MAJOR ARTICLE

A Large, Travel-Associated Outbreak of Legionellosis among Hotel Guests: Utility of the Urine Antigen Assay in Confirming Pontiac Fever

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(See the editorial commentary by Edelstein on pages 229–31)

Background. During March 2004, a large outbreak of legionnaires disease and Pontiac fever occurred among hotel guests in Oklahoma. An investigation was conducted to identify the source and evaluate the utility of the *Legionella* urine antigen assay and serologic testing for the identification of Pontiac fever.

Methods. A retrospective cohort investigation of hotel guests and employees and an environmental evaluation were performed. Participants were interviewed, and clinical specimens were collected from consenting individuals.

Results. Six cases of legionnaires disease and 101 cases of Pontiac fever were identified. Exposure to the indoor pool and hot tub area was associated with legionellosis (relative risk, 4.4; 95% confidence interval, 2.8–6.9). Specimens from the pool and hot tub tested positive for *Legionella pneumophila* serogroup 1 by polymerase chain reaction. For Pontiac fever, the sensitivity and positive predictive value were 35.7% and 100%, respectively, for the urine antigen assay, and 46.4% and 90%, respectively, for serologic testing. The specificity and negative predictive value were 100% and 47.8%, respectively, for the urine antigen assay, and 89.3% and 45.5%, respectively, for serologic testing.

Conclusions. Urine antigen testing, with or without serologic testing, can be used to confirm outbreak-associated cases of Pontiac fever caused by *L. pneumophila* serogroup 1.

Legionellosis can present as 2 distinct clinical entities: Pontiac fever, a self-limited, nonpneumonic, flulike illness; and legionnaires disease, a more severe form involving pneumonia [1–5]. Between 8000 and 18,000 persons are hospitalized in the United States each year with legionnaires disease [6]. Legionellae are ubiquitous in natural and manmade aquatic environments and multiply in the presence of warm water temperatures (25°C–42°C) inside free-living amoebae [1, 7]. Sources previously identified in outbreak investigations include hot tubs, building air-handling systems, decorative fountains, and industrial operations [2, 8–26].

It is important to recognize isolated cases of legionellosis, because they may lead to the identification of more cases of Pontiac fever or legionnaires disease [9, 14]. In outbreaks of infection in which a point source is identified rapidly, environmental water samples can be tested to confirm the presence of legionellae, although laboratory confirmation of human disease has been much more challenging. Serologic testing has been used in previous outbreak investigations to confirm that *Legionella* species are the etiologic agent. The *Legionella* urinary antigen test is the most widely used diagnostic test for diagnosis of legionnaires disease; however, it has not been useful in previous outbreaks of Pontiac fever [11–14, 19, 24, 26–29]. This test has important implications for control of outbreaks of legionnaires disease, because it allows for rapid diagnosis and iden-

Received 1 May 2006; accepted 19 September 2006; electronically published 8 December 2006.

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Clinical Infectious Diseases 2007; 44:222–8

1058-4838/2007/4402-0010

tification of *Legionella pneumophila* serogroup 1 (Lp1), the most common serogroup identified among patients.

On 20 March 2004, Oklahoma public health officials were notified of a cluster of persons who were ill with symptoms of fever, myalgias, shortness of breath, and fatigue, including 1 hospitalized person with a positive *Legionella* urinary antigen test result. All ill persons were attending a national youth basketball tournament and were guests at the same hotel during the week of 15 March through 21 March 2004. We investigated this outbreak of travel-associated legionellosis, which occurred among a well-defined cohort. A retrospective cohort study was conducted to identify the source(s) of transmission for *Legionella* infection, to institute measures to prevent additional cases, and to evaluate the sensitivity and specificity of the urinary antigen assay and the serologic indirect fluorescent antibody test for the diagnosis of Pontiac fever.

