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Editor's Note: This special issue features *Journal* articles that were pre-published digitally in 2012 on NEHA's Members Only Web site.

ABOUT THE COVER

"State Public Health Laboratory Biomonitoring Programs: Implementation and Early Accomplishments," our cover feature this month, discusses the implementation of three Centers for Disease Control and Prevention–funded state pilot biomonitoring programs. The authors interviewed program officials in each state (California, New York, and Washington) to determine challenges, successes, and lessons learned in order to prepare for an eventual National Biomonitoring Plan. The authors point out that biomonitoring is an important tool as environmental health moves forward to address questions such as lifetime exposures, health impacts of chemical mixtures, and cumulative risks.

See page 90.

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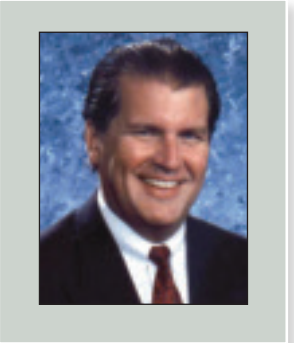


Brian Collins,
MS, REHS, DAAS

Lessons Learned for Future Leaders

NEHA published two outstanding articles in 2006 and 2007 titled, "Profiles in Leadership, Part I : *JEH* Goes on a Quest," and "Profiles in Leadership, Part II: The 15 Faces of Environmental Health Leadership." (*Journal of Environmental Health*, *69*[5], December 2006; *Journal of Environmental Health, 69*[6], January/February 2007). Author Rebecca Berg, PhD, did a fabulous job in a "quest" to find out what leadership means to environmental health practitioners and then put a face to the "profile."

At the time Dr. Berg interviewed me for the article, I tried to rationalize why those of us in environmental health struggle to become leaders. I was quoted as saying, "Many people who go into science, candidly, are not exactly well versed in people skills. They chose science because it's finite. There's an adventure and discovery and an outcome. And they haven't necessarily had to depend on others in those relationships to make things happen." My focus at the time was that leaders in our profession needed people skills in addition to a strong technical background.

Even before that interview and certainly since, I have been a student of leadership. Attempting a degree of modesty, I have been enabled to "practice" leadership in various environments. Armed with education and experience, I can now attest that there are certain qualities or attributes that facilitate leadership thought and practice. Here are some lessons learned for aspiring and future leaders.

A number of years ago I read an article by Carl Neu, an executive vice president and general manager of a company that provides resources and services for local governments. The article was entitled, "Leadership: Awakening the Best in People" (*Texas Town and City*, February 2003). Many of its vignettes stayed with me.

> *Leadership will define our success and the success of our profession.*

In his article, Neu stated that leaders engage people by touching the imagination and consciousness of others. He described this as reaching out to people to bring them into a community. I believe he was describing the active use of **vision**: communication of a clear, succinct picture that provides not only the goal or endgame, but also a path as to how to attain the vision. He was describing a leadership tool. Establishing a vision requires incorporating it into the very fabric of the community, organization, or team. It requires buy in and commitment from the top down and bottom up.

Leaders must inspire themselves and others to achieve the vision—to make things happen—to change. Leaders must have **passion**! You generally cannot inspire others if you are not bought in and almost evangelical in your communication and actions. Those whom you lead must know you are taking them to a better place, personally and professionally.

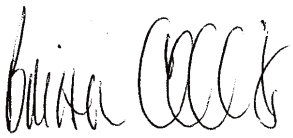
In the September 2012 issue of the *JEH*, I wrote of **ethics** and **integrity**. It bears repeating that leaders require a higher standard of honesty and integrity than that expected of others. I expect honesty, integrity, and ethical behavior from those whom I choose to follow and in turn, team members expect it of me and I expect it of myself!

Leaders are facilitators that empower and support **teamwork**. Most people cannot achieve what they want or need by themselves. Leaders need the contributions of others and leaders need teams to get things done. Well-composed teams are generally comprised of those who have similar drive, attitudes, and skills. They are bought in and committed to the vision. They are also generally competitive—everyone wants to be on the best team!

Leadership and leading means one must **embrace change**. In fact, leadership is all about change! There is no need for leadership if no change is needed. (I think that's called a paraprosdokian—a figure of speech in which the latter part of a sentence or phrase is surprising or humorous. Winston Churchill was a master of paraprosdokians!) Embracing change in this day and time also means **embracing technology**. It is not necessary to be a technical or technology expert. It is important to understand that technology and the evolution of technology will get and keep you in the game!

Finally, I believe **emotional intelligence** (EI) is critical to leadership. Daniel Goldman of Rutgers University wrote about EI in *Harvard Business Review* in his 1998 article, "What Makes a Leader." Goldman proposed that "IQ and technical skills are important, but emotional intelligence is the *sine qua non* (indispensable or essential characteristic) of leadership." He described five components of EI: (1) self-awareness—an ability to recognize and understand your moods, emotions, and drives, and their impact on others; (2) self-regulation—the ability to control or redirect behaviors and actions that are not constructive and the ability to think before acting; (3) motivation—passion and energy that goes beyond money and status; (4) empathy—listen to and understand others and treat them appropriately; and (5) social skills—managing relationships, building networks, and finding common ground.

I believe if we incorporate these attributes, and let experience serve as lessons for thought and practice, the type of leadership we provide as environmental health professionals, no matter the venue in which we practice, will define our success and the success of our profession.



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The 2013 Walter F. Snyder Award will be presented during NEHA's 77th Annual Educational Conference (AEC) & Exhibition to be held in Washington D.C., July 9 - 11, 2013.

For more information or to download nomination forms, please visit www.nsf.org or www.neha.org or contact Stan Hazan at NSF at 734-769-5105 or hazan@nsf.org.

Fish Consumption Patterns and Mercury Exposure Levels Among Women of Childbearing Age in Duval County, Florida

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Abstract **Consumption of fish containing methylmercury can pose serious health concerns including neurotoxic effects in adults and toxicity to the fetuses of mothers exposed during pregnancy. In the study described in this article, the authors examined fish consumption patterns and measured hair mercury levels of women of childbearing age in a coastal county in Florida. Women from the community participated in a risk factor assessment survey (*N* = 703). Hair samples (*n* = 698) were collected and analyzed for mercury. The authors identified 74.8% below detection limit; 25.2% had detectable limits of mercury, while 7% exceeded 1 µg/g. Hair mercury levels increased with fish consumption and age. Race, income, and education levels were also associated with increased hair mercury levels. Women of Asian/Pacific Islander origin had the highest levels. Although reported fish consumption exceeded the recommendations for women of childbearing age, the study population had lower mercury levels than other comparative studies in Florida and at national levels.**

Introduction

Fish is a rich source of protein and other essential nutrients including omega-3 fatty acids. Because of its nutritional properties, fish should be considered part of a healthy diet. Fish and shellfish contaminated with methylmercury, however, can raise serious human health concerns particularly for women of childbearing age, nursing mothers, pregnant women, and their developing fetuses (Agency for Toxic Substances and Disease Registry, 1999).

Florida offers the longest saltwater coastline of any state in the continental U.S., providing abundant opportunities for fresh, local fish consumption by its residents and visitors. From 2000 to 2008, however, the National Atmospheric Deposition Program (NADP) consistently ranked Florida as the leading state in the southeast with the highest mercury wet deposition and rainfall (NADP, 2008). These high mercury levels can be washed out of the atmospheric environment into streams, lakes, and oceans, converted by microorganisms into methylmercury and biomagnified in the food chain. Human fish consumption and mercury exposure to particularly vulnerable populations such as pregnant women or women of childbearing age are concerns that warrant further epidemiologic exploration.

As part of an environmental public health surveillance project, we identified the potential risks of mercury exposure by evaluating fish consumption habits and measuring hair mercury levels in women of childbearing age (18–49 years) in a northeastern coastal county in Florida.

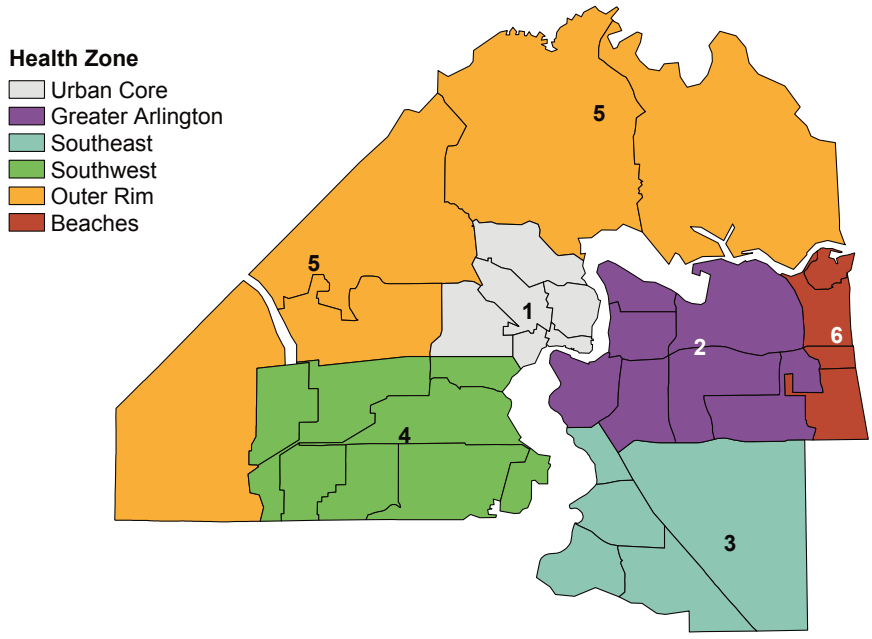
Background

Over the past decade, concerns have been growing over mercury exposure related to fish consumption in the U.S. In 2007, a report issued by the U.S. Environmental Protection Agency (U.S. EPA) indicated that approximately 80% of fish consumption advisories in the U.S. were related to mercury, with 35 states having statewide advisories due to mercury (U.S. EPA, 2007). In August 2009 the U.S. Geological Survey marked its most comprehensive examination of mercury contamination in fish, bed sediment, and water from streams across the U.S. and found that the fish mercury concentration exceeded the U.S. EPA human health criterion of 0.3 µg/g (3 parts per million) weight at 27% of the sampled sites. The mercury levels were high enough to exceed what U.S. EPA considers a safe average for humans who eat fish (Scudder et al., 2009).

Numerous studies have measured mercury levels in both fish and humans from various regions throughout the U.S. and the world. Several of these studies suggest that mercury exposure varies widely and is influenced by factors including population demographics, cultural fish consumption habits, local sources of pollution, and commonly eaten fish species (Oken et al., 2005).

FIGURE **1**

Health Zones in Duval County



Prepared by: Duval County Health Department/Institute for Public Health Informatics and Research, October 2012

The Florida Department of Health (FDOH) periodically publishes fish advisory guidelines to alert consumers about the possibility of chemically contaminated fish in Florida waters. The advisories are meant to inform the public of potential health risks of specific fish species from specific water bodies. In 2004, 218 statewide advisories were issued for fresh, coastal, and other water bodies. In 2009, FDOH issued a fish advisory for mercury on 27 different fish species, located in all coastal waters, Florida Bay, Biscayne Bay, or Florida Keys (Florida Department of Health [FDOH], 2009a).

Florida receives much of its mercury deposition from both domestic and global sources. The southeastern states may be more heavily influenced by long-range global transport resulting from large convective summer storms that scavenge mercury from the middle and upper troposphere (Butler et al., 2008). Chalmers and co-authors note increasing Asian mercury emissions may have a greater impact in the southeastern U.S. than elsewhere in North America (Chalmers et al., 2010).

The greatest source of mercury in the environment is industrial pollution. Mercury is released by several types of industrial facilities including waste incinerators, coal-fired power plants, chlorine plants, and auto scrap processing facilities. Coal-burning power plants are the largest contributors of mercury air pollution in the U.S. accounting for over 50% of total domestic human-made mercury emissions (U.S. EPA, 2005). These plants release an estimated 51.6 tons of mercury into the air each year. Data from a 2005 report published by the U.S. Public Interest Research Group ranks Florida as 11th highest in the nation for power plant mercury emissions, with 91% of mercury air emissions resulting from power plants. Within the state of Florida, Duval County ranked as having the highest mercury emissions from power plants in the state (U.S. Public Interest Research Group, 2005).

Duval County is located along the coast in northeast Florida. It is a popular destination primarily because of its abundant commercial and recreational fishing opportunities. Because of the consistent availability of year-round fresh fish, fish consumption among coastal communities is generally higher than in inland areas (Knobeloch, Anderson, Imm, Peters, & Smith, 2005). According to a recent study, the southeastern portion of the country has been experiencing upward trends of mercury in fish compared to other regions of the U.S. (Chalmers et al., 2010).

The popularity of fishing in Florida, coupled with high mercury emissions in the southeast, raises concerns about consumption of fish with elevated mercury contamination among vulnerable populations of Duval County residents, specifically women of childbearing age. Based on these facts, environmental public health officials in Florida were interested in assessing fish consumption patterns of vulnerable populations.

This project was conducted as part of Florida's ongoing surveillance activities under its federally funded Centers for Disease Control and Prevention Environmental Public Health Tracking Program. More specifically, we carried out this surveillance project to evaluate fish consumption patterns of women of childbearing age (18–49 years) who were residents of Duval County. The aim of the activity was twofold: 1) to gain a better understanding of the mercury levels of the participants by measuring and evaluating baseline mercury levels of hair samples in the aforementioned selected women, and 2) to identify which groups of women were at an increased risk for mercury exposure with expectations to develop future intervention measures, such as educational and outreach mechanisms towards specific populations.

Methods

Participant Selection

The FDOH institutional review board approved our study on April 14, 2009. Recruitment took place from May to July 2009. Eligibility criteria required that participants be 1) a woman of childbearing age (18–49 years) and 2) a resident of Duval County, Florida, for at least one year. In order to capture a representative demographic and socioeconomic cross-sectional sample, recruitment was conducted throughout the six health zones (HZs) of the city of Jacksonville. As shown in Figure 1, zones include the urban core (HZ 1), greater Arlington (HZ 2), southeast (HZ 3), southwest (HZ 4), outer rim (HZ 5), and beaches (HZ 6).

Convenience sampling was used to recruit women from each HZ, as these zones represent distinct geographical areas of the city. Recruitment announcements were posted in county health department (CHD) clinics, private physician offices, local libraries,

newspapers, agency Web sites, women's fitness centers, beauty salons, and other local businesses in Duval County. In addition, participants were actively recruited at county health department clinics; Women, Infants, and Children (WIC) offices; health fairs; and community events throughout Duval County.

Questionnaire and Hair Sampling

A study questionnaire was developed by a group of state and local health department staff including toxicologists, environmental health specialists, and survey design consultants. The survey was tested among a small group of local women.

The questionnaire collected data on demographics, socioeconomic information, pregnancy status, mercury exposure history (unrelated to fish consumption), knowledge of fish consumption advisories, and fish consumption habits (frequency, type, and source). To assess recent consumption of high-risk fish (one or more meals in 60 days), species high in mercury according to state and federal advisories were listed by name. A fish identification chart was provided to assist in recognizing each fish species.

Eligible participants signed an informed consent form and completed the self-administered questionnaire. Interpreters assisted Spanish-speaking participants by verbally translating English language study documents. Each participant also provided a scalp hair sample (approximately 50 mg) for mercury analysis. Samples were collected by CHD personnel on site at field locations behind a privacy screen. The hair sample was gathered from the occipital region of the head and cut at the scalp using stainless steel scissors. The hair sample was placed in a folded Post-It note and secured with a plastic coated paper clip. Each sample was then placed in a plastic bag, labeled, and stored at room temperature until transport to the laboratory.

TABLE **1**

Demographics of Study Population Compared to General Population

Demographic	Study Population, *n* (%) (*N* = 703)	Duval County Female Population
Age (years)		
Median age	30	36
18–24	162 (23)	19.6%*
25–34	289 (41.1)	29.8%*
35–49	252 (35.9)	50.6%*
Race		
White/Caucasian	371 (52.8)	63.4%
Black/African-American	177 (25.2)	29.1%
Asian/Pacific Islander	50 (7.1)	3.4%
Other	82 (11.7)	4.1%
Unreported	23 (3.3)	–
Ethnicity		
Hispanic	132 (18.8)	6.2%
Non-Hispanic	561 (79.8)	93.8%
Unreported	10 (1.4)	–
Annual household income		
Less than $25,000	305 (43.4)	65.82%
$25,000–$34,999	134 (19.1)	11.65%
$35,000–$49,999	83 (11.8)	12.38%
$50,000–$74,999	56 (8)	6.89%
$75,000 or greater	69 (9.8)	3.26%
Unreported	56 (8)	–
Education		
High school grad or less	223 (31.7)	45.3%
Beyond high school	471 (67)	54.7%
Unreported	9 (1.3)	–
Health zone (HZ)		
HZ 1—Urban core	93 (13.2)	14%
HZ 2—Greater Arlington	197 (28.02)	31.3%
HZ 3—Southeast	63 (8.96)	16.9%
HZ 4—Southwest	249 (35.42)	20.7%
HZ 5—Outer rim	45 (6.40)	10%
HZ 6—Beaches	56 (7.97)	7.1%
Pregnancy status		
Pregnant	90 (12.8)	–
Gave birth in past 60 days	15 (2.2)	–

*% of female population aged 18–49.

Laboratory Analysis

Hair samples were analyzed for total mercury by combustion-gold amalgamation-atomic absorption spectroscopy with a mercury analyzer at the FDOH Bureau of Laboratories using a modified procedure (Legrand, Passos, Mergler, & Chan, 2005). The linear calibration curve (10–200 ng of mercury) was constructed from a mercury standard diluted with 0.001% L-cysteine and 0.2% nitric acid. The linear dynamic range was created so that a 20 mg hair sample would encompass concentrations ranging from 0.5 to 10 µg/g. Laboratory quality controls were prepared similarly at low (20 ng of mercury) and high (180 ng of mercury) levels from a mercury standard from an alternate source. Additionally, a hair reference material QMEQAS 07H-07, mean value 7.71 µg/g was prepared as a hair sample, but was not acetone rinsed per instructions, since the material was spiked with mercury. Details of the laboratory analysis are available upon contacting the corresponding author.

Data Analysis

Data from the hair mercury laboratory analysis and the fish consumption questionnaire

TABLE **2**

Fish Consumption Habits Among Sample of Women of Childbearing Age in Duval County (*N* = 703)

Characteristic	*n*	%
Include fish or shellfish in their diet	640	91.1
Eat canned tuna	447	69.8
Eat albacore or white canned tuna	240	53.7
Eat fish prepared at a restaurant	417	65.2
Eat fish bought from the store/market	451	70.5
Eat sport-caught fish from local waters	197	30.8
Eat shellfish	543	84.8
Eat fish	568	88.8
Ate high-risk fish in past 60 days	221	31.4
Pregnant women who ate high-risk fish in past 60 days	13/90	14.4

Note. High-risk fish include Chilean sea bass, golden snapper, jack, king mackerel, Spanish mackerel, marlin, shark, swordfish, tilefish, tuna steaks, and orange roughy.

TABLE **3**

Average Number of Fish Meals Consumed per Month (*n* = 640)

Demographic	Arithmetic Mean	Range
n = 640	12.41	1–93
Age (years)		
18–24	10.83	1–48
25–34	11.63	1–80
35–49	14.24	1–93
Race		
White/Caucasian	12.52	1–93
Black/African-American	13.22	1–80
Asian/Pacific Islander	13.49	4–48
Other	9.84	1–46
Annual household income		
Less than $25,000	12.66	1–84
$25,000–$34,999	12.40	1–53
$35,000–$49,999	11.18	1–45
$50,000–$74,999	10.64	2–40
$75,000 or greater	15.51	2–93
Health zone (HZ)		
HZ 1—Urban core	15.64	1–70
HZ 2—Greater Arlington	10.42	1–93
HZ 3—Southeast	12.17	2–48
HZ 4—Southwest	12.34	1–84
HZ 5—Outer rim	13.60	1–35
HZ 6—Beaches	13.86	1–48

were analyzed using SAS (v. 9.1). ANOVA and *t*-tests were used to identify associations between fish consumption and age, race, ethnicity, pregnancy status, knowledge of fish consumption advisories, and socioeconomic factors. Chi-square tests were used to identify associations between mercury awareness and fish consumption, age, race, ethnicity, pregnancy status, and socioeconomic factors. Probability plots demonstrated that the normal and lognormal distributions did not provide a good fit to the hair mercury observations, precluding parametric analyses.

Nonparametric methods from PROC LIFETEST, with data transformed for left-censoring, were used to account for results below the detection limit (Helsel, 2005). Nonparametric estimates of mean hair mercury levels were calculated using the area under survival curves obtained by the product-moment method. The nonparametric Wilcoxon test, adjusted for multiple comparisons, was used to identify associations between hair mercury levels and fish consumption, age, race, ethnicity, pregnancy status, knowledge of fish consumption advisories, and socioeconomic factors.

Results

General Characteristics

A total of 703 participants met the study criteria. Characteristics of the population are shown in Table 1. Approximately 62% of women were recruited from Duval County Health Department clinics or WIC programs; about 38% were recruited from local organized community events. The median number of years of residency in Duval County was 10 years (range: 1–49 years).

Over half of the participants reported their race as white/Caucasian (52.8%), followed by black/African-American (25.2%). The study population included a greater proportion of other minority groups (Asian, Hispanic, and other) than the general population of Duval County. Also reported were higher education levels and annual household income than the general population. These population differences provided an increased sample size among demographic groups that have been previously reported to have increased mercury levels or fish consumption.

Fish Consumption Patterns

Of the 703 women surveyed, 640 (91.1%) included fish or shellfish in their diet. Table 2 describes the types of fish consumed, with 88.8% of the 640 women reporting fish consumption, 84.9% reporting shellfish consumption, and 69.8% reporting consumption of canned tuna. Of those who eat fish, 70.5% prepare

store-bought fish at home and 65.2% eat fish prepared at a restaurant. Additionally, 30.8% of women eat fish that they or someone they know caught from local waterways. Of those who eat canned tuna, over half (53.7%) eat albacore or white tuna fish.

Consumption of "high-risk" fish was defined as eating any of the following fish species within the past 60 days: Chilean sea bass, golden snapper, jack, king mackerel, Spanish mackerel, marlin, orange roughy, shark, swordfish, tilefish, or tuna steak. These fish oftentimes contain high levels of mercury and are not recommended for consumption by nursing women and women who may become pregnant (FDOH, 2009b). Among the study group, 31.4% of women reported eating a high-risk fish species in the past 60 days. Among the 90% of women who were surveyed, 14.4% consumed a high-risk fish species.

As shown in Table 3, mean fish consumption was calculated based on monthly number of fish meals consumed. For women who reported fish consumption on a weekly basis, values were multiplied by four to give a monthly estimate.

The mean monthly fish consumption for the total study population was 12.41 fish meals per month. This exceeds the Food and Drug Administration's (FDA's) recommendation regarding fish consumption for women and young children, which is eight fish meals per month (or average of two meals per week) (U.S. EPA & FDA, 2004). In our study, fish consumption appeared to increase with the age of women. Among racial groups, Asian/Pacific Islanders reported the highest level of fish consumption. For income groups, fish consumption appeared to have a bimodal distribution with the highest among women reporting the greatest annual household income, followed by women reporting the lowest annual household income. Women of HZs 1, 5, and 6 reported higher fish consumption than other areas of Jacksonville.

TABLE **4**

Awareness of Mercury Advisories Among Women of Childbearing Age (*N* = 703)

Demographic	% Who Heard About Limiting Fish Consumption Due to Mercury	% Aware of Local Fish Advisories
Total	63.4	15.7
Age (years)		
18–24	55.4	11.4
25–34	64.5	16.4
35–49	67.1	17.9
Race		
White/Caucasian	73.1	21.4
Black/African-American	57	5.8
Asian/Pacific Islander	65.3	22.5
Other	37	6.2
Education		
≤High school graduate	51.6	10.4
Beyond high school	68.6	18.4
Have a valid Florida fishing license	70.4	29.6
Pregnant women	56.7	11.1
Eat local sport-caught fish	61.7	21.3
Health zone (HZ)		
HZ 1—Urban core	57.6	13
HZ 2—Greater Arlington	65.6	16.5
HZ 3—Southeast	58.7	22.2
HZ 4—Southwest	58.9	12.1
HZ 5—Outer rim	75	20.5
HZ 6—Beaches	80.4	23.2

Mercury Awareness

Of all women surveyed, 63.4% reported having heard about limiting fish consumption due to potential mercury exposure (Table 4). This percentage was lower among pregnant women than nonpregnant women. Only 15.7% of the study population reported being aware of the local fish consumption guidelines for Florida waterways. This percentage was higher among women who had a fishing license (29.6%) and women who consumed locally caught fish (21.3%).

Knowledge of mercury and its related fish consumption advisories increased with age and education. Whites/Caucasians and Asians/Pacific Islanders reported a greater level of awareness than blacks/African-Americans and other racial groups. Mercury awareness was highest in HZ 6 (beaches) and lowest in HZ 1 (urban core).

Mercury Levels

Each woman completed a questionnaire and submitted a hair sample. Three hair samples were not tested for mercury content—two samples included artificial hair and one sample was interrupted during testing due to a power outage. Two samples had mercury levels >5 µg/g, meeting Florida's case definition for possible acute mercury poisoning and were referred for follow-up. These cases were not representative of the general population and were excluded as outliers. These five records were excluded for the mercury analysis, leaving a total $n = 698$.

Hair mercury levels ranged from below the detection limit (BDL) to 3.03 µg/g. Of the 698 hair samples, 522 (74.8%) were BDL. Nonparametric mean estimates were calculated that accounted for values below the detection limit without making a distributional assumption. The overall mean hair mercury level of the study population was 0.33 µg/g (confidence interval [*CI*] = 0.30, 0.37). Nonparametric tests of significance and estimated means by subgroup are displayed in Table 5.

Overall, hair mercury levels were associated with fish consumption, age, race, ethnicity, health zone, income, and education level. When comparing race groups, the highest hair mercury levels were seen among Asians/Pacific Islanders. The health zone with highest hair mercury levels was HZ 6 (beaches). Higher hair mercury levels were found among women who report knowledge of the recommendation

TABLE **5**

Average Mercury Levels (µg/g) by Subgroup

Demographic	*n*	Nonparametric Mean Mercury Level	Wilcoxon *p*-Value
Total	698	0.334	–
Weekly fish consumption			<.0001
None	63	0.152	
1–2 meals/week	292	0.295	
3–4 meals/week	204	0.418	
More than 4 meals/week	139	0.530	
Age (years)			.0005
18–24	159	0.252	
25–34	287	0.361	
35–49	252	0.405	
Race			<.0001
White/Caucasian	371	0.393	
Black/African-American	174	0.481	
Asian/Pacific Islander	49	0.640	
Other	81	0.277	
Ethnicity			.0085
Hispanic	131	0.334	
Non-Hispanic	557	0.351	
Annual household income			<.0001
Less than $25,000	302	0.262	
$25,000–$34,999	132	0.319	
$35,000–$49,999	83	0.355	
$50,000–$74,999	56	0.498	
$75,000 or greater	69	0.602	
Education level			<.0001
≤High school graduate	221	0.235	
Beyond high school	468	0.384	
Health zone (HZ)			<.0001
HZ 1—Urban core	92	0.295	
HZ 2—Greater Arlington	195	0.397	
HZ 3—Southeast	63	0.398	
HZ 4—Southwest	248	0.289	
HZ 5—Outer rim	44	0.450	
HZ 6—Beaches	56	0.656	
Pregnancy status			.0915
Pregnant	90	0.269	
Not pregnant	597	0.350	
Heard about limiting mercury			.0007
Yes	446	0.371	
No	252	0.336	
Fishing license			.0121
Yes	142	0.476	
No	556	0.314	

to limit fish consumption because of mercury. Women from households with a Florida fishing license had higher hair mercury levels than women from households without fishing licenses. Pregnancy status was not found to be a statistically significant variable.

Discussion

U.S. EPA recommends that women of childbearing age include low-mercury fish as part of their diets but also limit fish consumption to two fish meals per week, or eight fish meals per month (U.S. EPA & FDA, 2004). Women surveyed in this Duval County study population consume more than this recommended amount, reporting an average of more than 12 meals per month. Interestingly, despite higher fish consumption, this study population had lower hair mercury levels when compared to other studies. The average mercury levels (geometric mean [GM] = 0.11 µg/g, *CI* = 0.09–0.14 and nonparametric mean = 0.33 µg/g, *CI* = 0.30–0.37) of the current study were significantly less than the average mercury hair levels (GM = 0.25 µg/g, *CI* = 0.22–0.28, arithmetic mean [AM] = 0.56 µg/g, *CI* = 0.46–0.64) found among selected women of childbearing age in a 2008 Florida panhandle study (Karouna-River et al., 2008) and those women (GM = 0.20 µg/g, AM = 0.47 µg/g) who participated in the 1999–2000 National Health Nutrition Examination Survey. Similarly, only 7% of the current study population had mercury levels that are above 1 µg/g level of concern, compared to 16% in the Florida panhandle and 12% nationally (Karouna-Renier, et al., 2008; McDowell et al., 2004). One possible explanation is that the types of fish commonly eaten in Duval County may be low in mercury. Less than a third of those surveyed reported eating a high-risk fish species. About 85% of the study population reported eating shellfish, which has very low levels of mercury when compared to finfish (FDA, 2004). Additionally, the majority of women reported eating commercially bought fish from a grocery store, market, or restaurant, as opposed to recreationally caught fish. Studies have shown that commercially bought fish, which can come from a variety of regional sources, may have lower mercury levels than fish caught from local contaminated waterways (Burger, Stern, & Gochfeld, 2005; Lincoln et al., 2011).

Our study also found that increased hair mercury levels were associated with increased fish consumption. The risk groups for increased mercury levels mirrored the risk groups for increased fish consumption. One of these risk groups was Asians/Pacific Islanders, which is consistent with other studies (Mahaffey, Clickner, & Jeffries, 2009; McKelvey et al., 2007; Patch, Maas, & Sergent,

2005). This could be attributed to cultural traditions that include more fish in the diet.

Our study also showed that women with the highest household income had the highest mercury levels, which has been demonstrated in other studies as well (Hightower & Moore, 2003; Mahaffey et al., 2009; McKelvey et al., 2007). One explanation is that fish costs more than other dietary sources of protein. Another factor is that higher-mercury fish species, such as swordfish or Chilean sea bass, typically cost more than other types of fish such as canned tuna. Women with higher incomes may be better able to afford more frequent and higher-mercury fish meals.

Finally, residents of HZ 6 (beaches) in Duval County were found to have the highest hair mercury levels and second-highest average fish consumption. This is consistent with other studies that show increased fish consumption among coastal communities (Lincoln et al., 2011; Liu et al., 2008; Mahaffey et al., 2009). This may be attributed to the proximity of coastal fishing boats, which leads to the increased availability of fresh fish and opportunities for recreational fishing charters.

One important finding of our study was that awareness of mercury contamination and fish advisories among study participants was low, and even lower among higher-risk pregnant women. This finding appears consistent with other studies. Even more concerning is that awareness was lower among pregnant women, who are at increased risk. For example, Ney and Ney reviewed fish consumption advisories in six states and reported that awareness among the general public and high-risk women was low (20%–40%). Their findings suggest that this may stem from poor perceptions of adverse health risks from eating fish as well as inconsistent and complex messaging of the advisory (Ney & Ney, 2008).

This demonstrates the need to improve education for women, especially women who are pregnant, breastfeeding, or trying to get pregnant. It is interesting that groups with the highest mercury levels are also groups reporting higher levels of mercury awareness. For example, women in HZ 6 (beaches) were most knowledgeable about fish advisories and mercury contamination but also had the highest hair mercury levels and reported the second-highest frequency of consuming fish meals. This is unlike other studies that indicate an association between low awareness and higher mercury levels (Karouna-Renier et al., 2008; Knobeloch et al., 2005). This could mean that knowledge of the association between mercury and fish consumption alone may not influence the frequency of fish consumption or deter women from eating high-risk fish. More complex behavior change strategies may be necessary when developing educational messages for at-risk women.

Our study provided useful public health data, but not without limitations. First, the study population was not chosen at random. Rather, it was a self-selected convenience sample. Women who eat fish frequently may have had concerns about their individual mercury levels, making them more likely to participate. This could have skewed mercury levels, and the results may not be generalized to the greater population. Next, many women approached for the study refused to participate because they did not want to provide a hair sample. Some did not want to cut their hair. Others wore wigs or hair weaves, which made it difficult to easily obtain a natural hair sample. These barriers led to a lowered response rate.

The possibility of recall bias also exists. Participants were asked to estimate the number of seafood meals they had over a period of 60 days. They may have over or underestimated what they actually ate. In addition, "fish meals" was not defined in terms of ounces. Participants may have different concepts of what constitutes a fish meal. Finally, the laboratory methods used to analyze the hair mercury levels could not detect very small levels of mercury. Therefore, many subjects had results that were below the detectable limit. Using a statistical method that accounts for values below the detection limit still results in a loss of information compared to obtaining actual measured values by using a laboratory method that detects very small levels of mercury.

Conclusion

Our study accomplished its objectives by evaluating the potential risk of mercury exposure by examining fish consumption patterns among women of childbearing age. Given that Florida has the highest mercury emission levels in the southeast, accompanied by commercial and recreational fishing, our study signifies contributory importance to public health practice. By collecting survey and hair data we helped to characterize fish consumption habits and mercury levels in hair among women of childbearing age in Duval County.

With minor modifications, a similar study could be expanded to other counties or incorporated into public health surveillance programs. We found that a need exists to improve mercury education, especially among pregnant women and those at increased risk for elevated mercury levels. Providing accurate and consistent educational materials will require collaboration with many partners, including health care providers, food retailers, nutritionists, and the seafood industry. The goal should be to promote the many health benefits of eating fish, while providing specific guidelines for consumption frequency and fish species to avoid. Future studies may include looking more closely at specific species of fish being consumed, investigating mercury education practices among health care providers, and surveying seafood retailers about their views on providing mercury education to consumers.

In summary, fish continue to be an important part of a human diet, high in protein, rich in nutrients, and low in saturated fatty acids and cholesterol. Because mercury levels vary according to fish species, nutrition and health experts should provide sound advice by asking women of childbearing age to consume fish in moderation and follow state and federal advisories on local and national fish consumption.

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References

Agency for Toxic Substances and Disease Registry. (1999). Toxicological profile for mercury. Retrieved from http://www.atsdr.cdc.gov/toxprofiles/tp46.pdf

Burger, J., Stern, A.H., & Gochfeld, M. (2005). Mercury in commercial fish: Optimizing individual choices to reduce risk. *Environmental Health Perspectives, 113*(3), 226–271.

Butler, T.J., Cohen, M.D., Vermeylen, F.M., Likens, G.E., Schmeltz, D., & Artz, R.S. (2008). Regional precipitation mercury trends in the eastern USA, 1998–2005: Declines in the Northeast and Midwest, no trend in the Southeast. *Atmospheric Environment, 42*(7), 1582–1592.

Chalmers, A.T., Argue, D.M., Gay, D.A., Brigham, M.E., Schmitt, C.J., & Lorenz, D.L. (2010). Mercury trends in fish from rivers and lakes in the U.S., 1969–2005. *Environmental Monitoring and Assessment, 175*(1–4), 175–179.

Florida Department of Health. (2009a). *Your guide to eating fish caught in Florida.* Retrieved from http://www.doh.state.fl.us/floridafishadvice/Final%202009%20Fish%20Brochure.pdf

Florida Department of Health. (2009b). *Fish for your health: Advice for pregnant or nursing women, women who may become pregnant, and children (2–6 years).* Retrieved from http://www.doh.state.fl.us/floridafishadvice/PrintableWalletCard.pdf

Food and Drug Administration. (2004). *Mercury levels in commercial fish and shellfish.* Retrieved from http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115644.htm

Helsel, D.R. (2005). *Nondetects and data analysis: Statistics for censored environmental data.* New York: John Wiley & Sons.

Hightower, J.M., & Moore, D. (2003). Mercury levels in high-end consumers of fish. *Environmental Health Perspectives, 111*(4), 604–608.

Karouna-Reiner, N., Rao, R., Lanza, J., Rivers, S., Wilson, P., Hodges, D., Levine, K., & Ross, G. (2008). Mercury levels and fish consumption practices in women of childbearing age in the Florida Panhandle. *Environmental Research, 108*(3), 320–326.

Knobeloch, L., Anderson, H.A., Imm, P., Peters, D., & Smith, A. (2005). Fish consumption, advisory awareness, and hair mercury levels among women of childbearing age. *Environmental Research, 97*(2), 220–227.

Legrand, M., Passos, C.J., Mergler, D., & Chan, H.M. (2005). Biomonitoring of mercury exposure with single human hair strand. *Environmental Science and Technology, 39*(12), 4594–4598.

Lincoln, R.A., Shine, J.P., Chesney, E.J., Vorhees, D.J., Grandjean, P., & Senn, D.B. (2011). Fish consumption and mercury exposure among Louisiana recreational anglers. *Environmental Health Perspectives, 119*(2), 245–251.

Liu, X., Cheng, J., Song, Y., Honda, S., Wang, L., Liu, Z., Sakamoto, M., & Liu, Y. (2008). Mercury concentration in hair samples from Chinese people in coastal cities. *Journal of Environmental Health* (China), *20*(10), 1258–1262.

Mahaffey, K.R., Clickner, R.P, & Jeffries, R.A. (2009). Adult women's blood mercury concentrations vary regionally in the U.S.: Association with patterns of fish consumption (NHANES 1999–2004). *Environmental Health Perspectives, 117*(1), 47–53.

McDowell, M.A., Dillon, C.F., Osterloh, J., Bolger, P.M., Pellizzari, E., Fernando, R., Montes de Oca, R., Schober, S.E., Sinks, T., Jones, R.L., & Mahaffey, K.R. (2004). Hair mercury levels in U.S. children and women of childbearing age: Reference range data from NHANES 1999–2000. *Environmental Health Perspectives, 112*(11), 1165–1171.

McKelvey, W., Gwynn, R.C., Jeffery, N., Kass, D., Thorpe, L.E., Garg, R.K., Palmer, C.D., & Parsons, P.J. (2007). A biomonitoring study of lead, cadmium, and mercury in the blood of New York City adults. *Environmental Health Perspectives, 115*(10), 1435–1441.

National Atmospheric Deposition Program. (2008). *Annual data summary.* Retrieved from http://nadp.sws.uiuc.edu/lib/dataReports.aspx

Ney, J.J., & Ney, J.P. (2008). Risky business: Evaluation of U.S. consumption advisories for freshwater sport fish. In M.S. Allen, S. Sammons, & M.J. Maceina (Eds.), *Balancing fisheries management and water uses for impounded river systems*. Bethesda, MD: American Fisheries Society.

Oken, E., Wright, R.O., Kleinman, K.P., Bellinger, D., Amarasiriwardena, C.J., Hu, H., Rich-Edwards, J.W., & Gillman, M.W. (2005). Maternal fish consumption, hair mercury, and infant cognition in a U.S. cohort. *Environmental Health Perspectives, 113*(10), 1376–1380.

