data suggest that such initiatives, when they are undertaken, might well take place in STD, prenatal, and contraceptive clinics which serve teenagers who are exposed to early coitus.

REFERENCES

- Green DE: Teenage Smoking: Immediate and Long-Term Patterns. Washington, DC: Department of Education, National Institute of Education, November 1979.
- Bachman JG, Johnson LD, O'Malley PM: Smoking, drinking and drug use among American high school students: correlates and trends, 1975– 1979. Am Public Health 1981; 71:59-68.
- McAlister A, Perry C, et al. Pilot study of smoking, alcohol and drug abuse prevention. Am J Public Health 1980; 70:719-721.
- Perry C, Killen J, et al: Modifying smoking behavior of teenagers: a school-based intervention. Am J Public Health 1980; 70:722-725.
- Royal College of General Practitioners', Oral Contraceptive Study: Further analyses of mortality in oral contraceptive users. Lancet 1981; 1:541-546.

- Shapiro S, Slone D, et al: Oral-contraceptive use in relation to myocardial infarction. Lancet 1979; 1:747.
- Population Information Program: Oral Contraceptives. Population Reports 1982; Series A, No. 6:A204-A209.
- Jessor R, Jessor SL: Problem Behavior and Psychosocial Development, A Longitudinal Study of Youth. New York: Academic Press, 1977.
- Zabin LS, Clark SD: Why they delay: a study of teenage family planning clinic patients. Fam Plann Perspect 1981; 13:205-217.
- Hunter SM, Webber LS, Bevenson GS: Cigarette smoking and tobacco usage behavior in children and adolescents: Bogalusa Heart Study. Prev Med 1980; 9:701-712.
- 11. Zabin LS, Kantner JF, Zelnik M: The risk of adolescent pregnancy in the first months of intercourse. Fam Plann Perspect 1979; 11:215-222.

ACKNOWLEDGMENTS

This paper is based on data collected under a grant from the Brush Foundation, Cleveland, Ohio. The author is grateful to them, and to Samuel D. Clark, Jr., whose work with the data was an essential part of this study. Thanks are also due to the 32 contraceptive clinics who took part in the study, to their staffs, and to their teenage patients.

A Community Waterborne Gastroenteritis Outbreak: Evidence for Rotavirus as the Agent

RICHARD S. HOPKINS, MD, G. BARRY GASPARD, BS, FREDERICK P. WILLIAMS, JR., BS, RICHARD J. KARLIN, BS, PE, GEORGE CUKOR, PHD, AND NEIL R. BLACKLOW, MD

Abstract: A community waterborne nonbacterial gastroenteritis outbreak occurred in Eagle-Vail, Colorado in March 1981. Illness (defined as vomiting and/or diarrhea) was statistically associated with water consumption (χ^2 for linear trend = 7.07, p < .005). Five of seven persons associated with the outbreak were infected with rotavirus as shown by virus detection or serological methods. Bacterial pathogens, Giardia lamblia, and Norwalk virus were excluded as responsible agents. Rotavirus should be looked for as a cause of waterborne outbreaks. (Am J Public Health 1984; 74:263–265.)

Waterborne gastroenteritis outbreaks have been associated with Salmonella (including typhi), Shigella, Campylobacter, hepatitis A, Giardia lamblia, Entameba histolytica, Norwalk virus, 1.2 a similar virus, the Snow Mountain Agent,3 and other agents. The outbreak of gastrointestinal illness described here was clearly waterborne and was initially approached as a fairly routine Norwalk-like outbreak. It may have been associated with rotavirus, however.

Background

Eagle-Vail and Avon are residential communities adjacent to a major tourist and skiing center in central Colorado.

Address reprint requests to Richard S. Hopkins, MD, Chief, Communicable Disease Control Section, Epidemiology Division, Colorado Department of Health, 4210 E. 11th Avenue, Denver, CO 80220. Mr. Gaspard is also with that Division and Mr. Karlin is with the Water Quality Division, Colorado Department of Health; Mr. Williams is with the Microbiology Branch, Toxicology & Microbiology Division, Health Effects Research Laboratory, USEPA, Cincinnati; Dr. Cukor and Dr. Blacklow are with the Division of Infectious Diseases, Department of Medicine, U-Mass Medical School, Worcester. This paper, submitted to the Journal February 3, 1983, was revised and accepted for publication June 1, 1983.

