; **43** (53): 3.
2. Gust ID. Epidemiological patterns of hepatitis A in different parts of the world. Vaccine 1992; **10** (Suppl 1): S56–8.
3. Siegl G, Lemon SM. Recent advances in hepatitis A vaccine development. Virus Res 1990; **17**: 75–92.
4. Lemon SM. Type A viral hepatitis. New developments in an old disease. N Engl J Med 1985; **313**: 1059–67.
5. Shapiro CN, Coleman PJ, McQuillan GM, Alter MJ, Margolis HS. Epidemiology of hepatitis A: seroepidemiology and risk groups in the USA. Vaccine 1992; **10** (Suppl 1): S59–62.
6. Shaw FE, Sudman JH, Smith SM, et al. A community-wide epidemic of hepatitis A in Ohio. Am J Epidemiol 1986; **123**: 1057–65.
7. Shaw FE, Shapiro CN, Welty TK, Dill W, Reddington J, Hadler SC. Hepatitis transmission among the Sioux Indians of South Dakota. Am J Public Health 1990; **80**: 1091–4.

8. Majeed FA, Stuart JM, Cartwright KAV, et al. An outbreak of hepatitis A in Gloucester, UK. Epidemiol Infect 1992; **109**: 167–73.
9. Pavia AT, Nielsen L, Armington L, Thurman DJ, Tiernay E, Nichols CR. A community-wide outbreak of hepatitis A in a religious community: impact of mass administration of immune globulin. Am J Epidemiol 1990; **131**: 1085–93.
10. Werzberger A, Mensch B, Kuter B, et al. A controlled trial of a formalin-inactivated hepatitis A vaccine in healthy children. N Engl J Med 1992; **327**: 453–7.
11. Margolis HS, Shapiro CN. Who should receive hepatitis A vaccine? Considerations for the development of an immunization strategy. Vaccine 1992; **10** (Suppl 1): S85–7.
12. Dean AG, Dean JA, Burton AH, Dicker RC. Epi Info, Version 5: a word processing, database, and statistics program for epidemiology on microcomputers. USD, Incorporated, Stone Mountain, Georgia, 1990.
13. Gebski V, Leung O, McNeil D, Lunn D. SPIDA Version 6.0. Statistical Computing Laboratory Pty. Ltd., Australia, 1992.
14. Hosmer DW, Lemeshow S. Applied logistic regression. New York: John Wiley and Sons, 1989: 129–31.
15. Ispen J, Donovan WR, James G. Sociologic factors in the spread of epidemic hepatitis in a rural school district. J Hyg 1952; **50**: 457–70.
16. Lilienfeld AM, Bross IDJ, Sartwell PE. Observations of an outbreak of infectious hepatitis in Baltimore during 1951. Am J Public Health 1953; **43**: 1085–96.
17. Barondess JA, Drake ME, Bashe WJ, et al. Epidemic of infectious hepatitis. Some notes on delineation of high–risk groups and protection of exposed susceptibles by gamma globulin. Arch Intern Med 1995; **95**: 633–45.
18. Mosley WH, Speers JF, Chin TDY. Epidemiologic studies of a large urban outbreak of infectious hepatitis. Am J Public Health 1963; **53**: 1603–17.
19. Crusberg TC, Burke WM, Reynolds JT, Morse LE, Reilly J, Hoffmann AH. The reappearance of a classical epidemic of infectious hepatitis in Worcester, Massachusetts. Am J Epidemiol 1978; **107**: 545–51.
20. Desenclose JA, MacLafferty L. Community wide outbreak of hepatitis A linked to children in day care centres and with increased transmission in young adult men in Florida 1988–9. J Epidemiol Commun Health 1993; **47**: 269–73.
21. Ashley A. Gamma globulin. Effect on secondary attack rates in infectious hepatitis. N Engl J Med 1954; **250**: 412–7.
22. Maguire HC, Handford S, Perry KR, et al. A collaborative case control study of sporadic hepatitis A in England. Commun Dis Rep Rev 1995; **5**: R33–40.
23. Hadler SC, Webster HM, Erben JJ, Swanson JE, Maynard JE. Hepatitis A in day-care centers. A community-wide assessment. N Engl J Med 1980; **302**: 1222–7.
24. Hadler SC, Erben JJ, Francis DP, Webster HM, Maynard JE. Risk factors for hepatitis A in day-care centers. J Infect Dis 1982; **145**: 255–61.
25. Storch G, McFarland LM, Kelso K, Heilman CJ, Caraway CT. Viral hepatitis associated with day-care centers. JAMA 1979; **242**: 1514–8.
26. Klein BS, Michaels JA, Rytel MW, Berg KG, Davis JP. Nosocomial hepatitis A. A multinursery outbreak in Wisconsin. JAMA 1984; **252**: 2716–21.
27. Hoff RS, Galambos J. Viral hepatitis. In: Schiff L, Schiff ER, eds. Diseases of the liver. 5th edn. Philadelphia: JB Lippincott, 1982: 498–9.
28. Green MS, Zaaide Y. Sibship size as a risk factor for hepatitis A infection. Am J Epidemiol 1989; **129**: 800–5.
29. Frosner GG, Papaevangelou G, Butler R, et al. Antibody against hepatitis A in seven European countries. I. Comparison of prevalence data in different age groups. Am J Epidemiol 1979; **110**: 63–9.
30. Szmuness W, Dienstag JL, Purcell RH, et al. The prevalence of antibody to hepatitis A antigen in various parts of the world: A pilot study. Am J Epidemiol 1977; **106**: 392–8.
31. Szmuness W, Dienstag JL, Purcell RH, Harley EJ, Stevens CE, Wong DC. Distribution of antibody to hepatitis A antigen in urban adult populations. N Engl J Med 1976; **295**: 755–9.
32. Landrigan PJ, Huber DH, Murphy GD, Creech WB, Bryan JA. The protective efficacy of immune serum globulin in hepatitis A. A statistical approach. JAMA 1973; **223**: 74–5.
33. Hall WT, Madden DL, Mundon FK, Brandt DEL, Clarke NA. Protective effect of immune serum globulin (ISG) against hepatitis A infection in a natural epidemic. Am J Epidemiol 1977; **106**: 72–5.
34. Gellis SS, Stokes J, Brother GM, et al. The use of human immune serum globulin (gamma globulin) in infectious (epidemic) hepatitis in the Mediterranean theater of operations. I. Studies on propylaxis in two epidemics of infectious hepatitis. JAMA 1945; **128**: 1062–3.
35. Krugman S, Ward R, Giles JP, Jacobs AM. Infectious hepatitis. Studies on the effect of gamma globulin and on the incidence of inapparent infection. JAMA 1960; **174**: 823–30.
36. Hadler SC, Erben JJ, Matthews D, Starko K, Francis DP, Maynard JE. Effect of immunoglobulin on hepatitis A in day-care centers. JAMA 1983; **249**: 48–53.
37. Purcell RH, D'Hondt E, Bradbury R, Emerson SU, Govindarajan S, Binn L. Inactivated hepatitis A vaccine: active and passive immunoprophylaxis in chimpanzees. Vaccine 1992; **10** (Suppl 1): S148–51.
38. McMahon BJ, Beller M, Williams J, et al. A program to control an outbreak of hepatitis A in Alaska using an inactivated hepatitis A vaccine. Presented at the 34th Interscience Conference on Antimicrobial Agents and Chemotherapy, Orlando, Florida, October 1994.