Patch, S.C., Maas, R.P., & Sergent, K.R. (2005). *An investigation of factors related to levels of mercury in human hair*. Asheville, NC: Environmental Quality Institute, University of North Carolina. Retrieved from http://www.greenpeace.org/usa/Global/usa/report/2007/7/mercury-report.pdf

Scudder, B.C., Chasar, L.C., Wentz, D.A., Bauch, N.J., Brigham, M.E., Moran, P.W., & Krabbenhoft, D.P. (2009). *Mercury in fish, bed sediment, and water from streams across the U.S., 1998–2005: U.S. Geological Survey scientific investigations report 2009–5109*. Retrieved from http://pubs.usgs.gov/sir/2009/5109/pdf/sir20095109.pdf

U.S. Environmental Protection Agency. (2005). *2005 national emissions inventory*. Retrieved from http://www.epa.gov/ttn/chief/net/2005inventory.html

U.S. Environmental Protection Agency. (2007). *Fact sheet: Bioaccumulative contaminants*. Retrieved from http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/archive/2006_index.cfm

U.S. Environmental Protection Agency, & Food and Drug Administration. (2004). *What you need to know about mercury in fish and shellfish*. Retrieved from http://www.fda.gov/Food/ResourcesForYou/Consumers/ucm110591.htm

U.S. Public Interest Research Group. (2005). *Made in the USA: Power plants and mercury pollution across the country*. Washington, DC: Author. Retrieved from http://cdn.publicinterestnetwork.org/assets/HD9rUiz0KrbzbCoMUZoKMA/Made_In_The_USA.pdf

Lead Detection in Food, Medicinal, and Ceremonial Items Using a Portable X-Ray Fluorescence (XRF) Instrument

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Abstract **The authors evaluated a Niton XLp303A X-ray fluorescence (XRF) instrument, used to identify lead hazards in housing, to determine its effectiveness to screen food, medicinal, and ceremonial items during lead poisoning investigations. Fifty-eight suspect exposure items were tested for lead by XRF and then sent to the laboratory for confirmation. A lead content cut-point of 10 parts per million (ppm; the lower level at which the XRF model could reliably determine the presence of lead) was used to evaluate the results. The Niton consistently identified the presence of lead spectra emissions and gave quantitative readings above 10 ppm for the nine samples with lead content that exceeded 10 ppm in laboratory testing. The authors' study suggests that the Niton XLp303A is an effective screening method for food and similar items with lead content ≥10 ppm, provided the operator is trained to identify lead spectra. Rapid, on-site identification of lead exposure sources allows an investigator to inform the family of immediate steps they can take to decrease their child's lead exposure.**

Introduction

Assessing lead in paint, dust, and soil is an important element of every childhood lead poisoning case investigation. The Centers for Disease Control and Prevention (CDC) have stated that "the most common sources of high-dose lead exposure for U.S. children are lead-based paint and lead-contaminated house dust and soil (CDC, 2005)." Handheld X-ray fluorescence (XRF) instruments have become one of the essential components of lead exposure assessment, due to the advantages of the technology: rapid, on-site screening capability, nondestructive testing, and immediate results.

The California Department of Public Health (CDPH) Childhood Lead Poisoning Prevention Branch (CLPPB) has previously validated the use of portable XRFs for lead in paint testing and lead in soil testing (Reames & Lance, 2002) and has developed a series of guidance documents with XRF testing procedures for local environmental health jurisdiction staff. CLPPB administers an XRF loan program that makes Niton model XLp303A instruments available to local county and city health jurisdictions throughout California. These instruments have been approved by the U.S. Environmental Protection Agency (U.S. EPA) and the U.S. Department of Housing and Urban Development (HUD) for lead-based paint testing (U.S. EPA, 2004).

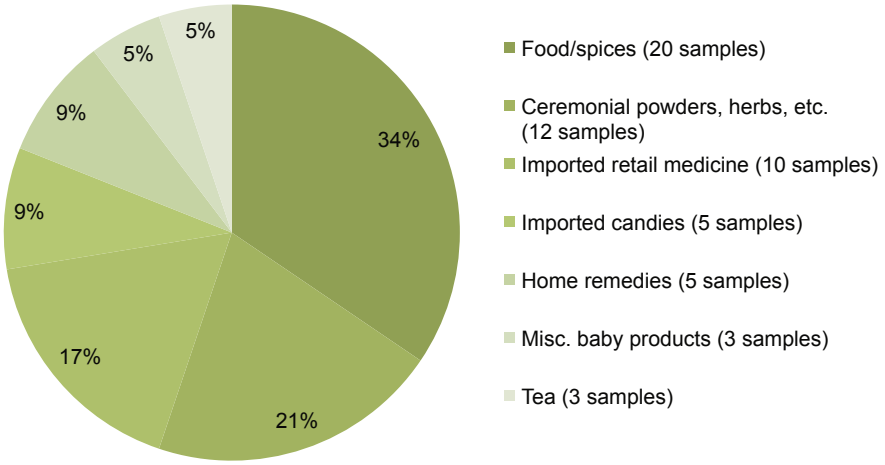
Attempting to identify as many of the sources of lead exposure as possible is critical for a childhood lead poisoning case in order to reduce or eliminate all of the sources. Portable XRF instrument usage has allowed California lead programs to quickly inform the family of a child lead poisoning case of lead exposure hazards in paint, dust, and soil. Although established state (California Department of Public Health [CDPH], 2008) and federal guidance documents (Lead, 2001; U.S. Department of Housing and Urban Development [HUD], 1995) are available on field sampling and identification of housing-based lead hazards, guidance is lacking on using XRFs to screen nonhousing items.

Discussions with Food and Drug Administration (FDA) officials and review of their previous work with XRF analysis led CLPPB to conduct a study to determine if the on-site screening capability of the CLPPB XRF instruments could be expanded to include food, medicinal, and similar relatively homogeneous samples. Staff affiliated with FDA have previously highlighted the suitability of XRF for rapid screening of toxic elements such as lead (Palmer, Jacobs, Baker, Ferguson, & Webber, 2009), and issued a laboratory information bulletin on the same topic for FDA staff (Palmer, Webber, Ferguson, & Jacobs, 2007). Some prior studies that concentrated on specific foods have also been performed, such as spices (Al-Bataina, Maslat, & Al-Kofahi, 2003) and Indian spices and cultural powders (Lin, Schaider, Brabander, & Woolf, 2010). Other than the FDA evaluations, however, previous XRF studies have involved the use of bench-top laboratory-grade XRF instruments.

CLPPB undertook an evaluation to focus on a handheld XRF that could easily be brought into homes and used both for test-

FIGURE **1**

Categories of Items Tested Using Niton XRF



ing lead-based paint and other environmental hazards and for screening lead in food and medicinal items. CLPPB's objective was to confirm that the XRF used for identifying environmental lead hazards could also be used as a screening tool for food, medicinal, and ceremonial items. If the XRF was found to be a valid screening methodology, the future addition of on-site food and medicinal item testing would be a major advance in rapid evaluation of suspect lead exposure sources for lead-poisoned children.

Methods

Sample Collection and Testing

Fifty-eight test samples were acquired from seven local California childhood lead programs and through childhood lead poisoning investigations by CLPPB staff over a 10-month period (June 2009 through March 2010). The majority of samples were collected because they were suspected lead exposure sources. Some additional samples were purchased at ethnic grocery stores. These samples were similar to those recalled by FDA due to food labeling concerns (FDA, 2008) and those tested in a prior lead in spices study (Lin et al., 2010). Samples consisted of seven general categories: imported candies, miscellaneous baby products (powder, lotion, and astringent), imported retail medicines, home remedies, tea, foods and spices, and ceremonial items (powders, herbs, incense, and camphor associated with Hindu worship). Foods and spices constituted the majority of samples tested (Figure 1).

Test Methods

The lead content of the samples included in our study was unknown prior to testing. Since the goal of testing was to develop a rapid screening method, the samples were not altered by grinding or other practices used by laboratories to further homogenize a sample. The majority of samples were tested in the original packaging, plastic bags, and XRF test cups. When initial testing suggested that lead-containing ink might be present in the packaging, some items were removed from the packaging and evaluated separately. Some bulk powders and liquids were transferred to an XRF test cup with a thin Mylar covering. An instrument quality control sample, consisting of a National Institute for Standards and Technology (NIST) bulk lead in soil standard, was tested prior to XRF testing of samples. XRF sample testing proceeded only if the quality control XRF result was within plus or minus 10% of the NIST lead value in parts per million (ppm).

After XRF testing, samples were submitted to either a commercial laboratory or the CDPH Environmental Health Laboratory Branch (CDPH EHLB) following appropriate chain-of-custody procedures. Five samples that appeared to be low in lead when tested by XRF were sent to the CDPH EHLB for analysis, which employs a more rigorous modified sample analysis technique that results in lower detection limits. An imported candy sample was sent from CDPH EHLB to the CDPH Food and Drug Branch to determine if the sample exceeded the California Health and Safety Code Section 110552 level of 0.1 ppm, the California standard for lead in candies (CDPH, 2005). The majority of the samples were analyzed using U.S. EPA reference method 3050B/7421 (graphite furnace atomic absorption). The remaining samples that contained higher concentrations of lead were analyzed using U.S. EPA reference method 3050B/7420 (flame atomic absorption).

Data Analysis Plan

Review of previous findings by FDA indicated that quantitative XRF results derived from proprietary algorithms may not always be accurate due to a variety of factors including sample homogeneity, sample density, depth of the XRF readings, interferences from unexpected elements, the exact focal point of the X-ray beam, and limitations of preprogrammed algorithms used to calculate quantitative results. Therefore, two screening criteria were used to evaluate the Niton XRF lead detection results. Both criteria were based on a cut-point of 10 ppm, which is significantly lower than the lead-based paint standard of 5,000 ppm (Lead, 2001). This cut-point was thought to represent the lowest lead level for which results could reliably be obtained for the CLPPB model of XRF, based on prior FDA work and initial pilot testing by CLPPB.

The Niton XRF provides a test result for a given element in a bulk sample in two ways: a reading with units in ppm and a graph of the spectra of the elemental peaks that are present in the sample. For the first criterion of our evaluation, we hypothesized that if the laboratory sample result was ≥10 ppm lead then the unique spectral emissions produced by lead should be observed. Graphing software on the XRF and a companion proprietary PC software program (Thermo Scientific NDT© Software Suite) were used to observe whether both characteristic L-shell peaks of lead were present at specific energy levels: 10.5 kiloelectron

volts (KeV) for the alpha peak and 12.6 KeV for the beta peak, respectively. For the second criterion, we hypothesized that if the laboratory sample result for lead was ≥10 ppm, then the XRF lead reading should also be ≥10 ppm.

Results

Lead Content of Samples

Fifty-eight samples were included in the data analysis. These samples were first tested by XRF and then analyzed by the laboratory (Table 1). Both the Niton readings and the corresponding laboratory values were fairly low for the majority of samples, with 74% (43/58) of Niton readings reported as below method detection limits (<4.0 ppm–<17.3 ppm). Niton results that were preceded by a "less than" sign were classified as a nondetectable lead result. Seventy percent (41/58) of laboratory results were reported as below method detection limits (<0.1 ppm–<7.0 ppm). Detectable Niton lead readings ranged from 12.3 ppm to values that exceeded detector algorithm limits. Two samples were very high in lead, which caused the XRF to report values higher than one million ppm. This is due to an assumption made in the Niton analyzer's soil mode calibration that the sum total of metallic content is below 10% and that the metals present in the sample are not expected to affect one another. Since both samples contained over 10% lead, the numeric values provided by the analyzer exceeded the maximum value for ppm results, demonstrating that the XRF algorithm was oversaturated. Laboratory sample results above method detection limits ranged from 0.2 ppm to 340,000 ppm. One outlier was omitted from the data set because when tested by XRF, the sample appeared to have a very large amount of mercury (very large spectra peaks that overshadowed the area of the lead peaks) that caused the instrument to give a meaningless value (<91,018) for lead that could not be interpreted as being above or below the cut-point of 10 ppm.

TABLE **1**

Summary of Niton XRF and Laboratory Results

Category of Items Tested	# of Items Tested	Range of Niton XRF Results (ppm[a])	Range of Lab Results (ppm)
Imported candies	5	<4.3–<8.3	<0.1–<0.6
Misc. baby products (powder, formula, and astringent)	3	<4.6–<7.0	<0.1–<3.0
Imported retail medicines	10	<4.0–721.9	<0.1–1,500
Home remedy	5	<6.7–5.9 x 10^{6}*	<3.0–1.9 x 10^{5}
Tea	3	<7.9–9.4	<2.0–6.0
Food/spice	20	<4.5–<13.1	<0.1–<3.0
Ceremonial (powders, herbs, camphor, face chalk, incense)	12	<7.7–4.1 x 10^{5}	<5.0–3.4 x 10^{5}
Summary for all samples	58	<4.0–5.9 x 10^{6}*	<0.1–3.4 x 10^{5}

Note. Niton and lab results reported with a "less than" sign were classified as below method detection limits.
[a]ppm = parts per million.
*Niton results shown as reported, although exceeding one million ppm is incorrect mathematically (the Niton detector algorithm was oversaturated due to the high lead content of the item).

Evaluation of Results Based on Screening Criteria

Laboratory sample results were grouped according to the cut-point of 10 ppm. Nine out of 58 samples (16%) were ≥10 ppm. Samples that exceeded the cut-point consisted of home remedies, imported retail medicine, and ceremonial items (Table 2). Based on the first criterion (confirmation of the presence of the alpha and beta L-shell elemental peaks of lead using spectra graphing software), lead peaks were observed for all samples that contained ≥10 ppm (9/9). Conversely, lead peaks did not appear to be present in all of the samples with laboratory results that were <10 ppm (49/49). This demonstrates that the Niton XRF could be used to reliably classify samples as having lead above the cut-point of 10 ppm by observation of the presence of lead peaks.

For the second criterion (quantitative agreement relative to the 10 ppm cut-point), all of the samples containing ≥10 ppm lead by laboratory analysis corresponded with Niton readings of ≥10 ppm (9/9). Six Niton XRF results, however, were ≥10 ppm for samples that were <10 ppm by laboratory analysis (false positive results). This finding demonstrates that the observation of the spectra is the most accurate means to determine whether lead is present in samples above 10 ppm.

Since the objective of the Niton evaluation study was to determine whether the instrument could be used as a field screening tool using the two screening criteria, no additional statistical analyses were performed. The Niton XRF detection limits were as much as two orders of magnitude greater than those obtained by the laboratory, particularly for samples with lead content below 10 ppm, which comprised the majority of samples in our study. The Niton detection limits were considerably higher than the laboratory detection limits in part because the XRF is a screening methodology and lacks the rigor of sample preparation, further homogenization, and acid digestion such as that employed in laboratory analysis methods for lead.

Discussion

Our study, although limited in scope, demonstrates the potential food and medicinal item screening capability of XRFs such as the Niton XLp303A. The instrument consistently identified the presence of the characteristic lead spectra for samples with ≥10 ppm of lead (10 ppm cut-point). Lead spectra were absent for samples with <10 ppm of lead. Although the XRF is a screening methodology, instrument readings were ≥10 ppm for all laboratory sample results of ≥10 ppm. For some samples with laboratory results <10 ppm, however, the XRF readings were ≥10 ppm. These results were considered to be false positive results relative to the 10 ppm

TABLE **2**

Niton Results for Laboratory Sample Results With ≥10 Parts per Million (ppm) Lead (Nine Samples)

Item Tested	Niton L-shell Spectra Observed	Niton Result ≥10 ppm	Niton Result (ppm)	Lab Result (ppm)
Mexican home remedy powder (orange/red)	Yes	Yes	1.3×10^{6}*	1.1×10^{5}
Mexican home remedy powder (yellow)	Yes	Yes	5.9×10^{6}*	1.9×10^{5}
Imported Vietnamese commercially made aspirin (package)	Yes	Yes	722	1,500
Mexican home remedy (herbal mixture)	Yes	Yes	82	70
Hindu ceremonial item ("Puja samagri") camphor (package)	Yes	Yes	1,147	5,200
Hindu ceremonial item (cosmetic chalk)	Yes	Yes	4.1×10^{5}	3.4×10^{5}
Hindu ceremonial item ("Vibhuti" sacred ash)	Yes	Yes	47	31
Hindu ceremonial item (Devi picture)	Yes	Yes	244	1,400
Hindu ceremonial item (incense sticks)	Yes	Yes	12	20

*Niton results shown as reported, although exceeding one million ppm is incorrect mathematically (the Niton detector algorithm was oversaturated due to the high lead content of the item).

cut-point used for evaluation purposes. This supports the findings cited by FDA (Palmer et al., 2007; Palmer et al., 2009) that spectra results should be given precedence when determining if a metal such as lead is present in the sample. Given this limitation, it should be noted that the instrument appears to be able to correctly classify samples that may be a lead exposure concern (≥10 ppm), which supports the public health goal of rapid screening to identify lead-contaminated food and medicinal items.

Use of this methodology will allow childhood lead poisoning investigators to quickly identify food and medicinal items with ≥10 ppm lead that may be contributing to a child's lead exposure. The main limitation of the XRF is that the limits of detection are not low enough to determine if a given food or medicine is not a significant contributor to a child's elevated blood lead level. A food item that is tested by XRF with a result that is reported as below the detection limit of <10 ppm could still contain enough lead to be a concern. If the food item actually contains 7 ppm (mcg/g) lead and the child eats a gram a day, this would exceed the FDA 6 mcg per day provisional tolerable total daily intake for children (Provisional Tolerable Total Daily Intake for Children, 1993). Laboratory analysis is therefore still required for samples with low XRF lead screening results. The two strengths of XRF technology, however, are the ability to view and categorize the unique spectra of various elements and to provide immediate feedback to the lead-poisoned child's family regarding food and medicinal items with high lead-exposure potential.

As testing procedures were developed for our evaluation study, it became clear that this screening method requires operators to become experienced in spectra identification. It is essential for XRF users to learn to recognize the presence of elemental lead peaks. This can be learned by testing samples with known lead content and "blank" samples. It is also important for operators to recognize elements that can overlap with lead spectra, resulting in inconclusive or inaccurate results. The outlier sample that had a high level of mercury required the operator to readily identify that the sample could not be adequately characterized in the field.

Although a relatively small number of lead samples ≥10 ppm were tested (nine samples, 16% of the samples in the study) this reflects CLPPB experience of testing these types of items during childhood lead poisoning investigations. The majority of suspect items appear to have relatively low lead content, while occasionally a very high lead item is identified. The distribution of lead results in our study provided an opportunity to determine if the XRF was able to distinguish high lead-level items from among the suspect items tested. The capability demonstrated in our study shows that XRF results can inform the family of a lead-poisoned child so that they can immediately remove high lead-level items from their child's environment.

The usefulness of this methodology is best illustrated with an example from our study. Two different types of ceremonial cosmetic chalks brought from India were evaluated to determine if either one was a lead exposure source in a childhood lead poisoning case. One chalk was white, the other yellow. Neither chalk had any packaging or other information regarding the ingredients. The XRF quickly identified the yellow chalk as a very high lead exposure source (large elemental lead peaks and 414,000 ppm quantitative result). No lead peaks were confirmed for the white chalk, although a quantitative XRF result exceeded 10 ppm (34.5 ppm). The child's family was told to discontinue use of the chalks, pending the laboratory results. The laboratory results for these chalks were 340,000 ppm and 8.6 ppm, respectively.

Conclusion

Our study illustrates that it is possible to expand the capability of an XRF used to identify lead exposure hazards in paint, dust, and soil to screen food and medicinal items. Although the XRF has higher limits of detection than a

laboratory, the instrument used in our study was able to consistently classify samples using a cut-point of 10 ppm. The effectiveness of this screening methodology is dependent on the operator's ability to discern the presence of the characteristic elemental lead peaks. Rapid identification of suspect lead exposure items enhances the ability of a childhood lead poisoning investigator to inform the family of immediate steps they can take to decrease their child's lead exposure.

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References

Al-Bataina, B.A., Maslat, A.O., & Al-Kofahi, M.M. (2003). Element analysis and biological studies on ten oriental spices using XRF and Ames test. *Journal of Trace Elemental Biology*, *17*(2), 85–90.

California Department of Public Health. (2005). *Sherman food, drug, and cosmetic law*, California Health and Safety Code, §110552.

California Department of Public Health. (2008). *Accreditation, certification, and work practices for lead-based paint and lead hazards* (California Code of Regulations, Title 17, Division 1, Chapter 8). Sacramento: Author.

Centers for Disease Control and Prevention. (2005). *Preventing lead poisoning in young children.* Atlanta: Author.

Food and Drug Administration. (2008). *Raja Foods issues nationwide recall on Swad brand abil, gulal, kanku, kum kum, lagan samagri and pooja samagri containing high levels of lead.* Retrieved from http://www.fda.gov/Safety/Recalls/ArchiveRecalls/2008/ucm112341.htm

Lead; Identification of Dangerous Levels; Final Rule (U.S. Environmental Protection Agency), 40 C.F.R. Part 745, 66 (2001).

Lin, C.G., Schaider, L.A., Brabander, D.J., & Woolf, A.D. (2010). Pediatric lead exposure from imported Indian spices and cultural powders. *Pediatrics, 125*(4) 828–835.

Palmer, P.T., Jacobs, R., Baker, P.E., Ferguson, K., & Webber, S. (2009). Use of field-portable XRF analyzers for rapid screening of toxic elements in FDA-regulated products. *Journal of Agricultural and Food Chemistry, 57*(7), 2605–2613.

Palmer, P., Webber, S., Ferguson, K., & Jacobs, R. (2007). On the suitability of portable X-ray fluorescence analyzers for rapid screening of toxic elements. *Laboratory Information Bulletin, 4376,* 1–15.

Provisional Tolerable Total Daily Intake for Children (U.S. Food and Drug Administration). *Federal Register, 58,* 33860 (1993).

Reames, G., & Lance, L.L. (2002). Childhood lead poisoning investigations: Evaluating a portable instrument for testing soil lead. *Journal of Environmental Health, 64*(8), 9–13, 25.

U.S. Department of Housing and Urban Development, Office of Lead Hazard Control. (1995). *Guidelines for the evaluation and control of lead-based paint hazards in housing.* Washington, DC: Government Printing Office.

U.S. Environmental Protection Agency. (2004). *Performance characteristic sheet for Niton LLC model series XLp300 (1st ed.).* Kansas City, MO: Midwest Research Institute.

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Evaluation of Metal Impurities in Foods Preserved With Sodium Lactate

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Abstract **The public is being bombarded by the media almost daily with real and potential food health concerns leading to a public sentiment that questions the vulnerability and quality of our food. Sodium lactate is a food-grade product that in recent years has been used in bioremediation to stimulate microbial growth and contaminant breakdown processes. In previous work, impurities including arsenic and chromium were discovered to be present in the sodium lactate concentrate. The study described in this article was performed to determine whether arsenic and chromium were at detectable levels, posing a potential concern in food products preserved with sodium lactate available to the general public. A pilot sampling of three sodium-lactate-preserved food products was obtained from a local market and used to determine the commercial laboratory's detection and reporting limits for arsenic and chromium for these food products. Once these limits were established, a random sampling and analyses of 17 food products was performed. Arsenic was not reported above the detection limits in either the pilot or subsequent study, but chromium was detected at concentrations up to 0.30 parts per million in a pilot test sample and lower concentrations in the subsequent study. This study suggests that the sodium lactate in the sampled products was diluted enough for the arsenic concentration to be below the laboratory detection limit. Chromium was detected and may be an unaccounted source of chromium in diets of vulnerable populations.**

Introduction

Common food-grade product additives, such as sodium lactate, ethyl lactate, and soybean oil, are used during *in situ* bioremediation of contaminated groundwater. During the evaluation of data associated with these bioremediation activities, elevated levels of impurities (metals, alcohols, and ketones) were discovered in undiluted sodium lactate (Chiang, Carter, & Woodward, 2008) at concentrations that raise the issue of a potential health concern. The same undiluted sodium lactate is also used by food products manufacturers, primarily in packaged meats. Arsenic was identified at significant concentrations (up to 2 mg/L) in samples of sodium lactate, and chromium up to 2.3 mg/L. In earlier unpublished work, sodium lactate was found to contain ketones (acetone on the order of 2.6 mg/L, 2-butanone [methyl ethyl ketone, MEK]) on the order of 0.0380 mg/L, and 2-hexanone (methyl butyl ketone [MBK] on the order of 0.0073 mg/L) based on laboratory analyses and confirmed by vendors. In addition to ketones, ethanol was found at 880 mg/L and isobutyl alcohol at 0.02 mg/L in samples of sodium lactate used for bioremediation. Furthermore, sodium lactate free of impurities could not be secured following contacts with numerous distributors and manufacturers.

Based on a review of the available health data for the identified impurities in sodium lactate, arsenic would pose the greatest potential health risk if present at even low concentrations in food products. Chromium was selected because this metal was detected at higher concentrations in the undiluted sodium lactate solution. The purpose of our study was to determine whether two impurities—arsenic and chromium—were at levels of health significance in food products preserved with sodium lactate available to the general public.

Several regulatory controls are in place to protect the food supply from potential impurities/contaminants introduced through food additives such as sodium lactate. The U.S. Department of Agriculture (USDA) regulates meat products but not the preservatives that are added to these products. According to USDA, it is the "sole responsibility of the manufacturing plant to provide documentation that the lactate serves the purpose intended in the product" and test the finished consumer-ready package according to "their own testing frequency" using their own lab if available (personal communication, Technical Service Center Staff Officer, Food Safety and Inspection Service Technical Service Center, USDA, July 21, 2008). A spokesperson for a leading company that produces meats preserved with sodium lactate notes that "FDA [U.S. Food and Drug Administration] tested all ingredients and determined them to be safe. The meat has been approved by the USDA. The food is preserved by the plant according to strict government regulations."

Furthermore, FDA does not regulate meat products (personal communication with the Communication & Coordination Branch, FDA, July 11, 2008), but it does regulate the use of specific substances used as food additives (personal communication with the

Communication & Coordination Branch, FDA, July 17, 2008). For example, the regulation that addresses sodium lactate can be found in Title 21, Code of Federal Regulations (Sodium Lactate, 2010), which classifies sodium lactate as GRAS (generally recognized as safe). This classification is exempted from FDA's testing and approval process, although the additive could be removed from the list if tests indicate that it is not safe for human consumption. Therefore, our study provides an important contribution to understanding and evaluating arsenic and chromium in sodium lactate, a food additive that currently is exempt from further testing and approval by FDA.

Review of Literature

Sodium Lactate

Sodium lactate (NaL) is a food-grade sodium salt derived from the fermentation of lactic acid (JRW Bioremediation, 2009). Sodium lactate is used as a food flavor enhancer and preservative. It is manufactured in India, China, and the U.S. (Chemical Register, 2007). Sodium lactate has been used to control *Clostridium perfringens* spores in pork (Reddy Velugoti, Rajagopal, Juneja, & Thippareddi, 2007) and chicken (Juneja, 2006); *Listeria* and *Salmonella* in beef (Serdengecti, Yildirim, & Gokoglu, 2006); to potentiate enterocin AS-48 to control toxicogenic *Bacillus cereus* in rice gruel (Grande et al., 2006); and many more uses in food preservation.

Arsenic

Arsenic is a naturally occurring element in soil and minerals (Agency for Toxic Substances and Disease Registry [ATSDR], 2007). It has long been a top priority concern for FDA's regulation of the U.S. food supply (Jelinek & Corneliussen, 1977). Human exposure to arsenic is primarily through dietary and drinking water ingestion (ATSDR, 2007). Total dietary arsenic exposures vary, ranging from 1.01–1,081 µg/day (mean of 50.6 µg/day) for females and 0.21–1,276 µg/day (mean of 58.5 µg/day) for males (ATSDR, 2007). Grains and produce are considered primary sources of inorganic arsenic in the U.S. diet, estimated to range from 1 to 20 µg/day. Although drinking water contains an average of 2 µg/L of arsenic, water supplies in particular U.S. regions have levels exceeding 20 µg/L (ATSDR, 2007). Arsenic has been identified in seafood, carrots, and rice, and extraction methods have been researched for these food sources (Heitkemper, Kubachka, Halpin, Allen, & Schockey, 2009).

Arsenic can be exhibited in a variety of chemical species. Arsenic III and arsenic V are the main species in the environment. Under most conditions, arsenic III is found in its neutral form (arsenite), and arsenic V is found as the anion arsenate. Arsenate is the main species in contaminated soil, and bacterial and other environmental occurrences can transform arsenate into more mobile and toxic forms of arsenic (Melamed, 2004). Preserving the arsenic conditions *in situ* during sampling is difficult and costly, and laboratory analysis of arsenic speciation is also expensive (Melamed, 2004).

Little is known about the relationship of organic arsenic compounds and humans (ATSDR, 2007). Animal studies suggest a lower toxicity in simple organic arsenic compounds when compared to inorganic ones. For example, "methyl and dimethyl compounds can cause diarrhea and damage to the kidneys (ATSDR, 2007)." Multisystem symptoms result from acute ingestion of several hundred milligrams of soluble arsenic, such as sodium arsenite or arsenic trioxide, with initial symptoms occurring within 30 minutes of exposure (Kosnett, 2004).

Ingestion of low levels of arsenic can cause gastrointestinal distress with nausea and vomiting resulting in hypotension and metabolic acidosis (ATSDR, 2007; Kosnett, 2004). Symptoms progress within one to seven days to cardiovascular complications, including congestive heart failure, noncardiogenic pulmonary edema, and ventricular arrhythmias (Kosnett, 2004). Less is known about chronic arsenic exposure, although hypertension, ischemic heart disease, and decreased birth weight have been suggested in international studies (Kosnett, 2004). Inorganic arsenic is identified as a human carcinogen (U.S. Environmental Protection Agency [U.S. EPA], 2000) primarily affecting the skin, liver, bladder, and lungs (ATSDR, 2007) although it also can be used clinically to treat cancer (Kosnett, 2004).

ATSDR (2007) sets the minimal risk level for arsenic at 0.005 mg/kg/day for acute oral exposure with a gastrointestinal endpoint and 0.0003 mg/kg/day for chronic oral exposure with a dermal endpoint. The U.S. Environmental Protection Agency (U.S. EPA) standards for nonoccupational oral exposure to arsenic at a conservative reference dose of 0.0004 mg/kg/day. The concern is that arsenic could be present at low concentrations in food long before symptoms of arsenic-induced chronic health problems are evident. The World Health Organization says long-term arsenic exposure can lead to arsenicosis, a chronic illness that produces skin disorders, gangrene, and cancer of the kidneys and bladder (Northoff, 2007). Populations whose drinking water is contaminated by arsenic have higher rates of dermal lesions, peripheral neuropathy, skin cancer, and peripheral vascular disease (Otles & Cagindi, 2010).

Studies that specifically examine the presence of arsenic in products available to the U.S. population through commercial supermarkets are rare. One study examining arsenic in beverages and broths purchased through a Tucson, Arizona, chain supermarket found variability between lots and brands, and also identified arsenic levels higher than groundwater standards set by U.S. EPA (Roberge et al., 2009). Arsenic was included as one of the analytes in the Total Diet Study that annually evaluated the publicly accessible food sources for a number of contaminants (FDA, 2010). Hughes and co-authors (2007) describe the current lack of critical information regarding the identification of the chemical species that are active toxicants and potentially susceptible populations. Our study contributes to the dearth of literature that focuses on the presence of arsenic in commercially available food sources.

Chromium

Chromium is a naturally occurring element, placed in the first transitional level of the periodic table (ATSDR, 2008). The three most stable forms of chromium are 0 (metals and alloys), III (trivalent chromium), and VI (hexavalent chromium) (U.S. EPA, 1998). Chromium IV and chromium V are the intermediate forms of chromium III and VI. Gastrointestinal absorbency of trivalent chromium is dependent on dietary practices, age, and dose of chromium intake (U.S. EPA, 1998). Trivalent chromium clears quickly from the bloodstream, binding to amino acids, other organic acids, and plasma proteins. It remains in the tissues

for months or more, most significantly in bone, liver, kidney, and spleen (U.S. EPA, 1998). Hexavalent chromium is more toxicologically active than trivalent chromium. It more readily crosses cell membranes, and once inside the cell is suspected to reduce to trivalent chromium (U.S. EPA, 1998). Chromium crosses the placental wall and is found in breast milk. It is primarily excreted through urine (ATSDR, 2008).

Dietary ingestion is the primary nonoccupational exposure route for chromium (ATSDR, 2008). Trivalent chromium potentiates insulin in peripheral tissues, and it is essential for lipid, protein, and fat metabolism (U.S. EPA, 1998). Chromium deficiencies have been linked to maturity-onset diabetes, cardiovascular diseases, and neurological disorders (U.S. EPA, 1998).

Chromium can be found throughout the food supply in small amounts (less than 0.002 mg), and it is a popular dietary supplement. Suggested daily intake by the National Research Council for chromium is 50–200 µg/day, corresponding to 0.71–2.9 µg/kg/day for a 70-kg adult (U.S. EPA, 1998). Adult men in the U.S. exceed the recommended adequate chromium intake level (National Institutes of Health [NIH] Office of Dietary Supplements, 2005). Although many health benefits are identified with low-dose chromium intake, the cumulative effect of overall chromium intake for children is not well studied. Additionally, certain medications interact with chromium; therefore, people should be aware of the presence of chromium in their foods if they are taking medications such as antacids, corticosteroids, H2 blockers, proton-pump inhibitors, beta-blockers, corticosteroids, insulin, nicotinic acid, nonsteroidal anti-inflammatory drugs, and prostaglandin inhibitors (NIH, Office of Dietary Supplements, 2005). Minimum risk levels (which do not consider carcinogenicity) have been set for chromium ingestion at ≥0.036 mg/kg (ATSDR, 2008). Most information for risk levels, however, has been determined by case reports for fatal ingestion.

Increasing doses of chromium have been shown to have toxic and even fatal results (ATSDR, 2008). No human studies of intermediate exposure (15–364 days) were identified, and ATSDR (2008) established the intermediate minimum risk level for exposure to hexavalent chromium at 0.005 mg/kg/day. A chronic (more than one year) exposure minimum risk level of 0.001 mg/kg/day has been established for hexavalent chromium. Overexposure to chromium through ingestion has been associated with gastritis, peptic ulcers, convulsions, kidney and liver damage, and death (ATSDR, 2008).

TABLE **1**

Pilot Study Analysis Results of Arsenic and Chromium in Foods Preserved With Sodium Lactate

Analysis	Level Found	Detection Limit	Method
Sample 07230801			
Arsenic (total)	n.d.[a]	0.01 ppm[b]	ICP-MS[c]
Chromium (total)	0.18 ppm	0.10 ppm	ICAP[d]
Sample 07230802			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.30 ppm	0.10 ppm	ICAP
Sample 07230803			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.12 ppm	0.10 ppm	ICAP

Note. Chromium values were analyzed by ICAP and confirmed by ICP-MS.
[a]Not detected.
[b]Parts per million.
[c]Inductively Coupled Plasma—Mass Spectrometry.
[d]Inductively Coupled Argon Plasma.

Methods

During the presampling walk-through survey of a retail consumer market of a national grocery chain, 60 food products were identified to contain sodium lactate from packaging ingredient lists in the following store refrigerators: luncheon meat, marinated meat, chicken, dinner ham, deli, and breakfast freezer. The majority of the labels indicated that these foods contained less than 2% sodium lactate in the package; however, the foods selected for the pilot sampling and analyses did not identify the percentage of sodium lactate. The samples for the study were subsequently obtained from the same store.

A pilot sampling of three food products (chicken nuggets, cooked ham, and smoked link sausage) of varying textures and moisture content was conducted to determine if the commercial laboratory could meet the requested low detection and reporting limits for arsenic and chromium. This request was made based on the concentrations of arsenic and chromium in the undiluted sodium lactate product, apparent amount of dilution, and available health data for these two compounds. Once the detection limits were established, a computer-generated random sampling of 17 food products that were labeled as containing sodium lactate was performed. The food samples were inserted in laboratory-provided sample bottles, placed in a cooler, and shipped to an accredited commercial food-testing laboratory along with accompanying chain of custody and custody seals. Midwest Laboratory, Inc., performed analytical testing of the selected food products for arsenic (total) and chromium (total). The sample preparation methods were derived from AOAC 985.01 (American Association of Analytical Chemists). The sample was then analyzed using Inductively Coupled Argon Plasma (ICAP) and confirmed by Inductively Coupled Plasma—Mass Spectrometry (ICP-MS).

The presence of arsenic is difficult to determine because of potential interference from other compounds. Also, detecting arsenic at such low concentrations is at the limits of the analytical instruments, which increase the difficulty in quantification. Due to the low detection limits, additional data quality assurance methods were used. The laboratory conducted duplicate analyses of

TABLE **2**

Arsenic and Chromium in 17 Random Samples of Foods Preserved With Sodium Lactate

Analysis	Level Found	Detection Limit	Method
Sample 08240801			
Arsenic (total)	n.d.[a]	0.01 ppm[b]	ICP-MS[c]
Chromium (total)	0.01 ppm	0.01 ppm	ICAP[d]
Sample 08240802			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.01 ppm	0.01 ppm	ICAP
Sample 08240803			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.03 ppm	0.01 ppm	ICAP
Sample 08240804			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.02 ppm	0.01 ppm	ICAP
Sample 08240805			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.04 ppm	0.01 ppm	ICAP
Sample 08240806			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	n.d.	0.01 ppm	ICAP
Sample 08240807			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.02 ppm	0.01 ppm	ICAP
Sample 08240808			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.01 ppm	0.01 ppm	ICAP
Sample 08240809			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.02 ppm	0.01 ppm	ICAP
Sample 08240810			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.04 ppm	0.01 ppm	ICAP
Sample 08240811			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.03 ppm	0.01 ppm	ICAP
Sample 08240812			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.01 ppm	0.01 ppm	ICAP
Sample 08240813			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.04 ppm	0.01 ppm	ICAP
Sample 08240814			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.02 ppm	0.01 ppm	ICAP
Sample 08240815			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	n.d.	0.01 ppm	ICAP
Sample 08240816			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.03 ppm	0.01 ppm	ICAP
Sample 08240817			
Arsenic (total)	n.d.	0.01 ppm	ICP-MS
Chromium (total)	0.03 ppm	0.01 ppm	ICAP

Note. Chromium values were analyzed by ICAP and confirmed by ICP-MS.
[a]Not detected.
[b]Parts per million.
[c]Inductively Coupled Plasma—Mass Spectrometry.
[d]Inductively Coupled Argon Plasma.

each sample with two different analytical methods, ICAP and ICP-MS, for confirmation and quality control.

The laboratory completed the pilot sample for chromium with a reporting concentration limit of 0.10 parts per million (ppm) per laboratory standards for chromium. For the next run with 17 samples, the laboratory reduced the reporting limit to 0.01 ppm at our request. The pilot sample results were significantly higher than the second set of samples, and the laboratory conducted a confirmatory analysis which supported the accuracy of the results for both samples.

Results

Arsenic was the primary impurity of concern in the sodium lactate. In the pilot study (Table 1) and the expanded study (Table 2), total arsenic for each of the samples was reported as not detected above the instrument detection limits. The highest arsenic concentration detected in the undiluted sodium lactate additive was on the order of 2 ppm (Chiang et al., 2008). For the majority of the food products researched, the packaging stated that sodium lactate was less than 2%. A 2% sodium lactate solution containing 2 ppm of arsenic would result in an arsenic concentration in the range of 0.04 ppm in food products if the highest concentration of arsenic and percentage of sodium lactate is assumed in the food. The laboratory's detection limit for arsenic was 0.01 ppm.

Chromium was reported to be above the detection limit in all three of the pilot test samples (Table 1) and in 88% of the subsequent samples (15/17) ranging in levels from non-detect to 0.30 ppm in the study samples (Table 2). The chromium detected in the pilot test samples (with labels not identifying the percentage of sodium lactate in the food) were significantly higher, from 0.12 ppm to 0.30 ppm compared to the second group of samples (highest concentration = 0.04 ppm). The ratio between the arsenic and chromium concentration in the undiluted sodium lactate was variable between batches and vendors; therefore, conclusions about whether the source of the chromium was from sodium lactate cannot be made.

Discussion

The lack of detected arsenic in the preserved foods may be a result of a combination of variables, including the available detection

limits of current laboratories, the amount of sodium lactate dilution, and the concentration of arsenic in the undiluted sodium lactate. The data suggest that although undiluted sodium lactate contains arsenic, once diluted for use as a food additive, the potential risk for exposure to arsenic is minimized to less than current detection limits.

Although chromium is a popular dietary supplement, the cumulative effects are not well understood in children, and its interactions with medications are of concern. Therefore, it is important to know the presence of chromium in food additives that may elevate the total chromium intake in daily diets. This is an especially important concern for children and persons taking antacids, corticosteroids, H2 blockers, proton-pump inhibitors, beta-blockers, corticosteroids, insulin, nicotinic acid, nonsteroidal anti-inflammatory drugs, and prostaglandin inhibitors. Many of the foods tested are popular, especially with children. These foods include but are not limited to cooked ham, chicken nuggets, skinless beef franks, sausage, and turkey. The additive effects of frequent intake of these foods by children is unknown and is an area of further study for the environmental health professional.

