

work suggests that, with the exception of men whose military occupation entailed handling herbicides, most US Army combat troops who served in Vietnam were not heavily exposed to dioxin.¹² Since no other factor in the Vietnam experience has been linked to the increased NHL risk, reasons for the excess found in this study and two other studies remain unclear. □

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References

1. Committee on the Effects of Herbicides in Vietnam: The effects of herbicides in South Vietnam. Part A—Summary and conclusions. Washington, DC: National Academy of Sciences, 1974; 111–117.
2. Hardell L, Ericksson M, Lenner P, *et al*: Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: A case-control study. *Br J Cancer* 1981; 43:169–176.
3. Hoar SK, Blair A, Holmes FF, *et al*: Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. *JAMA* 1986; 256:1141–1146.
4. Breslin P, Kang HK, Lee Y, Burt V, Shepard BM: Proportionate mortality study of US Army and US Marine Corps veterans of the Vietnam war. *JOM* 1988; 30:412–419.
5. Selected Cancers Cooperative Study Group. The association of selected cancers with service in the US military in Vietnam. I. Non-Hodgkin's Lymphoma. *Arch Intern Med* 1990; 150:2473–2483.
6. Centers for Disease Control Vietnam Experience Study. Postservice mortality among Vietnam veterans. *JAMA* 1987; 257:790–795.
7. Centers for Disease Control Vietnam Experience Study: Health status of Vietnam veterans. I. Psychosocial characteristics. *JAMA* 1988; 259:2701–2707.
8. National Cancer Institute: Surveillance, epidemiology, and end results: Incidence and mortality data, 1973–77. National Cancer Institute Monograph 57. NIH Pub. No. 81-2330. Washington, DC: Govt Printing Office, 1981.
9. Heath CW Jr: The leukemias. In: Schottenfeld D, Fraumeni JF Jr (eds): *Cancer Epidemiology and Prevention*. Philadelphia: WB Saunders, 1982; 728–738.
10. Decouffé P: Occupation. In: Schottenfeld D, Fraumeni JF Jr (eds): *Cancer Epidemiology and Prevention*. Philadelphia: WB Saunders, 1982; 318–335.
11. Dalager N, Kang H, Burt V: Non-Hodgkin's lymphoma among Vietnam-era veterans (abstract). *Am J Epidemiol* 1989; 130:815.
12. Centers for Disease Control Veterans Health Studies: Serum 2,3,7,8-tetrachlorodibenzo-p-dioxin levels in US Army Vietnam-era veterans. *JAMA* 1988; 260:1249–1254.

Endemic Giardiasis and Municipal Water Supply

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ABSTRACT

To test the hypothesis that endemic giardiasis may be transmitted by unfiltered municipal water supplies, the incidence of laboratory-confirmed giardiasis was studied in a natural experiment due to the arrangement of the public water supply of Dunedin, New Zealand. The incidence rate ratio was 3.3 (90% CI = 1.1, 10.1) for the population receiving unfiltered (microstrained) water relative to that using sand filtered water. In a parallel case-control study of incident cases, the odds ratio for giardiasis and unfiltered (microstrained) water supply was 1.8 (90% CI = 0.5, 6.9). (*Am J Public Health*. 1991; 81:760–762)

Introduction

Epidemics of waterborne giardiasis originating from water supplies have been clearly associated with surface catchments, treatment systems that do not eliminate *Giardia* cysts, and downstream contamination of the reticulation system.^{1–5} In contrast, there have been few tests of the hypothesis that surface catchment waters contribute to endemic giardiasis where treatment systems cannot eliminate cysts.^{1,6,7}

Giardia cysts have only recently been isolated from water supplies in New Zealand. No substantive epidemics attributable to giardiasis have yet been documented. Many municipal supplies use surface waters and simple treatment methods that would not reliably filter or deactivate giardia cysts.^{3,8}

The present study took advantage of a natural experiment—the divided water supply of the city of Dunedin, New Zealand—to test the hypothesis that endemic giardiasis might be transmitted by unfiltered municipal water supplies.

Methods

Dunedin (population 89,000) is supplied almost entirely by water from surface catchments. Most water is filtered by mechanical microstrainers (screen size 23 µm). Part of the city water is treated at a modern station (Mount Grand) using coagulation/flocculation and direct dual media filtration (anthracite and silica sand) that would normally be expected to remove any *Giardia* cysts present.^{3,8} All water supplies are chlorinated and fluoridated.