METHODS

Case definitions. A confirmed case of legionnaires disease was defined as radiography-confirmed pneumonia and laboratory-confirmed *Legionella* infection in a hotel guest or employee who had an onset of illness within 10 days after visiting the hotel. Laboratory confirmation consisted of at least 1 of the following events: isolation of *Legionella* from lung tissue, respiratory secretion, pleural fluid, blood, or other sterile site specimen; detection of Lp1 antigen in urine; demonstration of a $\geqslant$4-fold increase in the antibody titer to at least 1:128 between acute- and convalescent-phase serum specimens; or demonstration of an antibody titer $\geqslant$1024. A probable case of legionnaires disease was defined as radiography-confirmed pneumonia, absence of laboratory confirmation of *Legionella* infection, and at least 1 reported symptom (fever, cough, shortness of breath, or wheezing) in a hotel guest or employee who had onset of illness within 10 days after being at the hotel. A case of Pontiac fever was defined as documented or subjective fever or chills and at least 1 other symptom (headache, cough, shortness of breath, muscle aches, vomiting, or diarrhea) in a hotel guest or employee who had onset of illness within 3 days after visiting the hotel.

Retrospective cohort study. The cohort study included persons registered at the hotel at least 1 evening and employees who worked at least 1 shift from 15 March through 22 March. A standardized questionnaire was administered to obtain demographic data, to identify potential sources for exposure, and to elicit information about the clinical characteristics of the illness.

We requested urine and paired serum samples from consenting symptomatic and asymptomatic persons. Acute-phase serum specimens were collected from symptomatic persons within 14 days after symptom onset and within 14 days after hotel departure for asymptomatic persons. Convalescent-phase serum specimens were obtained 3–6 weeks after acute specimen collection. Urine specimens were collected from symptomatic persons within 10 days after symptom onset and within 10 days after hotel departure for asymptomatic persons. Serum and urine specimens were tested at the Centers for Disease Control and Prevention (CDC; Atlanta, GA).

Environmental investigation. A standard pool inspection was conducted, which included a review of the facility's operation and maintenance records, as well as water quality testing of the pool and hot tub. We measured bromine concentrations and pH levels and interviewed the pool service representative contracted by the hotel regarding recent pool and hot tub maintenance activities.

Water samples and biofilm swab specimens were collected on 22 March from multiple sites, including the swimming pool, the hot tub, and the dehumidifier unit. Specimens were placed in sterile containers and submitted to the Oklahoma State Department of Health Public Health Laboratory (Oklahoma City, OK). All specimens were plated on standard growth media for the cultivation of *Legionella* species [30]. Specimens were also forwarded to the CDC and tested for the presence of *Legionella* species using direct fluorescent antibody testing and PCR, because conditions for environmental specimens were suboptimal for culture as a result of high levels of disinfectant in the water at the time of collection. Biofilm specimens taken from the cartridge filter of the hot tub were examined by direct fluorescent antibody testing. The specimen was stained with an antibody specific to Lp1 and examined microscopically for the presence of the bacteria. Environmental specimens were extracted using the Qiagen DNA kit and tested using the TaqMan PCR, with primers and probes specific for *Legionella* species [31].

Clinical laboratory methods. Serum samples were tested at the CDC using an indirect immunofluorescent antibody assay for combined IgG, IgM, and IgA antibodies using an antigen of Lp1 [32]. Urine specimens were tested at the CDC for Lp1 antigen using the Binax ELISA immunoassay in accordance with manufacturer instructions.

Statistical analysis. Data were entered into a Microsoft Access 2000 database, and analyses were performed with EpiInfo, version 3.03 (CDC), and SAS software, version 9.1 (SAS Institute). Relative risks (RRs) and 95% CIs were calculated for exposures. Age group differences and duration of exposures were compared using the Wilcoxon rank sum test. Dose-response relationships were assessed using the χ^2 test for trend analysis. Multivariate logistic regression was performed and included all statistically significant ($P < .05$) variables identified during univariate analysis.

Sensitivity was defined as the proportion of positive test results among persons whose cases met the definition for Pontiac fever. Specificity was defined as the proportion of negative test results among persons whose cases did not meet the Pontiac

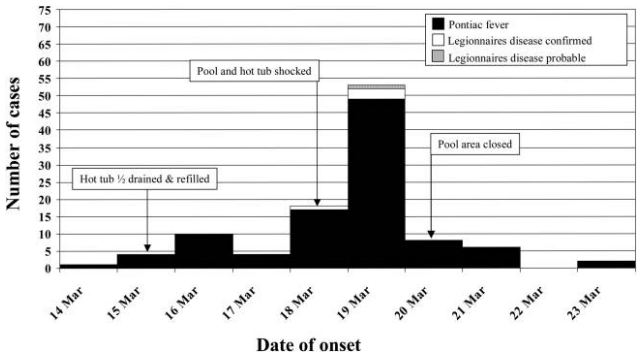

Figure 1. Illness onset for 107 patients with legionellosis and pool maintenance timeline at an Oklahoma hotel, March 2004.

fever case definition. The positive predictive value was calculated as the proportion of Pontiac fever cases among the total number of positive test results. The negative predictive value was calculated as the proportion of non–Pontiac fever cases among the total number of negative test results.