The foods found to have the highest levels of chromium included chicken nuggets and ham in the pilot sample, sausage links in both the pilot and random samples, and turkey breast in the random sample. The turkey breast contained 0.04 ppm of chromium, which equals 2 µg in one serving (4 shaved slices). Chromium in the chicken nugget sample in the pilot study equaled 27 µg in a single serving (five nuggets). Using the National Research Council–recommended intake of chromium of 50–200 µg/day, corresponding to 0.71–2.9 µg/kg/day for a 70-kg adult (U.S. EPA, 1998), these data do not support concern of a risk for chromium overexposure due to ingestion of sodium-lactate-preserved foods for the intake of a normal adult. More study is warranted for children and those on medications, as the health effects for chromium exposure can be serious.

Conclusion

Periodic replication of this study would be warranted to verify that the dilution results remain at acceptable levels. Further investigation may also be directed toward the presence of ketones, specifically acetone, methyl ethyl ketone (2-butanone), and ethanol, which also have been identified in food-grade sodium lactate. Ideally, further analysis of the food-grade sodium lactate used in processing is warranted. Because Roberge and co-authors (2009) identified variations between lots in their study of arsenic in beverages, future studies should expand to include an analysis of multiple lots of the same food sources.

Arsenic and chromium are present in the undiluted food-grade sodium lactate at concerning levels. Our study, however, yielded nondetectable results for arsenic. Chromium was detectable in the samples of food studied, although it cannot be directly inferred that the origin of the chromium was from sodium lactate. The arsenic and chromium data from our study suggest that dilution is adequate at this time for adults, but monitoring of proper dilution will remain a concern for the environmental health professional. Improper dilution could pose risk to the population consuming these products, especially the more vulnerable populations of children, older adults, and those on medications. Future studies by environmental health professionals are warranted to focus on the sources of impurities in the concentrated sodium lactate, such as soil fumigation, other environmental factors, or biologic processes.

Foods and components used to preserve foods are produced throughout the world, but not necessarily under strict controls on what eventually is sold in the local supermarket (Roberge et al., 2009). Although sodium lactate is classified as GRAS, the environmental health professional has a responsibility to be aware of and vigilant for potential contaminants that may threaten the safety of the food supply.

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References

Agency for Toxic Substances and Disease Registry. (2007). *Toxicological profile for arsenic*. Atlanta, GA: Author. Retrieved from http://www.atsdr.cdc.gov/ToxProfiles/tp2.pdf

Agency for Toxic Substances and Disease Registry. (2008). *Toxicological profile for chromium*. Atlanta, GA: Author. Retrieved from http://www.atsdr.cdc.gov/ToxProfiles/tp7.pdf

ChemicalRegister. (2011). *Sodium lactate CAS 72-17-3 suppliers*. Retrieved from http://www.chemicalregister.com/Sodium_Lactate/Suppliers/pid12707.htm

Chiang, D., Carter, G., & Woodward, D. (2008, May). *Potential complications from the injection of organic substrate for enhanced reductive dechlorination*. Paper presented at the Sixth International Conference on Remediation of Chlorinated and Recalcitrant Compounds, Monterey, California.

Grande, M.J., Lucas, R., Abriouel, H., Valdivia, E., Omar, N.B., Maqueda, M., Martinez-Bueno, M., Martinez-Canamero, M., & Galvez, A. (2006). Inhibition of toxicogenic *Bacillus cereus* in rice-based foods by enterocin AS-48. *International Journal of Food Microbiology, 106*(2), 185–194.

Heitkemper, D., Kubachka, K., Halpin, P., Allen, M., & Schockey, N. (2009). Survey of total arsenic and arsenic speciation in U.S.-produced rice as a reference point for evaluating change and future trends. *Surveillance Communications, 2*(2), 112–120.

References

Hughes, M., Kenyon, E., & Kitchin, K. (2007). Research approaches to address uncertainties in the risk assessment of arsenic in drinking water. *Toxicology & Applied Pharmacology, 222*(3), 399–404.

Jelinek, C.F., & Corneliussen, P.E. (1977). Levels of arsenic in the United States food supply. *Environmental Health Perspectives, 19,* 83–87.

JRW Bioremediation, LLC. (2009). *WilClear lactate concentrate specifications*. Retrieved from http://www.jrwbioremediation.com/pdf/WILCLEARCutSheet1-09.pdf

Juneja, V.K. (2006). Delayed *Clostridium perfringens* growth from a spore inocula by sodium lactate in sous-vide chicken products. *Food Microbiology, 23*(2), 105–111.

Kosnett, M.J. (2004). Arsenic, an old poison rediscovered. *Journal of Toxicology—Clinical Toxicology, 42*(4), 423–424.

Melamed, D. (2004). *Monitoring arsenic in the environment: A review of science and technologies for field measurements and sensors* (U.S. EPA Document #542/R-04/002). Cincinnati, OH: U.S. EPA, National Service Center for Environmental Publications. Retrieved from http://www.epa.gov/tio/download/char/arsenic_paper.pdf

National Institutes of Health, Office of Dietary Supplements. (2005). *Dietary supplement fact sheet*. Retrieved from http://ods.od.nih.gov/factsheets/Chromium-HealthProfessional

Northoff, E. (2007, December). *Arsenic threat in rice*. FAO Newsroom, Food and Agriculture Organization of the United Nations. Retrieved from http://www.fao.org/newsroom/en/news/2007/1000734/index.html

Otles, S., & Cagindi, O. (2010). Health importance of arsenic in drinking water and food. *Environmental Geochemistry and Health, 32*(4), 367–371.

Reddy Velugoti, P., Rajagopal, L., Juneja, V., & Thippareddi, H. (2007). Use of calcium, potassium, and sodium lactates to control germination and outgrowth of *Clostridium perfringens* spores during chilling of injected pork. *Food Microbiology, 24*(7–8), 687–694.

Roberge, J., Abalos, A., Skinner J., Kopplin, M., Harris, R., Zuckerman, M., & Zuckerman, E. (2009). Presence of arsenic in commercial beverages. *American Journal of Environmental Sciences, 5*(6), 688–694.

Serdengecti, N., Yildirim, I., & Gokoglu, N. (2006). Investigation of inhibitory effects of several combinations of sodium salts on the growth of *Listeria* monocytogenes and *Salmonella enterica* serotype *Enteritidis* in minced beef. *Journal of Food Safety, 26*(3), 233–243.

Sodium Lactate (U.S. Food and Drug Administration), 21 C.F.R. § 184.1768 (2010).

U.S. Environmental Protection Agency. (1998). *Toxicological review of trivalent chromium* (CAS #16065-83-1). Retrieved from http://www.epa.gov/iris/toxreviews/0028tr.pdf

U.S. Environmental Protection Agency. (2000). *Engineered approaches to* in situ *bioremediation of chlorinated solvents: Fundamentals and field applications* (U.S. EPA Document #542-R-00-008). Retrieved from http://www.clu-in.org/download/remed/engappinsitbio.pdf

U.S. Environmental Protection Agency. (2007). *Arsenic, inorganic. Integrated risk information system* (CASRN #7440-38-2). Retrieved from http://www.epa.gov/iris/subst/0278.htm

U.S. Food and Drug Administration. (2010). *Total diet study*. Retrieved from http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/default.htm

INTERNATIONAL PERSPECTIVES

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Mutagenicity and Genotoxicity of Water Treated for Human Consumption Induced by Chlorination By-products

Although most of the information presented in the Journal *refers to situations within the United States, environmental health and protection know no boundaries. The* Journal *periodically runs International Perspectives to ensure that issues relevant to our international membership, representing over 20 countries worldwide, are addressed. Our goal is to raise diverse issues of interest to all our readers, irrespective of origin.*

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Abstract **Water used for human consumption may contain mutagens and carcinogens generated during the disinfection process with chlorine. In the study described in this article, the mutagenicity and genotoxicity of water samples taken from the San Cristobal treatment plant in Medellin, Colombia, were evaluated. Short-term mutagenic and genotoxic assays using the Ames test and comet assay, respectively, were employed to examine the genotoxic activity of the extracts of these water samples. Two samples were taken before and after the chlorination process. The treated water samples without chlorination did not show mutagenic effects using the Ames test, while the chlorinated samples produced mutagenic activity in both strains. A dose-response relationship for the comet assay was obtained only in the chlorinated samples. MX (3-chloro-4-[dichloromethyl]-5-hydroxy-2[5H]-furanone), E-MX ([E]-2-chloro-3-[dichloromethyl]-4-oxobutenoic acid), and some trihalomethanes were detected at low concentrations. These concentrations were enough, however, to cause detectable mutagenic and genotoxic activity in the extracts of chlorinated water samples.**

Introduction

Most of the routine analyses performed for drinking water quality are focused on physicochemical and microbiological tests. Based on these analyses, some criteria for water used for human consumption have been established. In this way, with the introduction of water disinfection, the population can be ensured that their drinking water is likely to be free of waterborne infectious diseases (Boorman et al., 1999). Alternatives methodologies exist, but none of them offers continuous protection against pathogens through the distribution network.

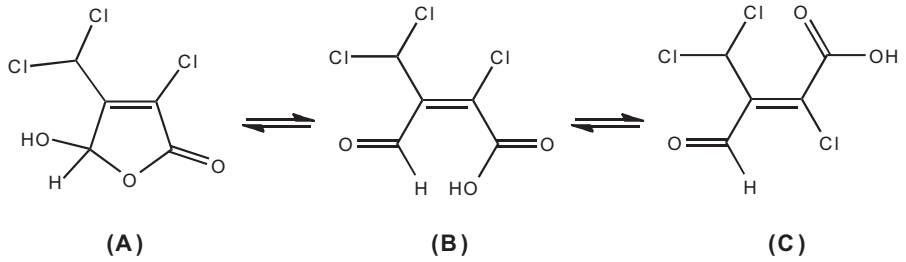
Since the 1970s, a new risk for human health appeared in drinking water, as findings showed that it can contain mutagenic and carcinogenic compounds known as disinfection by-products (DBPs) (Hemming, Holmbom, Reunanen, & Kronberg, 1986; Kusamran et al., 1994; Meier, Blazak, & Knohl, 1987). When chlorine reacts with humic and fulvic acids present naturally in the water, it can produce several compounds such as trihalomethanes, halofuranes, haloacetic acids, halophenols, halopropanones, and others that are well known for their mutagenic and carcinogenic properties (Langvik & Holmbom, 1994; Richardson, Plewa, Wagner, Schoeny, & DeMarini, 2007; Richardson, Simmons, & Rice, 2002; Shi et al., 2009). A large number of those compounds have been isolated from chlorinated waters (McDonald & Komulainen, 2005; Richardson et al., 2007). The trihalomethanes (THMs) include chloroform ($CHCl_3$), dibromochloromethane ($CHBr_2Cl$), bromodichloromethane ($CHBrCl_2$), and bromoform ($CHBr_3$); these compounds represent between 5% and 20% of the total DBPs (Fayad, 1993).

Other compounds with similar properties have been identified and quantified in chlorinated water, and it is believed that they are responsible for the rest of the mutagenic activity. Within the compounds detected are the haloacetic acids (HAAs) (Krasner et al., 2006) and chlorohydroxyfuranones (MXs) (Kronberg & Vartiainen, 1988; Smeds, Vartiainen, Maki-Paakkanen, & Kronberg, 1997). Hemming and co-authors (1986) identified and quantified 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX), one of the most potent bacterial mutagens that is the product of the reaction of chlorine with the organic material present in water. Figure 1 shows the structure of MX and its open isomeric forms, Z-MX ([Z]-2-chloro-4-[dichloromethyl]-4-oxo-butenoic acid) and E-MX ([E]-2-chloro-3-[dichloromethyl]-4-oxobutenoic acid) (Franzen & Kronberg, 1994; Richardson et al., 2007).

MX can be found at low levels (2–310 ng/L) in drinking water (Kronberg, Christman, Singh, & Ball, 1991; McDonald & Komulainen, 2005; Wright et al., 2002). It has been estimated, however, that this compound

FIGURE **1**

Structure of MX and Its Isomeric Forms: (A) MX, (B) Z-MX, and (C) E-MX



MX = 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone, Z-MX = (Z)-2-chloro-4-(dichloromethyl)-4-oxo-butenoic acid, E-MX = (E)-2-chloro-3-(dichloromethyl)-4-oxobutenoic acid.

may contribute about 3% to 67% of the total mutagenicity of chlorinated waters, inducing a wide spectrum of mutations in bacterial and mammalian cells (Hyttinen, Myohanen, & Jansson, 1996; Jansson & Hyttinen, 1994; Maki-Paakkanen & Hakulinen, 2008; Wright et al., 2002). In *Salmonella typhimurium* strains TA100 and TA102, MX induces damage of the DNA by base-pair substitution (Hemming et al., 1986; Kronberg & Vartiainen, 1988) and in *Salmonella typhimurium* TA98, a bacterial strain sensitive to frameshift mutations, MX produces loss or gain of a pair of bases (DeMarini, AbuShakra, Felton, Patterson, & Shelton, 1995). TA98 and TA100 have been widely used to test a numerous series of chemical mutagens and carcinogens.

MX also induced a wide variety of DNA damage in mammalian cells *in vitro* (Jansson & Hyttinen, 1994; Maki-Paakkanen & Hakulinen, 2008) including human cells (Chang, Daniel, & Deangelo, 1991) such as sister chromatid exchange (SCE), chromosomal aberrations (Hyttinen et al., 1996; Jansson et al., 1993), DNA strand breaks and different kinds of mutations (Hyttinen et al., 1996; Richardson et al., 2007), and other effects (King, Hester, Warren, & DeMarini, 2009). MX has been classified by the International Agency of Research on Cancer (IARC) as a possible carcinogen in humans (type 2B) (International Agency of Research on Cancer [IARC], 2004).

Several studies have concluded that a potential risk of cancer is associated with the consumption of chlorinated water (Cantor, 1997; Tao, Zhu, & Matanoski, 1999). IARC (1995) conducted research that concluded that a positive correlation exists between chlorinated water consumption and the development of kidney and bladder cancer. Nevertheless, for the amount of factors to be considered, IARC considers that not enough evidence exists to classify DBPs as carcinogenic agents in humans. By contrast, the World Health Organization (WHO) recommendation prioritized the latter for authorities wishing to meet the disinfection by-products and the microbiological guidelines (World Health Organization [WHO], 2004).

According to WHO, MX concentrations of 1.8 µg/L are associated with 10^{-5} cancer risk for a 60 kg adult drinking 2 L of water per day (WHO, 2004). In 2001, some studies were initiated in Colombia to understand the mutagenic effect of chlorinated and nonchlorinated waters in one of the principal water treatment plants (Villa Hermosa) in the city of Medellin (Melendez, Zuleta, Marín, Calle, & Salazar, 2001). The authors found mutagenicity before and after the chlorination process and concluded that the pollution from the river that supplies water to the plant and DBP formation from the chlorination process were responsible for the mutagenicity.

In our study we evaluated the mutagenic and genotoxic effect of water extracts taken from San Cristobal water treatment plant in Medellín, Colombia, increasing the number of treatment plants previously evaluated for mutagenicity by DBPs in Colombia. The main goal of our study was to identify the potential risk to the population due to the presence of these compounds in drinking water.

Methods

Water Sampling and Sample Workup Procedure

Water samples were obtained from two different areas of the San Cristobal plant: (1) immediately before chlorination and (2) after the chlorination process but before water distribution. Samples were taken manually at a total volume of 80 L. The pH was adjusted to 2 with concentrated HCl. The samples were passed through columns filled with XAD-2 and XAD-7 (1:1) sorbants at a flow rate of 15 mL/min., according to procedure described by Meier and co-authors (Meier, Knohl, et al., 1987) and Stahl (1991), with some modifications. The elution was performed with 300 mL of acetone and 300 mL of methanol. The volume of the eluent was concentrated on a roto-evaporator at 55°C. The samples were weighed and kept at -20°C for further mutagenic and genotoxic assays.

Mutagenic Test (Ames Test)

The mutagenic activity of water extracts and MX were determined by means of the Ames test (Maron & Ames, 1983), using two strains (TA98 and TA100) of *Salmonella typhimurium*, with metabolic activity (with mixture S9, made from a fraction of rat liver homogenate) and without metabolic activity (without mixture S9) to detect indirect mutagenic activity. The 2-aminofluorene (2-AF, 10 µg/plate) was used as a positive control. Sterile distilled water was used as a negative control. The tests were conducted using three doses in duplicate, with a minimum of three independent experiments. The answer is positive when the number of mutations is at least doubled in contrast with the negative control, according to the criteria suggested by WHO (2004).

Genotoxic Test (Comet Assay)

To determine the level of damage of the DNA of human lymphocytes, the technique single cell gel electrophoresis (SCGE) or comet assay was utilized according to the protocol described by Singh and co-authors (1988) with slight modifications. Between 5,000 and 50,000 human lymphocytes were isolated from total blood by Ficoll density gradient centrifugation (Duthie,

Ross, & Collins, 1995), which were incubated with the water extracts.

The viability of lymphocytes was determined with the trypan blue (0.2%) exclusion test, before and after the treatment showing values greater than 91%. Hydrogen peroxide (50 µM) was used as a positive control; dimethyl sulphoxide (DMSO) and phosphate buffered saline (PBS) as negative control.

After the treatment, 10µL of the cellular suspension was mixed with 75µL of low-melting-point agarose (LMA) 0.5% (w/v) at 37°C and placed onto microscope slides precoated with normal-melting-point agarose (NMA). The cellular suspension was covered with a cover slip and maintained at 4°C for five minutes. The cover slip was removed and a third layer of agarose was added and cooled up to 4°C. The slides were immersed in a lysis solution adjusted to pH 10 (NaCl 2.5 molars [M], Na_2EDTA 100 millimolar [mM], TRIS 10 mM, Sarcosinate 1%, Triton X-100 1%, and DMSO 10%) at 4°C for 90 minutes.

The layers were placed in a buffer solution (Na_2EDTA 1 mM, NaOH 300 mM, pH 13) for electrophoresis to allow DNA unwinding. The electrophoresis was conducted at 4°C for 30 minutes at 25 volts and 300 milliamperes. After this procedure, the slides were rinsed with a neutralizing buffer (tris-HCl 0.4 M, pH 7.5) for 15 minutes and dehydrated with methanol. The slides were stained with 40 µL of acridine orange (5µg/mL) and examined with a fluorescence microscope equipped with an excitation filter of 450–490 nm, using a magnification of 10x and 40x.

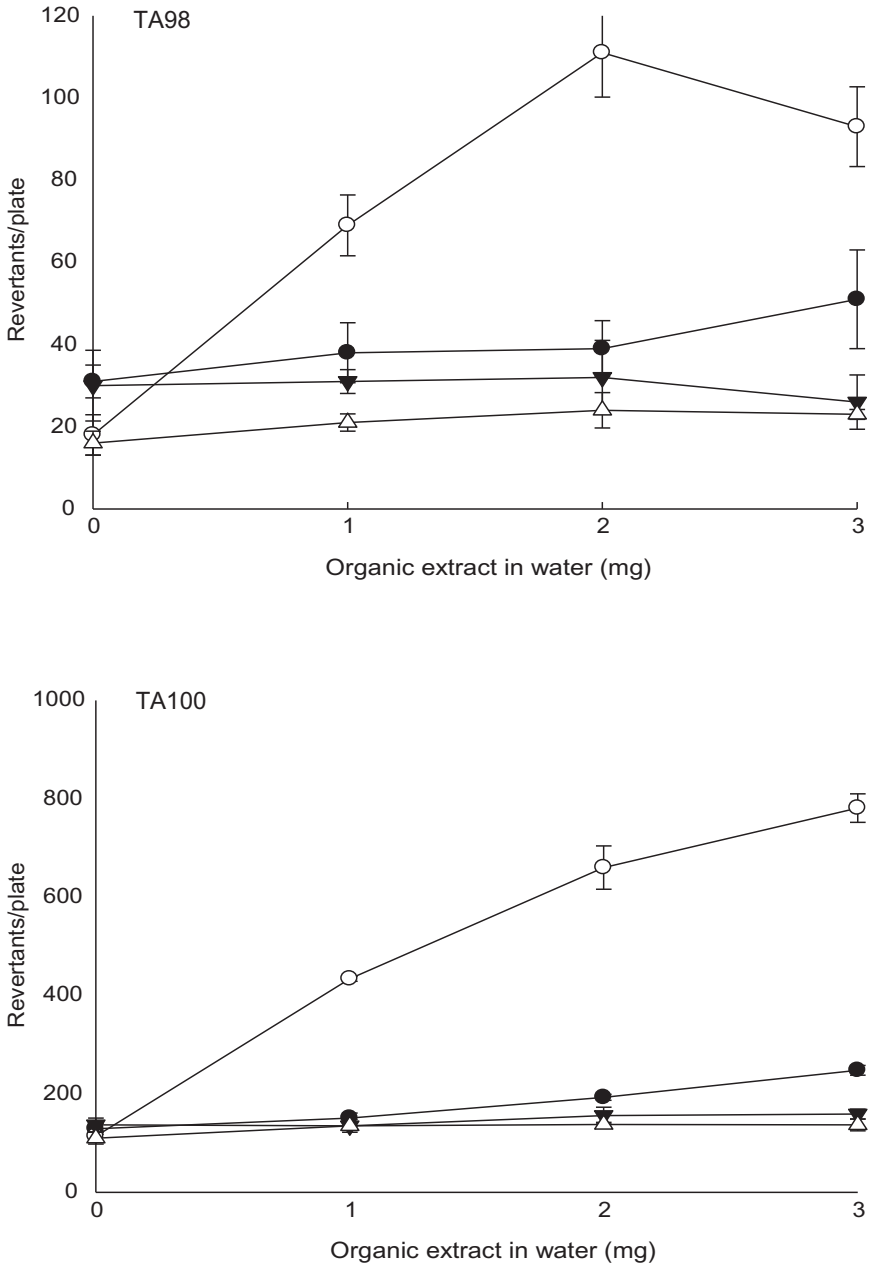
The slides were ready for image analysis using a fluorescence microscope with a camera. Twenty-five randomly selected comet cells from each slide were analyzed with an ocular-micrometer; two slides were done by doses. The DNA damage was evaluated by measuring the length of the resulting image (nuclei diameter plus migrated DNA comet tail) in microns and an average was calculated. The effect of the doses over the migration of the DNA was analyzed by the Duncan test with $\alpha = .05$.

Gas Chromatography-Mass Spectrometry (GC-MS) Analysis of MX and E-MX

Twelve liters of each water sample were concentrated on a column of 12 x 40 cm filled with XAD-2 and XAD-7 (1:1) with a

FIGURE **2**

Mutagenic Effects of the Water Extract Before and After the Chlorination in *Salmonella typhimurium*, Strain TA98 and TA100 With and Without S9



Mutagenic effects of the water extract before and after the chlorination in *Salmonella typhimurium*, strain TA98 and TA100 with and without S9. Water before chlorination without S9 (—△—), water before chlorination with S9 (—▼—), water after chlorination without S9 (—○—), water after chlorination with S9 (—●—).

TABLE **1**

Mutagenicity of the Water Extracts With and Without Chlorine in the Strains TA98 and TA100

Doses (mg/Plate)	Mixture S9	Average Mutagenicity (Rev/Plate) ± *SD* TA98		Average Mutagenicity (Rev/Plate) ± *SD* TA100	
		Chlorine +	Chlorine -	Chlorine +	Chlorine -
1.0	+	38 ± 7.3	31 ± 2.9	151 ± 10	135 ± 0.5
1.0	-	69 ± 7.4	21 ± 2.1	434 ± 5	135 ± 13
2.0	+	39 ± 6.8	32 ± 9	193 ± 6	156 ± 17
2.0	-	111 ± 10.8	24 ± 4.3	660 ± 44	138 ± 4
3.0	+	51 ± 12	26 ± 6.6	248 ± 9.9	159 ± 10
3.0	-	93 ± 9.7	23 ± 1.2	781 ± 29	137 ± 12
C-*	+	31 ± 4.0	30 ± 8.6	129 ± 11	137 ± 14
C-*	-	18 ± 4.9	16 ± 2.9	114 ± 12	110 ± 12

*Negative control: distilled water.

flow rate of 20 mL/min. The adsorbed organics were eluted with 300 mL of ethyl acetate (EtAc). The EtAc extracts were concentrated in a roto-evaporator. The MX and E-MX were determined in the extract after derivatization with methanol at 70°C for 2 hours in a H_2SO_4 2% (v/v) (sulfuric acid) solution.

The GC-MS analyses for non-volatile organochlorinated compounds were performed on a gas chromatograph Agilent G1530A (HP 6890A) equipped with a selective mass detector 5973N MSD. The separation of the components in the GC-MS analyses was performed on a HP-5MS fused silica column (30 m x 0.25 mm i.d., film thickness 0.25 µm). The carrier gas was helium at a flow rate of 1 mL/min. The oven temperature was programmed from 80°C to 230°C at a rate of 6°C/min.; the injector temperature was kept at 250°C. Electron impact (EI^+) ionization was at 70 electron volts.

GC/Electron Capture Detector (ECD) Analyses for THMs

The standard solutions for calibration were prepared from a standard calibration mix. They were prepared in iso-octane for chloroform and n-pentane for bromoform, bromodichloromethane and dibromochloromethane.

For the quantification of THMs, 1 mL of fresh water treated from the water treatment plant San Cristobal was extracted with 0.5 mL of n-pentane or iso-octane for chloroform (David, Sandra, & Klee, 1997). Chloroform was extracted in iso-octane because of the co-elution with n-pentane during the chromatographic analysis.

The analyses were preformed in a gas chromatograph Shimadzu 15-A, with an injector SPL-G9 and ECD. The system was equipped with a DB-1 column (30 m, 0.53 mm i.d., film thickness 1.5 µm). The carrier gas was helium (5.7 psi) with an isothermal temperature of 45°C for bromoform, bromodichloromethane, and dibromochloromethane and for chloroform a temperature program of 45°C for 2.6 minutes with a ramp of 20°C/min. up to 125°C for 3.0 min. The temperature of the injector and the detector were 250°C and 310°C, respectively.

Results and Discussion

Mutagenicity of the Water Extracts Before and After Chlorination

Figure 2 shows the mutagenic activity of the water extracts before and after chlorination in *Salmonella typhimurium*, in strains TA98 and TA100, in presence and absence of the mixture S9. The water extracts for human consumption in the San Cristobal plant exhibited mutagenicity to the TA98 and TA100 strains of *Salmonella typhimurium* in absence of the mixture S9 (Table 1, Figure 2).

Table 1 shows the mutagenic activity of water extracts of *Salmonella* TA98 and TA100. The water extracts with chlorine and without mixture S9 exhibited a significant mutagenic activity in both strains. For the TA98 strain, the three concentrations tested (1, 2, 3 mg/plate) produced four-, six-, and five-fold increases, respectively, in mutant frequency relative to the negative control without mixture S9. Regarding the same control, the TA100 strain, the three concentrations tested (1, 2, 3 mg/plate) produced four-, six- and seven-fold increases, respectively. Those results indicate that the extracts contain a strong mutagenic activity, according to the criteria established by WHO (Coulston & Dunne, 1980). Furthermore, due to the fact that the extract presented mutagenic activity to both strains suggests that the mechanism to damage the DNA involves frameshift mutations (loss or gain of a pair of bases) as substitution of a pair of bases (Benigni, 2005; King et al., 2009).

The mutagenic activity for both extracts of chlorinated water for both strains (TA98 and TA100) was reproducible with a relatively low standard deviation (10.3% and 3.8% coefficient of variation, respectively) over six assays developed in a period of four months.

The TA98 strain presented a linear mutagenic response until 2 mg of extract (Figure 2), where it reaches the maximal number of revertants/plate. The highest dose used (3 mg/plate) decreased by about 16% the number of revertants/plate regarding the previous dose, which can be explained by a toxic effect to this level of dose. For the TA100 strain (Figure 2) a linear effect was observed for all doses tested.

In contrast, the water extract without chlorination did not present mutagenic activity for

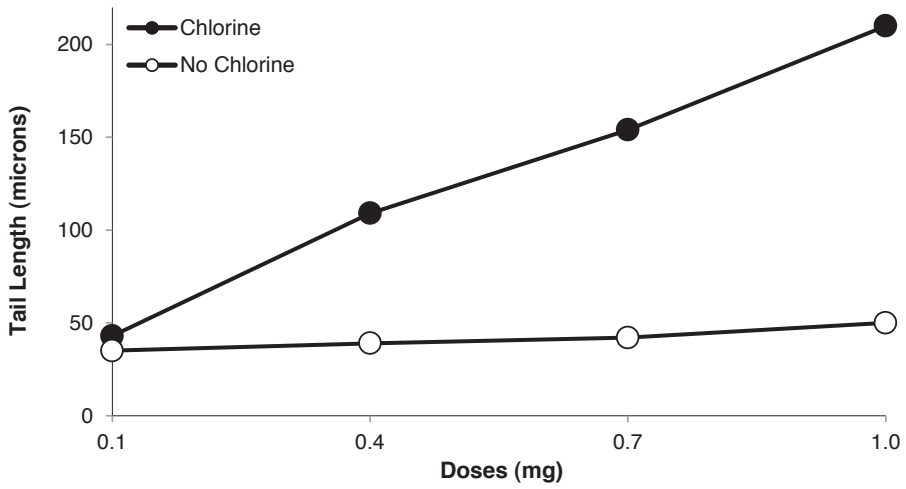
any of the strains used in condition of presence or absence of S9 (Table 1 and Figure 2); this indicates that the substances responsible for the mutagenic activity of the extracts are formed during the chlorination process. Similar results were found in water extracts collected from Finland and Russia (Smeds et al., 1997).

Table 1 shows that the addition of the mixture S9 that contains microsomal enzymes responsible for the metabolic activity, i.e., cytochrome P450, lessens almost completely the mutagenicity of the extracts of chlorinated waters. These results are consistent with those of other researchers that report a reduction of the mutagenic activity in chlorinated waters in the presence of S9 in *Salmonella typhimurium,* strain TA100 (Backlund, Kronberg, Pensar, & Tikkanen, 1985). These results indicate that the presence of S9 reduces markedly the activity of the mutagenic substances that are present in the extracts; the average reduction of the mutagenicity was 51% for the strain TA98 and 68% for the strain TA100, suggesting that the mutagenic substances present in the extracts have a direct-acting mutagenicity. Previous work demonstrated that MX is a directly acting mutagen and its mutagenic affects *in vitro* are greatly reduced in the presence of liver enzymes-S9 mix (Franzen, Goto, Tanabe, & Morita, 1998).

According to the mutagenicity obtained for the extracts, it is possible to infer that a good recovery of the mutagenic compounds occurred using the procedure of absorption by XAD resins and acidifying previously the water samples at pH 2. This is in agreement with the fact that the resins are made of relative nonpolar materials (styrene-divinylbenzene copolymers) and at this pH the nonionic form of the MX is favored (Rezemini, Vaz, & Carvalho, 2008). That apparently is one of the compounds that is contributing to the main part of the mutagenic activity of the extracts.

FIGURE **3**

Length of Migration of DNA (Microns) of Human Lymphocytes Treated in Function of the Concentration of the Extracts (mg) of Water Before and After Chlorination, Evaluated for the Comet Assay



Each point represents the average of 150 cells evaluated in three different experiments.

Genotoxic Effect of the Water Extracts of the DNA of Human Lymphocytes

Figure 3 shows that the extracts of chlorinated water produced a significant migration of the DNA (tail of the comet) compared with the nonchlorinated extracts. Figures 4 and 5 show photomicrographs obtained during the comet assay for different doses used with the extracts of chlorinated water 0.1, 0.4, 0.7, and 1.0 mg; the negative and positive controls and a representative dose of the water extracts without chlorination (0.4 mg). The remaining doses of these extracts are not shown as they are very similar to those presented. The photomicrographs show how the extracts of chlorinated water produce migration of the DNA in the electrophoresis gel in a progressive way according to the dose, indicating that the extracts of chlorinated water contain compounds that are able to induce strand breaks and labile sites in the DNA of human lymphocytes. The damage is dependent on the dose, with the higher concentration of the extract, the greater the DNA migration, which is equivalent to greater damage. In contrast, the same concentrations 0.1, 0.4, 0.5, and 0.7 mg of the water extract without chlorination (only the photomicrograph of the dose 0.4 mg is shown) did not produce visible genotoxic damage (Figure 5C).

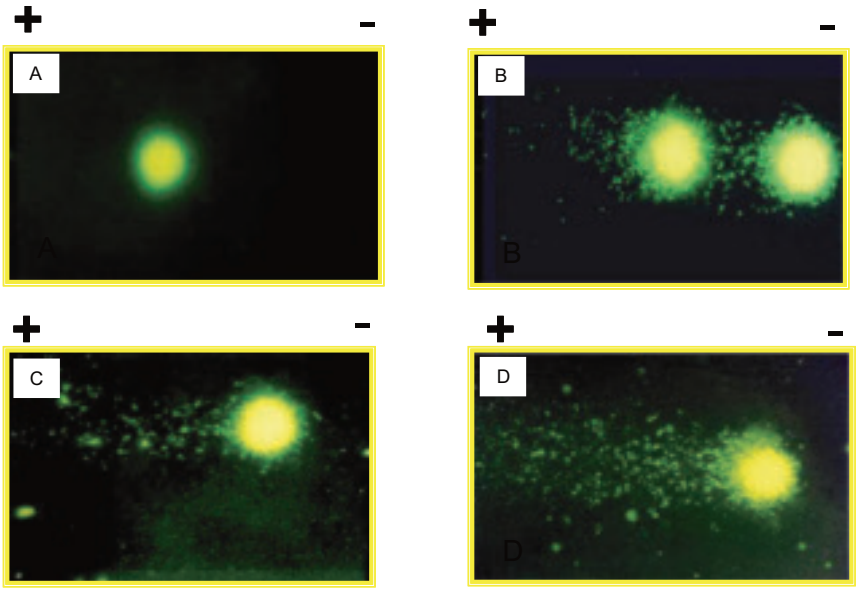
The DNA strand break evaluated with the comet assay could be produced for an adduct reparation mechanism possibly formed by the reaction of MX with DNA bases. According to Lindahl & Andersson (1972) the DNA adducts are repaired by N-glycosylases originating at an apurinic/apyrimidinic site (AP site) that can be repaired by AP-endonucleases or hydrolyzed by alkalis producing a DNA break, that is visualized in the length of the "tail of the comet."

Analytical Detection of THMs and MX/E-MX

Using the GC-MS technique, MX, and E-MX were identified in extracts of chlorinated water. Using scan mode the compounds of interest coeluted with some other compounds, however using single ion monitoring (SIM) the compounds were clearly identified by looking at the fragments (m/z) 198.9, 200.9, and 202.9 for MX, corresponding to the isotopic group OCH_3 in the MX; and the fragments 241.0 and 243.0 for the loss of chlorine in the E-MX molecule and the resulting molecule due to the loss of the group E-MX with the fragments 244.9 and 246.9. These fragments were used by other authors to identify these molecules in chlorinated waters (Kronberg, Holmbom, Reunanen, & Tikkanen, 1988; Smeds, Vartiainen, Maki-Paakkanen, & Kronberg, 1997). In Table 2 it is possible to observe a correspondence in the abundances for both the standards and the

FIGURE **4**

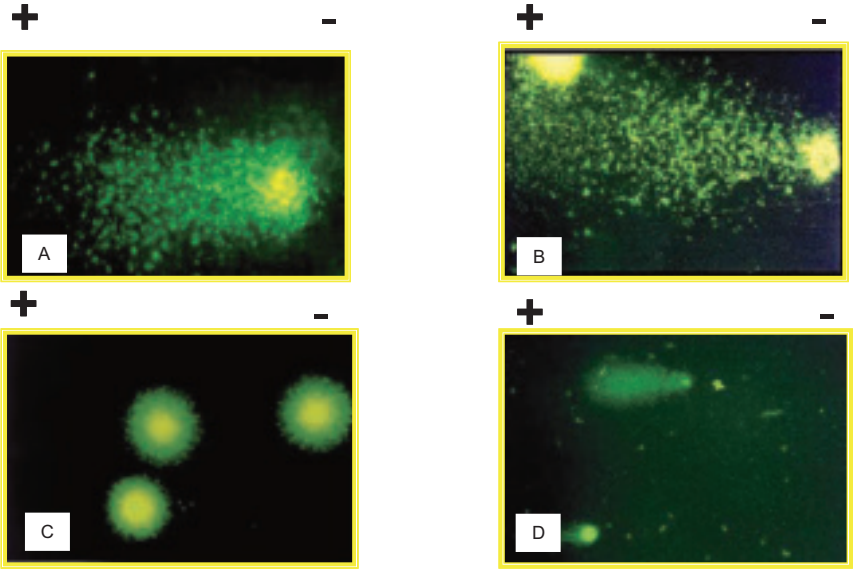
Microphotographs (40x) of Migration Patterns of DNA in Human Lymphocytes Treated With Chlorinated Water Extracts



Negative control (A), doses 0.1 mg (B), 0.4 mg (C), and 0.7 mg (D).

FIGURE **5**

Microphotographs (40x) of Migration Patterns of DNA in Human Lymphocytes Treated With Chlorinated Water Extracts



Doses 1.0 mg (A) and (B), nonchlorinated water dose 0.4 mg (C), and positive control (D) in 10x.

water extracts that confirms the presence of MX and E-MX in the samples. Quantification of these compounds was not possible at the time of the analysis due to unavailability of the standard when the analysis was performed.

The GC-ECD analysis was used to quantify four trihalomethanes: chloroform, bromoform, bromodichloromethane, and dibromochloromethane. Table 3 reports the quantity detected for each. The average concentration in the samples were 1.34 µg/L for $CHBrCl_2$, 0.24 µg/L for $CHBr_2Cl$, 0.30 µg/L for $CHBr_3$ and 5.77 µg/L for $CHCl_3$. The sum of the four THMs studied was 7.65 µg/L, an amount that is very small compared to the amount permitted by international regulations of 80 µg/L (National Primary Drinking Water Regulations, 2006). Nevertheless, depending on the period of sampling and some physicochemical conditions, the concentration found in drinking water may vary (Loyola-Sepulveda, Lopez-Leal, Munoz, Bravo-Linares, & Mudge, 2009).

Conclusion

The purpose of this research was to evaluate the mutagenic as well genotoxic effects of water treated with chlorine in the San Cristobal plant. The results show the following:

a) The concentrated water extracts after the chlorination process were mutagenic to bacteria and genotoxic for mammal cells (human lymphocytes), evaluated through the Ames test and comet assay, respectively. The use of both protocols in the same study allows a correlation between mutagenic and genotoxic events that are complementary, constituting a good tool for the evaluation of the sequential steps that can lead to a carcinogenic process.

b) The mutagens formed during the chlorination process are of direct action and are inhibited by enzymes of metabolic activation as the one contained in the mixture S9.

c) The detection by GC-MS of MX and its isomer E-MX in the extracts of chlorinated water suggests that part of the mutagenicity and carcinogenicity of these extracts might be attributed to the presence of these compounds classified by WHO (Coulston & Dunne, 1980) as potent mutagens of direct action.

d) The quantification of the THMs in the water extracts indicates that these compounds are present in a minimal amount, less than the permitted by the international regulations,

and consequently their contribution to the mutagenicity and genotoxicity of the water extracts may not be significant.

Further studies should be conducted to evaluate the mechanisms for what these compounds such as the MX and its isomers produce damage to the DNA. Also the data could be of interest for future study to specifically understand the mechanism involved in the genotoxicity and it could also be helpful to implement regulation to set criteria for acceptable limits of these compounds in water treated by chlorination. It will also open room to introduce mandatory testing for the presence of these compounds in drinking water.