© 1984 American Journal of Public Health 0090-0036/84 \$1.50

Three other communities lie upstream from Eagle-Vail and Avon: Minturn and Redcliff on the Eagle River, and Vail on Gore Creek, a tributary of the Eagle River. In early 1981, Eagle-Vail and Avon shared a common water supply and distribution system. Raw water was obtained either from a small stream in the mountains south of the area, or from an intake on the Eagle River downstream from its confluence with Gore Creek. The Eagle River was ordinarily an auxiliary water source, but dry warm conditions in early 1981 forced its use starting in January as the primary source when the usual source was insufficient to meet demand.

On Friday, March 13, 1981, several citizens independently alerted the Colorado Department of Health to a possible outbreak of gastrointestinal disease in Eagle-Vail and Avon. Telephone contact with the principal medical facility serving the five communities described above showed that office and emergency-room visits for gastrointestinal disease had risen sharply March 6-11; review of emergency room records showed that the increase was entirely in residents of Eagle-Vail and Avon, not of Vail itself or the other two communities in the service area of the medical facility.

Methods

Eagle-Vail and Avon share a unique three-digit telephone exchange. A household telephone survey was performed on March 13 and 14 by calling every 6th listed residential number in that exchange until 48 households were reached; about 80 households in the sample did not answer on multiple attempts in a 24-hour period, and 20 were disconnected. This relatively low completion rate may be due to intermittently occupied residences in a winter resort area. There were no refusals among those who were contacted.

All persons at home at the time of the call were

interviewed about illness in the previous 30 days, including symptoms and onset date, average daily consumption of tap water and beverages made with tap water before the outbreak period (controls) or onset of illness (cases), meals eaten away from home, and social events attended. They were also asked to answer the same questions, to the best of their knowledge, for household members who were not at home.

We calculated χ^2 for trend and confidence intervals around relative risks using the calculator programs of Rothman and Boice.⁴

Laboratory records of the Vail Mountain Medical Center for the period March 1-25, 1981 were reviewed. In addition, five ill adults seen between March 12-22 at the medical center for diarrheal illness with onsets March 7-20 submitted stools that were examined for the presence of virus particles by routine electron microscopy (EM) at the Health Effects Research Laboratory, US Environmental Protection Agency, Cincinnati, Ohio. Acute and convalescent (three weeks post illness) sera from six persons who were ill during the same time period were tested for viral antibody by immune electron microscopy (IEM) using a rotavirus preparation from the stool of one individual containing large numbers of particles. The individual sera were examined under code after overnight incubation with the rotavirus preparation. Each serum was rated 0-4+ according to the amount of aggregation and antibody coating of particles observed.5 The serum pairs were identified after examination and a significant sero response was indicated if a convalescent serum was rated at least 1+ higher than the corresponding acute serum. The sera were also tested under code by radioimmunoassay (RIA) for antibody to rotavirus and to Norwalk virus at the University of Massachusetts Medical School using procedures previously described. 6,7 Significant sero responses were indicated by a four-fold or greater increase in antibody titer against the virus tested.

The same five stools from ill adults were tested for heatlabile and heat-stable enterotoxigenic *Escherichia coli* at the Centers for Disease Control by described techniques.⁸⁻¹⁰

The water treatment plant at Eagle-Vail and the sewage treatment plant at Vail were inspected within a few days after the outbreak came to attention.

Results

Forty-eight households were reached, with 128 residents, of whom over 80% were adults. All but five residents were over two years old and only a handful were over age 50. Of these 128 residents, 56 (43.8 per cent) had been ill in the previous 30 days with diarrhea (three or more loose stools per day) and/or vomiting. (This is the case definition used through the rest of this analysis.)

The peak of the epidemic curve was on March 6. New cases gradually tapered off March 7 through March 11. Mean duration of illness among 41 cases with onset during the epidemic period (March 6–13) was 2.6 days (range 1 to 6). Of the 41 cases, 34.1 percent had diarrhea only, 26.8 per cent had vomiting only, and 39.0 per cent had both. Cramping was noted by 34.1 per cent, and fever by 21.9 per cent.