Records of all laboratory-proven cases of giardiasis from persons residing within the Dunedin municipal water sup-

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TABLE 1—Incidence of Giardiasis in Dunedin: Laboratory-Confirmed Primary Household Cases by Age and Sex*

Age (years)	Cases			Incidence Rate**
	Male	Female	Total	
0-4	5	3	8	7.8
5-14	0	2	2	0.9
15-34	11	15	26	4.3
35-64	5	5	10	2.1
65+	0	1	1	0.5
Total	21	26	47	2.9

*Three cases excluded from this table, as age or sex unspecified.
**Rate per 10,000 person-years.

TABLE 2—Incidence of Laboratory-Confirmed Giardiasis by Water Supply Filtration Method

Filtration Method	Cases*	Incidence**	Relative Risk (90% CI)
Direct dual media granular filtration	2	0.99	1.0 (reference group)
Microstrained	47	3.29	3.3 (1.1, 10.1)

*Primary household cases only. One case could not be classified, as domicile received a blend of both kinds of supply.
**Rate per 10,000 person-years.

TABLE 3—Giardiasis and Exposure to Microstrained Water: Case-Control Study

Exposed to Microstrained Water	Cases	Controls
Ever	19	53
Never	2	10
Odds Ratio (90% CI)	1.8 (0.5, 6.5)	

ply area were obtained from the two medical laboratories in the city for two periods (February to December 1986 and April 1987 to February 1988). Each record included age, sex, residential address, presence of *Giardia* or other pathogens, and the date of receipt of the fecal specimen. The laboratory diagnosis was made by direct microscopy following formalin-ethyl-acetate sedimentation and iodine staining. The denominators for rates were obtained from the 1986 Census of the usually resident populations of the respective water supply areas.⁹

A case-control study was also conducted using cases in the 1987-88 period. Controls were selected from the population referred for laboratory parasitology examination who had negative findings for giardiasis. Three controls were selected

per case; cases and controls were matched for age, sex, and season of occurrence of infection. Cases and controls were classified blindly by city council water engineers according to whether their residences were "ever" exposed to microstrained water during the presumed period of infection (defined as seven days to four weeks before the receipt of the fecal specimen).¹⁰

Relative risks, odds ratios, and confidence limits were computed using the programs of Rothman and Boice.¹¹

Results

Fifty primary household cases of giardiasis were diagnosed in the two study periods. Subsequent cases in a household were excluded from the analysis (13 cases). Four further cases were excluded through insufficient or absent information on domicile.

The incidence of laboratory-confirmed giardiasis was greatest in preschool children and in the 15-34 age group (Table 1). The incidence of laboratory-proven giardiasis for residents of the two water supply areas are given in Table 2. In the first study period, 28 of the 29 cases were residents in areas supplied by microstrained water; the remaining case was in an area traditionally supplied by a blend of the two

supplies. In the second study period, 19 cases resided in the microstrained areas and two cases in areas that received the Mount Grand supply. The relative risk of laboratory-confirmed giardiasis for residents of areas supplied by microstrained water was estimated at 3.3 (one-tailed $P = 0.04$). The risk ratio was essentially unchanged following indirect standardization for age.

In the case-control study the odds ratio for exposure to microstrained water was 1.8 (90% CI = 0.5, 6.5) (Table 3).

Discussion

The present findings suggest that endemic giardiasis can originate from the municipal water supply. Both the incidence estimates and the case-control analysis indicate a greater risk of laboratory-diagnosed giardiasis among residents of areas whose supply was treated only by mechanical filtration and chlorination.

The study was small and of correspondingly weak statistical power. As it was limited to the analysis of laboratory reports, information on some possible confounding factors was not available. However, some potential confounders are either unlikely to have had a significant effect, or would have operated as negative confounders. Age differences were not sufficient to account for differences in rates observed. The area with low rates included the most mobile young adult population. The two population areas may have differed in physician practice styles in the investigation of gastrointestinal symptoms, but this should have been accounted for by the case-control study. As most workplaces are supplied by Mount Grand water, misclassification from consumption of cold water at work would be expected to bias the relative risk toward unity. A small proportion of the controls may have had giardiasis (due to the intermittent nature of *Giardia* excretion) but this would bias the odds ratio toward unity.