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TABLE **2**

Selected Ions for the Identification of MX and E-MX and Comparison of Their Relative Abundances With the Standards

Compound	Fragment	m/z	Relative Abundance	
			Standard	Sample
MX	M-OCH_3	198.9	0.54	0.53
		200.9	1.00	1.00
		202.9	0.64	0.68
E-MX	M-Cl	241.0	0.62	0.70
		243.0	0.43	0.48
	M-OCH_3	244.9	1.00	1.00
		246.9	0.90	0.94

TABLE **3**

Height and Concentrations (µg/L) of Four Trihalomethanes in the Chlorinated Water Samples From the San Cristobal Treatment Plant

Sample	$CHBrCl_2$		$CHBr_2Cl$		$CHBr_3$		$CHCl_3$	
	H	µg/L	H	µg/L	H	µg/L	H	µg/L
1	7833	1.29	914	0.24	250	0.27	14,029	5.86
2	8055	1.33	900	0.24	300	0.32	12,484	6.34
3	8435	1.39	917	0.24	287	0.31	5796	5.11
Average	–	1.34	–	0.24	–	0.30	–	5.77

References

Backlund, P., Kronberg, L., Pensar, G., & Tikkanen, L. (1985). Mutagenic activity in humic water and alum flocculated humic water treated with alternative disinfectant. *Science of the Total Environment, 47*, 257–264.

Benigni, R. (2005). Structure-activity relationship studies of chemical mutagens and carcinogens: Mechanistic investigations and prediction approaches. *Chemical Reviews, 105*(5), 1767–1800.

Boorman, G.A., Dellarco, V., Dunnick, J.K., Chapin, R.E., Hunter, S., Hauchman, F., Gardner, H., Cox, M., & Sills, R.C. (1999). Drinking water disinfection by-products: Review and approach to toxicity evaluation. *Environmental Health Perspectives, 107*(Suppl. 1), 207–217.

Cantor, K.P. (1997). Drinking water and cancer. *Cancer Causes & Control, 8*(3), 292–308.

Chang, L.W., Daniel, F.B., & Deangelo, A.B. (1991). DNA strand breaks induced in cultured human and rodent cells by chlorohydroxy-furanones-mutagens isolated from drinking-water. *Teratogenesis Carcinogenesis and Mutagenesis, 11*(2), 103–114.

Coulston, F., & Dunne, J.F. (1980). *The potential carcinogenicity of nitrosatable drugs*. Geneva: World Health Organization.

David, F., Sandra, P., & Klee, M. (1997). *Analysis of trihalo-methanes by micro liquid-liquid extraction and capillary gas chromatography with the 6890 Micro ECD* (Publication #23 5965-8014E). Santa Clara, CA: Agilent Technologies.

DeMarini, D.M., AbuShakra, A., Felton, C.F., Patterson, K.S., & Shelton, M.L. (1995). Mutation spectra in *Salmonella* of chlorinated, chloraminated, or ozonated drinking water extracts: Comparison to MX. *Environmental and Molecular Mutagenesis, 26*(4), 270–285.

Duthie, S.J., Ross, M., & Collins, A.R. (1995). The influence of smoking and diet on the hypoxanthine phosphoribosyltransferase (HPRT) mutant frequency in circulating t-lymphocytes from a normal human-population. *Mutation Research-Fundamental and Molecular Mechanisms of Mutagenesis, 331*(1), 55–64.

Fayad, N.M. (1993). Seasonal variations of THMs in Saudi Arabian drinking water. *Journal of American Water Works Association, 85*(1), 46–50.

Franzen, R., Goto, S., Tanabe, K., & Morita, M. (1998). Genotoxic activity of chlorinated butenoic acids in *Salmonella typhimurium*

References

strains TA98, TA100, and TA104. *Mutation Research-Genetic Toxicology and Environmental Mutagenesis, 417*(1), 31–37.

Franzen, R., & Kronberg, L. (1994). Determination of chlorinated 5-methyl-5-hydroxyfuranones in drinking water, in chlorinated humic water, and in pulp bleaching liquor. *Environmental Science & Technology, 28*(12), 2222–2227.

Hemming, J., Holmbom, B., Reunanen, M., & Kronberg, L. (1986). Determination of the strong mutagen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone in chlorinated drinking and humic water. *Chemosphere, 15*(5), 549–556.

Hyttinen, J.M.T., Myohanen, S., & Jansson, K. (1996). Kinds of mutations induced by 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) in the HPRT gene of Chinese hamster ovary cells. *Carcinogenesis, 17*(5), 1179–1181.

International Agency for Research on Cancer. (1995). *Monographs on the evaluation of carcinogenic risks to humans, vol. 63*. Lyons, France: Author.

International Agency for Research on Cancer. (2004). *Monographs on the evaluation of carcinogenic risks to humans, vol. 84*. Lyons, France: Author.

Jansson, K., & Hyttinen, J.M.T. (1994). Induction of gene mutation in mammalian-cells by 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5h)-furanone (MX), a chlorine disinfection by-product in drinking water. *Mutation Research-Genetic Toxicology, 322*(2), 129–132.

Jansson, K., Maki-Paakkanen, J., Vaittinen, S.L., Vartiainen, T., Komulainen, H., & Tuomisto, J. (1993). Cytogenetic effects of 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5h)-furanone (MX) in rat peripheral lymphocytes in vitro and in vivo. *Mutation Research, 299*(1), 25–28.

King, L.C., Hester, S.D., Warren, S.H., & DeMarini, D.M. (2009). Induction of abasic sites by the drinking-water mutagen MX in *Salmonella* TA100. *Chemico-Biological Interactions, 180*(3), 340–343.

Krasner, S.W., Weinberg, H.S., Richardson, S.D., Pastor, S.J., Chinn, R., Sclimenti, M.J., Onstad G.D., & Thruston, A.D. (2006). Occurrence of a new generation of disinfection by-products. *Environmental Science & Technology, 40*(23), 7175–7185.

Kronberg, L., Christman, R.F., Singh, R., & Ball, L.M. (1991). Identification of oxidized and reduced forms of the strong bacterial mutagen (z)-2-chloro-3-(dichloromethyl)-4-oxobutenoic acid (mx) in extracts of chlorine-treated water. *Environmental Science & Technology, 25*(1), 99–104.

Kronberg, L., Holmbom, B., Reunanen, M., & Tikkanen, L. (1988). Identification and quantification of the Ames mutagenic compound 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5h)-furanone and of its geometric isomer (e)-2-chloro-3-(dichloromethyl)-4-oxobutenoic acid in chlorine-treated humic water and drinking-water extracts. *Environmental Science & Technology, 22*(9), 1097–1103.

Kronberg, L., & Vartiainen, T. (1988). Ames mutagenicity and concentration of the strong mutagen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5h)-furanone and of its geometric isomer e-2-chloro-3-(dichloromethyl)-4-oxo-butenoic acid in chlorine-treated tap waters. *Mutation Research, 206*(2), 177–182.

Kusamran, W.R., Wakabayashi, K., Oguri, A., Tepsuwan, A., Nagao, M., & Sugimura, T. (1994). Mutagenicities of Bangkok and Tokyo river waters. *Mutation Research Letters, 325*(2–3), 99–104.

Langvik, V.A., & Holmbom, B. (1994). Formation of mutagenic organic by-products and AOX by chlorination of fractions of humic water. *Water Research, 28*(3), 553–557.

Lindahl, T., & Andersson, A. (1972). Rate the chain breakage at apurinic sites in double-stranded deoxyribonucleic acid. *Biochemistry, 11*(19), 3618–3623.

Loyola-Sepulveda, R., Lopez-Leal, G., Munoz, J., Bravo-Linares, C., & Mudge, S.M. (2009). Trihalomethanes in the drinking water of Concepción and Talcahuano, Chile. *Water and Environment Journal, 23*(4), 286–292.

Maki-Paakkanen, J., & Hakulinen, P. (2008). Assessment of the genotoxicity of the rat carcinogen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) in rat liver epithelial cells in vitro. *Toxicology in Vitro, 22*(2), 535–540.

Maron, D.M., & Ames, B.N. (1983). Revised methods for the *Salmonella* mutagenicity test. *Mutation Research, 113*, 173–215.

McDonald, T.A., & Komulainen, H. (2005). Carcinogenicity of the chlorination disinfection by-product MX. *Journal of Environmental Science and Health Part C-Environmental Carcinogenesis & Ecotoxicology Reviews, 23*(2), 163–214.

Meier, J.R., Blazak, W.F., & Knohl, R. (1987). Mutagenic and clastogenic properties of 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone: A potent bacterial mutagen in drinking water. *Environmental Molecular Mutagenesis, 10*(4), 411–424.

Meier, J.R., Knohl, R.B., Coleman, W.E., Ringhand, H.P., Munch, J.W., Kaylor, W.H., Streicher, R.P., & Kopfler, F.C. (1987). Studies on the potent bacterial mutagen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone: aqueous stability, XAD recovery and analytical determination in drinking water and in chlorinated humic acid solutions. *Mutation Research, 189*(4), 363–373.

Melendez, I., Zuleta, M., Marín, I., Calle, J., & Salazar, D. (2001). Actividad mutagénica de aguas de consumo humano antes y después de clorar en la planta de Villa Hermosa, Medellín [Article in Spanish]. *Iatreia, 14*(3), 167–175.

National Primary Drinking Water Regulations: Stage 2 disinfectants and disinfection by-products rule (U.S. Environmental Protection Agency), 40 C.F.R. § 9 141 and 142, (2006).

Rezemini, A.L., Vaz, J.M., & Carvalho, L.R.E. (2008). Solid-phase microextraction for determination of 3-chloro-4-(dichloromethyl)-5-hydroxy-2[5H]-furanone in water. *Journal of the Brazilian Chemical Society, 19*(5), 922–928.

Richardson, S.D., Plewa, M.J., Wagner, E.D., Schoeny, R., & DeMarini, D.M. (2007). Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection by-products in drinking water: A review and roadmap for research. *Mutation Research-Reviews in Mutation Research, 636*(1–3), 178–242.

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References

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Richardson, S.D., Simmons, J.E., & Rice, G. (2002). Disinfection by-products: The next generation. *Environmental Science & Technology, 36*(9), 198A–205A.

Shi, Y., Cao, X.W., Tang, F., Du, H.R., Wang, Y.Z., Qiu, X.Q., Yu, H.P., & Lu, B. (2009). In vitro toxicity of surface water disinfected by different sequential treatments. *Water Research, 43*(1), 218–228.

Singh, N.P., McCoy, M.T., Tice, R.R., & Schneider, E.L. (1988). A simple technique for quantitation of low-levels of DNA damage in individual cells. *Experimental Cell Research, 175*(1), 184–191.

Smeds, A., Vartiainen, T., Maki-Paakkanen, J., & Kronberg, L. (1997). Concentrations of Ames mutagenic chlorohydroxyfuranones and related compounds in drinking waters. *Environmental Science & Technology, 31*(4), 1033–1039.

Stahl, R.G. (1991). The genetic toxicology of organic-compounds in natural-waters and wastewaters. *Ecotoxicology and Environmental Safety, 22*(1), 94–125.

Tao, X.G., Zhu, H.G., & Matanoski, G.M. (1999). Mutagenic drinking water and risk of male esophageal cancer: A population-based case-control study. *American Journal of Epidemiology, 150*(5), 443–452.

World Health Organization. (2004). *MX in drinking water*. Retrieved from http://www.who.int/water_sanitation_health/dwq/chemicals/mx.pdf

Wright, J.M., Schwartz, J., Vartiainen, T., Maki-Paakkanen, J., Altshul, L., Harrington, J.J., & Dockery D.W. (2002). 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) and mutagenic activity in Massachusetts drinking water. *Environmental Health Perspectives, 110*(2), 157–164.

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Mercury, Lead, and Cadmium in Umbilical Cord Blood

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Abstract **The study described in this article aimed to determine if measurable levels of mercury, lead, and cadmium are detected in the umbilical cord blood specimens collected in a community hospital in Rhode Island and if prenatal exposure correlates with prematurity or fetal growth indicators. Total mercury, lead, and cadmium concentrations were measured in 538 specimens of cord blood and correlated with demographic characteristics and pregnancy outcomes for each mother-infant pair. Lead concentrations determined in the cord blood of Rhode Island women (geometric mean 0.99 µg/dL) were similar to those reported in U.S. biomonitoring studies. The overall geometric mean for mercury concentration (0.52 µg/L) was slightly lower than in other comparable studies. Cadmium concentrations were generally below the limit of detection. A statistically significant correlation was detected between elevated mercury concentrations and racial and ethnic characteristics of the study participants. Non-Hispanic African-American mothers were 9.6 times more likely to have a mercury concentration ≥5.8 µg/L compared to women of other racial/ethnic backgrounds. No association was detected between elevated mercury levels and adverse birth outcomes.**

Introduction

Mercury, lead, and cadmium are found in the environment either as naturally occurring metals or as a result of anthropogenic activities. All have the ability to cross the maternal circulation into the placental and fetal circulation (National Research Council [NRC], 2006). The presence of these metals in cord blood has been extensively documented (Butler Walker et al., 2006; Korpela, Loueniva, Yrjänheikki, & Kauppila, 1986; Kuhnert, Kuhnert, & Erhard, 1981; Lauwerys, Buchet, Roels, & Hubermont, 1978; Ong et al., 1993; Soong, Tseng, Liu, & Lin, 1991; Truska et al., 1989), and has been frequently postulated as having harmful effects on child development (Andrews, Savitz, & Hertz-Picciotto, 1994; Bellinger, Leviton, Waternaux, Needleman, & Rabinowitz, 1987; Dietrich, 1991; Gao et al., 2008; Jedrychowski et al., 2006; Ramirez et al., 2003; Salpietro et al., 2002).

Mercury exists in several forms, each of which differs in bioavailability and toxicity. In contrast to elemental and inorganic mercury, methylmercury (MeHg) accumulates in erythrocytes at a wide range of exposure levels (Kershaw, Clarkson, & Dhahir, 1980). Ingested MeHg is almost completely absorbed and crosses the placenta and blood-brain barrier (Xue, Holzman, Rahbar, Trosko, & Fischer, 2007). Pregnant women who consume fish, especially large species such as shark, swordfish, and tuna, may expose the fetus to MeHg. International health disasters, including those in Minimata, Japan; Tagum, Philippines; and Iraq demonstrated that high levels of MeHg are neurotoxic, and cause seizures, microcephaly, mental retardation, and cerebral palsy, especially in children whose mothers were exposed in the prenatal period (Cox et al., 1989; Harada, 1968; Marsh et al., 1980). The susceptibility of the infant's developing brain to MeHg is due to the ability of lipophilic MeHg to cross the placenta and concentrate in the fetal central nervous system, where it can inhibit developmental functions such as neuronal cell division and migration (Choi, Lapham, Amin-Zaki, & Saleem, 1978).

While blood lead levels have decreased dramatically in the past decade, estimates show that 4.4% of children between the ages of one and five in the U.S. still have elevated blood lead levels (Centers for Disease Control and Prevention [CDC], 2005a). Lead crosses the placenta and the infant blood lead concentration reflects that of maternal blood (Goyer, 1990). Maternal exposure to lead during gestation contributes to the infant's lead burden at birth. In pregnancy, lead exposure has been associated with diminished neurological function, low birth weight, and premature birth (Andrews et al., 1994; Dietrich, 1991). Higher lead levels in cord blood are also correlated with deteriorating performance on tests of infant development up to two years of age (Bellinger et al., 1987).

TABLE **1**

Lead Concentrations (µg/dL) in Cord Blood by Race, Ethnicity, and Age of Mother

Demographic	*N*	%	Mean	*CI*[a]	*GM*[a]	*CI*
Total population	538	100.0	1.36	1.26–1.47	0.99	0.92–1.06
By race						
African-American	46	8.6	1.82	1.46–2.18	1.41	1.10–1.79
White	344	63.9	1.22	1.13–1.31	0.92	0.84–1.01
Other	123	22.9	1.37	1.20–1.54	1.00	0.85–1.17
Missing	25	4.6				
By ethnicity						
Hispanic	121	22.5	1.43	1.24–1.62	1.06	0.90–1.24
Non-Hispanic	383	71.2	1.32	1.22–1.41	0.99	0.91–1.07
Missing	34	6.3				
By age of mother						
<24	233	43.3	1.30	1.11–1.48	0.94	0.84–1.05
25–34	226	42.0	1.46	1.31–1.60	1.07	0.96–1.20
35–44	66	12.3	1.23	1.01–1.46	0.88	0.70–1.11
Missing	13	2				

[a]*CI* = 95% confidence interval; *GM* = geometric mean.

In the U.S., the dominant source of human exposure to cadmium is from cigarette smoking (CDC, 2005b). Cadmium can accumulate in the placenta and cause changes in placental morphology (Bush et al., 2000), which is subsequently linked to preterm labor, decreased birth weight (Salpietro et al., 2002), and decreased length at birth (Zhang et al., 2004).

Studies investigating the effects of heavy metals on pregnancy outcomes yield conflicting results. Two studies (Galicia-García, Rojas-López, Rojas, Olaiz, & Rios, 1997; Salpietro et al., 2002) found that birth weight was inversely correlated with the amount of cadmium in maternal and cord blood specimens. Zentner and co-authors (2006) also found an inversely proportional correlation of cord blood lead and newborn weight, but Greene and Ernhart (1991) found no such association. Lederman and co-authors (2008) found no association between levels of mercury in the cord blood and birth weight, head circumference, or gestational age. Our study sought to further investigate and clarify the relationship between the concentrations of lead, cadmium, and mercury in cord blood and the newborns' gestational age, birth weight, and head circumference.

Methods

Site and Subjects

Our study was conducted at Memorial Hospital in Pawtucket, Rhode Island, a teaching affiliate of The Warren Alpert Medical School of Brown University. This hospital participates in approximately 400 births each year, most of them to Medicaid recipients.

Institutional review boards at Memorial Hospital and at the Rhode Island Department of Health approved the study design prior to subject enrollment and data collection. The option to participate in the study was offered to all women admitted to the hospital in early labor. Women who were admitted in active labor and who were too uncomfortable at the time of admission to consent to the study were offered participation in the study after the delivery was complete.

Specimen and Data Collection

A sample of cord blood from a clamped umbilical cord was collected in an ethylenediaminetetraacetic acid (EDTA)–treated tube after the birth. Specimens were then sent for analysis to the State Health Laboratory in Providence, Rhode Island. Information on primary care providers for the mother and the baby was collected so that the medical care provider could be notified in the event of an elevated result. Head circumference, birth weight, and gestational age of the neonate were obtained through a chart review.

Laboratory Methods

Cord blood specimens were stored refrigerated at 0ºC–4ºC until testing. Total mercury, lead, and cadmium concentrations were measured in whole blood using a PerkinElmer ELAN series DRC Inductively Coupled Plasma Mass Spectrometer (ICP/MS) using a previously described analytical method (CDC, 2004). The instrument was calibrated daily for mercury, lead, and cadmium using matrix-matched calibration standards.

Calibration standards were from High Purity Standards. The quality control (QC) materials, covering the anticipated range of exposures, were from SPEX Certiprep. The internal quality control protocol involved running blood blanks and three QC samples (at a low, medium, and high concentration) at the beginning of each run. Each specimen was analyzed in duplicate. Repeat analyses were also performed for all specimens exceeding 10 µg/L for mercury and cadmium and 10 µg/dL for lead. Method detection limits were determined by analyzing seven replicates of base (blank) blood and were calculated to be 0.10 µg/L for cadmium, 0.20 µg/L for mercury, and 0.31 µg/dL for lead.

Statistical Analysis

In cases where the metal concentration in the cord blood sample was below the limit of detection, one-half the limit of detection was substituted into the dataset and used in further analyses. Gestational age was determined following the guidelines of the American College of Obstetrics and Gynecology (2009) and was characterized as preterm (<35 weeks), late preterm (35–37 weeks), and term (>37 weeks gestation). Head circumference was characterized as >90th percentile, 10th–90th percentile, and <10th percentile, according to the standards developed by the National Center for Health Statistics.

To describe characteristics of the study sample, the mean, median, geometric mean, and 95th percentiles were calculated for each of the metals for the total population, and for distinct age, racial, and ethnic groups. Preva-

lence of elevated mercury levels (at or above 5.8 µg/L) was then determined among the total population, and selected age, racial, and ethnic groups. Odds ratios were calculated to estimate the odds of having elevated mercury levels (≥5.8 µg/L) among the following pairs: non-white women vs. white women; African-American women vs. non-African-American women; Hispanic women vs. non-Hispanic women; non-Hispanic white women vs. other women; and non-Hispanic African-American women vs. other women. Differences in elevated mercury levels across racial/ethnic groups were determined to be statistically significant based on Chi-square or Fisher's exact tests. All statistical analyses were done using SAS version 9.1.

Results

Study Participants

Study participants (*N* = 538) were principally residents of Pawtucket, Rhode Island, and surrounding communities. Six hundred sixty-one births occurred at Memorial Hospital during the study period, and 538 specimens were collected over this time period, representing 81.4% of all births. The population was predominantly white (63.9%), and a small percentage (8.6%) was African-American. Almost one-quarter of the population (22.5%) was Hispanic. All women participating in the study were between the ages of 18 and 44.

Lead and Cadmium Concentrations

The geometric mean lead concentration in umbilical cord blood was 0.99 µg/dL (95% Confidence Interval [*CI*] 0.92–1.06). The measured concentrations ranged from below the limit of detection to 18.9 µg/dL. Only one of the 538 cord blood specimens had a concentration above 10 µg/dL, the public health level of concern, and five were between 5 and 10 µg/dL. The 95th percentile level for lead was 3.29 µg/dL. The cord blood lead results are described in Table 1.

The mean lead concentration in cord blood tracks very closely with the geometric mean calculated for females ages one and older (1.19 µg/dL) in the *Third National Report on Human Exposure to Environmental Chemicals* (CDC, 2005b). A statistically significant difference existed between the geometric mean cord blood concentrations for white women (1.22 µg/dL) compared to African-American women (1.82 µg/dL). Whether this difference has clinical significance is uncertain, but it is consistent with slightly higher lead exposures in African-Americans vs. whites (CDC, 2005b). No statistically significant associations were found between ethnic groups or age ranges and cord blood lead concentrations.

The geometric mean cadmium concentration in cord blood was not calculated. The proportion of results below the limits of detection (528 of 538) was too high to permit the calculation of valid geometric mean values. This result was also very similar to that obtained in the *Third National Report on Human Exposure to Environmental Chemicals* (CDC, 2005b).

TABLE **2**

Mercury Concentrations (µg/L) in Cord Blood by Race, Ethnicity, and Age of Mother

Demographic	*N*	%	Mean	*CI*[a]	*GM*[a]	*CI*
Total population	538	100.0	2.00	1.62–2.38	0.52	0.45–0.59
By race						
African-American	46	8.6	5.78	3.42–8.15	2.10	1.26–3.48
White	344	63.9	1.15	0.91–1.39	0.38	0.33–0.44
Other	123	22.9	3.03	1.94–4.11	0.77	0.56–1.06
Missing	25	4.6				
By ethnicity						
Hispanic	121	22.5	1.83	1.14–2.53	0.53	0.40–0.71
Non-Hispanic	383	71.2	2.10	1.63–2.57	0.52	0.44–0.62
Missing	34	6.3				
By age of mother						
<24	233	43.3	1.24	0.96–1.51	0.43	0.36–0.52
25–34	226	42.0	2.26	1.62–2.91	0.55	0.44–0.69
35–44	66	12.3	3.79	2.01–5.57	0.88	0.56–1.40
Missing	13	2.4				

[a]*CI* = 95% confidence interval; *GM* = geometric mean.

Mercury Concentrations and Association With Age and Race/Ethnicity

The geometric mean cord blood mercury concentration was 0.52 µg/L (95% *CI* 0.45–0.59). The arithmetic mean was 2.0 µg/L. Detected concentrations ranged from below the limit of detection to 39.9 µg/L. Nearly half (42.8%) of the women had cord blood mercury levels below the limit of detection. Almost 93% of women had mercury concentrations below 5.8 µg/L. Nineteen (3.5%) had levels over 10 µg/L. The summary of mercury results is presented in Table 2.

Analysis of the correlation of mercury levels and race and age groups indicated significant differences in the prevalence of elevated (≥5.8 µg/L) concentrations of mercury. The geometric mean calculated for African-American women was 2.10 µg/L, four times higher than the geometric mean for the study population. African-American women were 8.5 times more likely to have elevated mercury concentrations than non-African-American women ($p < .0001$). Further, non-Hispanic African-American mothers had a 9.6 higher chance of having a mercury concentration ≥5.8 µg/L compared to women of other racial/ethnic backgrounds ($p < .0001$). No significant differences existed between Hispanic and non-Hispanic women. The summary of these results is presented in Table 3. A statistically significant difference existed between prevalence of elevated mercury levels in women 25–34 years of age compared to those younger than 24 years (9.3% vs. 3.0%, $p < .0049$). The difference is even more pronounced when age groups 35–44 and <24 are compared (13.6% vs. 3.0%, $p < .0007$).

TABLE **3**

Odds Ratios of Mercury Levels ≥5.8 µg/L Among Different Racial/Ethnic Groups

Comparison Groups	Odds Ratio	95% Confidence Interval	*p*-Value
Non-white vs. white	6.5	3.0–14.1	<.0001
African-American vs. non-African-American	8.5	4.0–18.1	<.0001
Hispanic vs. non-Hispanic	0.9	0.4–2.0	.8257
Non-Hispanic white vs. other	0.2	0.1–0.4	<.0001
Non-Hispanic African-American vs. other	9.6	4.3–21.3	<.0001

Cord Blood Mercury Concentrations and Fetal Development Indicators

Gestational age, birth weight, and head circumference were collected, and their correlation with mercury levels in cord blood was evaluated. A statistically significant association was not detected in the cord blood between elevated mercury concentrations and the neonate's weight, head circumference, or gestational age ranges.

Discussion

Mercury, lead, and cadmium concentrations can be reliably measured in umbilical cord blood and provide the most direct estimate of fetal exposure to these metals. Consistent with national biomonitoring studies (CDC, 2005b), no significant cadmium concentrations were detected in umbilical cord bloods in our study. This finding is in contrast to the New York City biomonitoring study (McKelvey et al., 2007), which detected a geometric mean of 0.79 µg/L of cadmium in adult females.

The geometric mean concentration of lead in our study (0.99 µg/dL) correlates closely with the values in recent biomonitoring studies of adult females: 1.19 µg/dL (CDC, 2005b) and 1.54 µg/dL (McKelvey et al., 2007).

Total blood mercury concentrations are a combination of inorganic, elemental, and MeHg, with the last comprising approximately 90% of the total blood mercury in populations not occupationally exposed (Mahaffey, Clickner, & Bodurow, 2004). MeHg exposure occurs mainly through the consumption of mercury-contaminated fish. Fetal exposure to MeHg has been shown to lead to neuropsychological deficits (NRC, 2000), and the risk of adverse health effects increases as exposure to MeHg rises. Risk assessment studies standardized a blood mercury threshold below which no adverse neurological effects are expected (U.S. Environmental Protection Agency, 2001) and suggested 5.8 µg/L concentration of MeHg in cord blood as a level of public health concern.

Our study found a geometric mean cord blood mercury concentration of 0.52 µg/L, which is comparable to the cord blood levels measured in a study conducted in Poland (mean of 0.88 µg/L) (Jedrychowski et al., 2006), a country where seafood consumption is relatively low. The cord blood geometric mean found in our study, however, is lower than those found in a World Trade Center (WTC) cohort (4.4 µg/L) (Lederman et al., 2008) and lower than the geometric mean blood mercury concentration detected in females aged 16–49 (0.83 µg/L) reported on a national scale (CDC, 2005b).

A comparison of the ethnic and racial compositions of these cohorts offers a plausible explanation for the observed differences between our study results and other comparable biomonitoring studies. The WTC population has a large percentage of Asians, both U.S. and foreign born (Lederman et al., 2008). This broad ethnic group, which had the highest mercury concentrations of all ethnic groups in the New York City study (McKelvey et al., 2007), was almost completely absent in our study. In addition, almost a quarter of all women in our study were Hispanic, a group consistently shown to have lower blood mercury levels than other ethnic groups (CDC, 2005b). While we did not explicitly gather income information, the majority of women delivering babies at Memorial Hospital are Medicaid recipients. Lower family income correlated with lower blood mercury concentrations in the New York City study (McKelvey et al., 2007).

The results obtained in our study indicate consistently elevated mercury levels in African-American women. African-American women constituted only 8.6% of the study population, but accounted for 36.8% of the mercury concentrations over 5.8 µg/L. The geometric mean mercury concentration for African-American women was 2.10 µg/L, four times the value for the entire study population and 5.5 times the value for white women. An African-American woman in our study was 8.5 times more likely to have a mercury concentration ≥5.8 µg/L than a non-African-American woman. This finding was consistent with the National Exposure Report, which reported a higher mean blood concentration of mercury in African-American women of childbearing age than in white women of childbearing age; however, the magnitude of the race effect is higher in our study. In contrast, the New York City Health and Nutrition Examination Survey and WTC cohort studies indicated mercury levels in African-American adults that were slightly lower than those in white adults (Lederman et al., 2008; McKelvey et al., 2007).

We have also observed the effect of age on blood mercury concentrations. Older women had higher mean concentrations of mercury than did younger women. This trend was also observed in the New York City study.

We were not able to find a statistically significant correlation between elevated mercury levels (at or higher than 5.8 µg/L) and any of the infant biometrics measures (weight or head circumference) or gestational age. This is not surprising in view of a relatively low prevalence of elevated cord blood mercury measurements. A WTC cohort study (Lederman et al., 2008) showing a higher prevalence of elevated results also did not find a statistically significant relationship between these same variables.

Limitations

Our study has potential limitations. The study population was from a single geographic location and from one hospital, which limits the ability to generalize the results to the population at large, on the state or national level. Although most women presented with the option to participate in the study did so, no available data exist regarding the small number of women who declined participation. We have no reason to believe

that this group would be different in terms of risk of exposure to heavy metals compared to the study group. In addition, the majority of women presenting in preterm labor (<34 weeks) are sent to Women and Infants Hospital, a tertiary care birthing hospital in the state. It is unknown if the women who were diverted to the other hospital might differ from those delivering at the study site in terms of heavy metal exposure.

Smoking status of the study participants was not collected. Tobacco smoke is a major source of exposure for heavy metals, especially cadmium; however, cadmium was not detected in most of the cord blood samples.

Race and ethnicity were self-reported variables, and these data points were missing in a small percentage of our samples. Therefore, associations regarding race and higher levels of heavy metals in cord blood need to be made cautiously. Lastly, biometry was grouped according to gestational age, and essentially was characterized as early preterm (<35 weeks), preterm (35–37 weeks), and term (>37 weeks). Data analysis might be limited by the fact that there could be significant variation in biometry values within each of these groups.

Conclusion

Our study indicates that relatively low geometric mean concentrations of mercury were detected in cord blood of women giving birth at a community hospital in Rhode Island. While prenatal exposure to mercury on average was low in this population, children of African-American women were shown to have an increased chance of being exposed to MeHg prenatally. While specific public health implications of mercury found in cord blood are currently unknown, some recent studies suggest developmental delays in children are possible at cord blood levels of mercury as low as 0.8 µg/L (Jedrychowski et al., 2006). Further studies regarding the effect of measurable concentrations of heavy metals in cord blood are needed. Measurements of environmental toxicants in cord blood give the most direct estimate of prenatal exposure and can more accurately guide local public health education efforts aimed at reducing prenatal exposure to toxic substances.

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References

American College of Obstetrics and Gynecology. (2009). Ultrasonography in pregnancy (Practice Bulletin No. 101). *Obstetrics & Gynecology, 113,* 451–461.

Andrews, K.W., Savitz, D.A., & Hertz-Picciotto, I. (1994). Prenatal lead exposure in relation to gestational age and birth weight: A review of epidemiological studies. *American Journal of Industrial Medicine, 26*(1), 13–32.

Bellinger, D., Leviton, A., Waternaux, C., Needleman, H., & Rabinowitz, M. (1987). Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *The New England Journal of Medicine, 316*(17), 1037–1043.

Bush, P.G., Mayhew, T.M., Abramovich, D.R., Aggett, P.J., Burke, M.D., & Page, K.R. (2000). A quantitative study on the effects of maternal smoking on placental morphology and cadmium concentration. *Placenta, 21*(2–3), 247–256.

Butler Walker, J., Houseman, J., Seddon, L., McMullen, E., Tofflemire, K., Mills, C., Corriveau, A., Weber, J.P., LeBlanc, A., Walker, M., Donaldson, S.G., & Van Oostdam, J. (2006). Maternal and umbilical cord blood levels of mercury, lead, cadmium, and essential trace elements in Arctic Canada. *Environmental Research, 100*(3), 295–318.

Centers for Disease Control and Prevention. (2004). *Blood cadmium, lead, and mercury using ICPMS*. Retrieved from http://www.cdc.gov/NCHS/data/nhanes/nhanes_09_10/PbCd_F_met.pdf

Centers for Disease Control and Prevention. (2005a). Blood lead levels, United States, 1999–2002. *Morbidity and Mortality Weekly Report, 54*(20), 513–516.

Centers for Disease Control and Prevention. (2005b). *The third national report on human exposure to environmental chemicals* (NCEH Publication No. 05-0570). Retrieved from http://www.cphfoundation.org/documents/thirdreport_001.pdf

Choi, B.H., Lapham, L.W., Amin-Zaki, L., & Saleem, T. (1978). Abnormal neuronal migration, deranged cerebral cortical organization and diffuse white matter astrocytosis of human fetal brain: A major effect of methylmercury poisoning in utero. *Journal of Neuropathology and Experimental Neurology, 87*(6), 719–733.

Cox, C., Clarkson, T.W., Marsh, D.O., Amin-Zaki, L., Tikriti, S., & Myers, G.J. (1989). Dose-response analysis of infants prenatally exposed to methylmercury: An application of a single compartment model to single strand hair analysis. *Environmental Research, 49*(2), 318–332.

Dietrich, K.N. (1991). Human fetal lead exposure: Intrauterine growth, maturation, and postnatal neurobehavioral development. *Fundamental and Applied Toxicology, 16*(1), 17–19.

Galicia-García, V., Rojas-López, M., Rojas, R., Olaiz, G., & Ríos, C. (1997). Cadmium levels in maternal, cord and newborn blood in Mexico City. *Toxicology Letters, 91*(1), 57–61.

Gao, Y., Yan, C.H., Tian, Y., Wang, Y., Xie, H.F., Zhou, X., Yu, X.D., Yu, X.G., Tong, S., Zhou, Q.X., & Shen, X.M. (2007). Prenatal exposure to mercury and neurobehavioral development of neonates in Zhoushan City, China. *Environmental Research, 105*(3), 390–399.

Goyer, R.A. (1990). Transplacental transport of lead. *Environmental Health Perspectives, 89*, 101–108.

References

Greene, T., & Ernhart, C.B. (1991). Prenatal and preschool age lead exposure: Relationship with size. *Neurotoxicology and Teratology, 13*(4), 417–427.

Harada, Y. (1968). Congenital (or fetal) Minamata disease (pp. 93–118). In Study Group of Minamata Disease (Eds.), *Minamata disease*. Kurokami Kumamoto, Japan: Kumamoto University.

Jedrychowski, W., Jankowski, J., Flak, E., Skarupa, A., Mroz, E., Sochacka-Tatara, E., Lisowska-Miszczyk, I., Szpanowska-Wohn, A., Rauh, V., Skolicki, Z., Kaim, I., & Perera, F. (2006). Effects of prenatal exposure to mercury on cognitive and psychomotor function in one-year-old infants: Epidemiologic cohort study in Poland. *Annals of Epidemiology, 16*(6), 439–447.

Kershaw, T.G., Clarkson, T.W., & Dhahir, P.H. (1980). The relationship between blood levels and dose of methylmercury in man. *Archives of Environmental Health, 35*(1), 28–36.

Korpela, H., Loueniva, R., Yrjänheikki, E., & Kauppila, A. (1986). Lead and cadmium concentrations in maternal and umbilical cord blood, amniotic fluid, placenta and amniotic membranes. *American Journal of Obstetrics and Gynecology, 155*(5), 1086–1089.

Kuhnert, P.M., Kuhnert, B.R., & Erhard, P. (1981). Comparison of mercury levels in maternal blood, fetal cord blood, and placental tissues. *American Journal of Obstetrics and Gynecology, 139*(2), 209–213.

Lauwerys, R., Buchet, J.P., Roels, H., & Hubermont, G. (1978). Placental transfer of lead, mercury, cadmium, and carbon monoxide in women. I. Comparison of the frequency distributions of the biological indices in maternal and umbilical cord blood. *Environmental Research, 15*(2), 278–289.

Lederman, S., Jones, R., Caldwell, K., Rauh, V., Sheets, S., Tang, D., Viswanathan, S., Becker, M., Stein, J.L., Wang, R.Y., & Perera, F.P. (2008). Relation between cord blood mercury levels and early child development in a World Trade Center cohort. *Environmental Health Perspectives, 116*(8), 1085–1091.

Mahaffey, K.R., Clickner, R.P., & Bodurow, C.C. (2004). Blood organic mercury and dietary mercury intake: National Health and Nutrition Examination Survey, 1999 and 2000. *Environmental Health Perspectives, 112*(5), 562–570.

Marsh, D.O., Myers, G.J., Clarkson, T.W., Amin-Zaki, L., Tikriti, S., & Majeed, M. (1980). Fetal methylmercury poisoning: Clinical and toxicological data on 29 cases. *Annals of Neurology, 7*(4), 348–353.

McKelvey, W., Gwynn, C., Jeffery, N., Kass, D., Thorpe, L., Garg, R., Palmer, C.D., & Parsons, P.J. (2007). A biomonitoring study of lead, cadmium and mercury in the blood of New York City adults. *Environmental Health Perspectives, 115*(10), 1435–1441.

National Research Council. (2000). *Toxicological effects of methylmercury*. Washington, DC: National Academies Press.

National Research Council. (2006). *Human biomonitoring for environmental chemicals*. Washington, DC: National Academies Press.

Ong, C.N., Chia, S.E., Foo, S.C., Ong, H.Y., Tsakok, M., & Liouw, P. (1993). Concentrations of heavy metals in maternal and umbilical cord blood. *Biometals, 6*(1), 61–66.

Ramirez, G.B., Pagulayan, O., Akagi, H., Francisco Rivera, A., Lee, L.V., Berroya, A., Vince Cruz, M.C., & Casintahan, D. (2003). Tagum study II: Follow-up study at two years of age after prenatal exposure to mercury. *Pediatrics, 111*(3), e289–295.

Salpietro, C.D., Gangemi, S., Minciullo, P.L., Briuglia, S., Merlino, M.V., Stelitano, A., Cristani, M., Trombetta, D., & Saija, A. (2002). Cadmium concentration in maternal and cord blood and infant birth weight: A study on healthy nonsmoking women. *Journal of Perinatal Medicine, 30*(5), 395–399.

Soong, Y.K., Tseng, R., Liu, C., & Lin, P.W. (1991). Lead, cadmium, arsenic, and mercury levels in maternal and fetal cord blood. *Journal of the Formosan Medical Association, 90*(1), 59–65.

Truska, P., Rosival, L., Balázová, G., Hinst, J., Rippel, A., Palusová, O., & Grunt, J. (1989). Blood and placental concentrations of cadmium, lead, and mercury in mothers and their newborns. *Journal of Hygiene, Epidemiology, Microbiology, and Immunology,* 33(2), 141–147.

U.S. Environmental Protection Agency. (2001). Methylmercury oral reference dose assessment (CASRN22967-92-6). Retrieved from www.epa.gov/iris/subst/0073.htm

Xue, F., Holzman, C., Rahbar, M.H., Trosko, K., & Fischer, L. (2007). Maternal fish consumption, mercury levels, and risk of preterm delivery. *Environmental Health Perspectives, 115*(1), 42–47.

Zentner, L.E., Rondó, P.H., & Mastroeni, S.S. (2006). Lead contamination and anthropometry of the newborn baby. *Journal of Tropical Pediatrics, 52*(5), 369–371.

Zhang, Y.L., Zhao, Y.C., Wang, J.X., Zhu, H.D., Liu, Q.F., Fan, Y.G., Wang, N.F., Zhao, J.H., Liu, H.S., Ou-Yang, L., Liu, A.P., & Fan, T.Q. (2004). Effect of environmental exposure to cadmium on pregnancy outcome and fetal growth: A study on healthy pregnant women in China. *Journal of Environmental Science and Health, A39(9),* 2507–2515.

