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Reviewed work(s):
Source: *The Journal of Infectious Diseases*, Vol. 176, No. 3 (Sep., 1997), pp. 815-818
Published by: Oxford University Press
Stable URL: http://www.jstor.org/stable/30107374
Accessed: 09/11/2011 16:11

A Prolonged Outbreak of *Escherichia coli* O157:H7 Infections Caused by Commercially Distributed Raw Milk

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A protracted outbreak of *Escherichia coli* O157:H7 infections was caused by consumption of unpasteurized ("raw") milk sold at Oregon grocery stores. Although it never caused a noticeable increase in reported infections, the outbreak was recognized because of routine follow-up interviews. Six of 16 Portland-area cases reported between December 1992 and April 1993 involved people who drank raw milk from dairy A. By pulsed-field gel electrophoresis (PFGE), *E. coli* O157:H7 isolates from these cases and from the dairy A herd were homologous (initially, 4 of 132 animals were *E. coli* O157:H7-positive). Despite public warnings, new labeling requirements, and increased monitoring of dairy A, retail sales and dairy-associated infections continued until June 1994 (a total of 14 primary cases). Seven distinguishable PFGE patterns in 3 homology groups were identified among patient and dairy herd *E. coli* O157:H7 isolates. Without restrictions on distribution, *E. coli* O157:H7 outbreaks caused by raw milk consumption can continue indefinitely, with infections occurring intermittently and unpredictably.

Escherichia coli O157:H7 outbreaks are most commonly caused by consumption of undercooked beef or other foods that are cross-contaminated with beef. Such outbreaks are usually detected because of a marked increase in illness within a group or community. Common-source outbreaks are typically short-lived, limited by the quantity and shelf life of the contaminated product(s), and are preventable with adequate kitchen hygiene and cooking.

While often contaminated with enteric organisms during collection, before sale almost all milk is pasteurized, which kills pathogenic microbes. Some consumers, however, choose to drink unpasteurized milk, despite the well-documented risk of enteric infection [1]. Milk has a limited shelf life, but dairy animals can be productive for years, and the output of a pathogen-colonized dairy herd may be contaminated indefinitely. Thus, depending on product distribution and frequency of contamination, raw milk–associated infections can be widely scattered in both time and place, making it difficult to recognize a common source.

Mandatory reporting of *E. coli* O157:H7 infections in Oregon was initiated in 1990. Reports are first investigated by local health department personnel. During the first few weeks of April 1993, 3 ostensibly unrelated patients in one county mentioned consuming raw milk from a single dairy farm (dairy A). The retail sale of raw milk is legal in Oregon; dairy A milk was sold at supermarkets and other grocery stores in the tricounty Portland area. Here we present the results of our investigation and their implications for disease surveillance and outbreak control.

Methods

Case finding and definitions. We defined cases as persons from whose stools *E. coli* O157:H7 was cultured or persons who developed diarrhea (≥3 loose stools within a 24-h period) within 7 days of the symptom onset of a culture-confirmed household member. Cases were identified from routine surveillance reports. For this investigation, we reviewed cases with symptom onsets from January 1992 through June 1994 among residents of the three counties (Multnomah, Washington, and Clackamas) comprising greater Portland. When initially reported, cases (or household informants) had been interviewed, using a standardized questionnaire, about possible exposures. We defined raw milk–associated cases as those who reported drinking raw milk within the 10 days before symptom onset. Persons whose illness began ≥2 days after that of another household member were considered secondary cases.

Statistics. The probability of finding as many raw milk drinkers as we did among reported cases was estimated using a standard binomial model. We used an arbitrarily chosen interval extending from 1 month before the first raw milk–associated case was reported to the date the cluster was first identified (1 December 1992 to 20 April 1993).

Received 19 March 1996; revised 13 April 1997.

Grant support: Washington State Agricultural Research Center (10A-3072-0835 to D.D.H.).

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The Journal of Infectious Diseases 1997;176:815–8

0022–1899/97/7603–0045$02.00

Dairy sampling and microbiology. Agriculture inspectors routinely (every 8 weeks) visit Oregon dairies that sell retail raw milk; milk samples are collected and assayed by standard methods for total coliform count and other parameters. We reviewed milking operations and test results for dairy A from January 1992 through June 1994. Milk samples collected from the bulk tank at least every 2 weeks after April 1993 were assayed for *E. coli* O157:H7 by two methods: 1 aliquot was cultured directly on sorbitol–MacConkey's agar; another was enriched overnight and screened by EIA (EHEC-Tek; Organon Teknika; Durham, NC).