The attack rate for diarrhea and/or vomiting during the epidemic period was closely related to amount of tap water consumption with a χ^2 for linear trend of 7.07, p = .0039 (Table 1). Overall attack rate was 41/128 (32.0 per cent). Those who drank no tap water drank bottled water for the

TABLE 1—Attack Rates for Vomiting and/or Diarrhea among Eagle-Vail/
Avon (Colorado) Phone Survey Respondents, and Average
Daily Tap Water Consumption

Daily Water Consumption (8 oz. glasses)	Illness Onset 2/15–3/5	Illness Onset 3/6–3/13	Total	Per Cent Attack Rate 2/15-3/5	Per Cent Attack Rate 3/6-3/13
0	5	6	32	15.6	18.7
1–2	3	11	36	8.3	30.6
3-4	4	13	26	15.4	50.0
5 or more	3	10	21	14.3	47.6
unknown	_	1	13		7.7
TOTAL	15	41	128	11.7	32.0

most part. The relative risk for those who drank one or more glasses of tap water a day compared to those who drank no tap water was 2.18 (95 per cent confidence interval 1.10–4.32). Water consumption for 15 ill persons with onset February 5 to March 5 was not different from that for well persons.

Persons who lived in a household with a case occurring in the peak of the outbreak (March 6-7) were more likely to be ill in the period March 8-13 (39 per cent) than persons not so exposed (22 per cent). Three of the six ill persons with no tap water consumption were exposed to an ill water-drinking household member two to four days before their own illness. Distribution of ill and well persons in multiple-person households, however, was consistent with chance.

Six ill persons who either lived or worked in Eagle-Vail or Avon, and a seventh ill person exposed to an ill roommate who worked in Eagle-Vail, were studied virologically. Three of four individuals developed a seroconversion to rotavirus by RIA, while the fourth had a high titer on both acute and convalescent sera. Three of five persons developed a seroconverion to rotavirus by IEM while two had high titers on both acute and convalescent sera. Rotavirus particles were detected by EM in one person from whom paired sera were lacking. Altogether, five of the seven ill individuals were infected with rotavirus as shown by virus detection or serological methods, and a sixth had stable high titers by both RIA and IEM 13 and 32 days after onset.

All six individuals from whom paired serum samples were available were studied for seroconversions to Norwalk virus by RIA; none were found.

From March 8 through March 21, the Vail Mountain Medical Center laboratory examined 10 stools of ill persons for ova and parasites and six for enteric bacterial pathogens (Salmonella, Shigella and Campylobacter). They found none. The usual number of stools examined is two per week. The five stools examined at the Centers for Disease Control were all negative for enterotoxigenic E. coli.

Four coincident physical factors probably contributed to the outbreak:

- The upstream Vail Water & Sanitation District sewage treatment plant could not handle the load being presented to it and was discharging sludge solids into Gore Creek, upstream from the Eagle River water intake for the Eagle-Vail plant;
- There was no chemical or physical pretreatment of water prior to filtration (no coagulation or polymer addition) at the Eagle-Vail water treatment plant;
- Severely channeled filter beds were found in the water treatment plant at Eagle-Vail; and

• The Eagle-Vail chlorinator failed for an undetermined period of up to 24 hours ending March 5.

Cost of emergency repairs to the Eagle-Vail water treatment plant in the 30 days after the outbreak was \$92,400. Over the 18 months following the outbreak, costs of improvements to ensure a reliable safe water supply are estimated at over \$1 million. The plant will have much greater storage for treated water and a third filter, so the filters can be run at a steady rate, and chemical treatment will precede filtration. These improvements will bring the water treatment plant in compliance with current Colorado Department of Health requirements, which have become more stringent since the original plant was designed and built.

Discussion

The epidemiologic and engineering evaluation together show that this was a waterborne gastroenteritis outbreak whose immediate cause was a chlorinator breakdown March 4 and 5. On March 4, 1981, the chlorinator was the only effective barrier between the toilets of Vail and the watertaps of Eagle-Vail and Avon. The other two barriers designed to ensure safe drinking water were compromised: raw water quality and inadequate pretreatment and filtration. The simultaneous existence of these defects opened the way for a waterborne outbreak.

Attack rates for illness during the epidemic period rose with increasing self-reported water consumption, while illness during the month before the outbreak could not be related to water consumption. While it is possible that cases selectively recalled higher water consumption than well controls, the persons with earlier onsets of illness were also convinced that their illnesses were water-related and recall bias should have been just as common. The clinical syndrome, with fever rare, duration short, and vomiting prominent, was characteristic of viral gastroenteritis and the laboratory data are consistent with, but do not establish, a rotaviral etiology.