Community-Associated Methicillin-Resistant *Staphylococcus aureus* in College Residential Halls

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Abstract **Methicillin-resistant *Staphylococcus aureus* (MRSA) was once a predominantly hospital-acquired organism but community-associated MRSA (CA-MRSA) has become a concern in athletics, prisons, and other nonclinical closed populations. As such, college residential hall occupants and workers may be at elevated risk of spreading or contracting MRSA.**

Environmental samples were obtained to identify the occurrence of MRSA on surfaces in bathrooms of 15 university residential halls. Sterile swabs and BBL CHROMagar plates were used to sample seven categories of potentially contaminated surfaces in each location. Frequencies and descriptive statistics were prepared. All sites had at least one positive sample for MRSA, and shower floors displayed the greatest prevalence (50%). These results indicate areas for heightened sanitation, and illustrate CA-MRSA potential from such surfaces. The need for hygiene education of affected persons about skin and soft tissue infections like MRSA, and intervention opportunities for public health professionals, are discussed.

Introduction

Each year approximately 12 million Americans visit a physician to be examined for *Staphylococcus aureus* or methicillin-resistant *S. aureus* (MRSA) infections (Centers for Disease Control and Prevention [CDC], 2008). MRSA infections total approximately 90,000 deaths and $6 billion in health care costs per year, which makes them the sixth leading cause of death nationally in the U.S. (Klein, Smith, & Laxminarayan, 2007).

MRSA is an evolving pathogen that has morphed into several potentially infectious strains (Shukla, 2006). The Centers for Disease Control and Prevention (CDC) define community-associated MRSA (CA-MRSA) as a strain of MRSA acquired by those who have not been hospitalized or undergone a medical procedure within the past year. CA-MRSA has unique microbiologic and genetic properties relative to health care–associated MRSA (HA-MRSA), which allow the bacteria to spread more easily and therefore cause more skin infections (CDC, 2005). Seventy percent of all MRSA infections are caused by five major strains of MRSA. The most predominant strain in the U.S. is USA 300 (Sampathkumar, 2007). Ninety-seven percent of infections reported from 11 different hospitals were of the USA 300 clone (Herman, Kee, Moores, & Ross, 2008). MRSA is able to survive on a range of surfaces for extended periods of time and can infect hosts as a result of only limited exposure (Salgado, Farr, & Calfee, 2003; Shukla, 2006).

MRSA has been found to be capable of penetrating intact skin, allowing the bacteria to infect deeper layers of tissue (Shukla, 2006). MRSA colonization can persist for months and sometimes years, with a half-life of 40 months (Salgado et al., 2003). Previous studies have indicated that MRSA is commonly transferred through skin-to-skin contact with an infected person, but little is known about a person's likelihood of becoming infected through contact with MRSA-contaminated surfaces (Cohen, 2005). Many risk factors for developing MRSA exist within athletics, including the sharing of clothing, sports equipment, towels, balms, lubricants, razors, and soaps; improper care of skin lesions; and direct skin-to-skin contact with MRSA lesions (Beam & Buckley, 2006).

Of the total cases of *S. aureus* diagnosed annually, the proportion of those infected with MRSA has risen from 29% in 2001–2002 to 64% in 2003–2004 (McKenna, 2008). A CDC analysis found that 8% to 20% of all MRSA infections reported in hospitals were of the community strain (McKenna, 2008). Thus CA-MRSA is not only of interest to health department sanitarians and hospital infection control personnel, but to housekeeping and environmental services personnel as well.

MRSA cases among athletes are most common in sports involving high-physical contact, such as wrestling, football, and rugby (Kirkland & Adams, 2008). Cases have also been reported, however, among athletes participating in soccer, basketball,

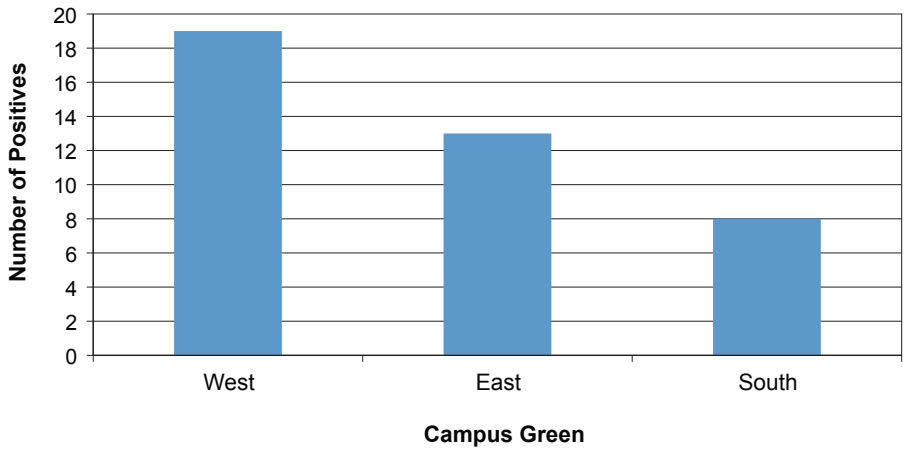
TABLE **1**

Rank-Ordered Occurrence of Methicillin-Resistant *Staphylococcus aureus* by Surface Category

Surface Category	Site	Positive Results	
	N	*n*	%
Shower floors	30	15	50
Shelf below mirror	30	11	37
Toilet seats	30	9	30
Sink faucet handles	30	3	10
Shower faucet handles	30	2	7
Stall door handles	30	0	0
Controls (high on wall, just below ceiling)	30	0	0

FIGURE **1**

Frequency of Positive Methicillin-Resistant *Staphylococcus aureus* Samples by Campus Green



field hockey, volleyball, rowing, martial arts, fencing, and baseball (CDC, 2005). Few if any studies have investigated the presence of MRSA in college living environments, especially those housing collegiate athletes. Studies that relate to college residential housing include those done in military and jail environments, entailing captive populations. A recent jail study concentrated on the use of antibiotics and the effect it had on an inmate's ability to resist contracting the MRSA bacterium (David, Mennella, Mansour, Boyle-Vavra, & Daum, 2008). Although many genetic factors are related to the antibiotics, one of the main explanations for their findings of a high prevalence in the facilities was said to be crowding and suboptimal hygienic practices along with a rapid turnover of detainees (David et al., 2008).

During military service troops are commonly exposed to numerous infections and diseases; among the most common is MRSA (Roberts & Kazragis, 2009). It has been reported that the close living quarters, unsanitary living conditions due to deployment, and the use of communal bathrooms have a great impact on the contraction of the bacteria. From previous studies it has been seen that close living quarters and shared hygiene utensils are some of the main sources of MRSA contraction: both have characteristics in common with residential halls and college students. To date, few if any studies have been published examining the prevalence of MRSA in college residential halls (i.e., dormitories). The purpose of our study was to survey bathroom shower floors, toilet seats, shower handles, stall door handles, shelves, and sink faucet handles to better characterize such environments and their potential for MRSA spread.

Methods

The institutional review committee exempted formal review and approval of this study as no human testing took place.

Fifteen residential halls at a large college campus were sampled for the presence of MRSA. Sampling occurred early in the morning before custodial cleaning took place but after the majority of use for that time period. Of these facilities, five were considered to service a larger-than-usual student athlete population (i.e., West Green with football, soccer, swimming, wrestling, and volleyball athletes). The remaining 10 halls on East and South Green housed only the general student population, with a minimal number of athletes. In each of the 15 halls two bathrooms (one male, one female) with seven similar categorical surfaces were sampled using sterile swabs. Composite samples (i.e., multiple swab contact on similar category) were collected from 1) the surface of stall door handles, 2) toilet seats, 3) shower floors, 4) shower faucet handles, 5) sink faucet handles, 6) shelves below bathroom mirrors, and 7) a surface high on the bathroom walls. This bathroom wall sample was taken from the tile directly below the ceiling to be used as a control. In some areas door handles were not as abundant as others so toilet handles were sampled in their absence.

The MRSA analytical method employed in this study is identical to that used previously (Montgomery, Ryan, Krause, & Starkey, 2010; Stanforth, Krause, Starkey, & Ryan, 2010). The reader is referred to those studies for full details of sampling techniques and colony identification. The same laboratory was used to culture and grow the samples in all studies.

Briefly, laboratory surfaces were disinfected before and after so as to prevent personal or cross contamination. The field-

swab samples were streaked onto sterile BBL CHROMagar MRSA plates within 24 hours of collection and prior to the manufacturer's expiration date of the plates. Plates were incubated at 35°C for 24–48 hours with minimal exposure to light. BBL CHROMagar MRSA is a selective and differential medium that uses cefoxitin in order to identify MRSA. Mauve-colored colonies are indicative of positive MRSA samples due to the hydrolysis of the chromogenic substrate (Becton, Dickenson, & Company, 2008). After incubation for 24 hours the plates were checked for mauve colonies, and those lacking any were incubated for an additional 24 ± 4 hours. Plates not demonstrating mauve colonies by 48 hours were reported as negative for MRSA. The agar plates have a reported 96%± accuracy rate for MRSA when mauve colonies are detected within the first 24 hours of grow out, although this specificity drops to 93.5%–94.9% if counted after 24 hours (but before 48 hours) of incubation (Flayhart et al., 2005). The accuracy of the BBL CHROMagar plates used here has recently been called into question (Roberts, Meschke, Soge, & Reynolds, 2010). Their use here was as in previous studies in order to maintain cross-study consistency until such time as any accuracy questions are definitively resolved.

TABLE **2**

Prevalence of Methicillin-Resistant *Staphylococcus aureus* in College Residential Hall Bathrooms

% Positive for Each Gender[a]		% Positive Total (Male + Female)
Male	Female	
14.3	14.3	14.3
28.6	28.6	28.6
28.6	14.3	21.4
28.6	42.9	35.7
28.6	42.9	35.7
0	42.9	21.4
28.6	0	14.3
28.6	14.3	21.4
28.6	14.3	21.4
14.3	14.3	14.3
14.3	14.3	14.3
14.3	0	7.1
14.3	14.3	14.3
14.3	0	7.1
0	28.6	14.3
Avg. = 19%	Avg. = 19%	–
n = 7	*n* = 7	*n* = 14

[a]Percentage positive by gender (*n* = 7) by dorm and by total positive samples (*n* = 14).

Results

CA-MRSA was detected on bathroom surfaces in all 15 (100%) of the sites tested with at least one positive result for MRSA at each location. The shower floors displayed the greatest occurrence of MRSA (50%) while the stall door handles and controls (i.e., high wall surfaces) were found to have none (Table 1). Of the 70 available sites sampled on West Green (5 halls x 7 categorical sample types x 2 genders of bathrooms), 19 (27%) were positive for CA-MRSA; on South Green only 8 (11%) of the 70 and on East Green only 13 (19%) of the 70 samples produced positive results. Pooling all positives according to sampling category indicated that shower floors were the most likely location for exposure to MRSA, followed by the bathroom toiletry shelf (Figure 1).

The prevalence of MRSA by residential hall did not vary greatly, ranging from a low of 7% to a high of 36% (Table 2). At the residential halls with the least frequent MRSA detection ("L & N," Table 3), only the shower floors and sink faucet handles were positive. The range of positive surfaces within the dormitories was 7.1% to 35.7% of all surfaces tested. Data indicate that the following three surfaces have the highest percentage of MRSA detections: shower floors, shelves below bathroom mirrors (alternately directly above sinks or toiletry shelves), and toilet seats. It was found that gender had no significant difference, with both females and males averaging 19% of their surfaces positive for MRSA (Table 2). As can be seen in Figure 2 a higher count of MRSA positives in the athletic residential halls (A–E) occurred than in the nonathletic halls (F–O), but these differences were not statistically significant.

Discussion

Based on our data, West Green, which is highly populated with student athletes, carried a slightly higher but insignificant number of MRSA positives as compared to East and South Greens (housing nonathlete students). In comparing specific sampling locations, great variability is not evident. Two residential halls had five out of 14 surfaces test positive for the presence of MRSA (36%), while two others had only one of 14 (7%) surfaces test positive (Table 2). This demonstrates considerable variability in the presence of MRSA among residential halls. Although MRSA was present in greater numbers in the residence halls housing athletes, this elevated occurrence was not significant. Since cleaning and decontamination procedures at the residential halls were not examined as part of this project, it is not clear what precisely explains this wide range of MRSA prevalence on campus. Plausible explanations include sampling variability, hygiene practices within the residential halls, time between sampling and last cleaning at the residential bathrooms, as well as actual MRSA prevalence differences.

A high prevalence of MRSA on the shower floors (50%) was observed, strongly suggesting that residents are exposed to an elevated risk of surface-contact MRSA if they do not use proper shower attire such as shower sandals. Possible explanations for this high percentage of MRSA found on the shower

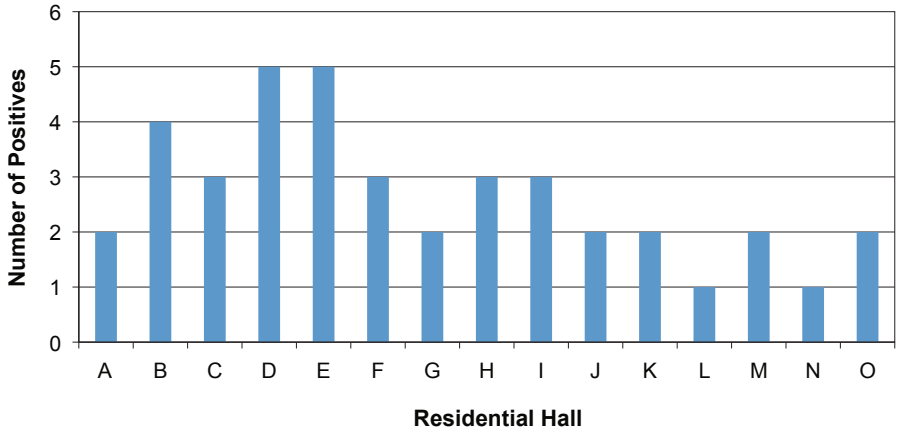
TABLE **3**

Surfaces Positive (x) by Category by Residential Hall by Gender

Residential Hall	Stall Door Handles		Toilet Seats		Shower Floors		Shower Faucet Handles		Sink Faucet Handles		Shelf Below Mirror		Control (High on Wall)	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
A					x	x								
B					x	x					x	x		
C			x	x	x									
D			x	x		x					x	x		
E				x	x	x	x					x		
F			x		x						x			
G					x						x			
H			x		x	x								
I						x			x		x			
J									x			x		
K				x	x									
L					x									
M											x	x		
N							x							
O				x				x						

FIGURE **2**

Frequency of Positive Methicillin-Resistant *Staphylococcus aureus* Samples by Residential Hall Tested



floors include the fact that samples were taken around the drains, which would hold all bacteria that would come off of an individual. Since it is highly recommended by the CDC to shower after physical activity, this is one possible explanation for the findings. Results from this location bolster common advice that shower sandals should always be worn to ensure that the MRSA bacterium is not contracted. All restrooms are on a weekly cleaning schedule with the custodial department. Restrooms are not cleaned daily, however, but every other day during the week and once on the weekends. The fact that these bathrooms were visited by 20 to 60 residents daily suggests that maintaining cleanliness is not just something that the custodial services must provide but is also a responsibility for the occupants of the building. To illustrate this point it was observed that disposal cans are provided in all bathrooms but were not always used.

The toiletry shelves below the bathroom mirrors also displayed a high prevalence of MRSA (37%) compared to other surfaces (Table 1). As this is a commonly used area of all residential bathrooms for personal items such as razors, tweezers, clippers, etc., it is important to use a proper disinfectant to eliminate the risk of contracting the MRSA bacterium. Residents should also be advised to regularly clean their toiletries and other items that they may place on this shelf while performing daily hygiene activities such as brushing their teeth or washing their face.

CDC recommendations to prevent MRSA infections include proper personal hygiene, washing hands often, showering immediately following exercise, and washing clothing after each use. It is also important to not share any personal items and to take proper care of skin, including wearing protective clothing and covering all abrasions and lacerations (CDC, 2003, 2005). The awareness level of facility users relative to the CDC guidelines was not determined in this study, although a lack of hygienic warnings was evident in all areas surveyed. Future stud-

ies might explore residential knowledge of MRSA risks and prevention safeguards and residential hall staff attitudes regarding the need for proper hygiene. The opportunity exists for the study of sanitarian or health educator interventions directed at the general MRSA-affected population in residential facilities, especially those highly populated by athletes and—by extension—campus recreation centers.

In order to prevent MRSA from spreading via personal toiletries, shower sandals, use of athletic equipment, and other personal items in a residential hall, it is imperative to follow proper cleaning regimens. Residential halls should be kept clean and cleaning procedures should be reviewed with custodial staff and residents to ensure CDC guidelines are being met. Surfaces that are most commonly touched should receive more frequent cleaning. Detergents and disinfectants registered by the U.S. Environmental Protection Agency as effective against MRSA should be used to clean surfaces. It is important to follow all instruction labels of all cleaners and disinfectants, paying particular attention to the amount of contact time each product must have on surfaces in order to be effective. The rigor with which such practices are followed would make for a timely and significant follow-up study to the findings reported here. Finally, the bathrooms studied here saw a high volume of users between cleanings and so it is perhaps worth considering whether user-education relative to MRSA contact avoidance might be considered to avoid infection with this as well as other direct contact agents.

Conclusion

Our findings support the premise that college residential hall occupants may be frequently exposed to several MRSA contact points in the bathrooms. All of the 15 residential halls sampled had at least one positive result for MRSA; at two residential halls, 36% of the locations tested were positive for MRSA. Another residential hall was positive at 29% of the locations, and four were positive at three of the 14 locations tested (21%), when male and female bathroom data were pooled. Of the locations that residents were most likely to have bare skin contact with—shower floors, toiletry shelves below mirrors, and toilet seats—an elevated number were positive for MRSA in the majority of the samplings.

Individuals living in the residential halls interact with each other on a daily basis within their living environment. Further research could look more specifically at residential halls populations to determine where athletes may live and consider MRSA locations relative to athlete use. This information could further aid in the prevention and reduction of outbreaks, and could be of use to campus sanitarians, associated public health officials, and campus resident life departments for both managing CA-MRSA cases and controlling the chain of infection for similar agents within residential halls.

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References

Beam, J.W., & Buckley, B. (2006). Community-acquired methicillin-resistant *Staphylococcus aureus*: Prevalence and risk factors. *Journal of Athletic Training, 41*(3), 337–340.

Becton, Dickinson, & Company. (2008). *The product center for BBL CHROMagar MRSA page*. Retrieved from http://www.bd.com/ds/productCenter/215084.asp

Centers for Disease Control and Prevention. (2003). Methicillin-resistant *Staphylococcus aureus* infections among competitive sports participants in competitive sports participants—Colorado, Indiana, Pennsylvania, and Los Angeles County, 2000–2003. *Morbidity and Mortality Weekly Report, 52*(33), 793–795.

Centers for Disease Control and Prevention. (2005). *Community-associated MRSA information for clinicians*. Retrieved from www.cdc.gov/ncidod/dhqp/ar_mrsa_ca_clinicians.html

Centers for Disease Control and Prevention. (2008). *National MRSA education initiative: Preventing MRSA skin infections*. Retrieved from www.cdc.gov/mrsa/mrsa_initiative/skin_infection/index.html

Cohen, P.R. (2005). Cutaneous community-acquired methicillin-resistant *Staphylococcus aureus* infection in participants of athletic activities. *Southern Medical Journal, 98*(6), 596–602.

David, M.Z., Mennella, C., Mansour, M., Boyle-Vavra, S., & Daum, R.S. (2008). Predominance of methicillin-resistant *Staphylococcus aureus* among pathogens causing skin and soft tissue infections in a large urban jail: Risk factors and recurrence rates. *Journal of Clinical Microbiology, 46*(10), 3222–3227.

Flayhart, D., Hindler, J.F., Bruckner, D.A., Hall, G., Shrestha, R.K., Vogel, S.A., Richter, S.S., Howard, W., Walther, R., & Carroll, K.C. (2005). Multicenter evaluation of BBL CHROMagar MRSA medium for direct detection of methicillin-resistant *Staphylococcus aureus* from surveillance cultures of the anterior nares. *Journal of Clinical Microbiology, 43*(11), 5536–5540.

Herman, R.A., Kee, V.R., Moores, K.G., & Ross, M.B. (2008). Etiology and treatment of community-acquired methicillin-resistant *Staphylococcus aureus*. *American Journal of Health-System Pharmacy, 65*(3), 219–225.

Kirkland, E.B., & Adams, B.B. (2008). Methicillin-resistant *Staphylococcus aureus* and athletes. *Journal of the American Academy of Dermatology, 59*(3), 494–502.

Klein, E., Smith, D.L., & Laxminarayan, R. (2007). Hospitalizations and deaths caused by methicillin-resistant *Staphylococcus aureus*, United States, 1999–2005. *Emerging Infectious Diseases, 13*(12), 1840–1846.

McKenna, M. (2008). The many faces of MRSA: Community-acquired infection knows no bounds. *Annals of Emergency Medicine, 51*(3), 285–288.

References

Montgomery, K., Ryan, T., Krause, A., & Starkey, C. (2010). Assessment of athletic health care facility surfaces for MRSA in the secondary school setting. *Journal of Environmental Health, 72*(6), 8–11.

Roberts, M.C., Meschke, J.S., Soge, O.O., & Reynolds, K.A. (2010). Comment on MRSA studies in high school wrestling and athletic training facilities [Letter to the editor]. *Journal of Environmental Health, 72*(9), 48–49.

Roberts, S.S., & Kazragis, R.J. (2009). Methicillin-resistant *Staphylococcus aureus* infections in U.S. service members deployed to Iraq. *Military Medicine, 174*(4), 408–411.

Salgado, C.D., Farr, B.M., & Calfee, D.P. (2003). Community-acquired methicillin-resistant *Staphylococcus aureus*: A meta-analysis of prevalence and risk factors. *Clinical Infectious Diseases, 36*(2), 131–139.

Sampathkumar, P. (2007). Methicillin-resistant *Staphylococcus aureus*: The latest health scare. *Mayo Clinic Proceedings, 82*(12), 1463–1467.

Shukla, S.K. (2006). CA-MRSA triangulation: Virulent strains, susceptible hosts, and contaminated environments. *Wisconsin Medical Journal, 105*(6), 21–23.

Stanforth, B.A., Krause, A., Starkey, C., & Ryan, T.J. (2010). Prevalence of community-associated methicillin-resistant *Staphylococcus aureus* in high school wrestling environments. *Journal of Environmental Health, 72*(6), 12–16.

Did You Know?

The Centers for Disease Control and Prevention have a methicillin-resistant *Staphylococcus aureus* (MRSA) Web site (www.cdc.gov/mrsa/) that provides information on symptoms, causes, prevention, people at risk, treatment, environmental cleaning, and educational resources.

INTERNATIONAL PERSPECTIVES

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Prevalence of Methicillin-Resistant Staphylococci Species Isolated From Computer Keyboards Located in Secondary and Postsecondary Schools

Although most of the information presented in the Journal *refers to situations within the United States, environmental health and protection know no boundaries. The* Journal *periodically runs International Perspectives to ensure that issues relevant to our international membership, representing over 20 countries worldwide, are addressed. Our goal is to raise diverse issues of interest to all our readers, irrespective of origin.*

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Abstract **Methicillin-resistant *Staphylococcus aureus* (MRSA) is a public health threat within the general community, thereby warranting identification of MRSA reservoirs within the community. Computer terminals in schools were sampled for *S. aureus* and methicillin-resistant staphylococci. The overall prevalence of MRSA on computer keyboards was low: 0.68% for a postsecondary institution and 2% and 0% for two secondary institutes. The MRSA isolate from the postsecondary institution did not correspond to the Canadian epidemic clusters, but is related to the USA 700 cluster, which contains strains implicated in outbreaks within the U.S.**

The isolate from the secondary institute's keyboard was typed as CMRSA7 (USA 400), a strain that has been implicated in both Canadian and U.S. epidemics. Methicillin-resistant *S. haemolyticus* and *S. epidermidis* were also isolated from keyboards, indicating that a mixed community of methicillin-resistant staphylococci can be present on keyboards. Although the prevalence was low, the presence of MRSA combined with the high volume of traffic on these student computer terminals demonstrates the potential for public-access computer terminals and computer rooms at educational institutes to act as reservoirs.

Introduction

Staphylococcus species are commonly divided into two groups: pathogenic *S. aureus* and coagulase-negative staphylococci (CoNS). CoNS include multiple species and are generally regarded as only opportunistically pathogenic. The frequency of methicillin resistance in CoNS is notably high and it has been suggested that this may provide a reservoir to propagate methicillin resistance into other *Staphylococcus* species (Lindsay & Holden, 2004).

The occurrence of methicillin-resistant *Staphylococcus aureus* (MRSA) in hospitals was first reported in 1961 (Jevons, 1961). Numerous nosocomial MRSA outbreaks occur annually due to the widespread prevalence of MRSA within hospitals (Klein, Smith, & Laxminarayan, 2007). Recently, highly virulent strains of MRSA have been identified in individuals with no history of recent hospitalizations and no evidence of having predisposing risk factors. These strains have been referred to subsequently as community-associated MRSA (CA-MRSA) and have become a global infectious threat (reviewed in Diep & Otto, 2008). In the U.S., 33% of current MRSA infections are due to infections of community origin (Klevens et al., 2007).

Compared to the U.S., Australia, and other nations, MRSA rates in Canada have been relatively low; however, they have increased 16-fold from 1995 to 2005 from 0.46 per 1,000 hospital admissions to 7.6 per 1,000 hospital admissions (Webster, Rennie, Brosnikoff, Chui, & Brown, 2007). Two main strains have been implicated in the majority of CA-MRSA infections in Canada: CMRSA 7 (also known as USA 400/MW2) and CMRSA 10 (also known as USA 300) (Christianson, Golding, Campbell, & Mulvey, 2007).

Identifying reservoirs for pathogenic organisms is an important step in implementing intervention methods to prevent the spread of disease. Studies examining routes of transmission of hospital-associated MRSA (HA-MRSA) have shown that hospital keyboards can represent a significant reservoir; the incidence of keyboard contamination by MRSA in these studies ranged from 8% to 42% (Bures,

Fishbain, Uyehara, Parker, & Berg, 2000; Devine, Cooke, & Wright, 2001; Fellowes, Kerstein, & Azadian, 2006; Neely et al., 2005). The high number of users on computer terminals in public settings such as libraries and computer labs at schools creates an opportunity for the transmission of bacteria (Anderson & Palombo, 2009), suggesting they may be a possible reservoir for CA-MRSA.

MRSA prevalence on keyboards within a community setting was recently investigated by researchers at the University of Toledo (Kassem, Siglar, & Esseili, 2007). Twenty-four public-access computer keyboards were sampled and two of the keyboards were found to be contaminated with MRSA. The presence of MRSA combined with the high volume of traffic on public computer terminals is a concern and may contribute to the spread of this pathogen in the community. Using selective and differential media we investigated the prevalence of *S. aureus* and methicillin-resistant staphylococci contamination on public-access computer terminals at the University of Regina and two secondary schools (grades 10–12) within the Regina area.

Methods

Specimen Collection

Keyboards in the Archer Library at the University of Regina were sampled repeatedly over several nonconsecutive days with a minimum of seven days between sampling dates during the months of October–November 2007 and January–February 2008. Both high traffic (n = 7) and low traffic keyboards (n = 13) were included in the sampling. High-traffic computers are standing terminals located at the main entrance of the library and are used by many individuals for short periods of time, whereas low-traffic computers are sit-down terminals used for longer periods of time resulting in fewer users on any given day.

Computer keyboards were also sampled by high school students at two high schools in the Regina area on March 5 (HS #1) and March 27 (HS #2), 2009. A total of 50 individual keyboards from two computer labs were sampled one time from HS #1 while 71 individual keyboards were sampled one time from three computer labs at HS #2. These computer labs are accessed by the majority of the student population and are in use throughout the day.

Sterile cotton swabs dipped in sterile phosphate buffered saline (PBS, Fluka) were passed over the entire surface of all letter keys, space bar, and enter key. Swabs were cut so that only the cotton swab was placed directly into tryptic soy broth (TSB) and incubated overnight at 37°C with agitation. A control swab dipped in phosphate buffered saline and briefly exposed to the air was also incubated in TSB along with the keyboard samples.

Isolation and Identification of *Staphylococcus* Colonies

After incubation, turbid TSB tubes were subcultured onto mannitol salt agar (MSA) medium, a selective medium used to isolate putative *Staphylococcus* species and differentiate *S. aureus* (Chapman, 1943), and incubated for 48 hours at 37°C. One hundred µL of the turbid TSB culture was also inoculated into TSB supplemented with oxacillin (2 mg L-1) and incubated overnight at 37°C with agitation (Jonas, Speck, Daschner, & Grundmann, 2002) prior to plating onto MSA and Baird-Parker agar (Baird-Parker, 1962; Oxoid). Oxacillin, which is in the same class of drugs as methicillin, is used since methicillin is no longer commercially available. Additionally, oxacillin maintains its activity during storage better than methicillin. Colonies arising on MSA and Baird-Parker agar exhibiting morphology appropriate to *S. aureus* were further characterized using gram-staining, testing for catalase, and coagulase testing (Pastorex Staph-Plus kit, Bio-Rad). Catalase and coagulase positive isolates were subcultured onto MRSASelect medium (Bio-Rad) and oxacillin screen agar (OSA) medium.

Isolates that grew on OSA and MRSASelect were inoculated onto lysogeny broth plates and sent to the Saskatchewan Disease Control Laboratory (Regina, Saskatchewan) for automated identification and antibiotic susceptibility testing. Antimicrobial susceptibility testing was performed using automated instrumentation (MicroScan WalkAway plus System). Interpretive criteria for MIC values were applied as recommended by the Clinical and Laboratory Standards Institute (Clinical and Laboratory Standards Institute, 2011).

Genomic Profiling of MRSA and Other MRS Strains

Profiling of the MRSA strains involved *S. aureus* protein A gene (spa) typing (Shopsin et al., 1999), detection of Panton-Valentine leukocidin (PVL) toxin gene, and methicillin-resistance mecA gene detection by multiplex polymerase chain reaction (PCR) as described by McDonald and co-authors (2005). Pulsed-field gel electrophoresis (PFGE) as described by Mulvey and co-authors (2001) was used when necessary. Spa types and PFGE profiles of MRSA isolates were compared to local and national databases (Saskatchewan Disease Control Laboratory and Canadian Nosocomial Infections Surveillance Program) to determine if they were members of known clusters or match any previously observed clinical strains. Classification based on PFGE profile followed the recommendation of Tenover and co-authors (1997) whereby if the typical number of fragment differences compared to the outbreak pattern is greater or equal to seven, then they are not related. Indistinguishable, closely related, and possibly related strains have 0, 2–3, and 4–6 fragment differences from the outbreak pattern, respectively (Tenover et al., 1997).

Determining Survival of *Staphylococcus* spp. on Keyboards

Individual computer keyboard keys were removed from standard keyboards, cleaned, and autoclaved prior to inoculation with individual *Staphylococcus* strains. *Staphylococcus* species were provided by the Saskatchewan Disease Control Laboratory. The HA-MRSA was a CMRSA-2 (PVL-) strain while the CA-MRSA strain was a CMRSA-7 (PVL+) strain. Isolates were enriched overnight at 35°C on TSB (with oxacillin for MRSA isolates). Cells were adjusted to optical density of 0.9 at 620 nm (approximately 5 x 10^9 cells). Twenty µL of the cell suspension were inoculated onto individual keyboard keys. For each strain a total of 36 keys were inoculated, allowing each sampling day to be conducted in triplicate. A negative control (20 mL sterile PBS) was also inoculated onto 12 keys. The keys were kept in the laboratory at ambient temperature and humidity. On a daily basis for a period of 12 days bacteria were recovered from the keys, in triplicate, by swabbing the entire surface of each key with a sterile swab moistened in PBS. The swab was cut to ensure no cross contamination, the keyboard keys were both placed in a sterile 50 mL tube containing 5 mL TSB, and the tube was vortexed for one minute in order to recover all the cells. The bacteria were subsequently enumerated by spread plating serial dilutions onto TSA medium.

Statistical Analysis

The bacterial counts obtained for each strain were compiled and the Weibull type model (Marfart, Couvert, Gaillard, & Leguerine, 2002) was used to fit them:

$$(\log10\ (N) = \log10\ (N_0) - \left(\frac{t}{\delta}\right)^{p}$$

where N represents the bacterial density (CFUs per keyboard key) observed at time t (in days), N_0 is the initial bacterial density (in CFUs per keyboard key), and δ is the time (in days) for the first decimal reduction in bacterial cell number. The model was fitted using the nls function of the R software version 2.0.1 (Ihaka & Gentleman, 1996). A one-way ANOVA test was carried out in order to examine the influence of the different strains on the δ parameter values. Multiple comparisons of the δ values were then made using pairwise *t*-tests (Bonferroni correction).

TABLE **1**

Prevalence of *Staphylococcus aureus*, Oxacillin-Resistant Bacteria, and Methicillin-Resistant *S. aureus* (MRSA) on Computer Keyboards

Location[a]	Growth on Tryptic Soy Agar[b]	Growth in Tryptic Soy Agar Oxacillin[b]	Coagulase Positive[b]	MRSA[c]
UR-L[d]	70 (100)	29 (56*)	9 (13)	0 (0.0)
UR-H[d]	77 (100)	17 (61*)	17 (22)	1 (1.3) Spa = 664 PVL-ve
HS #1[d]	50 (100)	50 (100)	32 (60)	1 (2.0) Spa = 128 PVL-ve
HS #2[d]	71 (100)	66 (92)	27 (38)	0 (0.0)

[a]Keyboards were sampled as described in the methods section.
[b]The parentheses represent % prevalence.
[c]The spa type and presence of Panton-Valentine leukocidin (PVL) genes are indicated for each MRSA isolate.
[d]UR-L = University of Regina low-traffic computers; UR-H = University of Regina high-traffic computers; HS #1 = high school 1; HS #2 = high school 2.
*% calculated from the second period of sampling, first period was subcultured in Mannitol salt agar.

Results

The computer keyboards from the three schools experienced different levels of *S. aureus* contamination (Table 1). Higher incidences of *S. aureus* were observed on the high school keyboards. Two MRSA strains were isolated during this survey, one originating from a single high-traffic keyboard (Arch 7) at the University of Regina library and the other from a HS #1 keyboard. The University of Regina keyboards, including Arch 7, were sampled several times during October–November 2007 and January–February 2008; however, the MRSA strain was only detected once on the Arch 7 keyboard during the October–November sampling period.

The two MRSA isolates were further characterized using spa typing. The MRSA isolate from the University of Regina library (UR-1) has a spa type 664 and has a repeat succession 07-23-12-12-17-20-17-12-17. This spa type is not present in the Saskatchewan Disease Control Laboratory (SDCL) or the Canadian Nosocomial Infection Surveillance Program (CNISP) spa typing databases. It is found, however, within the Ridom SpaServer (Harmsen et al., 2003). Six isolates with this spa type are present in the database and all were originally isolated in Sweden. Because of the relatively uncharacterized nature of the isolate, PFGE was performed for further identification. The UR-1 isolate's PFGE pattern clustered with the CMRSA7 profile; however, it has greater then seven fragment differences compared to its closest related strain. Therefore, UR-1 is a distant relative to CMRSA7 (Figure 1).

Furthermore, the PFGE fingerprint of UR-1 did not correspond to any patterns from MRSA isolates obtained from Saskatchewan patients that were stored in the SDCL database. The PFGE profile was subsequently compared to the PFGE national database of CNISP. The PFGE pattern of isolate UR-1 did match to three clinical isolates in this database, 02S1336 (isolated in 2002), 06S1154 (isolated in 1995), and N08-00209 (isolated in 2008), indicating that this strain can be associated with human disease. The strains found in this cluster are related to the USA700 cluster, which has been found in both community and nosocomial settings (Tenover et al., 2008). The MRSA isolate from HS #1 (Lum1) has the spa type t128, which is the spa type found in the CA-MRSA strain lineage CMRSA7/USA400, one of the two prominent community acquired MRSA strains in the U.S. and Canada (Baba et al., 2002; Christianson et al., 2007). This lineage and USA300/CMRSA10 are considered highly clinically significant and together with the hospital associated MRSA strains, CMRSA 1 to 6 and 9, they represent over 80% of all reported MRSA infections in Canada (Simmonds, Dover, Louie, & Keays, 2008). CA-MRSA strains often carry the genes coding for the PVL toxin (Tenover et al., 2008). Both the MRSA strains isolated in this study tested negative for the presence of the PVL genes (data not shown).

The incidence of oxacillin-resistant bacteria contaminating the keyboards was particularly high in the high schools (Table 1), although the prevalence observed in the university library was also relatively high. The high frequency of growth in oxacillin-supplemented TSB was investigated further and several coagulase-negative *Staphylococcus* isolates obtained in the University of Regina sampling were screened for growth on OSA. Four coagulase-negative *Staphylococcus* isolates grew on OSA and were confirmed to be oxacillin resistant (Table 2) and a PCR assay verified the presence of the mecA gene (not shown). Biochemical typing identified the strains as *S. epidermidis* and *S. haemolyticus*.

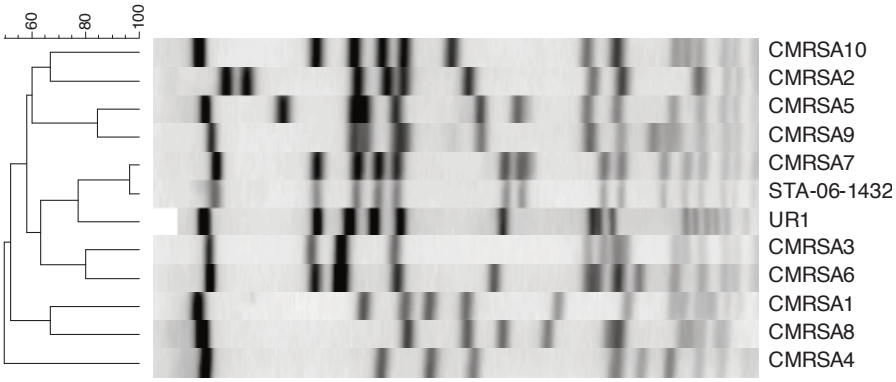
The two distinct MRSA isolates shared similar antibiotic resistance profiles (Table 2). The methicillin-resistant *S. epidermidis* and *S. haemolyticus* (MRSE and MRSH) isolates had distinctive profiles and were generally resistant to more antibiotics than the MRSA isolates.

To determine the length of time a keyboard may remain contaminated with MRSA, the survival of *Staphylococcus* strains on keyboards was also investigated. Figure 2a shows the survival curves for the *Staphylococcus* spp.