Rectal swabs were obtained from all cattle at dairy A and cultured [2]. Bovine isolates and isolates from human cases were confirmed as *E. coli* O157:H7 by standard biochemical tests, latex agglutination assay, and serologic testing [3].

Isolates were digested with *Xba*I and subtyped by pulsed-field gel electrophoresis (PFGE) [4]. Relative to one another, PFGE patterns were categorized as indistinguishable, homologous (one- or two-band difference), or unique.

Results

Initial epidemiologic findings. Sixteen primary cases had been reported in the Portland area from 1 December 1992 through 20 April 1993; 6 of these individuals reported drinking raw milk, all from dairy A (figure 1). The first raw milk–associated case became ill in late December 1992. By chance, the probability of finding at least 6 of 16 persons chosen at random to be dairy A milk drinkers is 0.0000000073, assuming that 1% of people drink that brand during a 10-day interval. (Dairy A's share of the Portland milk market was <0.5%.)

Dairy operation. Dairy A produced ~1400 L of whole milk, skim milk, and cream per day. Milk from the bulk tank was tested 26 times between December 1992 and June 1994, with total coliform levels ranging from undetectable to 17,000 cfu/mL; 5 samples had levels >150 cfu/mL. There was no obvious relationship between total coliform levels and the occurrence of infections. *E. coli* O157:H7 was never recovered from any dairy A milk sample.

Control measures and subsequent cases. Through media contacts on and after 21 April 1993, we warned the public of the hazards associated with raw milk, particularly from dairy A. Raw milk sales from dairy A were briefly suspended but resumed when *E. coli* O157:H7 was not recovered from a milk sample or a convenience sample of manure specimens collected at the dairy on 20 April. Legal analysis suggested that Oregon public health agencies lacked clear authority to restrict milk sales unless it could be demonstrated that specific containers destined for sale were contaminated.

Two more dairy A–associated cases were reported in June 1993, 1 with an April onset. In December, the Oregon Department of Agriculture introduced labeling requirements for raw milk products ("This product has not been pasteurized [and] may contain disease-producing organisms") and new standards for total coliform levels in retail raw milk (<10 cfu/mL). Dairy A agreed to suspend retail sales should additional illnesses be