RESULTS

Case finding. We contacted 144 (80%) of the 180 hotel registrants. Thirty-six (20%) could not be interviewed because of insufficient or inaccurate information. Questionnaires were administered to 317 people, including registrants ($n = 113$), guests who had stayed with a registrant ($n = 197$), and hotel employees ($n = 7$). A total of 107 persons' cases met the case definitions, including 5 persons with confirmed legionnaires disease, 1 person with probable legionnaires disease, and 101 persons with Pontiac fever (overall attack rate, 33.7%). Patients reported that the onset of symptoms occurred during the period from 14 March through 23 March (figure 1); 49.5% of onsets occurred on 19 March. Patients were residents of 8 states: 61 (57%) were from Texas, 29 (27.1%) were from Oklahoma, 10 (9.3%) were from Indiana, 2 (1.9%) were from Missouri, and 1 each was from Arkansas, Illinois, Michigan, Minnesota, and North Dakota.

The median age of persons with Pontiac fever was lower than that for persons without legionellosis ($P < .05$; table 1). Symptoms reported among persons with Pontiac fever included fever (90%), fatigue (90%), headache (90%), chills (87%), muscle aches (78%), cough (65%), shortness of breath (52%), vomiting (36%), and diarrhea (28%). Duration of illness ranged from 1 to 9 days, with a median duration of 4 days. The number of days of normal activities lost as a result of illness ranged from 0 to 12 days, with a median duration of 7 days. Twenty-seven patients with Pontiac fever underwent chest radiography; none had evidence of pneumonia. Three patients with Pontiac fever (2.9%) were hospitalized. One patient with legionnaires disease was hospitalized, and none died.

Retrospective cohort study. Univariate analysis of hotel exposures revealed several pool-related activities associated with legionellosis (legionnaires disease and Pontiac fever). Any visit to the indoor pool area was associated with an increased risk of legionellosis, compared with no visit to the pool area (RR, 4.4; 95% CI, 2.8–6.9). Persons who spent time in the pool area (RR, 3.4; 95% CI, 2.5–4.6), used the swimming pool (RR, 3.2; 95% CI, 2.4–4.4), or used the hot tub (RR, 2.8; 95% CI, 2.1–3.8) were more likely to become ill than were those who did not.

The risk of legionellosis increased with duration of exposure to the hotel pool area, time spent swimming, or time using the hot tub (table 2). The median time of cumulative pool exposures was 2 h, 24 min among patients with Pontiac fever and 0 h (mean duration, 28 min) among persons without legionellosis ($P < .05$). Compared with patients who had Pontiac fever, patients with legionnaires disease spent a significantly greater

Table 1. Demographic characteristics of patients with legionnaires disease, patients with Pontiac fever, and patients without legionellosis, Oklahoma, March 2004.

Characteristic	Patients with Pontiac fever ($n = 101$)	Patients with legionnaires disease ($n = 6$)	Patients without legionellosis ($n = 210$)
Age, median (range)[a]	15 (2–65)	6.5 (2–44)	36 (11 months–82 years)
Male sex	44 (43.6)	6 (100)	111 (52.9)
Race			
Asian	3 (3)	0 (0)	1 (0.5)
Black	1 (1)	0 (0)	8 (3.8)
Native American	3 (3)	0 (0)	10 (4.8)
Other	1 (1)	0 (0)	5 (2.4)
White	92 (91)	6 (100)	180 (85.7)
Hispanic ethnicity	1 (1)	0 (0)	0 (0)

NOTE. Data are no. (%) of subjects, unless otherwise indicated.

[a] Age is in years, unless otherwise indicated.

Table 2. Univariate dose response association of hotel exposures and legionellosis, Oklahoma, March 2004.