FIGURE **1**

a

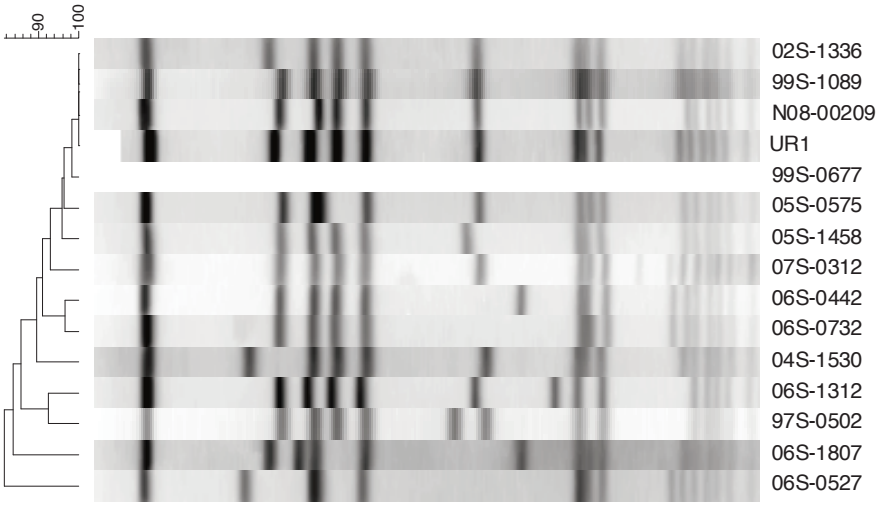
PFGE Fingerprint Comparison Between University of Regina Isolate (UR–1) and the 10 Canadian Epidemic Strains



PFGE = pulsed-field gel electrophoresis. STA-06-1432 is a clinical isolate related to CMRSA7 and was used as a control strain.

b

PFGE Fingerprint Comparison of UR-1 With Related PFGE Patterns Found in the Canadian Nosocomial Infections Surveillance Program Database



PFGE = pulsed-field gel electrophoresis. University of Regina Isolate clustered with isolates from the USA700 PFGE pattern.

used in the study. A large percentage of cells were inactivated rapidly during the first day of incubation. The rate of die off decreased, however, and persistent recovery of cells was possible following 12 days of incubation. Considering the 95% confidence interval overlap, no significant differences occurred between the mean δ values for the HA-MRSA (S2), *S. aureus* (S3), and *S. epidermidis* (S4) strains (Figure 2b). This statement was also confirmed by using the Bonferroni correction test ($p > .05$). The CA-MRSA (S1) strain had a significantly higher survival rate, however, when compared to the *S. aureus* and *S. epidermidis* strains (Bonferroni, $p < .01$).

Discussion

The primary mode of transmission of *S. aureus* is thought to be direct skin-to-skin contact (Miller & Diep, 2008). Computer keyboards have been recognized, however, as an alternative important reservoir for pathogenic bacteria, such as MRSA, within hospital and clinical settings (Fellowes et al., 2006; Shultz, Gill, Zubairi, Huber, & Gordin, 2003; Wilson et al., 2006). Moreover, recent attention has also focused on the potential role of public computer keyboard terminals as reservoirs for pathogens like MRSA (Anderson & Palombo, 2009; Kassem et al., 2007).

In our study, computer keyboards at educational institutions were selected since these keyboards receive relatively high volumes of users. The degree of contamination on the keyboards by *S. aureus* varied widely between institutions, with absolute prevalences ranging from 18% to 60%. These ranges are similar to other studies on public keyboard terminals at universities; for instance, Anderson and Palombo (2009) reported prevalences of *S. aureus* on multiple-user keyboards ranging from 40% to 60%. Kassem and co-authors (2007) reported a prevalence of 21% on multiple-user university keyboards.

The keyboard sampling at the University of Regina allowed for a comparison between high-traffic and low-traffic keyboards. Of the samples taken from low-traffic keyboard terminals, the prevalence of *S. aureus* was 13% (9/70) while the high traffic terminals the prevalence was 22% (17/77). Perhaps not surprisingly, the single MRSA strain from the University of Regina was isolated from one of the high-traffic computer keyboards. The keyboards at high schools are considered high traffic given the large numbers of students who access the computer labs on a daily basis, and this frequency likely contributes to the high incidence of *S. aureus* on these terminals. Intuitively it seems reasonable to expect higher contamination on multiple-user keyboards. The results of our study and that of Anderson and Palombo (2009) and Kassem and co-authors (2007) reinforce the emphasis that should be placed

on disinfection of high-traffic computer keyboards, especially, as well as placing hand sanitizers near high-traffic public computer keyboards.

MRSA was identified at two of the three institutions with an absolute prevalence of 0.68% and 2.0%. This result is in agreement with the limited data on MRSA prevalence on public computer terminals, where the incidence of MRSA on computer keyboards from another university setting was 8.3% (2/24) (Kassem et al., 2007). Brooke and co-authors (2009) did not detect any MRSA isolates from university keyboards (30 samples total).

Spa typing and PFGE profiling were used to characterize the MRSA isolates from this study. UR-1 is an uncommon CA-MRSA isolate while Lum-1 is an isolate within the prevalent CMRSA 7 group that is commonly implicated in CA-MRSA infections in both Canada and the U.S. Only sporadic cases of UR-1-like strains have been observed (Figure 1b) (Mulvey et al., 2005). The isolates were further characterized for the presence of the PVL genes. CA-MRSA strains isolated in clinical situations often carry the genes coding for the PVL toxin. PVL causes tissue necrosis and leukocyte destruction by forming pores in cellular membranes (Lina et al., 1999), and the PVL genes are commonly associated with CA-MRSA virulence (Diederen & Kluytmans, 2006; Diep & Otto, 2008; Etienne, 2005).

Interestingly, both the CA-MRSA strains isolated in our study did not possess the genes for PVL. Recent research comparing clinical isolates from the CA-MRSA USA400 (CMRSA 7) group indicated that only 22.3% of the isolates were PVL positive and the PVL-negative isolates shared similar clinical characteristics and virulence to the PVL-positive isolates, suggesting PVL may not be absolutely necessary for CA-MRSA virulence (Zhang, McClure, Elsayed, Tan, & Conly, 2008). UR-1 and Lum-1 were also characterized for additional antibiotic resistance phenotypes. Both isolates had a resistance profile typical of CA-MRSA strains (Chambers & Deleo, 2009).

The survival of MRSA on keyboards is an important consideration, as the duration of persistence will directly impact the potential risk for transmission of the pathogen to keyboard users. Our study found that MRSA and methicillin-susceptible *Staphylococcus aureus* (MSSA) can persist for at least 12 days on keyboards, thereby allowing for possible transmission to multiple users who access a contaminated keyboard. This is similar to reports of MRSA persisting on laminated tabletops for more than 12 days (Huang, Mehta, Weed, & Price, 2006). The slight but significantly higher survival rate in the CA-MRSA strain is noteworthy and merits further investigation.

The high prevalence of oxacillin-resistant bacteria on the keyboards and the subsequent isolation of MRSE and MRSH on the computer keyboards is worth noting. It is tempting to speculate that MSSA may gain resistance genes when colonizing environments that contain methicillin-resistant coagulase-negative staphylococci. In fact, it has been suggested that the SCCmec elements, which confer methicillin resistance to *Staphylococcus* species, are derived from coagulase-negative staphylococci (Lindsay & Holden, 2004). The mechanisms for the transfer of SCCmec elements are not well understood, however, and require further study. Notably MSSA and methicillin-resistant coagulase-negative staphylococci (*S. epidermidis* and *S. haemolyticus*) were isolated from the same keyboard on separate sample dates during the University of Regina sampling, and since *S. aureus* can survive on keyboards for extended periods of time (Duckworth & Jordens, 1990 and Figure 2) it is possible for co-contamination

TABLE **2**

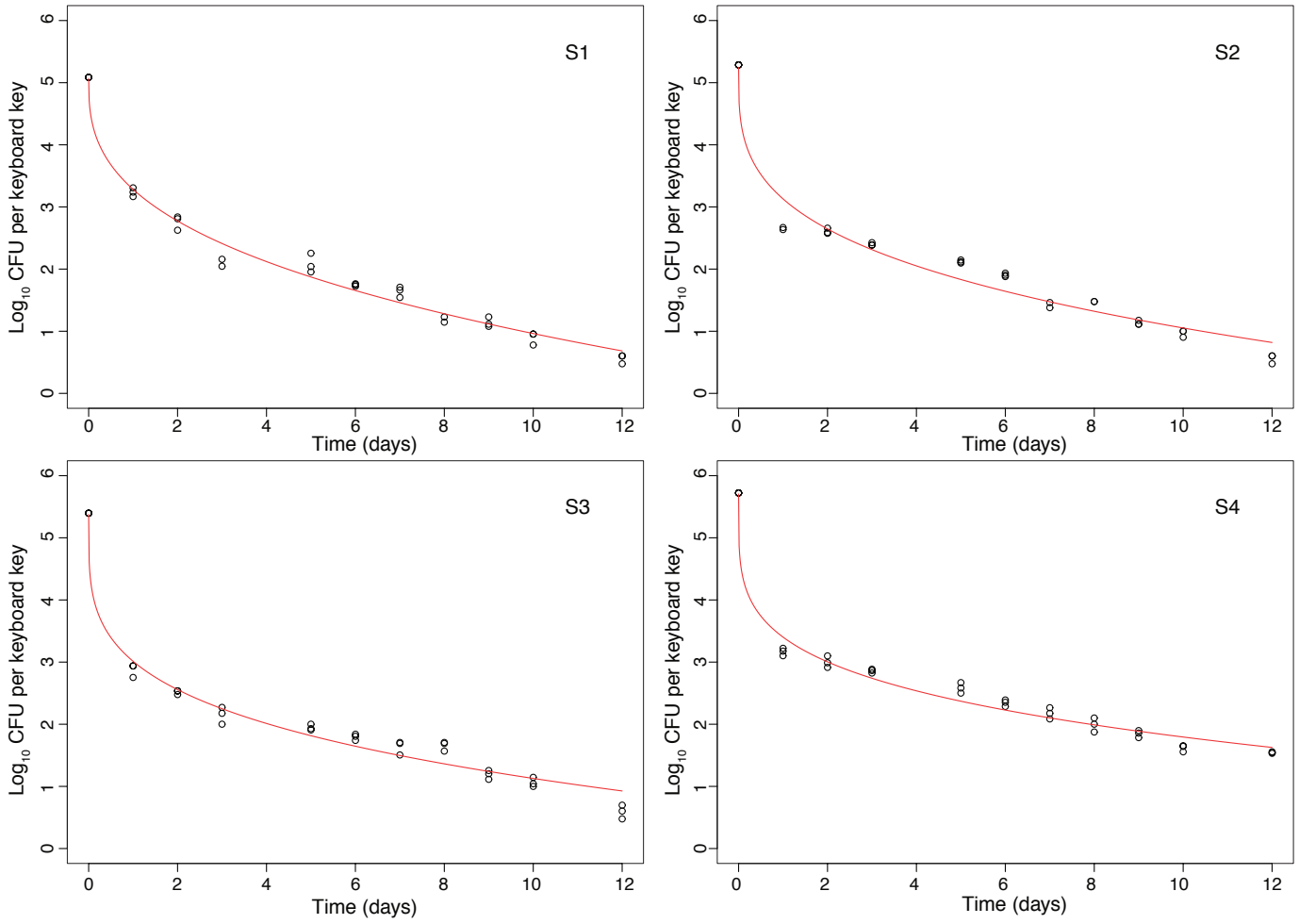
Antibiotic Resistance Profiles of the Methicillin-Resistant *Staphylococcus* spp. Isolates

Antibiotic Class	Antibiotic[a]	UR-1[b]	Lum-1[c]	MRSE-1[d]	MRSE-2	MRSH-1[d]	MRSH-2
Penicillins (B-Lactams)	Amox/K Clav	R	R	R	R	R	R
Penicillins (B-Lactams)	Amp/ Sulbactam	R	R	R	R	R	R
Penicillins (B-Lactams)	Ampicillin	BLAC	BLAC	BLAC	BLAC	BLAC	BLAC
Penicillins (B-Lactams)	Oxacillin	R	R	R	R	R	R
Penicillins (B-Lactams)	Penicillin	BLAC	BLAC	BLAC	BLAC	BLAC	BLAC
Cephalosporin 1 (B-Lactams)	Cefazolin	R	R	R	R	R	R
Cephalosporin 3 (B-Lactams)	Ceftriaxone	R	R	R	R	R	R
Aminoglycoside	Gentamicin	S	S	R	S	S	S
Dihydrofolate reductase inhibitor/sulfonamide	Trimeth/Sulfa	S	S	R	S	S	S
Fluoroquinolone	Ciprofloxacin	S	S	R	R	R	R
Fluoroquinolone	Gatifloxacin	S	S	S	S	S	S
Fluoroquinolone	Levofloxacin	S	S	S	S	S	S
Fluoroquinolone	Norfloxacin	S	S	S	S	S	S
Glycopeptide	Vancomycin	S	S	S	S	S	S
Lincosamide	Clindamycin	S	S	S	R	R	R
Macrolide	Erythromycin	S	S	S	R	R	R
Oxazolidinone	Linezolid	S	S	S	S	S	S
Rifamycin	Rifampin	S	S	S	S	S	S
Streptogramin	Quinupristin/ Dalfopristin (Synercid)	S	S	S	S	S	S
Tetracycline	Tetracycline	S	S	S	S	S	S

Note. R = resistant; S = susceptible.
[a]Amox/K Clav = amoxicillin-clavulanate; BLAC = B-lactamase positive.
[b]UR-1 = University of Regina isolate.
[c]Lum-1 = Isolate from HS #1.
[d]MRSE = methicillin-resistant *S. epidermidis*; MRSH = methicillin-resistant *S. haemolyticus*.

FIGURE **2a**

Survival of *Staphylococcus* Strains on Keyboard Keys as Determined by Viable Plate Counts



S1 = CMRSA7 (CA MRSA strain); S2 = CMRSA2 (HA MRSA strain); S3 = *S. aureus*; S4 = *S. epidermidis*.

to occur. Furthermore, the fact that MRSE and MRSH isolates were also resistant to selected macrolides and other antibiotics is notable. A mixed staphylococcal community of antibiotic-resistant genotypes occurring on the keyboards may contribute to development of newly acquired resistances in CA-MRSA isolates. Therefore, further studies on the transfer of antibiotic resistance from MR-CoNS to MSSA and MRSA in the environment is warranted.

Conclusion

In conclusion, computer terminals at the University of Regina and high schools within the Regina area were found to be contaminated with various staphylococci species, including normal flora, methicillin-resistant coagulase-negative staphylococci, and potentially pathogenic MRSA. Although the incidences of MRSA were low, the keyboards still presented a possible reservoir. Survival of *Staphylococcus* spp. were detected up to 12 days postinoculation of computer keyboards. Children have been noted as a population at risk for infection by CA-MRSA (Adcock, Pastor, Medley, Patterson, & Murphy, 1998), suggesting that further sampling of computer labs in elementary schools and promoting awareness to personal hygiene following use of multiuse computer keyboards across all educational institutes may have merit in helping to control the spread of CA-MRSA.

Reducing the risk of transmission from keyboards may benefit from the routine disinfection of keyboards, particularly on high-traffic computers in university and public libraries. Recent technologies have been developed that have been mainly deployed in hospital settings. For example, the use of keyboard designs that facilitate effective disinfection with chemical disinfectants have been considered for hospital settings (Rutala, White, Gergen, & Weber, 2006; Wilson, Ostro, Magnussen, & Cooper, 2008). Using ultraviolet light to sanitize

keyboards has also been tested for eliminating bacterial contamination of keyboards in hospital settings, although the efficiency of disinfection remains unclear (Martin, Qin, Braden, Migita, & Zerr, 2011; Sweeney & Dancer, 2009). In general, increasing public awareness about the risk of using public facilities and providing antimicrobial hand sanitizing stations in areas with open-access keyboards may help lessen the risk of transmittance and potential for infections.

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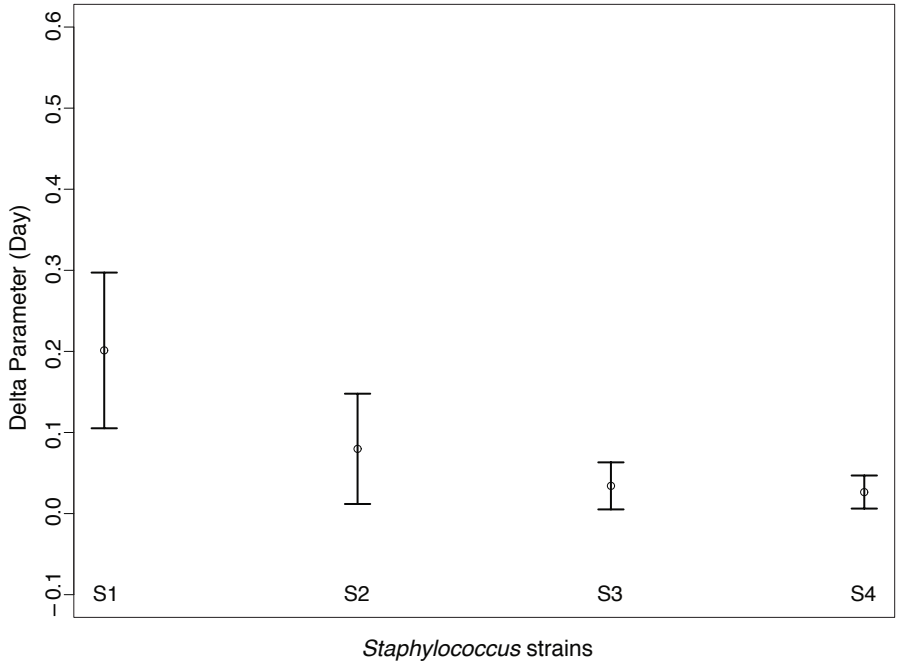
FIGURE **2b**

Mean δ Value With 95% Confidence Interval Obtained for Each *Staphylococcus* spp. Strain on Keyboard Keys



Based on three replicates. S1 = CMRSA7 (CA MRSA strain); S2 = CMRSA2 (HA MRSA strain); S3 = *S. aureus*; S4 = *S. epidermidis*.

References

Adcock, P.M., Pastor, P., Medley, F., Patterson, J.E., & Murphy, T.V. (1998). Methicillin-resistant *Staphylococcus aureus* in two child care centers. *Journal of Infectious Disease*, *178*(2), 577–580.

Anderson, G., & Palombo, E.A. (2009). Microbial contamination of computer keyboards in a university setting. *American Journal of Infection Control, 37*(6), 507–509.

Baba, T., Takeuchi, F., Kuroda, M., Yuzawa, H., Aoki, K., Oguchi, A., Nagai, Y., Iwama, N., Asano, K., Naimi, T., Kuroda, H., Cui, L., Yamamoto, K., & Hiramatsu, K. (2002). Genome and virulence determinants of high virulence community-acquired MRSA. *Lancet, 359,* 1819–1827.

Baird-Parker, A.C. (1962). An improved diagnostic and selective medium for isolating coagulase-positive staphylococci. *Journal of Applied Bacteriology, 25*(1), 12–19.

Brooke, J.S., Annand, J.W., Hammer, A., Dembkowski, K., & Shulman, S.T. (2009). Investigation of bacterial pathogens on 70 frequently used environmental surfaces in a large urban U.S. university. *Journal of Environmental Health, 71*(6), 17–22.

Bures, S., Fishbain, J.T., Uyehara, C.F., Parker, J.M., & Berg, B.W. (2000). Computer keyboards and faucet handles as reservoirs of nosocomial pathogens in the intensive care unit. *American Journal of Infectious Control, 28*(6), 465–471.

Chambers, H.F., & Deleo, F.R. (2009). Waves of resistance: *Staphylococcus aureus* in the antibiotic era. *Nature Reviews Microbiology, 7*(9), 629–641.

Chapman, G.H. (1943). Determination of the chromogenic property of staphylococci. *Journal of Bacteriology, 45*(4), 405–406.

Christianson, S., Golding, G.R., Campbell, J., Canadian Nosocomial Infection Surveillance Program, & Mulvey, M.R. (2007). Comparative genomics of Canadian epidemic lineages of methicillin-resistant *Staphylococcus aureus*. *Journal of Clinical Microbiology, 45*(6), 1904–1911.

Clinical and Laboratory Standards Institute. (2011). *Performance standards for antimicrobial susceptibility testing* (21st ed.). Wayne, PA: Author.

Devine, J., Cooke, R.P., & Wright, E.P. (2001). Is methicillin-resistant *Staphylococcus* (MRSA) contamination of ward-based computer

References

terminals a surrogate marker for nosocomial MRSA transmission and hand washing compliance? *Journal of Hospital Infection, 48*(1), 72–75.

Diederen, B.M., & Kluytmans, J.A. (2006). The emergence of infections with community-associated methicillin-resistant *Staphylococcus aureus*. *Journal of Infection, 52*(3), 157–168.

Diep, B.A., & Otto, M. (2008). The role of virulence determinants in community-associated MRSA pathogenesis. *Trends in Microbiology, 16*(8), 361–369.

Duckworth, G.J., & Jordens, J.Z. (1990). Adherence and survival properties of an epidemic methicillin-resistant strain of *Staphylococcus aureus* compared with those of methicillin sensitive strains. *Journal of Medical Microbiology, 32*(3), 195–200.

Etienne, J. (2005). Panton-Valentine leukocidin: A marker of severity for *Staphylococcus aureus* infection? *Clinical Infectious Diseases, 41*(5), 591–593.

Fellowes, C., Kerstein, K., Clark, J., & Azadian, B.S. (2006). MRSA on tourniquets and keyboards. *Journal of Hospital Infection, 64*(1), 86–88.

Harmsen, D., Claus, H., Witte, W., Rothgänger, J., Claus, H., Turnwald, D., & Vogel, U. (2003). Typing of methicillin-resistant *Staphylococcus aureus* in a university hospital setting using a novel software for spa-repeat determination and database management. *Journal of Clinical Microbiology, 41*(12), 5442–5448.

Huang, R., Mehta, S., Weed, D., & Price, C.S. (2006). Methicillin-resistant *Staphylococcus aureus* survival on hospital fomites. *Infection Control and Hospital Epidemiology, 27*(11), 1267–1269.

Ihaka, R., & Gentleman, R. (1996). R: A language for data analysis and graphics. *Journal of Computational and Graphical Statistics, 5*(3), 299–314.

Jevons, M.P. (1961). Celbenin-resistant staphylococci. *British Medical Journal, 1,* 124–125.

Jonas, D., Speck, M., Daschner, F.D., & Grundmann, H. (2002). Rapid PCR-based identification of methicillin-resistant *Staphylococcus aureus* from screening swabs. *Journal of Clinical Microbiology, 40*(5), 1821–1823.

Kassem, I.I., Siglar, V., & Esseili, M.A. (2007). Public computer surfaces are reservoirs for methicillin-resistant staphylococci. *ISME Journal, 1*(3), 265–268.

Klein, E., Smith, D.L., & Laxminarayan, R. (2007). Hospitalizations and deaths caused by methicillin-resistant *Staphylococcus aureus*, United States, 1999–2005. *Emerging Infectious Diseases, 13*(12), 1840–1846.

Klevens, R.M., Morrison, M.A., Nadle, J., Petit, S., Gershman, K., Ray, S., Harrison, L.H., Lynfield, R., Dumyati, G., Towners, J.M., Craig, A.S., Zell, E.R., Fosheim, G.E., McDougal, L.K., Carey, R.B., & Fridkin, S.K. (2007). Invasive methicillin-resistant *Staphylococcus aureus* infections in the United States. *Journal of the American Medical Association, 298*(15), 1763–1771.

Lina, G., Piemont, Y., Godail-Gamot, F., Bes, M., Peter, M.-O., Gauduchon, V., Vandenesch, F., & Etienne, J. (1999). Involvement of Panton-Valentine leukocidin-producing *Staphylococcus aureus* in primary skin infections and pneumonia. *Clinical Infectious Diseases, 29*(5), 1128–1132.

Lindsay, J.A., & Holden, T.G. (2004). Staphylococcus aureus: Superbug, super genome? *Trends in Microbiology, 12*(8), 378–385.

Mafart, P., Couvert, O., Gaillard, S., & Leguerinel, I. (2002). On calculating sterility in thermal preservation methods: Application of the Weibull frequency distribution model. *International Journal of Food Microbiology, 72*(1–2), 107–113.

Martin, E.T., Qin, X., Braden, H., Migita, R., & Zerr, D.M. (2011). Randomized double-blind crossover trial of ultraviolet light-sanitized keyboards in a pediatric hospital. *American Journal of Infection Control, 39*(5), 433–435.

McDonald, R.R., Antonishyn, N.A., Hansen, T., Snook, L.A., Nagle, E., Mulvey, M.R., Levett, P.N., & Horsman, G.B. (2005). Development of a triplex real-time PCR assay for detection of Panton-Valentine leukocidin toxin genes in clinical isolates of methicillin-resistant *Staphylococcus aureus*. *Journal of Clinical Microbiology, 43*(12), 6147–6149.

Miller, L.G., & Diep, B.A. (2008). Clinical practice: Colonization, fomites, and virulence: Rethinking the pathogenesis of community-associated methicillin-resistant *Staphylococcus aureus* infection. *Clinical Infectious Diseases, 46*(5), 752–760.

Mulvey, M.R., Chui, L., Ismail, J., Louie, L., Murphy, C., Chang, N., Alfa, M., & the Canadian Committee for the Standardization of Molecular Methods (2001). Development of a Canadian standardized protocol for sub typing methicillin-resistant *Staphylococcus aureus* using pulsed-field gel electrophoresis. *Journal of Clinical Microbiology, 39*(10), 3481–3485.

Mulvey, M.R., MacDougall, L., Cholin, B., Horsman, G., Fidyk, M., Woods, S., & the Saskatchewan CA-MRSA Study Group (2005). Community-associated methicillin resistant *Staphylococcus aureus*, Canada. *Emerging Infectious Diseases, 11*(6), 844–850.

Neely, A.N., Weber, J.M., Daviau, P., MacGregor, A., Miranda, C., Nell, M., Bush, P., & Lighter, D. (2005). Computer equipment used in patient care within a multi-hospital system: Recommendations for cleaning and disinfection. *American Journal of Infection Control, 33*(4), 233–237.

Rutala, W.A., White, M.S., Gergen, M.F., & Weber, D.J. (2006). Bacterial contamination of keyboards: Efficacy and functional impact of disinfectants. *Infection Control and Hospital Epidemiology, 27*(4), 372–377.

Schultz, M., Gill, J., Zubairi, S., Huber, R., & Gordin, F. (2003). Bacterial contamination of computer keyboards in a teaching hospital. *Infection Control and Hospital Epidemiology, 24*(4), 302.

Shopsin, B., Gomez, M., Montgomery, S.O., Smith, D.H., Waddington, M., Dodge, D.E., Bost, D.A., Reihman, M., Naidich, S., & Kreiswirth, B.N. (1999). Evaluation of protein A gene polymorphic region DNA sequencing for typing of *Staphylococcus aureus* strains. *Journal of Clinical Microbiology, 37*(11), 3556–3563.

Simmonds, K.A., Dover, D.C., Louie, M., & Keays, G. (2008). Epidemiology of community associated methicillin-resistant *Staphylococcus aureus*. *Canadian Communicable Disease Reports, 34,* 8.

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References *continued from page 57*

Sweeney, C.P., & Dancer, S.J. (2009). Can hospital computers be disinfected using a hand-held UV light source? *Journal of Hospital Infections, 72*(1), 92–94.

Tenover, F.C., Arbeit, R.D., & Goering, R.V. (1997). How to select and interpret molecular strain typing methods for epidemiological studies of bacterial infections: A review for healthcare epidemiologists. *Infection Control and Hospital Epidemiology, 18*(6), 426–439.

Tenover, F.C., McAllister, S., Fosheim, G., McDougal, L.K., Carey, R.B., Limbago, B., Lonsway, D., Patel, J.B., Kuehnert, M.J., & Gorwitz, R. (2008). Characterization of *Staphylococcus aureus* isolates from nasal cultures collected from individuals in the United States in 2001 to 2004. *Journal of Clinical Microbiology, 46*(9), 2837–2841.

Webster, D., Rennie, R.P., Brosnikoff, C.L., Chui, L., & Brown, C. (2007). Methicillin-resistant *Staphylococcus aureus* with reduced susceptibility to vancomycin in Canada. *Diagnostic Microbiology and Infectious Disease, 57*(2), 177–181.

Wilson, A.P., Hayman, S., Folan, P., Ostro, P.T., Birkett, A., Batson, S., Singer, M., & Bellingan, G. (2006). Computer keyboards and the spread of MRSA. *Journal of Hospital Infection, 62*(3), 390–392.

Wilson, A.P., Ostro, P., Magnussen, M., Cooper, B., & Keyboard Study Group. (2008). Laboratory and in-use assessment of methicillin-resistant *Staphylococcus aureus* contamination of ergonomic computer keyboards for ward use. *American Journal of Infection Control, 36*(10), e19–e25.

Zhang, K., McClure, J.-A., Elsayed, S., Tan, J., & Conly, J.M. (2008). Coexistence of Panton-Valentine leukocidin-positive and -negative community-associated methicillin-resistant *Staphylococcus aureus* USA400 sibling strains in a large Canadian health-care region. *Journal of Infectious Disease, 197*(2), 195–204.

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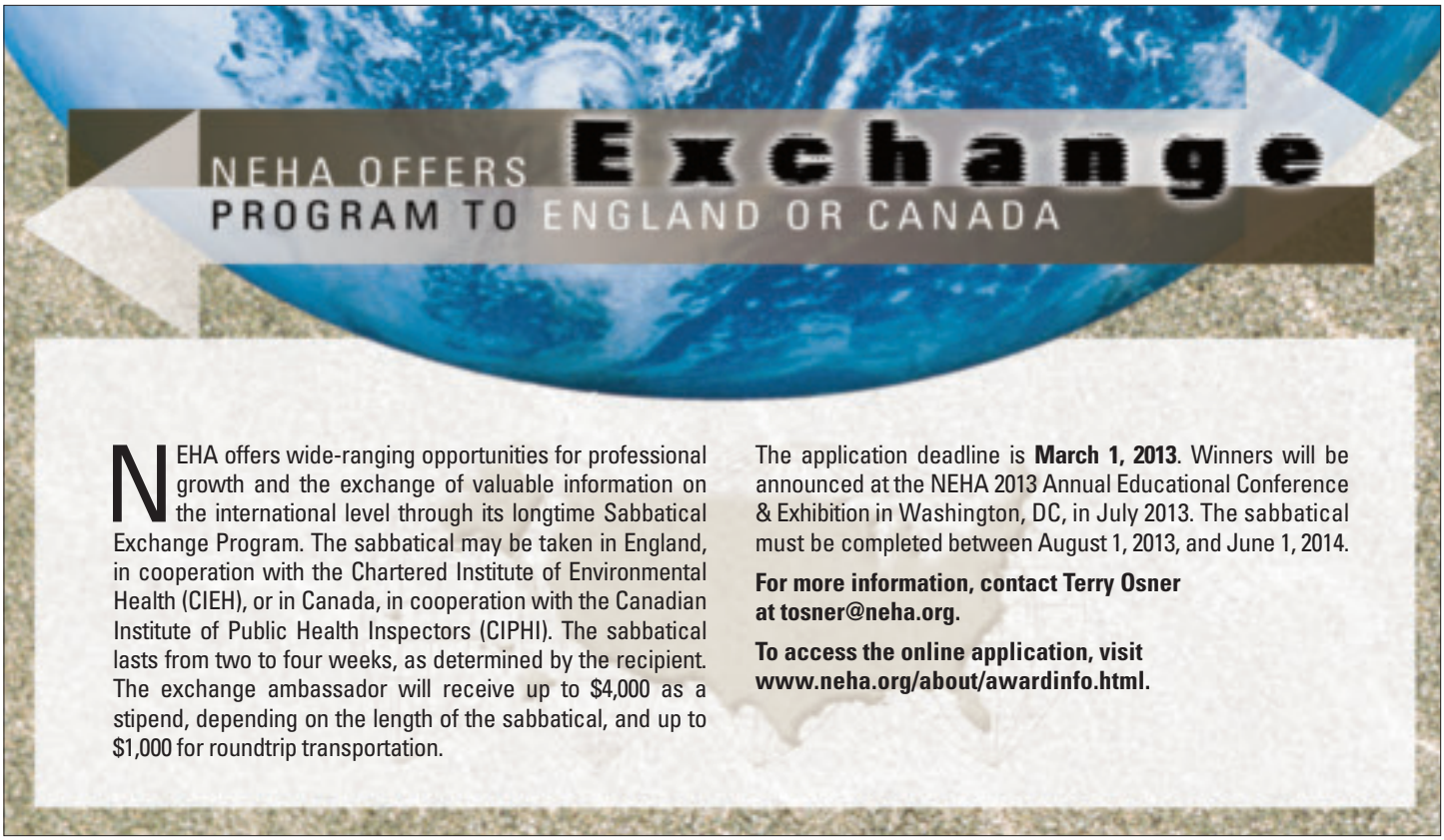

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Water Quality and Management of Private Drinking Water Wells in Pennsylvania

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National Environmental Health Association.

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Trout Unlimited

Abstract **Pennsylvania has over three million rural residents using private water wells for drinking water supplies but is one of the few states that lack statewide water well construction or management standards. The study described in this article aimed to determine the prevalence and causes of common health-based pollutants in water wells and evaluate the need for regulatory management along with voluntary educational programs. Water samples were collected throughout Pennsylvania by Master Well Owner Network volunteers trained by Penn State Extension. Approximately 40% of the 701 water wells sampled failed at least one health-based drinking water standard. The prevalence of most water quality problems was similar to past studies although both lead and nitrate-N were reduced over the last 20 years. The authors' study suggests that statewide water well construction standards along with routine water testing and educational programs to assist water well owners would result in improved drinking water quality for private well owners in Pennsylvania.**

Introduction

Over three million Pennsylvania residents rely on private wells for drinking water and approximately 10,000 new water wells are drilled annually throughout Pennsylvania. Residents utilizing private or small, semi-public wells are typically those most vulnerable to waterborne illnesses (Craun, 1986), and the presence of disease-causing bacteria has been documented in private wells throughout Pennsylvania (Lindsey, Rasberry, & Zimmerman, 2002; Swistock & Sharpe, 2005). Yet Pennsylvania remains one of the few states where private water wells are not regulated.

Various studies have linked water quality in groundwater wells to well construction (Swistock & Sharpe, 2005; Zimmerman, Zimmerman, & Lindsey, 2001), the proximity of wells to pollution sources (Swistock, Sharpe, & Robillard, 1993), climatic conditions (Swistock & Sharpe, 2005), and geology (New Jersey Department of Environmental Protection, 2004). These studies have reported that 15% to 50% of wells fail at least one safe drinking water standard.

The combination of a large population using private water wells along with the lack of statewide regulations and apparent prevalence of water quality problems have created a strong need for research and associated education efforts in Pennsylvania (Mancl, Sharpe, & Makuch, 1989; Swistock, Sharpe, & Dickison, 2001). As a result, Penn State Cooperative Extension created the Master Well Owner Network (MWON) program, a group of over 400 trained volunteers who have educated over 30,000 private water well owners in the state (Clemens, Swistock, & Sharpe, 2007). Despite these efforts, more information is needed to determine the prevalence and causes of water well quality problems along with current management strategies to assist lawmakers and water supply owners with proper strategies to protect groundwater supplies. The overall objective of our study was to investigate the causes of private well water contamination in Pennsylvania and evaluate the need for policy and education guidelines to improve drinking water quality.

Methods

MWON volunteers who had received training on sample collection and water testing methods collected samples from 450 private wells in 2006 and 251 wells in 2007, for a total of 701 water wells. Water wells were distributed regionally ranging from 61 in the sparsely populated northwest region of the state to 167 in the northeast region. A minimum distance of 1.6 km between water wells was used to maximize spatial distribution across the state. Selection of all study sites/participants was done by MWON volunteers, regional coordinators, Penn State Cooperative Extension educators, and project staff. Seventy-nine wells were owned by experienced MWON volunteers and 622 were owned by recently trained MWON volunteers or other homeowners. Experienced MWON volunteers may have improved their well management as a result of previous training; therefore, their results were analyzed separately from the results of other homeowners. Recently trained MWON volunteer and other homeowner wells

TABLE **1**

Variables Used in ANCOVA Statistical Models to Determine Causes of Contamination of Private Wells

Parameter	Description
Geology	Bedrock geology categorized as carbonate, inter-bedded sedimentary, sandstone/shale, igneous, or conglomerate
Soil moisture	Moisture conditions for the two-week period before each sample based on the Palmer soil moisture index ranging from -6 (dry) to +6 (wet)
Region	Regional location of water well based on six regions of Pennsylvania
Date	Date well water sample was collected (3/11/06 to 11/27/07)
Well depth	Estimated water well depth (feet below surface)
Well age	Years elapsed since water well was drilled
Well casing	Presence of a metal or plastic casing on water well
Buried casing	Water well casing extends above ground or entirely buried
Well grout	Visible evidence of grout or cement around the water well casing
Slope	Ground slope promotes movement of surface water toward or away from water well casing
Well cap	Presence of sanitary (vermin proof) well cap or nonsanitary well cap
Well score	Cumulative score of the previous five water well construction components ranging from 0 (none present) to 5 (all five present)
Plumbing type	Plastic versus metal plumbing components predominant in house (for lead analysis)
Plumbing age	Plumbing predominantly installed before or after 1991 (for lead analysis)
Wastewater	Type of wastewater disposal (on lot or public sewer)
Septic age	Estimated age of the wastewater system in years
Septic maintenance	Estimated pumping frequency (1 = yearly; 2 = 2–3 years; 3 = 4 years or more; 4 = never pumped)
Distance to land uses	Estimated distance category (1 = <50'; 2 = 50'–100'; 3 = 100'–500'; 4 = 500'–1000'; 5 = none visible) between the water well and cornfields, other crop fields, gardens, orchards, mines, gas/oil wells, dog kennels, barnyards, pastures, surface water, golf courses, and other water wells
Septic distance	Estimated distance between water well and septic system in feet

were considered representative of typical private wells throughout Pennsylvania.

Regional workshops were organized to efficiently collect water samples for delivery to the water testing laboratory at Penn State University. Volunteers collected water samples from each well on the morning of the workshop into sterile containers. Two water samples were collected from each home including a first draw sample from the kitchen faucet and a running water sample from an untreated tap. Volunteers received training on collecting raw water samples since more than 50% of the water supplies sampled had existing water treatment equipment (mostly softeners and sediment filters). Samples were stored on ice and returned to the workshop location where field measurements of pH and triazine pesticides were made by project personnel within a few hours of sample delivery. Triazine pesticides were measured using an immunoassay test kit with a detection limit equivalent to the drinking water standard of 3 µg/L while pH was measured using an Orion pH meter calibrated using 4.01 and 7.01 standards. A 100 mL aliquot of each sample was immediately filtered through sterile 0.45 µm filters and placed on growth agar for enumeration of total coliform bacteria and *E. coli* bacteria.

Upon completion of field water quality measures, water samples were delivered to the water laboratory at the Penn State Institutes of Energy and the Environment for analysis of lab pH, nitrate-N, and arsenic using standard methods. First draw samples collected from 251 water wells in 2007 were delivered to the same laboratory for measurement of lead. Fifty-eight quality control samples, including 12 reference samples, 20 duplicate samples, and 26 blanks were submitted to the water laboratory. All reference samples produced results within ±5% of the known concentration. All duplicate results were within ±25% with the exception of arsenic, which had very low concentrations and average absolute differences of 1 µg/L. Blank samples produced results below detection for all parameters except nitrate-N and arsenic, where very low detections were found in about 25% of the blank samples.

Volunteers completed a survey for each water well sample that included information about the water well characteristics, nearby land uses, and homeowner management practices. Since less than 10% of water supply owners had a water well completion report, water supply characteristics were based on volunteer observations and water supply owner responses. A follow-up survey was sent to the 450 water well owners sampled in 2006 within 12 months after the study to document actions taken by the well owner to solve water quality problems or better manage their water supply as a result of participating in our study. The follow-up survey had a 64.2% response rate.

Analysis of covariance (ANCOVA) and logistic regression models were used to determine well characteristics, land uses, and management activities that were important in explaining the occurrence of various pollutants. The various predictor variables used in statistical models are shown in Table 1. Many predictors were class variables where a "1" was used to indicate presence and a "0" indicated absence. For statistical comparisons, a "well score" was created from the presence/absence of five important well construction characteristics (presence of a casing; casing extended aboveground; evidence of grout or cement around the casing at the surface; ground sloping away from casing; and presence of a sealed, sanitary well cap). The sum of all well characteristics resulted in a range of well scores from 0 (a well containing zero well construction characteristics) to 5 (all five characteristics present). Comparisons of well characteristics and water quality parameters among the six Pennsylvania regions were made using the Tukey multiple means comparison statistical procedure.

Results

Of the 701 water wells in the study, 94% were drilled wells while 6% were hand dug. The mean and maximum well depths were 52 m and 305 m, respectively. Water well

construction characteristics between homeowner and MWON volunteer wells are shown in Table 2. Given the lack of statewide construction standards, the use of sanitary well caps and a grout seal around the casing were low. The presence of grout shown in Table 2 likely overestimates the proper use of grouting (from the surface to bedrock) because grout presence could generally only be determined based on homeowner memory or visual evidence of grout at the surface, since well logs were typically not available.