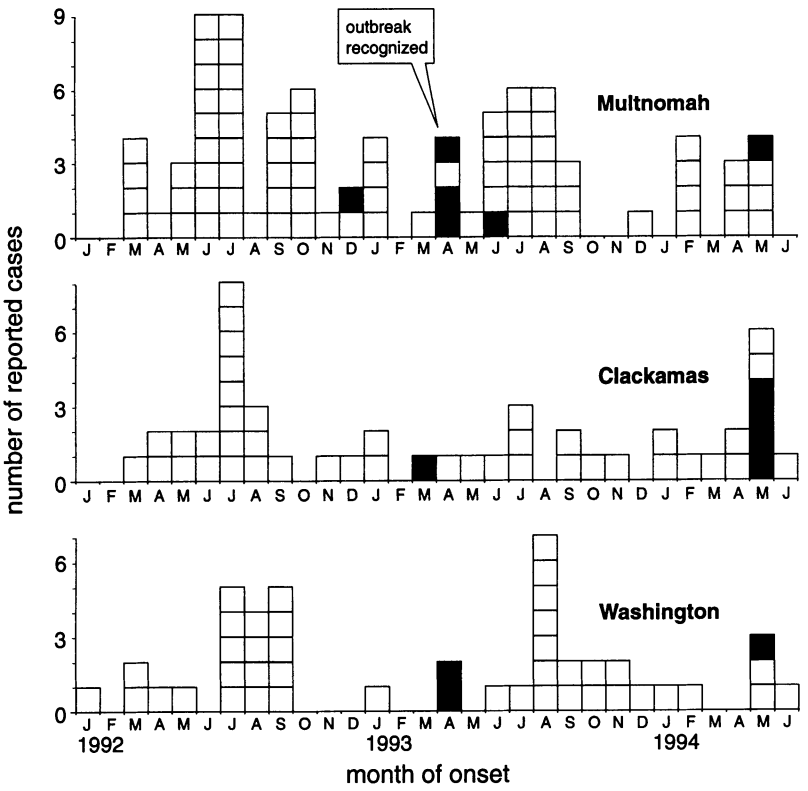

Figure 1. Reported monthly occurrence of primary *E. coli* O157:H7 infections in 3 Portland-area counties, January 1992 to June 1994. ■ = dairy A–associated *E. coli* O157:H7 cases; □ = other reported cases. Cases are shown by county of residence and month of symptom onset; within each month, cases are arranged (from bottom to top) in order of onset. Secondary cases (n = 7, including 3 associated with this outbreak) are not shown.

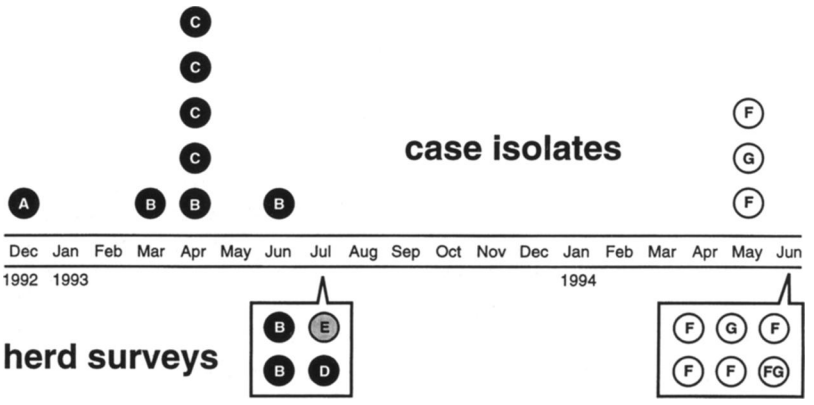

Figure 2. Subtyping of dairy A–associated *E. coli* O157:H7 isolates from primary cases and dairy herd animals, shown by month of symptom onset and month of culture, respectively. (Only 1 isolate was subtyped from 1 1994 household with 4 co-primary cases.) Homology (≤2 band difference by pulsed-field gel electrophoresis) is indicated by circle color; distinguishable patterns are indicated by letters.

linked to its products. In May 1994, 6 new cases were identified. An injunction was issued in June 1994 proscribing further distribution to consumers. Nevertheless, clandestine sales persisted until October 1995, when the dairy owner was fined and jailed for contempt of court. No dairy A–associated cases have been reported since June 1994.

Epidemiologic and clinical summary. In total, 14 (8%) of the 173 primary *E. coli* O157:H7 cases reported in the Portland area from January 1992 through June 1994 reported drinking dairy A milk (figure 1). Outbreak cases were between 1 and 73 years old; 8 were ≤10 years old. None developed the hemolytic uremic syndrome; 2 were hospitalized. Nine patients (64%) reported drinking raw milk for <1 month before they became ill, including 6 who reported only a single exposure. Only 4 characterized themselves as regular consumers of long standing. Three secondary cases were identified in these 11 households, representing either person-to-person spread or longer incubations.

Herd testing. Dairy A first allowed herd testing in July 1993, when *E. coli* O157:H7 was cultured from 4 (3%) of 132 cattle. When resampled in June 1994, 6 (5%) of 124 animals were culture-positive. No animal tested positive on both occasions.

Bacteria subtyping. The results of PFGE subtyping are shown in figure 2. Three distinguishable patterns were identified among the 8 dairy A–associated patient isolates from 1992–1993. The three patterns were homologous to each other and to 3 of the 4 herd isolates from 1993. Two of the bovine isolates were indistinguishable from 3 of the patient isolates (pattern B). One patient isolate from each of the 3 1994 case-households was subtyped. All were homologous to each other; 2 were indistinguishable. The same two patterns were seen in the 6 1994 cattle isolates from dairy A; 1 animal carried both (figure 2). There was no apparent relationship between dairy A–associated isolates (human or bovine) from 1994 and 1992–1993. None of the dairy-associated isolates appeared related to any other isolates tested from Oregon ($n = 10$) or elsewhere in the Northwest ($n = 101$).