Exposure	No. (%) of patients	No. (%) of subjects without legionellosis	OR	χ^2	*P*
Duration of exposure to the pool/hot tub area					
None	41 (38.3)	176 (84)	1.00		
≤2 h	27 (25.2)	28 (13)	4.14		
>2 h	39 (36.4)	6 (2.8)	27.90	83.05	<.01
Duration of time swimming in hotel pool					
None	44 (41.1)	173 (82)	1.00		
≤2 h	48 (44.9)	28 (13)	5.55		
>2 h	15 (14)	6 (2.8)	19.66	59.01	<.01
Duration of hotel hot tub use					
None	60 (56)	177 (84.3)	1.00		
≤1 h	42 (39.2)	29 (13.8)	4.27		
>1 h	5 (4.6)	4 (1.9)	3.69	26.21	<.01
Duration of pool exposures combined[a]					
None	19 (17.8)	137 (65.2)	1.00		
≤2 h	21 (19.6)	60 (28.6)	2.52		
>2 to 4 h	39 (36.4)	10 (4.8)	28.12		
>4 h	28 (26.2)	3 (1.5)	67.30	114.91	<.01
Duration of exposure to computer room					
None	75 (85.2)	183 (89.3)	1.00		
≤1 h	10 (11.4)	17 (8.3)	1.44		
>1 h	3 (3.4)	5 (2.4)	1.46	0.85	.35
Duration of exposure to breakfast area					
None	61 (65.6)	108 (53.7)	1.00		
≤1 h	19 (20.4)	79 (39.3)	0.43		
>1 h	13 (13.1)	14 (7)	1.64	0.343	.55
Duration of exposure to fitness area					
None	93 (92)	191 (93)	1.00		
≤1 h	5 (4.9)	12 (5.9)	0.86		
>1 h	3 (2.9)	2 (0.9)	3.08	0.5	.45

[a] Pool exposures combined includes exposure to the pool room, swimming in the hotel pool, and/or using the hotel hot tub.

amount of time in the pool area (median duration, 6 h, 17 min; $P < .05$). Length of time spent in other common areas of the hotel was not associated with legionellosis.

In multivariate analysis, increasing duration of exposure to the pool area and swimming were independently associated with increasing risk of legionellosis (table 3). Each hour spent in the pool area increased the odds of illness by 3.9 times (95% CI, 2.5–6.2). Each hour spent using the swimming pool increased the odds of illness by 1.6 times (95% CI, 1.1–2.3).

Laboratory results. Urine specimens were collected from 89 participants (28%) (table 4). Of 56 urine specimens collected from patients with Pontiac fever, 20 yielded a positive urinary antigen test result (sensitivity, 35.7%), whereas all 33 urine specimens submitted by persons without legionellosis yielded negative urinary antigen results (specificity, 100%; $P < .01$). The positive predictive value of the urinary antigen assay was 100%, and the negative predictive value was 47.8%.

Acute- and convalescent-phase serum specimens were collected from 84 participants (26.5%) (table 4). Of 56 paired serum specimens collected from patients with Pontiac fever, 26 had positive antibody titer test results (sensitivity, 46.4%), whereas 3 of 28 collected from persons without legionellosis yielded positive antibody titer test results (specificity, 89.3%) ($P < .01$). The positive predictive value of a positive antibody titer test result was 90%, and the negative predictive value was 45.5%.

Forty-five patients with Pontiac fever provided both serum and urine specimens; 10 (53%) of 19 cases with a positive serologic test result also had a positive urinary antigen test result. Eighty-four percent of persons with Pontiac fever who had a negative serologic test result also had a negative urinary antigen test result. Twenty-six persons without legionellosis provided both serum and urine specimens; the urinary antigen test result was negative for both of the persons without le-

Table 3. Risk factors associated with legionellosis, Oklahoma, March 2004.

Exposure	Adjusted OR (95% CI)	*P*
Age (per year)	0.98 (0.96–1.00)	.06
Time[a] in the pool area	3.9 (2.46–6.19)	<.05
Time[a] swimming	1.61 (1.11–2.33)	<.05
Time[a] in the hot tub	2.19 (0.89–5.41)	.08
Time[a] in the breakfast area	0.70 (0.33–1.48)	.36

NOTE. The multivariate model included variables that were significantly associated with illness, including age, exposure to the pool area, swimming in the pool, using the hot tub, and exposure to the breakfast area.