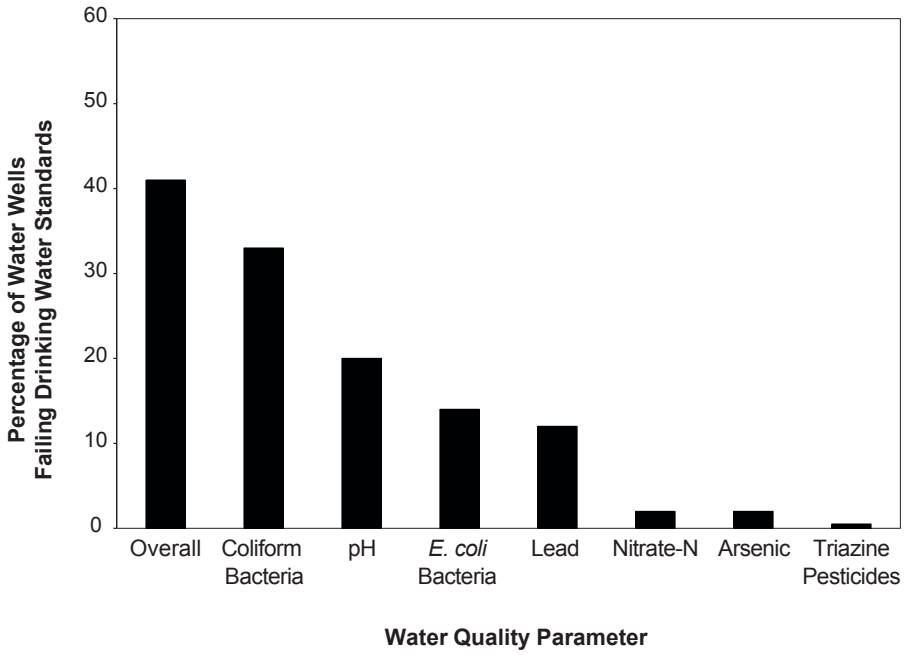
TABLE **2**

Well Construction Characteristics for Homeowner and Master Well Owner Network (MWON) Volunteer Water Wells Sampled

Well Construction Component	Homeowner Wells (*n* = 622)	MWON Volunteer Wells (*n* = 79)
Buried well casing	81 (13%)	7 (9%)
Extended casing with standard well cap	386 (62%)	39 (49%)
Extended casing with sanitary well cap	100 (16%)	27 (34%)
Extended casing with miscellaneous or no cap	56 (9%)	6 (8%)
Grout or cement around casing	112 (18%)	15 (19%)
Ground slopes away from well	224 (36%)	43 (54%)

FIGURE **1**

Overall Percentage of 701 Private Water Wells That Failed at Least One Drinking Water Standard and the Percentage That Failed Standards for Various Individual Water Quality Parameters



Prevalence and Regional Distribution of Contaminants

The percentage of water wells that failed safe drinking water standards for various water quality parameters are shown in Figure 1. Contamination rates in raw well water were similar between MWON volunteer wells and homeowner wells, so they are combined for this discussion. Overall, 41% of the water wells failed at least one health-based drinking water standard. Total coliform bacteria was the most common pollutant found in water wells (33%) and was significantly higher ($p \leq .01$) in wells located in the southeast region in comparison to the northwest or northeast regions. *E. coli* bacteria were detected in 14% of the private wells and showed regional trends similar to those found for coliform bacteria. Twenty percent of wells failed the recommended drinking water standard for pH, with most of these having a low pH below 6.5 that was most prevalent in the southeastern region of the state. Wells with a high pH (above the recommended level of 8.5) were rare (2%) and occurred sporadically across all regions.

Elevated lead levels occurred in first draw water from 12% of the 251 wells that were tested in 2007. Lead contamination was most prevalent in southeast and south-central regions where lower groundwater pH also occurred. Only 2% of the wells in our study exceeded the health-based drinking water standard for arsenic, and these occurred mostly in northern Pennsylvania. Three wells (<1%) located throughout the state contained triazine pesticides above 3 µg/L. While sampling showed that very few wells were above the triazine pesticide drinking water standard, the results do not allow an estimation of the number of wells that may have triazine pesticides present at lower detectable concentrations.

Nitrate-N occurred above the 10 mg/L drinking water standard in 2% of the private wells and varied strongly among regions. Mean nitrate-N concentrations were significantly higher in the intensive agricultural regions of the state (southeast and south-central), although sporadic nitrate-N concentrations above 10 mg/L were found in the central and northeast regions.

Variables Influencing Water Quality

Much of the variability in water quality parameters was attributable to geologic differences among the regions (Table 3). In the ANCOVA models, geology was statistically significant in explaining variation of all water quality parameters (*p*-values generally <.005), with the exception of arsenic, which had no statistically significant parameters. The carbonate rock type was correlated with significantly

TABLE **3**

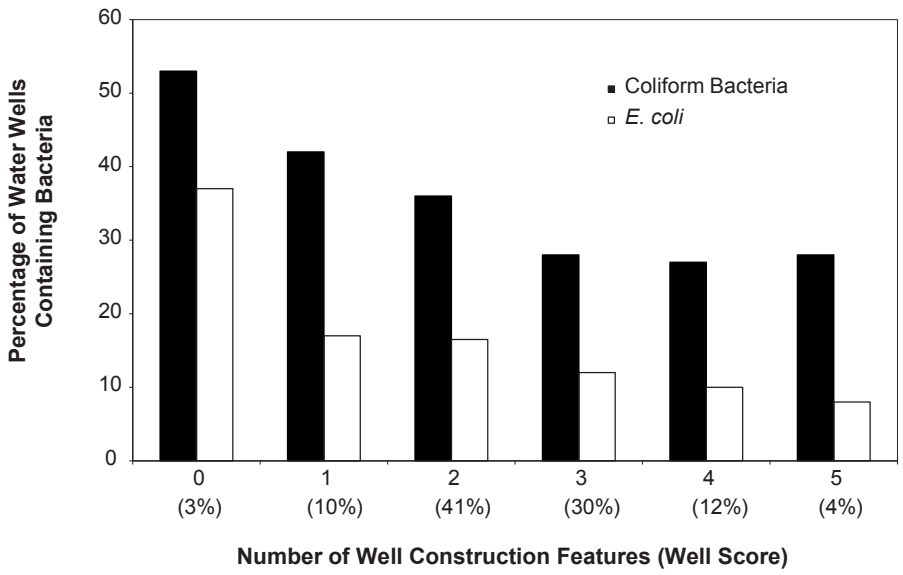
A Summary of Statistically Significant Variables ($p < .05$) in ANCOVA Models for Each Water Quality Parameter

Water Quality Parameter	Statistically Significant Variables
Coliform bacteria	Geology, well score, soil moisture
E. coli bacteria	Geology, well score, soil moisture
Nitrate	Geology, distance to cornfields and other crop fields, well age
pH	Geology
Lead	Geology, plumbing type
Arsenic	No statistically significant variables

Note. Variables are described in Table 1.

FIGURE **2**

Effect of the Number of Well Construction Features (Well Score) on the Percentage of Water Wells Containing Total Coliform and *E. coli* Bacterial Contamination



Percentages below each well score category show the percentage of all water wells in this study that had that well score.

higher bacteria levels, pH, and nitrate-N than most other rock types. Igneous rock, located in parts of southeast and south-central Pennsylvania, had significantly lower pH than other rock types. Sedimentary and sandstone/shale bedrock types, which are both comprised of various types of sandstone and shale, produced nearly identical water quality results.

Short-term soil moisture conditions were important in explaining the occurrence of both coliform and *E. coli* bacteria (Table 3). Of the wells that contained bacteria, 84% were tested during moist conditions (positive Palmer Z Index), while only 16% were tested during dry conditions (negative Palmer Z Index).

Water well construction was also statistically significant in explaining bacterial contamination. Wells with very poor construction (well score = 0) were twice as likely to have coliform bacteria and five times more likely to have *E. coli* bacteria compared to wells with excellent construction (well score = 5) (Figure 2).

Although many measures were made of land uses, activities, and other water supply characteristics (Table 1), these were generally not important in explaining water quality with a few exceptions. A statistically significant inverse correlation ($p < .05$) occurred between well water nitrate-N levels and the distance to various crop fields. Surprisingly, a statistically significant correlation did not occur between any water quality parameters and various septic system features (distance, maintenance, etc.) even though nearly all of these water wells were located on properties with septic systems and over half of water supply owners indicated that they rarely or never maintained their septic system. Nitrate-N concentrations were inversely correlated to the age of the water well ($p < .0001$) and well depth ($p = .08$). First draw lead concentrations were strongly related to the presence of metal versus plastic plumbing systems and to geology, which ultimately controls groundwater pH.

Water Supply Management

Previous water testing among the private water well owners in our study was rare with 30% indicating they had never tested their water quality and 44% indicating that it had been tested just once, usually just for coliform bacteria. Water testing among MWON volunteers was much more common (70%). This general lack of previous water testing resulted in a lack of awareness of health-related pollutants. Zero to 31% of homeowners with water supplies that contained unsafe levels of bacteria, nitrate-N, arsenic, or lead were already aware of these water quality problems. MWON volunteers who had been educated about proper water testing to detect pollutants were generally two to three times more likely to know about a health-related pollutant in their water supply.

The follow-up survey mailed in 2007 to the 450 water well owners who received testing in 2006 (responses received by 288 water well owners) showed that 76% of those who had wells that failed at least one health-based drinking water standard took at least one action to correct or better manage the problem (Table 4). A significant percentage of water well owners with no water quality problems also took actions to protect their water quality (Table 4). Water well rehabilitation (shock chlorination, improved

construction, etc.) and installation of water treatment equipment were the most frequent actions taken on water supplies.

In addition to the mailed survey in 2007, 60 water wells that tested positive for coliform bacteria in 2006 were resampled in 2007 to determine whether actions taken by water supply owners were successful in removing bacteria from the water supply. Over half of these water well owners had taken at least one action to directly reduce raw water bacterial contamination (shock chlorination, sanitary well cap, removing obvious source of contamination, etc.) and follow-up testing found that about 10% were successful in removing bacteria. This success rate and survey responses that showed other actions that were assumed to remove pollutants at the faucet (proper water treatment devices or use of bottled water) resulted in a high percentage (50% to 80%) of water well owners who avoided pollutants as a result of education gained during our study (Table 5).

TABLE **4**

Percentage of 288 Private Well Owners Who Took Various Actions to Improve Their Water Well*

Action Taken	Homeowners With No Water Quality Problems	Homeowners With Water Quality Problems
New water well or use of bottled water	4%	18%
Water treatment	7%	25%
Well rehabilitation	15%	54%
Pollution source(s) removed	13%	13%
Additional water testing	2%	19%
Totals	33%	76%

*As a result of having their well tested in the study. The columns distinguish between homeowners who were notified of water quality problems in their water well versus those who had no water quality problems.

TABLE **5**

Effect of Homeowner Actions on Water Contaminant Exposure Based Upon Follow-Up Testing and Survey of 60 Water Wells

Contaminant	Avoided Contaminant Before Study	Started or Improved Avoidance of Contaminant	Total Avoiding Contaminant 6–12 Months After Testing
Coliform bacteria	20 (33%)	26 (44%)	30 (50%)
E. coli bacteria	25 (42%)	41 (69%)	43 (71%)
Nitrate	7 (12%)	30 (50%)	37 (62%)
Lead	0 (0%)	45 (75%)	45 (75%)
Arsenic	12 (20%)	48 (80%)	48 (80%)

Note. Column 3, "Started or improved avoidance of contaminant," includes use of bottled water or installation of proper treatment equipment. Note that the total avoiding a contaminant (column 4) does not sum from columns 2 and 3 because some well owners simply improved on actions that were already having an effect.

Discussion

The lack of statewide construction standards for private wells in Pennsylvania presumably resulted in the low use of sanitary well caps and grout seals on water wells in this study (Table 2). Although grout or cement seals were evident around 18% of the water wells, the absence of well completion reports for most wells made it impossible to determine if grouting had been done along the entire well casing or just at the surface. Greater use of proper water well construction (sanitary well cap, grout, sloped ground, etc.) on MWON volunteer water wells in comparison to homeowner wells is likely the result of education during MWON training workshops. Additionally, the presence of sanitary well caps on twice the number of MWON wells than those of other homeowners is presumably due to the distribution of sanitary caps to volunteers during MWON training.

While other studies have demonstrated a slightly reduced incidence of bacteria with the presence of grout (Zimmerman et al., 2001), sanitary well caps (Swistock & Sharpe, 2005), and cased wells (Sharpe, Mooney, & Adams, 1985) this study demonstrates a clear connection between overall well construction and bacterial contamination (Figure 2). These results also confirm earlier work by Swistock and Sharpe (2005), which showed bacterial contamination in excess of 20% still occurs even in water wells with adequate construction, presumably due to larger-scale land use activities.

The prevalence of contamination reported for coliform bacteria, *E. coli* bacteria, pH, arsenic, and triazine pesticides were similar to past studies of private water wells in Pennsylvania (Francis et al., 1982; Sharpe et al., 1985; Swistock et al., 1993). While climate, water well construction, and nearby land uses occasionally impacted water quality, the geologic setting of each water well was clearly the most important factor. Some geological differences, however, may be the result of land uses that are most common on certain types of bedrock. For example, higher nitrate-N levels on carbonate and igneous bedrock are likely due to the predominance of these bedrock types in the regions of the state with intensive agricultural land use rather than differences in the bedrock chemistry.

The incidence of lead above the 15 µg/L action level in first draw water samples was lower in this study (12%) compared to 19% reported in a 1989–1991 study of over 1,600 private water wells in Pennsylvania (Swistock et al., 1993). This reduction may be a result of the 1991 Federal Lead and Copper Rule that required the use of lead-free solder and fixtures in home plumbing. Over 70% of the homes with elevated lead concentrations in our study had copper plumbing systems installed before the Lead and Copper Rule was passed and 93% also had acidic water (pH < 7.0). In fact, only one private well had a high lead level that could not be clearly linked to corrosion of metal plumbing (i.e.,

a new home with plastic plumbing and alkaline water).

The incidence of nitrate-N contamination in Pennsylvania well water of 2% in our study was also lower than past research, including 14% found in 1984 and 9% in 1991 (Sharpe et al., 1985; Swistock et al., 1993). These studies did not monitor the same water wells so some of this variation may be related to a larger number of wells sampled in the southeast region of the state where intensive agriculture is most prevalent. Additional reductions in groundwater nitrate-N levels are perhaps a result of education and mandated nutrient management plans that target better farm and home nitrogen management. Data reported in the 2002 Census of Agriculture by the U.S. Department of Agriculture (2004) suggest that applications of nitrogen by fertilizer and manure have dropped in southern Pennsylvania since the early 1990s. Given the ability of nitrate to move long distances through soil and rock, the correlation between nitrate-N and distance to nearest crop field is understandable. The link between nitrate-N concentrations and the combination of well depth and age characteristics suggests that older (typically shallower) wells are perhaps allowing shallow, nitrate-rich water to enter deeper groundwater aquifers.

Voluntary Well Testing

The most revealing results from our study were the lack of voluntary water testing done by private water well owners and the resulting lack of awareness of health-related water quality problems. Since many pollutants with health-based drinking water standards (coliform bacteria, lead, nitrate-N, arsenic, etc.) have no obvious symptoms, thorough water testing is critical to identify their presence in drinking water supplies. Clearly, education for private well owners is the key to increasing voluntary well testing and management in the absence of statewide regulations in Pennsylvania for well construction or maintenance. In our study, homeowners received Penn State Cooperative Extension fact sheets with their water test results to explain potential corrective measures for water quality problems. This approach resulted in a high percentage of water well owners taking action to reduce their exposure to health-related pollutants such as developing new water sources, installing water treatment devices, or reducing sources of pollutants.

Conclusion

Data from our study provide a wealth of information pertaining to the incidence of pollutants in private water wells throughout Pennsylvania, the causes of contamination, and appropriate measures to help water supply owners detect and avoid health-related pollutants. The prevalence of most water quality parameters was similar to past studies of water wells in Pennsylvania, although both lead and nitrate-N contamination rates were lower. The geologic setting of water wells was the most important factor in explaining water well quality but some parameters were also controlled by water well construction, nearby land uses, and climatic variables. The strong correlation between water well construction and the occurrence of both coliform and *E. coli* bacteria are important information for regulators considering water well construction standards in Pennsylvania. Most well construction features need to be included at the time of well drilling, but homeowners having new wells drilled are difficult to reach with educational programs and, as a result, the voluntary approach to encourage proper well construction has largely failed. Given the benefits of well construction and the difficulty in reaching the target audience for new wells, statewide regulations requiring well construction components appear to be warranted. Sixty percent of the water well owners surveyed in our study were in favor of such well construction and location standards.

Beyond proper water well construction, unsafe levels of pollutants in private wells can be removed, treated, or avoided through maintenance, water treatment, or alternate water supplies. The major barrier to successful avoidance of problems identified in our study was a lack of proper water testing strategies to detect water quality problems. Given the large target audience in Pennsylvania, it is not practical to require well owners to have their water tested routinely but thorough testing upon completion of new well construction and before finalization of any real estate transaction is reasonable. Most well owners in our study who were told of health-related water quality issues in their water supply voluntarily addressed the problem within one year. In the absence of both statewide water well construction standards and water testing requirements, comprehensive and unbiased educational programs are needed to educate water well owners.

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References

Clemens, S., Swistock, B.R., & Sharpe, W.E. (2007). The Master Well Owner Network: Volunteers educating Pennsylvania well owners. *Journal of Extension, 45*(4), 4–7.

Craun, G.F. (1986). *Waterborne diseases in the U.S.* Boca Raton, FL: CRC Press.

Francis, J.D., Brower, B.L., Graham, W.F., Larson, O.W., III, McCaull, J.L., & Vigorita, H.M. (1982). *National statistical assessment of rural water conditions*. Washington, DC: The Office of Drinking Water, U.S. Environmental Protection Agency.

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References *continued from page 65*

Lindsey, B.D., Rasberry, J.S., & Zimmerman, T.M. (2002). *Microbiological quality of water from noncommunity supply wells in carbonate and crystalline aquifers of Pennsylvania* (Water Resources Investigations Report 01-4268). New Cumberland, PA: U.S. Geological Survey.

Mancl, K., Sharpe, W.E., & Makuch, J. (1989). Educating the rural public about safe drinking water. *Water Resources Bulletin, 25*(1), 155–158.

New Jersey Department of Environmental Protection. (2004). *Private well testing act program: Initial results for September 2002 to March 2003*. Trenton, NJ: Division of Science, Research, and Technology, and Water Supply Administration (Bureau of Safe Drinking Water).

Sharpe, W.E., Mooney, D.W., & Adams, R.S. (1985). An analysis of ground water quality data obtained from private individual water systems in Pennsylvania. *Northeastern Environmental Science, 4*(3–4), 155–159.

Swistock, B.R., & Sharpe, W.E. (2005). The influence of well construction on bacterial contamination of private water wells in Pennsylvania. *Journal of Environmental Health, 68*(2), 17–23.

Swistock, B.R., Sharpe, W.E., & Dickison, J. (2001). Educating rural private water system owners in Pennsylvania using satellite vs. traditional programs. *Journal of Extension, 39*(3), 3–7.

Swistock, B.R., Sharpe, W.E., & Robillard, P.D. (1993). A survey of lead, nitrate, and radon contamination of private individual water systems in Pennsylvania. *Journal of Environmental Health*, *55*(5), 6–12.

U.S. Department of Agriculture, National Agricultural Statistics Service. (2004). *Pennsylvania state and county data, volume 1* (Geographical Area Series, Part 38, AC-02-A-38). Middletown, PA: Pennsylvania State Data Center.

Zimmerman, T.M., Zimmerman, M.L., & Lindsey, B.D. (2001). *Relation between well-construction practices and occurrence of bacteria in private household-supply wells, south-central and southeastern Pennsylvania* (U.S. Geological Survey Water-Resources Investigations Report No. 01-4206). New Cumberland, PA: U.S. Geological Survey.

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Relationship Between Food Safety and Critical Violations on Restaurant Inspections: An Empirical Investigation of Bacterial Pathogen Content

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Abstract

While various safety control measures exist within the U.S. food system, foodborne illness remains a costly and persistent problem. The purpose of the study described here was to examine the relationship between violations of critical restaurant inspection items ("critical items") and food safety as measured by the bacterial load of illness-causing pathogens. Specifically, the authors' study looked at bacterial pathogens present in foods of two groups of restaurants, those that consistently scored poorly on critical items as compared to restaurants that performed superiorly in the same types of evaluation in Jefferson County, Alabama. Laboratory analyses indicated that 35.7% of the foods tested had detectable levels of *Staphylococcus aureus*, but no difference occurred between the two groups of restaurants. No other bacterial pathogens were found in any of the tested samples. A total of 45.2% of the food samples were received outside of recommended temperatures. Findings draw attention to the ongoing need to improve temperature control and hygienic practices, specifically handwashing practices, in restaurants.

Introduction

It is estimated that foodborne illness costs the U.S. economy $10–$83 billion a year (Food and Drug Administration [FDA], 2004). Additionally, recent estimates indicate that contaminated food ultimately results in 48 million illnesses, 128,000 hospitalizations, and 3,000 deaths annually (Scallan et al., 2011). Laboratory-confirmed foodborne infections show that *Salmonella*, *Campylobacter*, *Shigella*, *Cryptosporidium*, and Shiga toxin-producing *E. coli* O157 are the top five foodborne pathogens affecting Americans (Centers for Disease Control and Prevention [CDC], 2009).

Although *Staphylococcus aureus* is believed to contribute to many cases of foodborne illness in the U.S., the true incidence of illness resulting from the toxin produced by *S. aureus* is unknown for a number of reasons, including the misdiagnosis of this illness and the lack of sample collection for laboratory testing (FDA, 2011a). A recent article reviewing the burden of foodborne illness in the U.S. highlighted the frequency with which Americans consume foods prepared outside the home as one of the five primary factors contributing to the occurrence of foodborne illness (Jones & Angulo, 2006; Nyachuba, 2010). Approximately 50% of funds budgeted for food by Americans are spent in restaurants (Creel, Sharkey, McIntosh, Anding, & Huber, 2008), where, according to the Centers for Disease Control and Prevention (CDC), half of foodborne outbreaks occur (CDC, 2006). While various safety control measures exist within the U.S. food system, foodborne illness remains a costly and persistent problem.

Local public health agencies routinely inspect restaurants for risks to human health by focusing on factors believed to be associated with food safety. Because it is difficult to measure the impact of these inspections on the reduction of risk to human health, the majority of food safety studies have focused on nonhealth outcomes (Cates et al., 2009; Chapman, Eversley, Fillion, Maclaurin, & Powell, 2010; Green & Selman, 2005; Kassa, Silverman, & Baroudi, 2010; Lee, Almanza, Nelson, & Ghiselli, 2009; Phillips, Elledge, Basara, Lynch, & Boatright, 2006; Reske, Jenkins, Fernandez, VanAmber, & Hedberg, 2007). In fact, much of the peer-reviewed research on food safety and restaurant inspections examines the validity and reliability of inspection scores (Klein & DeWall, 2008; Lee et al., 2009; Phillips et al., 2006; Reske et al., 2007) and the relationship between scores with other factors such as the presence of a food safety–trained kitchen manager (Cates et al., 2009; Kassa et al., 2010).

Although the frequency of foodborne illness can be assessed by the number of related hospitalizations and emergency department visits, these measures suffer from severe underreporting because people do not commonly seek medical care for mild cases (symptoms lasting 24–48 hours) of foodborne illness (FDA, 2011b; FDA Retail Food Program Steering Committee, 2000; Mead et al., 1999). Underreporting also results in inaccurate outbreak counts (CDC, 2006; Nyachuba, 2010). Despite these issues, one county-based study by Jin and Leslie measured foodborne illness hospitalizations in relation to changes in how restaurant inspections were scored and displayed for consumers (numerical versus letter scores) (Jin & Leslie, 2003). They found that mandating the display of letter scores was associated with a significant decrease in the number of foodborne illness hospitalizations.

Two additional studies examined the relationship between outbreaks and inspection scores but had contradictory findings (Irwin, Ballard, Grendon, & Kobayashi, 1989; Jones, Pavlin, LaFleur, Ingram, & Schaffner, 2004). In the absence of reliable and available estimates of foodborne illness, an examination of bacterial pathogens in food may shed light on the risk of foodborne illness in restaurants. Examining foods for bacterial pathogens may also provide information about the presence of bacteria, such as *S. aureus*, which are not commonly tested for in laboratory tests. Such pathogens cause acute cases of illness that people frequently endure without seeking medical care or, when they do seek care, physicians do not request specific testing (FDA, 2011a; Mead et al., 1999; Roberts, 2007; Scallan et al., 2006).

The purpose of our study was to examine the relationship between violations of critical restaurant inspection items ("critical items") and food safety as measured by the bacterial load of illness-causing pathogens. Specifically, we looked at bacterial pathogens present in foods of restaurants that consistently scored poorly on critical items as compared to restaurants that performed superiorly in the same types of evaluation. Our study simulates real-world scenarios by utilizing to-go food samples, measuring the temperatures at which they are received, and testing the samples for pathogens. By providing information relevant to our current public health food safety practices and their relationship to human health, our study will be of interest to practitioners and decision makers in public health.

Methods

Study Design and Population

We conducted a matched cohort study of 42 restaurants in Jefferson County, Alabama. The following section details the inclusion criteria, food sample collection and analysis, variables collected, and statistical methods employed.

Inclusion Criteria

Restaurant inclusion in the study was based on performance on recent public health inspections. In Jefferson County, restaurants are routinely inspected three times per year, unless they receive an inspection score below 85, in which case they receive a reinspection prior to the next scheduled one. Using retrospective Jefferson County Department of Health (JCDH) inspection data for the period of April 1 through October 31, 2010, we identified restaurants that lost points on the same critical human health–related violation during two back-to-back routine inspections (FDA Retail Food Program Steering Committee, 2000; U.S. Department of Health and Human Services, 2009). These restaurants were considered for inclusion in the cohort of Group A restaurants. The control cohort (Group B restaurants) was identified as restaurants that lost no points on critical violations across two routine food safety inspections during the study period. We matched Group A and Group B restaurants based on food type (American, fast food, Asian, or Mediterranean) and location (zip code). American food included barbeque and home-style restaurants, steak houses, and bar and grill restaurants. Fast food establishments included chain restaurants in which foods are regularly prepared and quickly available. Asian restaurants included those that serve sushi, Chinese, and Indian foods. Mediterranean restaurants included those that serve Greek and Italian foods. A total of 21 matched pairs were included in our study.

Food Sample Collection and Analysis

The same type of food samples were collected from each matched pair of restaurants on the same day. Food samples were collected as "to go" orders to mimic real-life food service scenarios. Immediately after collection, the temperature of each sample was systematically assessed following sterility protocols established to prevent contamination of study samples (Carson & Dent, 2007). All samples were collected during the same lunch period, deidentified, packed in dry ice, and shipped overnight to an independent laboratory for analysis. By deidentifying all samples the laboratory was blinded to restaurant groupings.

Food safety was determined by laboratory analysis of each individual food sample obtained from each study restaurant as follows. Samples that included chicken were tested for the presence of *Salmonella* and *Campylobacter*. Samples that included beef products were tested for *E. coli* O157 and *Clostridium perfringens*. Foods that contained rice and pasta were also tested for *Bacillus cereus*. Any meats that were possibly cooled and stored (e.g., chicken salad) were also tested for *Listeria*. Lastly, high protein foods that were likely to have been handled by hands during preparation (e.g., chicken salad, hamburgers, meatloaf, etc.) were tested for *S. aureus*. Due to the increased likelihood of the development of staphylococcal enterotoxins as *S. aureus* increases, higher levels of *S. aureus* are associated with greater human health risk (FDA, 2011a). In our study, any samples that contained *S. aureus* were also tested for staphylococcal enterotoxins.

Food samples were aseptically sampled and tested by the laboratory using approved scientific protocols. The following microbiological methods were used to test for the presence of pathogens: FDA-BAM Ch. 14 (*B. cereus*), ISO 16140 (*Campylobacter*), AOAC 976.30 (*C. perfringens*), AOAC RI 060903 (*E. coli* O157), AOAC RI 020901 (*Salmonella),* AOAC 975.55 (*S. aureus*), AOAC 070404 (staphylococcal enterotoxins), and AOAC 2004.02 (*Listeria monocytogenes*). Results of pathogen analyses were reported as negative or positive per 25 grams with the exception of *S. aureus*, which were reported as CFU/g. The laboratory issued a certificate of analysis upon completion of the testing. The study protocol was deemed exempt by our university institutional review board for not focusing on human subjects; nevertheless, all food samples, laboratory reports, and study findings were deidentified by name and location of restaurant.

Variables Collected

We collected the following variables for analysis: type of restaurant (e.g., American, fast food, Asian, or Mediterranean); type of food collected (e.g., hot-served chicken, cold-served chicken salad, hamburger, steak, hot dog, meatballs, meatloaf, sausage, rice, pasta, or mashed potatoes); food sample temperature; and total pathogen count for each pathogen present.

Statistical Methods

Descriptive statistical analyses were conducted to examine variable distributions. Chi squared or Fisher's exact tests indicated if differences existed between the two groups of restaurants with respect to presence of bacterial pathogens, food temperature, and whether foods were served at temperatures recommended by the Food and Drug Administration (FDA) (U.S. Department of Health and Human Services, 2009). FDA's 2009 *Food Code* recommends temperatures of ≥135°F for foods that are served hot and ≤41°F for foods that are served cold. Lastly, logistic regression analyses were used to examine the presence of any pathogens as it relates to restaurant and food characteristics. All analyses were computed in STATA version 11 and statistical significance was considered at the .05 level.

Results

Of the 42 restaurants sampled, 40.5% (n = 17) served American food, 23.8% (n = 10) served fast food, 19% (n = 8) served Asian foods, and 16.7% (n = 7) served Mediterranean foods (Table 1). A total of 42 primary food samples were collected and included such items as chicken, hamburgers, steaks, hot dogs, meatball dishes, sausages, meatloaf, rice, pasta, or mashed potatoes (Table 1).

Laboratory analyses indicated that 35.7% of the samples (n = 15) had detectable levels of *S. aureus*. Two of the 15 samples (4.8%) had *S. aureus* levels >10 CFU/g, indicating a greater potential for human health risk; one was chicken salad (70 CFU/g) and one was a hot dog (30 CFU/g). Both of these samples were tested for staphylococcal enterotoxin, but at the time of testing the colony had not produced toxins. One hundred percent of the chicken salad samples (n = 5) tested positive for *S. aureus*. Additionally, *S. aureus* was found in 100% of the hot dogs (n = 2), 100% of the meatloaf (n = 1), 62.5% of hamburger samples (n = 8), 50% of sausages (n = 2), and 5% of chicken (n = 20). None of the other bacterial pathogens (*E. coli* O157, *C. perfringens*, *Campylobacter*, *Salmonella*, *Listeria*, or *B. cereus*) were found in any of the tested samples.

No difference occurred between the percentage of Group A and Group B restaurants that contained *S. aureus* (33.3% vs. 38.1%, p = .75) (see Table 2). Additionally, Group A and Group B restaurants were not significantly different in regard to whether hot foods (57.9% vs. 55.6%, p = .89) or cold food items (50% vs. 66.7%, p = .71) were delivered at the recommended temperature. Moreover, restaurants that served foods outside of the recommended temperature were not associated with food samples containing *S. aureus* (p = .35).

A total of 42.9% (n = 18) of the 42 primary food samples were delivered at temperatures measuring below the recommended hot temperature (135°F) or above the recommended cold temperature (41°F) (see Table 3). Hot foods ranged from 84.9°F to 193°F with an average temperature of 142.6°F. Cold foods ranged from 36.9°F to 74.8°F with an average temperature of 49.1°F.

Regression analyses modeling the relationship between the outcome of detectable *S. aureus* as it relates to cuisine, food type, and recommended temperature found no significant differences.

Discussion

The key findings of our study are that no difference occurred in bacterial pathogen content or food temperatures between the restaurants in our two groups. These findings provide encouraging evidence regarding the public health restaurant inspection program, yet they also highlight ongoing challenges in restaurant food safety. While the overall findings suggest that the JCDH's current inspection program seems to be working, our findings also identify areas that may need more attention, including improved hand washing, safe holding temperatures, and ensuring timely food safety training to address risks associated with employee turnover.

Jefferson County follows the guidelines provided in the 2005 *Food Code* supplement, which prohibits bare hand contact with exposed, ready-to-eat food (U.S. Department of Health and Human Services, 2005). Also, when restaurant inspectors identify critical violations, restaurants are often required to complete a plan for remediating the problematic practices. Inspectors may also conduct repeat visits to ensure that such restaurants can demonstrate correct food safety practices, thereby increasing the likelihood that these restaurants provide foods equally as safe as restaurants without critical violations. Having found no difference in microbial colonization in the food samples from the matched cohorts in our study on the day of food collection, both cohorts provided foods that were equally safe.

Nevertheless, our study also indicated that several types of foods, most of which require extensive human hand contact to prepare, were contaminated with *S. aureus* regardless of restaurant group. The lack of a difference between groups may be due to the fact that poor hand washing and hygiene practices are difficult to identify during inspections, and as such, critical violations are often not directly related to these issues (Kassa et al., 2010). Consistent with the Hawthorne effect, food workers are more likely to practice good hand washing in the presence of inspectors (Kohli et al., 2009).

Despite not knowing the true incidence of illness caused by *S. aureus* (FDA, 2011a), the presence of *S. aureus* in 36% of food samples collected in our study suggests that it may be common in real-world food samples. Further, as *S. aureus* presence is related to poor hand washing and hygienic practices, this finding draws attention to the widespread need for improved emphasis and training on the importance of hygiene (Food Doctors, 2011; Franco, Hsu, & Simonne, 2010; Le Loir, Baron, & Gautier, 2003). In an effort to understand how to improve hand washing, previous researchers conducted a focus-group study with 11 groups of food service workers across five states (Green & Selman, 2005). They assessed perceptions on kitchen practices and foodborne risks and found that management emphasis and negative consequences were identified as facilitators to improving hand washing. Additionally, a recent study by Chapman and co-authors found that food safety info sheets (designed to initiate dialogue among food handlers) led to significant improvements in hand washing attempts (Chapman et al., 2010). Future research should examine the application of these interventions on hand washing in diverse "real-world" restaurant settings.

Though none of the 15 samples positive for *S. aureus* had levels above FDA's accept-

TABLE **1**

Sample Characteristics

Characteristic	# Sampled (%)
Restaurant types	
American	17 (40.5)
Fast food	10 (23.8)
Asian	8 (19.0)
Mediterranean	7 (16.7)
Total restaurants	42 (100)
Foods sampled	
Primary samples	
Hot-served chicken	20 (47.6)
Hamburger	8 (19.0)
Cold chicken salad	5 (11.9)
Steak	2 (4.8)
Hot dog	2 (4.8)
Meatball dish	2 (4.8)
Sausage	2 (4.8)
Meatloaf	1 (2.3)
Total primary samples	42 (100)
Additional samples	
Rice	13 (86.6)
Pasta	1 (6.7)
Mashed potatoes	1 (6.7)
Total additional samples	15 (100)

TABLE **2**

Presence of *Staphylococcus aureus* (SA) and Food Temperature by Restaurant Group

Parameter Tested	Group A Restaurants	Group B Restaurants	*p*-Value
Presence of SA			
% Samples with any level of SA	33.3	38.1	.75
% Samples with SA levels >10 CFU/g	4.8	4.8	1.0
Food temperature			
% Hot samples delivered at recommended temperatures	57.9	55.6	.89
% Cold samples delivered at recommended temperatures	50	66.7	.71

Note. Routine laboratory tests are sensitive to SA levels at 10 CFU/g. Group A includes those restaurants selected for our study that consistently lost points for critical violations in repeat food safety inspections. Group B refers to the cohort of restaurants that lost no points for critical violations during the two-year study period.

able level of 1,000 CFU/g, it is important to draw attention to the potential human health risk introduced by the mere presence of *S. aureus* in foods. First, small populations of *S. aureus* at the time of testing could be remnants of larger populations destroyed after cooking that were able to produce enterotoxins (which are not deactivated by heat); therefore, small populations at the time of testing are not necessarily an indication of a safe food (FDA, 2001). Second, as indicated in the following example, poor hygiene combined with the right conditions for *S. aureus* growth can create the potential for food poisoning. In an *S. aureus* outbreak involving chicken salad, 1,364 children suffered from foodborne illness (FDA, 2011a). Poor hygiene practices led to the contamination of *S. aureus* in the chicken deboning process, though improper cooling and holding temperatures created the conditions for *S. aureus* to grow. Once present, *S. aureus* colonies can grow when food is not held above 140°F or below 45°F (FDA, 2011a). Increased *S. aureus* leads to an increased likelihood of staphylococcal enterotoxin production, which causes vomiting and diarrhea in humans (Franco et al., 2010; Le Loir et al., 2003; Mead et al., 1999; FDA, 2011a). Staphylococcal enterotoxins are produced at temperatures ranging from 57.2°F to 111.2°F and once present cannot be inactivated by cooking or reheating (FDA, 2011a; Schmitt, Schuler-Schmid, & Schmidt-Lorenz, 1990). Since temperature is a key factor in the growth of bacteria in food and given that 45.2% of foods collected in our study were not served at the recommended temperature, greater attention should be given to ensuring safe holding and cooling practices in addition to improved food handling practices.

Because illness caused by *S. aureus* enterotoxins occurs relatively acutely (lasting 24–48 hours), people often do not to seek medical care and when they do the lack of laboratory confirmation complicates the ability to know the true incidence of cases (FDA, 2011a; Mead et al., 1999). Despite this, preliminary data presented herein reminds us of the ongoing need to address hygiene and hand washing practices throughout the restaurant industry. Improved hygienic practices would also impact the occurrence of norovirus in foods served to the public. Although laboratory testing for norovirus was unavailable for our study, previous research indicates that it has been the cause of 47% of laboratory-confirmed, outbreak-associated illnesses (Jones & Angulo, 2006). While recent headlines have focused on large-scale outbreaks stemming from problems in large-scale animal and farming practices, poor hand washing causes illness in proportionally more Americans and is often underemphasized despite being remediable (Hutchinson, 2010; Neuman, 2010).

Strengths and Limitations

To our knowledge, this is the first study to examine bacterial pathogens in food samples as an indication of risks to human health. Studies of this nature may be limited partly due to the costs associated with the laboratory testing of food samples. Even though this preliminary study examined a relatively small sample of restaurants, it included a diverse group of establishments and tested a wide variety of foods, adding to the representativeness of its findings. Another strength of our study design is that it was conducted in a real-world scenario, providing a reasonable assessment of the state of foods served to the public. Foods analyzed in our study were packed in dry ice immediately upon being collected; thus, little time elapsed for foods to be held at room temperature before being tested for pathogens. Since foods that are held at room temperatures have a greater likelihood of bacterial growth, the threat to human health becomes more apparent the longer foods are outside recommended temperatures. It is conceivable that many customers may not immediately consume their purchased food and in such instances the threat to human health may in fact be greater than indicated by our study. Finally, laboratories available for testing were unable to examine samples for the presence of viruses, including norovirus.

TABLE **3**

Food Temperatures by Type of Food

Type of Food	Delivered at Recommended Temperatures	Delivered at Nonrecommended Temperatures
Hot foods	**% ≥135°F**	**% <135°F**
Hot dogs (*n* = 2)	100	0
Meatloaf (*n* = 1)	100	0
Chicken (*n* = 20)	70	30
Sausage (*n* = 2)	50	50
Hamburgers (*n* = 8)	37.5	62.5
Meatball dish (*n* = 2)	0	100
Steak (*n* = 2)	0	100
Total hot foods	54.1	45.9
Cold foods	**% ≤41°F**	**% >41°F**
Chicken salad (*n* = 5)	60	40
Total all foods	**57.1**	**42.9**

Conclusion

While the current system seems to have strengths in preventing foodborne illness, both groups of restaurants had issues with bacterial contamination, suggesting that room for improvement exists. Further, even though the true incidence of foodborne illness caused by *S. aureus* toxins is unknown (FDA, 2011a), its presence in over a third of food samples collected in our study suggests that it may be common in real-world food samples and draws attention to the importance of proper hand hygiene and hot and cold holding temperatures. Future research should examine restaurant characteristics associated with critical violations related to poor hygiene, the lack of hand washing, and noncompliance with holding temperatures to better inform inspection and educational practices. Perhaps educational programs can be most effective if targeted to restaurants documented to have greater likelihood of such violations.