Rotavirus may be a cause of waterborne outbreaks, with disease in adults as well as children. 11-13 Three reports from outside the US have suggested the possibility of waterborne rotavirus infection. 14-16 Careful virologic and epidemiologic evaluation of future outbreaks will be necessary to establish how common waterborne rotavirus outbreaks are in comparison to those associated with other pathogens.

REFERENCES

- Taylor JW, Gary GW, Greenberg HB: Norwalk-related viral gastroenteritis due to contaminated drinking water. Am J Epidemiol 1981; 114:584

 592.
- Wilson R, Anderson LJ, Holman RC, Gary GW, Greenberg HB: Waterborne gastroenteritis due to the Norwalk agent: clinical and epidemiologic investigation. Am J Public Health 1982; 72:72-74.
- Morens DM, Zweighaft RM, Vernon TM, et al: A waterborne outbreak of gastroenteritis with secondary person-to-person spread: association with a viral agent. Lancet 1979; 1:964-966.
- Rothman KJ, Boice JD: Epidemiologic analysis with a programmable calculator. NIH Pub. No. 79-1649. Bethesda, MD: NIH, June 1979; 14– 15, 36-37.
- Kapikian AZ, Yolken RH, Greenberg HB, et al: Gastroenteritis viruses. In: Lennette EH, Schmidt NJ (eds): Diagnostic Procedures for Viral, Rickettsial and Chlamydial Infections, 5th Ed. Washington, DC: American Public Health Association, 1979; 927-995.
- Blacklow NR, Cukor G, Bedegian MK, et al: Immune responses and prevalence of antibody to Norwalk enteritis virus as determined by radioimmunoassay. J Clin Microbiol 1979; 10:903-909.
- Blacklow NR, Cukor G: Viral gastroenteritis agents. In: Lenette EH, Ballows A, Hausler WJ (eds): Manual of Clinical Microbiology, 3rd Ed. Washington, DC: American Society for Microbiology, 1980, 891-898.
- Sack DA, Sack RB: Test for enterotoxigenic Escherichia coli using Y/1 adrenal cells in miniculture. Infection and Immunity 1975; 2:334–336.
- Yolken RH, Greenberg HB, Merson MH, et al: Enzyme-linked immunosorbent assay for detection of Escherichia coli heat-labile enterotoxin. J Clin Microbiol 1977; 6:439–444.
- Dean AG, Ching Y, William RG, Harden LB: Test for Escherichia coli enterotoxin using infant mice: application in a study of diarrhea in Honolulu. J Inf Dis 1972; 125:407-411.
- Blacklow NR, Cukor G: Viral gastroenteritis. N Engl J Med 1981; 304:397-406.
- Wenman WM, Hinde D, Feltham S, Gurwith M: Rotavirus infection in adults. results of a prospective family study. N Engl J Med 1979; 301:303– 306
- Cubitt WD, Holzel H: An outbreak of rotavirus infection in a long-stay ward of a geriatric hospital. J Clin Pathol 1980; 33:306-308.
- Lycke E, Blomberg J, Berg G, Ericksson A, Madsen L: Epidemic acute diarrhoea in adults associated with infantile gastroenteritis virus. Lancet 1978; 2:1056-1057.
- Zamotin BA, Libiyainen LT, Bortnik FL, et al: Water-borne group infection of rotavirus etiology. Zh Mikro, Epid I Imm 1981; 11:100-103.
- Sutmoller F, Azeredo RS, Lacerda MD, et al: An outbreak of gastroenteritis caused by both rotavirus and Shigella sonnei in a private school in Rio de Janeiro. J Hyg (Camb) 1982; 88:285-293.

ACKNOWLEDGMENTS

We would like to thank Jane Merrit, RN, Eagle County Nursing Service; James Chubrillo, PE, Water Quality Division, Colorado Department of Health; Edwin C. Lippy, PE, Health Effects Research Laboratory, EPA, Cincinnati, Ohio; Nancy Nowak, University of Massachusetts Medical School; Joy Wells, Center for Infectious Diseases, Centers for Disease Control; and the staffs of Vail Mountain Medical Associates and Vail Valley Medical Center for their assistance with this work.

This research was supported in part by EPA Contract #68-03-2927 and by EPA Cooperative Agreement #CR-80880101. Although this study received intramural and extramural support from the USEPA, it has not been subjected to the agency's peer and policy review and, therefore, does not necessarily reflect the views of the agency and no official endorsement should be inferred.