[a] ORs are based on 1-h increments of time.

gionellosis who had positive serologic results and for all 24 of those who had negative results. Participants who provided specimens were significantly younger (median age, 16 years; range, 2–72 years) than those who did not provide specimens (median age, 35 years; range, 11 months to 82 years; $P = .0012$); however, this age difference was similar to the overall age distribution for patients with Pontiac fever versus persons without legionellosis.

Environmental investigation results. Disinfection of the pool and the hot tub was achieved by the addition of bromine tablets through an erosion feeder. According to Oklahoma regulations [33], a bromine concentration of 2–4 ppm and a pH level of 7.2–7.8 are required. The pool and hot tub were in heavy use during 15–20 March, because several large groups stayed at the hotel. Water quality was not monitored on a daily basis by hotel staff as required. On 15 March, the bromine concentrations in the pool and hot tub were 0.5 ppm and 2.5 ppm, respectively. On 18 March, there was no measurable bromine in either the pool or the hot tub, so both were hyperbrominated then shocked with lithium hypochlorite. An inspection conducted by investigators on 20 March found that bromine levels for both the pool and hot tub measured >50 ppm, which is 12.5 times higher than the maximum required level of 4 ppm. The pH levels in the pool and hot tub were measured at 7.1 and 5.7, respectively.

An environmental assessment of the facility revealed several problems with the ventilation and maintenance of the pool and hot tub area. The pool and spa filters were inappropriately cleaned, and the system monitoring the feed rates for water filtration and disinfection was found to be inaccurate. The ventilation system was not operating to the designed level of efficiency, resulting in inadequate airflow exchange. Direct fluorescent antibody testing of a biofilm swab specimen taken from a cartridge filter from the hot tub indicated the presence of a large number of *L. pneumophila* organisms. In addition, the following specimens tested positive for *L. pneumophila* by PCR: dehumidifying unit condensate, swab specimens from and a section of the hot tub filter, a swab of biofilm on the lid of the hot tub skimmer, and the neutralized pool water. All culture results were negative.

DISCUSSION

On the basis of our case definition of Pontiac fever, the sensitivity of the urinary antigen assay was 35.7%, and the specificity was 100%. In contrast to legionnaires disease, for which culture and the urine antigen assay are the acceptable gold standards, there is no gold standard for the diagnosis of Pontiac fever. Therefore, we relied on a necessarily nonspecific case definition, which likely led to a low estimated sensitivity of the urine antigen test. In future outbreaks of suspected legionellosis, the urine antigen test can be used to confirm that the outbreak is consistent with Pontiac fever caused by Lp1, but it should not be required to make the diagnosis in individual patients.

In the reported outbreaks of Pontiac fever, investigators confirmed the etiologic agent by a 4-fold increase in serologic titers among case patients or by isolation of *Legionella* species from environmental specimens. The sensitivity of serologic testing in this investigation was 46.4%, whereas the specificity was 89.3%. Among investigations in which a 4-fold increase in an-

Table 4. Comparison of frequencies of *Legionella pneumophila* serogroup 1 urinary antigen results and antibody titer results among patients with Pontiac fever and persons without legionellosis, Oklahoma, March 2004

	No. (%) of results					
	Patients with Pontiac fever			Persons without legionellosis		
Test	Positive	Negative	Total	Positive	Negative	Total
Urinary antigen testing ($n = 89$)[a]	20 (35.7)	36 (64.3)	56 (100)	0 (0)	33 (100)	33 (100)
Antibody titer test ($n = 84$)[b]	26 (46.4)	30 (43.6)	56 (100)	3 (10.7)	25 (89.3)	28 (100)

[a] A comparison of urinary antigen results between the 2 groups yielded the following results: $\chi^2 = 15.2$; $P < .01$; relative risk, 1.9 (95% CI, 1.5–2.4).