The consistency of our findings with those of other case-control and descriptive studies^{1,6,7} strongly suggests that transmission of endemic giardiasis can occur via municipal water supplies that do not use filtration technology capable of removing *Giardia* cysts. □

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References

1. Craun GF: Surface water supplies and health. *J Am Water Works Assoc* 1988; 80:40-52.
2. Craun GF: Waterborne giardiasis in the United States 1965-1984. *Lancet* 1986; 2:513-514.
3. Jakubowski W: Purple burps and the filtration of drinking water supplies. (Editorial) *Am J Public Health* 1988; 78:139-143.
4. Kent GP, Greenspan JR, Herndon JL, et al: Epidemic giardiasis caused by a contaminated public water supply. *Am J Public Health* 1988; 78:139-143.
5. Jephcott AE, Begg NT, Baker IA: Outbreak of giardiasis associated with mains water in the United Kingdom. *Lancet* 1986; 2:730-732.
6. Chute CG, Smith RP, Baron JA: Risk factors for endemic giardiasis. *Am J Public Health* 1987; 77:585-587.
7. Birkhead G, Vogt RL: Epidemiological surveillance for endemic *Giardia lamblia* infection in Vermont. *Am J Epidemiol* 1989; 129:762-768.
8. Ongerth JE: Evaluation of treatment for removing *Giardia* cysts. *J Am Water Works Assoc* 1990; 82:85-96.
9. NZ Department of Statistics: 1986 New Zealand Census of population and dwellings, series B. Wellington: Department of Statistics, 1987.
10. Benenson AS (ed): *Control of Communicable Disease in Man*. Washington, DC: American Public Health Association, 1986.
11. Rothman K, Boice JD: *Epidemiologic analysis with a programmable calculator*. Boston: Epidemiology Resources, Inc, 1978.

The Microbiologic Quality of Drinking Water in North Carolina Migrant Labor Camps

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ABSTRACT

A two-year study of the microbiological quality of drinking water in 27 randomly selected North Carolina migrant labor camps yielded total and fecal coliform prevalences of 44 percent and 26 percent, respectively in 1988 and similar but higher prevalences in 1989. Preoccupancy testing by county sanitarians had found virtually no total coliform contamination. These findings suggest that a potential source of contamination existed and that current testing protocols which rely on preoccupancy testing may be inadequate. (*Am J Public Health*. 1991;81:762-764)

Introduction

Although migrant farm workers are known to have a high prevalence of enteric infections,¹⁻⁷ many of which are water-borne, the potability of drinking water used by migrants has received little attention. This report presents the results of a two-year study of the microbiological quality of drinking water in North Carolina migrant labor camps.

Methods

In 1988, a random sample of 21 migrant camps was selected from a pooled list of camps in eastern North Carolina. Water was also tested in six additional camps, which were part of a related study of the incidence of enteric parasites in migrant farm workers, and 10 control sites (nearby rural businesses and residences). In 1989, all 23 inhabited study camps from the previous year were retested, along with seven additional nonrandomly selected camps. In all camps in both years, water was tested by county health departments prior to occupancy, the results of which were obtained after field study was completed.

Water samples were collected from taps (without aerators) into sterile 1 liter sampling bottles, kept chilled and analyzed within 24 hours. Microbiological testing of 100 ml sample volumes for total and fecal coliforms and *E. coli* was done

according to standard protocols using membrane filter methods.⁸ Sites with initial positive tests were retested when possible.

Fisher's Exact Test (two-tailed) and Chi-square tests were employed in data analysis.

Results

1988 Results

The bacteriological results for water samples from the 27 camps and 10 control sites are summarized in Table 1; 12 (44 percent) were positive for total coliforms with counts ranging from 1-186 colony forming units (CFU). Eight of these sites were retested and all were positive again. Seven samples (26 percent) were also positive for fecal coliforms. Two of 10 samples were positive for *E. coli*. All 10 control sites were negative for total and fecal coliforms. The presence of latrines (as opposed to flush toilets) was associated with total coliform occurrence in water ($p = 0.024$; Fisher's Exact Test).

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