Discussion

This outbreak never caused an obvious increase in disease reports from any one county or statewide. It was only recognized because potential exposures, including raw milk consumption, were reviewed during routine follow-up investigations. Such interviews, while labor-intensive, can be essential for identifying protracted or low-intensity outbreaks, particularly when background rates are high.

Suspicions were first voiced when 3 of the April cases in one county reported drinking raw milk—a relatively uncommon exposure. Other raw milk drinkers were then identified from a review of existing surveillance reports. All were confirmed to have consumed milk from dairy A. We then faced the dilemma of when to take public action. The binomial calculation, while using an arbitrarily selected time interval, suggested that a chance association between infection and consumption of dairy A milk was extremely unlikely. Given the ongoing nature of the outbreak, we elected to notify the public immediately rather than delay until a formal study could be done. In the absence of a distinct outbreak period, it was not clear how to delimit a case-control study without significant bias. A cohort study was also infeasible.

The association was eventually corroborated by PFGE subtyping results suggesting that the isolates from milk drinkers had a common origin with each other and with isolates from the dairy herd. Such subtyping can be of great value in outbreak investigations, although as this cluster illustrates, outbreaks are not necessarily marked by a single PFGE pattern.

Anecdotal reports and case-control studies suggest that raw milk is one cause of sporadic *E. coli* O157:H7 infections [5–8], but only one cluster due to raw milk consumption, an outbreak among Canadian schoolchildren visiting a dairy farm, has been well described [9]. Several other raw milk–associated *E. coli* O157:H7 clusters in Britain received brief mention [10, 11]. Pasteurized dairy products (yogurt [12] and milk [13]) were implicated in two other British outbreaks, apparently due to contamination with raw milk or improper processing.

Raw milk is an unsurprising vehicle for enteric infections [1], as it unavoidably contains fecal organisms from the milking herd. Occasionally, high levels of contamination can occur as a result of poor environmental sanitation or improper holding temperatures with attendant bacterial replication. Given their prevalence in cattle, these contaminants will sometimes include *E. coli* O157:H7, *Campylobacter* or *Salmonella* species, or other pathogens. Routine inspection of raw milk dairies by governmental agencies cannot prevent this contamination, and indeed may convey a misleading imprimatur of safety to unwary consumers.

While *E. coli* O157:H7 has been isolated from raw milk [11, 14], testing production lots for specific pathogens is an unreliable means of protecting consumers. Contamination may be intermittent or below the detection limit of available assays. Moreover, using a battery of pathogen-specific premarket tests would unrealistically delay distribution and would offer no protection against uncharacterized agents. Assaying for nonspecific indicators of fecal contamination would be similarly impractical, and such measures are probably unreliable predictors of risk [15].

Unlike most clusters of foodborne illness, raw milk–associated outbreaks have the potential to persist indefinitely, with cases occurring intermittently and unpredictably. Dairy A–associated infections were scattered over 18 months. Even when a source is identified, these outbreaks can be difficult to control. No known measures will eradicate *E. coli* O157:H7 from individual cattle, much less entire herds, or eliminate fecal contamination of milk as it is collected.

The only effective way to stop raw milk–associated disease is to stop people from drinking raw milk—easier said than done. Despite widespread publicity about the links between dairy A raw milk and potentially life-threatening illness, sales of this brand (and intermittent infections) continued until the dairy was forced out of the retail business. Many raw milk consumers remain skeptical about the inherent hazards of this product or are indifferent to the risks to themselves and their children. Short of an outright ban on sales, which has been enacted in at least 22 states (Klontz KC, personal communication), Canada, and Scotland, continuing consumer education and increasing financial risks for suppliers may be the only means to reduce raw milk consumption and associated illness. In response to this and another outbreak, legislation to outlaw the retail sale of raw milk in Oregon was introduced in 1995. It died in committee.

Acknowledgments

We thank Berdena Nickerson (Multnomah County Health Department) and Jim Postlewait, Jim Eyre, Jim Madden, and Jim Black (Oregon Department of Agriculture). Additional laboratory support was provided by Steve Mauvais (Oregon Health Division), Daniel H. Rice (Washington State University), and Stephen D. Weagant and Janelle Johnson (US Food and Drug Administration).

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