[b] Positive antibody titer result was defined as a ⩾4-fold increase (⩾128) in *Legionella* serogroup–specific antibody titer between acute- and convalescent-phase serum specimens. A comparison of antibody titer results between the 2 groups yielded the following results: $\chi^2 = 10.5$; $P < .01$; relative risk, 1.6 (95% CI, 1.2–2.1).

tibody titers was observed, seroconversion rates ranged from 11% to 100% [2, 3, 8–13, 18–20, 23, 25]. In this study, 4-fold increases were observed in paired serum samples obtained from 3 individuals without legionellosis. We hypothesize that these individuals represent acute exposure to *Legionella* species in the absence of clinical symptoms. Antibodies to *Legionella* species were detected in healthy persons who had been exposed to the implicated source in several previously documented outbreaks of legionellosis [13, 14, 18, 25].

The retrospective cohort study and environmental testing identified the hotel's indoor pool and hot tub area as the source of the outbreak. Because both the pool and the hot tub were contained in the same small area, exposures to either source were probably not entirely exclusive of each other. However, given that legionellosis is acquired through inhalation of aerosols, we believe that the hot tub was the most likely source of this outbreak. Importantly, immersion in the hot tub was not required for illness to develop. Epidemiologic findings of the retrospective cohort study were supported by environmental assessments that indicated a lack of daily monitoring and maintenance of the pool and hot tub. Laboratory testing of environmental samples confirmed the presence of *L. pneumophila.* Inadequate maintenance of the hot tub most likely compromised disinfectant concentrations, and this provided an opportunity for *Legionella* growth and transmission. Compliance with pool and hot tub maintenance regulations is necessary to prevent legionellosis.

Previous outbreak investigations have reported limited success in detecting Lp1 using the urine antigen assay among persons with Pontiac fever [11–14, 19, 24, 26, 28, 29]. It has been hypothesized that Pontiac fever may be caused by hypersensitivity to a cellular component of nonviable *Legionella* species [34]. Other theories regarding the cause of Pontiac fever and the marked differences in epidemiology, compared with legionnaires disease, include exposure to endotoxins produced from viable or nonviable *Legionella* species or a protozoan host of the bacteria [12, 29]. The peak in onset of illness occurred the day after shock bromination of the hot tub. Many of these cases were *Legionella* urine antigen test positive, suggesting that exposure to a large quantity of nonviable organisms can produce antigenuria. However, use of the indoor pool area exceeded bather load restrictions during the week, and most cases reported exposure to the pool area over multiple days. Therefore, prolonged exposure to live and nonviable organisms throughout the outbreak period may have resulted in the ability to detect antigenuria in patients with Pontiac fever.

There were limitations to our study. We did not have clinical and environmental *Legionella* isolates available for species or serogroup comparison or molecular characterization. All environmental culture results were negative; however, conditions for specimens were suboptimal because of the high levels of bromine at the time of specimen collection, thus decreasing the likelihood of obtaining a positive culture result.

Outbreaks of legionellosis are often detected by identifying links among individual cases in a community. Because travelers staying in hotels become ill after returning home to many different communities, individual cases may not be readily linked to the common source exposure. Physicians may not suspect the diagnosis of Pontiac fever among patients with influenza-like illness or the diagnosis of legionnaires disease among individual cases of pneumonia. Because a single case of legionellosis reflects an environmental source to which other persons can be exposed, a travel history should be obtained from all patients with symptoms consistent with legionellosis. Physicians assessing patients with respiratory illness and recent travel should consider legionellosis in the differential diagnosis. Finally, because of the importance of rapid detection of outbreaks of travel-associated legionellosis, state health departments, the Council of State and Territorial Epidemiologists, and the CDC have recently collaborated to improve the detection and reporting of cases of travel-associated legionellosis (http://www.cste.org).

Recognition of cases of Pontiac fever is essential for disease prevention; even 1 case may be the sentinel event that leads to the recognition of other cases and the contaminated point source. Identification of the source and prompt remediation will prevent additional cases of Pontiac fever. Because outbreaks of Pontiac fever and legionnaires disease have been identified together, identification of cases of Pontiac fever can lead to the identification of cases of legionnaires disease—the clinically more important presentation of legionellosis [35]. Although testing of several affected individuals may be required because of the test's low sensitivity, our findings demonstrate that urine antigen testing, with or without serologic testing, may assist public health officials in confirming outbreak-associated cases of Pontiac fever.

LEGIONELLOSIS OUTBREAK INVESTIGATION TEAM

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Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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