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References

Carson, P.A., & Dent, N. (Eds.). (2007). *Good clinical, laboratory, and manufacturing practices*. Cambridge, UK: The Royal Society of Chemistry.

Cates, S.C., Muth, M.K., Karns, S.A., Penne, M.A., Stone, C.N., Harrison, J.E., & Radke, V.J. (2009). Certified kitchen managers: Do they improve restaurant inspection outcomes? *Journal of Food Protection, 72*(2), 384–391.

Centers for Disease Control and Prevention. (2006). Surveillance for foodborne-disease outbreaks—United States, 1998–2002. *Morbidity Mortality Weekly Report, 55*(SS10), 1–34.

Centers for Disease Control and Prevention. (2009). Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food—10 States, 2008. *Morbidity Mortality Weekly Report, 58*(13), 333–337.

Chapman, B., Eversley, T., Fillion, K., Maclaurin, T., & Powell, D. (2010). Assessment of food safety practices of food service food handlers (risk assessment data): Testing a communication intervention (evaluation of tools). *Journal of Food Protection, 73*(6), 1101–1107.

Creel, J.S., Sharkey, J.R., McIntosh, A., Anding, J., & Huber, J.C., Jr. (2008). Availability of healthier options in traditional and nontraditional rural fast-food outlets. *BMC Public Health, 8*, 395.

Food and Drug Administration. (2001). *Bacteriological analytical manual*. Retrieved from http://www.fda.gov/Food/ScienceResearch/

References

LaboratoryMethods/BacteriologicalAnalyticalManualBAM/ucm071429.htm

Food and Drug Administration. (2004). *Produce safety from production to consumption: 2004 action plan to minimize foodborne illness associated with fresh produce consumption*. Retrieved from http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/FruitsVegetablesJuices/FDAProduceSafetyActivities/ProduceSafetyActionPlan/ucm129487.htm

Food and Drug Administration. (2011a). *Bad bug book: Foodborne pathogenic microorganisms and natural toxins handbook*. Retrieved from http://www.fda.gov/Food/FoodSafety/FoodborneIllness/FoodborneIllnessFoodbornePathogensNaturalToxins/BadBugBook/default.htm

Food and Drug Administration. (2011b). *What you need to know about foodborne illness–causing organisms in the U.S*. Retrieved from http://www.fda.gov/Food/ResourcesForYou/Consumers/ucm103263.htm

Food and Drug Administration Retail Food Program Steering Committee. (2000). *Report of the Food and Drug Administration retail food program database of foodborne illness risk factors*. Retrieved from http://www.fda.gov/downloads/Food/FoodSafety/RetailFoodProtection/FoodborneIllnessandRiskFactorReduction/RetailFoodRiskFactorStudies/ucm123546.pdf

Food Doctors. (2011, April 8) *The food safety file:* Staphylococcus aureus. Retrieved from www.fooddoctors.com/FSF/S_aureus.pdf

Franco, W., Hsu, W.Y., & Simonne, A.H. (2010). Survival of *Salmonella* and *Staphylococcus aureus* in Mexican red salsa in a food service setting. *Journal of Food Protection, 73*(6), 1116–1120.

Green, L.R., & Selman, C. (2005). Factors impacting food workers' and managers' safe food preparation practices: A qualitative study. *Food Protection Trends, 25*(12), 981–990.

Hutchinson, C. (2010, August 23). Cracks in the system: Egg recall reveals FDA flaws. *ABC News*. Retrieved from http://abcnews.go.com/Health/Wellness/salmonella-outbreak-egg-recall-shows-fda-flaws/story?id=11445664

Irwin, K., Ballard, J., Grendon, J., & Kobayashi, J. (1989). Results of routine restaurant inspections can predict outbreaks of foodborne illness: The Seattle-King County experience. *American Journal of Public Health, 79*(5), 586–590.

Jin, G.Z., & Leslie, P. (2003). The effect of information on product quality: Evidence from restaurant inspection grade cards. *The Quarterly Journal of Economics, 118*(2), 409–451.

Jones, T.F., & Angulo, F.J. (2006). Eating in restaurants: A risk factor for foodborne disease? *Clinical Infectious Diseases, 43*(10), 1324–1328.

Jones, T.F., Pavlin, B.I., LaFleur, B.J., Ingram, L.A., & Schaffner, W. (2004). Restaurant inspection scores and foodborne disease. *Emerging Infectious Diseases, 10*(4), 688–692.

Kassa, H., Silverman, G.S., & Baroudi, K. (2010). Effect of a manager training and certification program on food safety and hygiene in food service operations. *Environmental Health Insights, 4*, 13–20.

Klein, S., & DeWall, C.S. (2008). *Dirty dining: Have reservations? You will now*. Washington, DC: Center for Science in the Public Interest. Retrieved from http://cspinet.org/new/pdf/ddreport.pdf

Kohli, E., Ptak, J., Smith, R., Taylor, E., Talbot, E.A., & Kirkland, K.B. (2009). Variability in the Hawthorne effect with regard to hand hygiene performance in high- and low-performing inpatient care units. *Infection Control and Hospital Epidemiology, 30*(3), 222–225.

Lee, J.E., Almanza, B.A., Nelson, D.C., & Ghiselli, R.F. (2009). Using health inspection scores to assess risk in food services. *Journal of Environmental Health, 71*(7), 28–33.

Le Loir, Y., Baron, F., & Gautier, M. (2003). *Staphylococcus aureus* and food poisoning. *Genetics and Molecular Research, 2*(1), 63–76.

Mead, P.S., Slutsker, L., Dietz, V., McCaig, L.F., Bresee, J.S., Shapiro, C., Griffin, P.M., & Tauxe, R.V. (1999). Food-related illness and death in the United States. *Emerging Infectious Diseases, 5*(5), 607–625.

Neuman, W. (2010, May 8). Tainted lettuce linked to illness in three states. *The New York Times*, p. A10. Retrieved from http://www.nytimes.com/2010/05/08/us/08lettuce.html

Nyachuba, D.G. (2010). Foodborne illness: Is it on the rise? *Nutrition Review, 68*(5), 257–269.

Phillips, M.L., Elledge, B.L., Basara, H.G., Lynch, R.A., & Boatright, D.T. (2006). Recurrent critical violations of the food code in retail food service establishments. *Journal of Environmental Health, 68*(10), 24–30, 55.

Reske, K.A., Jenkins, T., Fernandez, C., VanAmber, D., & Hedberg, C.W. (2007). Beneficial effects of implementing an announced restaurant inspection program. *Journal of Environmental Health, 69*(9), 27–34, 76.

Roberts, T. (2007). WTP estimates of the societal costs of U.S. foodborne illness. *American Journal of Agricultural Economics, 89*(5), 1183–1188.

Scallan, E., Hoekstra, R.M., Angulo, F.J., Tauxe, R.V., Widdowson, M.A., Roy, S.L., Jones J.L., & Griffin, P.M. (2011). Foodborne illness acquired in the United States—major pathogens. *Emerging Infectious Diseases, 17*(1), 7–15.

Scallan, E., Jones, T.F., Cronquist, A., Thomas, S., Frenzen, P., Hoefer, D., Medus, C., Angulo, F.J., & FoodNet Working Group. (2006). Factors associated with seeking medical care and submitting a stool sample in estimating the burden of foodborne illness. *Foodborne Pathogen Disease, 3*(4), 432–438.

Schmitt, M., Schuler-Schmid, U., & Schmidt-Lorenz, W. (1990). Temperature limits of growth, TNase and enterotoxin production of *Staphylococcus aureus* strains isolated from foods. *International Journal of Food Microbiology, 11*(1), 1–19.

U.S. Department of Health and Human Services. (2005). *Food code 2005*. Retrieved from http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/Foodcode/FoodCode2005/default.htm

U.S. Department of Health and Human Services. (2009). *Food code 2009*. Retrieved from http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/FoodCode2009/default.htm

INTERNATIONAL PERSPECTIVES

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Common Phenotypic and Genotypic Antimicrobial Resistance Patterns Found in a Case Study of Multiresistant *E. coli* From Cohabitant Pets, Humans, and Household Surfaces

Although most of the information presented in the Journal *refers to situations within the United States, environmental health and protection know no boundaries. The* Journal *periodically runs International Perspectives to ensure that issues relevant to our international membership, representing over 20 countries worldwide, are addressed. Our goal is to raise diverse issues of interest to all our readers, irrespective of origin.*

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Abstract **The objective of the study described in this article was to characterize the antimicrobial resistance profiles among *E. coli* strains isolated from cohabitant pets and humans, evaluating the concurrent colonization of pets, owners, and home surfaces by bacteria carrying the same antimicrobial-resistant genes. The authors also intended to assess whether household surfaces and objects could contribute to the within-household antimicrobial-resistant gene diffusion between human and animal cohabitants. A total of 124 *E. coli* strains were isolated displaying 24 different phenotypic patterns with a remarkable percentage of multiresistant ones. The same resistance patterns were isolated from the dog's urine, mouth, the laundry floor, the refrigerator door, and the dog's food bowl. Some other multiresistant phenotypes, as long as resistant genes, were found repeatedly in different inhabitants and surfaces of the house. Direct, close contact between all the cohabitants and the touch of contaminated household surfaces and objects could be an explanation for these observations.**

Introduction

The use of antimicrobial drugs induces the increase of antimicrobial resistance, not just in the pathogenic bacteria but also in the endogenous commensal flora (Berge, Moore, & Sischo, 2006; Costa et al., 2008a; Dancer, 2004; Goossens, 2009; Goossens, Ferech, Stichele, & Elseviers, 2005; van den Bogaard & Stobberingh, 2000). During the last decade, an awareness has been increasing of the potential problems that selection for antimicrobially resistant bacteria among companion animals may cause on human health, due to the increasing utilization of the same antimicrobial substances in human medicine and to the close contact between pets and their human cohabitants (Guardabassi, Loeber, & Jacobson, 2004; Guardabassi, Schwarz, & Lloyd, 2004; Moyaert, de Graef, Haesebrouck, & Decostere, 2006; Schwarz, Kehrenberg, & Walsh, 2001). The growing number of household pets and their increasing health care standards has led to an augmented number of geriatric animals accompanied by extensive medical histories including antimicrobial drug administration and longer contact with owners increasing both the risk of antimicrobial resistance emergence and interspecies clonal spread.

The spread of antimicrobial-resistant bacteria can occur directly, by skin-to-skin contact and contact with bacteria-containing material (e.g. saliva, feces), or indirectly via the household environment (Guardabassi, Loeber, & Jacobson, 2004; Schwarz et al., 2001). When reaching the new host, resistant bacteria can either colonize and infect, or remain in that particular environment for only a very short period of time. During this period, the resistant bacteria can not only spread their resistance genes to other bacteria residing in the new host (commensals or pathogens), but also accept resistance genes from other bacteria (Livermore, 2003; Schwarz et al., 2001).

E. coli has a great ecological value in the assessment of resistance spreading not only because it plays an important role as acceptor and donor of transmissible drug-

TABLE **1**

Primers Used for Detection of Genes Encoding Antimicrobial Resistance in *E. coli* Isolates

Target Gene	Primer	Nucleotide Sequence (5'–3')	Size (Base Pair)	Reference
ampC	ampC-F	CCCCGCTTATAGAGCAACAA	634	Mendonça et al. (2007)
	ampC-R	TCAATGGTCGACTTCACACC		
bla_{TEM}	TEM-F	ATTCTTGAAGACGAAAGGGC	1150	Costa et al. (2008a); Sáenz et al. (2004)
	TEM-R	ACGCTCAGTGGAACGAAAAC		
bla_{OXA}	OXA1F	ACACAATACATATCAACTTCGC	813	Costa et al. (2008a); Sáenz et al. (2004)
	OXA1R	AGTGTGTTTAGAATGGTGATC		
bla_{SHV}	SHV-F	CACTCAAGGATGTATTGTG	885	Costa et al. (2008a); Sáenz et al. (2004)
	SHV-R	TTAGCGTTGCCAGTGCTCG		
bla_{CTX-M}	CTX-F	TTTGCGATGTGCAGTACCAGTAA	543	Mendonça et al. (2007)
	CTX-R	CGATATCGTTGGTGGTGCCATA		
$bla_{CTX-M-15}$	CTX15F	AGAATAAGGAATCCCATGGTT	875	Mendonça et al. (2007)
	CTX15R	ACCGTCGGTGACGATTTTAG		
aadA	AadA-F	GCAGCGCAATGACATTCTTG	282	Sáenz et al. (2004)
	AadA-R	ATCCTTCGGCGCGATTTTG		
strA	StrA-F	CTTGGTGATAACGGCAATTC	548	Srinivasan et al. (2007)
	StrA-R	CCAATCGCAGATAGAAGGC		
strB	StraB-F	ATCGTCAAGGGATTGAAACC	509	Srinivasan et al. (2007)
	StraB-R	GGATCGTAGAACATATTGGC		
gyrA	GyrA-F	TACACCGGTCAACATTGAGG	648	Costa et al. (2008a)
	GyrA-R	TTAATGATTGCCGCCGTCGG		
parC	ParC-F	AAACCTGTTCAGCGCCGCATT	395	Costa et al. (2008a)
	ParC-R	GTGGTGCCGTTAAGCAAA		
tetA	TetA-F	GTAATTCTGAGCACTGTCGC	937	Costa et al. (2008a)
	TetA-R	CTGTCCTGGACAACATTGCTT		
tetB	TetB-F	CTCAGTATTCCAAGCCTTTG	416	Costa et al. (2008a)
	TetB-R	CTAAGCACTTGTCTCCTGTT		
cml	CML-F	CCGCCACGGTGTTGTTGTTATC	698	Sidjabat et al. (2006)
	CML-R	CACCTTGCCTGCCCATCATTAG		
flo	FLO-F	TATCTCCCTGTCGTTCCAG	399	Sidjabat et al. (2006)
	FLO-R	AGAACTCGCCGATCAATG		
catA	M62822	AGTTGCTCAATGTACCTATAACC	547	Maynard et al. (2007)
	M62822	TTGTAATTCATTAAGCATTCTGCC		
sul1	Sul1-F	TGGTGACGGTGTTCGGCATTC	789	Costa et al. (2008a); Sáenz et al. (2004)
	Sul1-R	GCGAGGGTTTCCGAGAAGGTG		
sul2	Sul2-F	CGGCATCGTCAACATAACC	722	Costa et al. (2008a); Sáenz et al. (2004)
	Sul2-R	GTGTGCGGATGAAGTCAG		

resistant genes transferable to pathogenic bacteria (Sáenz et al., 2004; van den Bogaard & Stobberingh, 2000), but also because it is commonly found in the intestinal tract of humans and animals (Costa et al., 2008a). *E. coli* can also be implicated in various intestinal and extraintestinal diseases (Johnson, Owens, Gajewski, & Clabots, 2008; Johnson, Stell, & Delavari, 2001). Usually the host's own fecal flora is the immediate source of the extraintestinal pathogenic *E. coli* strains. The external reservoirs from which the hosts initially acquire such strains and the relevant transmission mechanisms, however, are poorly understood (Johnson et al., 2008).

The aim of our study was to characterize phenotypically and genetically the antimicrobial resistance profiles among *E. coli* strains isolated from cohabitant pets and humans, considering the concurrent colonization of pets, owners, and home surfaces by bacteria with the same resistance patterns and carrying the same antimicrobial-resistant genes.

Methods

Enrollment and Sampling

Case selection emerged from the universe of clients of the Institute of Biomedical Sciences Abel Salazar Companion Animals Veterinary Clinic (Porto University, Portugal). The participants were chosen taking into account that both the man and the dog had already been administered several antimicrobial treatments and that the dog was recently diagnosed with a recurrent urinary tract infection. A formal consent was signed and a complete questionnaire, including environment, human and veterinary medical records with antibiotic usage by themselves, family members, and their pets was completed.

A dog's oral swab and cystocentesis for urine collection were carried out immediately. Cystocentesis was performed by aseptic technique: prepubic hair was clipped and the skin was cleaned and disinfected with alcohol and clorhexidine before the insertion of a needle connected to a 10 mL syringe to collect urine directly from the dog's bladder. Fecal samples of the two adults (male and female owners), of their two-year-old grandchild (daily cohabitant), and of the household cat and dog were delivered the next morning.

Simultaneously, the following household environmental swabs were collected: two from light switches, one from the refrigerator door handle, two from door knobs, two from the dog's food and water bowls, one from the laundry floor, and one from each owner's hands.

E. coli Isolation

After reception at the laboratory, fecal samples were immediately diluted 1:10 in saline buffer and stored at room temperature for one hour. From this initial suspension, an aliquot of

TABLE **2**

Number of Antimicrobial Resistance Patterns in *E. coli* Isolates From Pets, Human Cohabitants, and Household Environment

Antimicrobial[a] Resistance Pattern	Pets				Human Cohabitants			Household Environment		
	Dog Feces	Dog Urine	Dog Mouth	Cat Feces	Male Feces	Female Feces	Child Feces	Laundry Floor	Refrig-erator Door	Dog Bowl
AMP AMC ATM CEF CAZ GEN STR TOB KAN CIP NAL TET CHL SXT	1									4
AMP ATM CEF CAZ GEN STR TOB KAN CIP NAL TET CHL SXT	19		5							
AMP ATM CEF CAZ STR CIP NAL TET CHL		6	4					2	1	4
None	8		6	4	3	3	2			
STR KAN NAL TET				3						
AMP ATM CEF CAZ				4						
AMP ATM CEF				2						
AMP ATM CEF CAZ CTX GEN STR KAN TET CHL SXT					1					
AMP ATM CEF CAZ CTX STR TOB TET CHL STX					5					
AMP CEF CTX STR KAN TET CHL SXT					1					
CEF KAN CIP NAL TET CHL SXT					11					
KAN CIP NAL TET CHL SXT						6				
AMP GEN TOB CIP NAL						1				
AMP STR TET SXT						2				
STR CIP TET SXT						1				
TET						1				
AMP ATM CEF STR KAN TET							1			
AMP STR KAN TET SXT							3			
AMP SRT KAN TET							3			
AMP STR NAL SXT							1			
TET SXT							1			
AMP AMC ATM CEF GEN SRT TOB KAN AMK TET CIP NAL CHL SXT										1
AMP ATM CEF GEN STR TOB KAN CIP NAL CHL SXT										1
AMP AMC ATM CEF CAZ STR CIP NAL TET CHL								2	1	

[a]Abbreviations: Ampicillin (AMP), amoxicillin-clavulanic acid (AMC), aztreonam (ATM), cephalothin (CEF), ceftazidime (CAZ), cefotaxime (CTX), gentamicin (GEN), amikacin (AMK), streptomycin (STR), tobramycin (TOB), kanamycin (KAN), ciprofloxacin (CIP), nalidixic acid (NAL), tetracycline (TET), chloramphenicol (CHL), and trimethoprim-sulfamethoxazol (SXT).

1 µL was streaked on Chromocult tryptone bile X-glucuronide (TBX) agar and 100 µL were spread on the same culture media containing cefotaxime (2 µg/mL). Oral swabs from the dog and house environmental swabs were put on buffered peptone water (BPW). After one hour at room temperature, unsupplemented and cefotaxime-supplemented TBX agar plates were inoculated with 100 µL from each sample. The urine was employed directly with 1 µL streaked on TBX agar and 100 µL spread on TBX containing cefotaxime at the same concentration.

Plates were incubated overnight at 37°C. Five colonies with the typical appearance of *E. coli* were selected from each plate and all colonies presenting different morphologies were additionally picked. Standard biochemical methods were used for the confirmation of *E. coli* isolates (Berge et al., 2006). The present procedure was adapted from standard protocols (Costa et al., 2008a; Simões, Poirel, Costa, & Nordmann, 2010) used in related studies as long as it is performed for getting the most reliable and accurate *E. coli* detection.

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility was performed using disk diffusion assay, following Clinical and Laboratory Standards Institute (CLSI, 2007) guidelines. Briefly, fresh bacterial colonies were inoculated on BPW suspension to a turbidity equivalent to 0.5 McFarland standard. With a sterile cotton swab the culture was swabbed on 150 mm depth Mueller-Hinton agar plates and standard discs (Oxoid antimicrobial susceptibility test discs) were applied using a disk dispenser. A total of 19 antimicrobial agents were tested: ampicillin, amoxicillin-clavulanic acid, aztreonam, cephalothin, ceftazidime, cefotaxime, cefoxitin, imipenem, gentamicin, amikacin, streptomycin, tobramycin, kanamycin, ciprofloxacin, nalidixic acid, tetracycline, chloramphenicol, trimethoprim-sulfamethoxazol, and nitrofurantoin.

The antimicrobials were selected because they, or their related antimicrobials, had been regularly used in both human and veterinary medicine and to provide diversity in representation of different antimicrobial classes (Elseviers, Ferech, Vander Stichele, Goossens, & ESAC Project Group, 2007; Goossens et al., 2005). The reference strain *E. coli* American Type Culture Collection 25922 was included as a control. After 10 hours of incubation at 37ºC, the diameters of the inhibition zones were measured using a caliper rounded up to the next millimeter and recorded. The interpretation of the inhibition zone length was made according to CLSI recommendations and breakpoints for Enterobacteriaceae.

According to several related studies (Costa et al., 2008a; Simões et al., 2010) quantitative analysis of antimicrobial resistance data was performed through a few basic descriptive statistic measures.

Polymerase Chain Reaction Amplification of Antimicrobial-Resistant Genes

Characterization of antimicrobial-resistant genes was performed in all strains displaying different antimicrobial resistance phenotypic patterns and strains with similar resistance patterns but isolated from different sources (humans, pets, or household environment). Bacteria were subcultured from glycerol stored cultures on tryptone soy agar medium overnight and DNA was extracted. Genomic DNA was extracted *in situ* by treatment with lysozyme (1 mg/mL) and proteinase K (0.5 mg/mL).

Genes for testing were selected taking into consideration the groups of antimicrobial drugs represented in the resistance phenotypes. Primers sequences and predicted sizes employed for polymerase chain reaction (PCR) amplification of the different antimicrobial-resistant genes are presented in Table 1 (Costa et al., 2008a; Costa et al., 2008b; Eckert et al., 2004; Mendonça, Leitão, Manageiro, Ferreira, & Caniça, 2007; Sáenz et al., 2004; Sidjabat et al., 2006; Srinivasan et al., 2007). In β-lactam resistant phenotypes, the presence of *amp*C, bla_{TEM}, bla_{OXA}, bla_{SHV}, bla_{CTX-M}, and $bla_{CTX-M-15}$ genes was studied. For aminoglycosides-resistant isolates, genes *str*A, *str*B, and *aad*A were investigated whereas phenotypes resistant to quinolones were explored for *gyr*A and *par*C genes. Tetracycline-resistant phenotypes were examined for *tet*A and *tet*B genes while the *cml*, *flo*, and *cat*A genes were explored in isolates showing resistance to phenicols. Genes *sul*1 and *sul*2 were scrutinized in isolates that showed resistance to trimethoprim-sulfamethoxazole.

Antimicrobial-resistant gene primers were obtained from previous studies (Costa et al., 2008a; Costa et al., 2008b; Eckert et al., 2004; Mendonça et al., 2007; Sáenz et al., 2004; Sidjabat et al., 2006; Srinivasan et al., 2007), as well as the amplification protocol. Primer sets were synthesized by Stab Vida. Amplification was performed in a DNA thermal cycler with 46-well PCR plates of 0.5 mL. The Taq polymerase kit used was from Fermentas Life Sciences. The reaction mixture consisted of 30 µL of sterile water; 5 µL of 10X Taq Buffer (100 mM Tris–HCl [pH 8.8 at 25ºC], 500 mM KCl, 0.8% Nonidet P40); 1.5 µL of 25 mM $MgCl_2$; 1 µL of deoxyribonucleoside triphosphates (2 mM each dATP, dCTP, dTTP, and dGTP); 0.5 µL of each primer (stock concentration, 40 µM); 10 µL of template; and 0.2 µL (5 U/µl) of TaqDNA polymerase. Preincubation was at 94ºC for five minutes.

Thirty PCR cycles were run under the following conditions: denaturation at 94ºC for 30 seconds, primer annealing at optimum temperature for 30 seconds, and DNA extension at 72ºC for 30 seconds in each cycle. After the last cycle, PCR tubes were incubated for seven minutes at 72ºC and held at 4ºC. The annealing temperature was optimized for all primer sets. *Salmonella typhi* G8518 CDC (ACCuST) and *Salmonella typhimurium* DT104 and DT193 were used as positive controls in all PCR reactions. With the exception of template DNA, sterile distilled water was used as the reagent control in the reaction mixture. The reaction mixture (20 µL) was analyzed by standard submarine gel electrophoresis (1.5% agarose; 5 V), and visualized by staining with ethidium bromide (0.5 mg/mL in the running buffer).

Results

The questionnaire submitted to the owners about themselves as well as the relationship with their pets, the respective human and animal medical records, in addition to some social interaction patterns and routines resulted in some useful information. Both husband and wife were 62 years old. The woman always worked at home as a seamstress and the man had an administrative job at the central mail services. The nine-year-old dog in question had a chronic, poorly controlled, allergic skin disease with recurrent pruritus and pyoderma. To control the secondary skin infections, several antimicrobial treatments had been prescribed: amoxicillin and clavulanic acid, cephalosporin, cefovecin, enrofloxacin, and ciprofloxacin.

The medical record of the man contained also an important detail: when hospitalized after a car accident (four years ago) he contracted a urinary tract infection treated according to the hospital protocol. The hospitalization lasted around two months, nearly the time he was under antimicrobial treatment. No relevant information was detected in the medical records of the woman, the child, or their exclusively indoor 12-year-old cat. The family lived in a small central apartment with apparently a medium economic level and good hygiene habits. The dog was dominant and an active element of the family with free access to all the divisions and items within the house. It was walked throughout the city center twice a day with a leash. Both pets, living with the owners since birth, were fed with specific canned dry food and drank water from the public system while their owners drank bottled water only.

A total of 124 *E. coli* isolates were recovered from the 17 samples collected from pets, hu-

TABLE **3**

Genes of Antimicrobial Resistance Found in *E. coli* Isolates From Pets, Human Cohabitants, and Household Environment

Resistance Genes	Pets				Human Cohabitants			Household Environment		
	Dog Feces	Dog Urine	Dog Mouth	Cat Feces	Male Feces	Female Feces	Child Feces	Laundry Floor	Refrig-erator Door	Dog Bowl
ampC	+	+	+	+	+	+	+	+	+	+
bla_{TEM}		+			+	+	+			
bla_{OXA}	+	+	+				+	+	+	+
bla_{SHV}	+	+	+	+				+	+	+
bla_{CTX-M}					+					
$bla_{CTX-M-15}$					+					
strA					+	+	+			
strB					+	+	+			
aadA	+	+	+	+	+	+	+	+	+	+
gyrA	+	+	+	+	+	+	+	+	+	+
parC	+	+	+	+	+	+	+	+	+	+
tetA					+	+	+			
tetB	+	+	+	+	+			+	+	+
cmI					+	+				
flo	+		+		+					+
catA	+	+	+					+		+
sul1	+	+			+	+	+			+
sul2	+	+			+	+	+			+

mans, and household environment. The number of isolates, their location, and resistance profiles are presented in Table 2. No cultivable *E. coli* was obtained from light switches, the dog's water bowl, or owners' hands.

Antimicrobial susceptibility testing displayed 24 different phenotypic patterns with a remarkable representation of multiresistant ones. Fifty-seven isolates (46%) displayed simultaneous resistance to at least nine different antimicrobials. Six *E. coli* isolates obtained from the dog's food bowl and feces were resistant to 14 out of the 19 antimicrobials tested. A considerable proportion of the *E. coli* isolates displayed resistance to tetracycline (75%), ampicillin (64%), streptomycin and chloramphenicol (60%), nalidixic acid (59%), cephalothin (58%), trimethoprim-sulfamethoxazole (53%), kanamycin (51%), ciprofloxacin (48%), and aztreonam (47%). The percentage of resistance to the other antimicrobial agents was below 28% and no resistance against cefoxitin, imipenem, or nitrofurantoin was detected.

It is noteworthy that the same resistance phenotype that displayed simultaneous resistance against nine antimicrobials was found in samples collected from the dog (urine and mouth swab) and in household environmental samples, namely from the laundry floor, the refrigerator door, and the dog's food bowl (Table 2). The resistance pattern of some other strains isolated from the dog (feces and urine) also matched some of those found in the dog's mouth, food bowl, laundry floor, and refrigerator door.

The results of the antimicrobial-resistant gene detection using PCR are presented in Table 3. The pool of antimicrobial resistance genes encountered in isolates obtained from the dog's feces, urine, mouth, and food bowl and the owners' feces comprised resistance to all of the tested antimicrobial groups. Phenicol was the only antimicrobial group to which no resistant genes were found in isolates from the grandchild's feces. Of the resistant genes tested

for, phenicol and trimethoprim-sulfamethoxazole resistance was absent in isolates from the cat and from the refrigerator door, and trimethoprim-sulfamethoxazole resistance was absent in isolates from the laundry floor.

From a total of 18 genes tested, *E. coli* isolated from the man's feces carried a total of 15 antimicrobial-resistant genes (Table 3). Isolates from feces, urine, mouth, and food bowl of the dog held 11, 11, 9, and 11 resistant genes, respectively. Strains isolated from feces of the woman and the grandchild carried 11 resistant genes. Analysis of the isolates from the laundry floor, the refrigerator door, and from the cat's feces resulted in 8, 7, and 6 resistant genes, respectively. The woman, the grandchild, and the cat, although never subjected to antimicrobial treatments, demonstrated to have multiresistant isolates with some common resistance patterns (Table 3).

Discussion

The aim of our study was to obtain a holistic picture of the in-house *E. coli* antimicrobial resistance profiles, accounting for the contribution of the different pet and human cohabitants as well as the household surfaces and objects.

Interesting results were achieved, namely the high level of antimicrobial resistance found in the majority of the isolates attained (Table 2), which is remarkable when compared with similar studies undertaken previously (Carattoli et al., 2005; Costa et al., 2008a; Machado et al., 2007; Mendonça et al., 2007; Moreno, Bello, Guggiana, Dominguez, & Gonzalez, 2008; Normand, Gibson, Reid, Carmichael, & Taylor, 2000). The finding of a higher prevalence of antimicrobial resistance among *E. coli* strains isolated from the dog and the male owner was somewhat expected considering their history of antimicrobial treatments, including the man's hospitalization, which is known to increase the risk for acquiring, temporary or permanently, multiresistant strains (Dancer, 2004; Mendonça et al., 2007).

It is also noteworthy that strains isolated from the household environment, besides being resistant to at least nine of the tested antimicrobials, were found to have similar resistance profiles when compared to those from the home inhabitants, particularly those from the dog (Tables 2 and 3). Others have already found that the virulent human pathogen *E. coli* serotype O157, of whom cattle are the primary reservoir, remain viable in soil fecal excretion greater than four months (Jones, 1999) or in wood samples from farmyard material (Williams, Avery, Killham, & Jones, 2005).

To our knowledge, this was the first time that *E. coli* from household environment samples was analyzed and its antimicrobial-resistant determinants compared with those isolated from the household inhabitants. These findings raise questions regarding the potential contribution of shared household surfaces in antimicrobial resistance transfer between animal and human cohabitants. Finally it was established that a pet can orally transport *E. coli* strains with the same antimicrobial resistance profile of their fecal and urinary strains, which could be explained by some frequent behavior of dogs such as rolling on feces, grooming, and perigenital licking. The presence of those resistant strains in the dog's mouth is likely to have played a key role in their spread.

Commensal flora of the grandchild, the woman, and the cat have never been directly exposed to antimicrobial drugs; however, several multiresistant *E. coli* were also recovered from their stool samples and, more importantly, those strains shared most of the resistant genes found in those recovered from the dog and the man (Table 3). This finding is not so surprising for the woman since she is the man's sex partner, which is known to have a risk of acquiring their commensal *E. coli* (Johnson et al., 2008) but results in an interesting picture if we hypothesize that the cat, who never lived outside the home, acquired antimicrobial resistances to ampicillin, aztreonam, cephalothin, and ceftazidime, all frequently expressed in isolates of the dog, the man, and the household, through the normal cohabitation contacts. The same could be speculated concerning the child's antimicrobial resistance patterns.

Conclusion

Although resistance patterns are not static the genotypic and phenotypic correspondences demonstrated in this applied study could suggest interspecies transmission. Furthermore, the finding that almost all of these resistant genes were also present among strains isolated from the household environment could be indicative of an in-home and through-home transmission.

While concurrent colonization with multiresistant *E. coli* has been identified in humans and animals (Guardabassi et al., 2004a; Johnson et al., 2001; Johnson et al., 2008), our study provides further information that supports the potential contribution of the household environment as a passive source of multiresistant *E. coli* that could be acquired by touching contaminated surfaces or objects. Thus, those strains could be repeatedly transmitted between humans and animals within the household aggregate. Further studies are needed to clarify how these strains were able to survive on physical surfaces (outside their natural environment), as this ability is one critical factor for indirect transmission to a new host or reinoculation on the original host.

Because resistance is becoming increasingly widespread without plausible relationships with the use of antimicrobials, it is necessary to consider other strategies to prevent the emergence of antimicrobial-resistant microorganisms. The phenotypic and genotypic correspondences found in our study could suggest interspecies transmission and support previous concerns that pets could become household reservoirs of multiresistant *E. coli* for subsequent infection (or reinfection) of susceptible household members. Johnson and co-authors (2001) corroborated these findings by confirming that canine feces can be regarded as a reservoir for virulent human clones of extraintestinal pathogenic *E. coli*. Frequent within-household sharing of *E. coli* strain was demonstrated among pets, humans, sex partners and non-sex partners (Johnson et al., 2008). This paradigm of in-home and through-home *E. coli* spreading patterns and antimicrobial-resistant genes transfer could influence the design of preventive measures against the diffusion of pathogenic organisms or antimicrobial-resistant genes throughout the population.

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References *continued from page 79*

Berge, A.C.B., Moore, D.A., & Sischo, W.M. (2006). Field trial evaluating the influence of prophylactic and therapeutic antimicrobial administration on antimicrobial resistance of fecal *Escherichia coli* in dairy calves. *Applied Environmental Microbiology, 72*(6), 3872–3878.

Carattoli, A., Lovari, S., Franco, A., Cordaro, G., Di Matteo, P., & Battisti, A. (2005). Extended-spectrum beta-lactamases in *Escherichia coli* isolated from dogs and cats in Rome, Italy, from 2001 to 2003. *Antimicrobial Agents and Chemotherapy, 49*(2), 833–835.

Clinical and Laboratory Standards Institute. (2007). *Performance standards for antimicrobial susceptibility testing; seventeenth informational supplement* (CLSI Doc. M100-S17). Wayne, PA: Author.

Costa, D., Poeta, P., Saenz, Y., Coelho, A.C., Matos, M., Vinue, L., Rodrigues, J., & Torres, C. (2008a). Prevalence of antimicrobial resistance and resistance genes in faecal *Escherichia coli* isolates recovered from healthy pets. *Veterinary Microbiology, 127*(1–2), 97–105.

Costa, D., Poeta, P., Saenz, Y., Vinue, L., Coelho, A.C., Matos, M., Rojo-Bezares, B., Rodrigues, J., & Torres, C. (2008b). Mechanisms of antibiotic resistance in *Escherichia coli* isolates recovered from wild animals. *Microbial Drug Resistance, 14*(1), 71–77.

Dancer, S.J. (2004). How antibiotics can make us sick: The less obvious adverse effects of antimicrobial chemotherapy. *The Lancet Infectious Diseases, 4*(10), 611–619.

Eckert, C., Gautier, V., Saladin-Allard, M., Hidri, N., Verdet, C., Ould-Hocine, Z., Barnaud, G., Delisle, F., Rossier, A., Lambert, T., Philippon, A., & Arlet, G. (2004). Dissemination of CTX-M-type beta-lactamases among clinical isolates of Enterobacteriaceae in Paris, France. *Antimicrobial Agents and Chemotherapy, 48*(4), 1249–1255.

Elseviers, M.M., Ferech, M., Vander Stichele, R.H., Goossens, H., & ESAC Project Group. (2007). Antibiotic use in ambulatory care in Europe (ESAC data 1997–2002): Trends, regional differences, and seasonal fluctuations. *Pharmacoepidemiology and Drug Safety, 16*(1), 115–123.

Goossens, H. (2009). Antibiotic consumption and link to resistance. *Clinical Microbiology and Infection, 15*(Suppl. 3), 12–15.

Goossens, H., Ferech, M., Stichele, R.V., & Elseviers, M. (2005). Outpatient antibiotic use in Europe and association with resistance: A cross-national database study. *The Lancet, 365*(9459), 579–587.

Guardabassi, L., Loeber, M.E., & Jacobson, A. (2004). Transmission of multiple antimicrobial-resistant *Staphylococcus intermedius* between dogs affected by deep pyoderma and their owners. *Veterinary Microbiology, 98*(1), 23–27.

Guardabassi, L., Schwarz, S., & Lloyd, D.H. (2004). Pet animals as reservoirs of antimicrobial-resistant bacteria. *Journal of Antimicrobial Chemotherapy, 54*(2), 321–332.

Johnson, J.R., Owens, K., Gajewski, A., & Clabots, C. (2008). *Escherichia coli* colonization patterns among human household members and pets, with attention to acute urinary tract infection. *Journal of Infectious Diseases, 197*(2), 218–224.

Johnson, J.R., Stell, A.L., & Delavari, P. (2001). Canine feces as a reservoir of extraintestinal pathogenic *Escherichia coli*. *Infection and Immunity, 69*(3), 1306–1314.

Jones, D.L. (1999). Potential health risks associated with the persistence of *Escherichia coli* O157 in agricultural environments. *Soil Use and Management, 15*(2), 76–83.

Livermore, D.M. (2003). Bacterial resistance: Origins, epidemiology, and impact. *Clinical Infectious Diseases, 36*(Suppl. 1), S11–S23.

Machado, E., Coque, T.M., Canton, R., Novais, A., Sousa, J.C., Baquero, F., & Peixe, L. (2007). High diversity of extended-spectrum beta-lactamases among clinical isolates of Enterobacteriaceae from Portugal. *Journal of Antimicrobial Chemotherapy, 60*(6), 1370–1374.

Maynard, C., Bekal, S., Sanschagrin, F., Levesque, R.C., Brousseau, R., Masson, L., Lariviere, S., & Harel, J. (2004). Heterogeneity among virulence and antimicrobial resistance gene profiles of extraintestinal *Escherichia coli* isolates of animal and human origin. *Journal of Clinical Microbiology, 42*(12), 5444–5452.

Mendonça, N., Leitão, J., Manageiro, V., Ferreira, E., & Caniça, M. (2007). Spread of extended-spectrum beta-lactamase CTX-M-producing *Escherichia coli* clinical isolates in community and nosocomial environments in Portugal. *Antimicrobial Agents and Chemotherapy, 51*(6), 1946–1955.

Moreno, A., Bello, H., Guggiana, D., Dominguez, M., & Gonzalez, G. (2008). Extended-spectrum beta-lactamases belonging to CTX-M group produced by *Escherichia coli* strains isolated from companion animals treated with enrofloxacin. *Veterinary Microbiology, 129*(1–2), 203–208.

Moyaert, H., De Graef, E.M., Haesebrouck, F., & Decostere, A. (2006). Acquired antimicrobial resistance in the intestinal microbiota of diverse cat populations. *Research in Veterinary Science, 81*(1), 1–7.

Normand, E.H., Gibson, N.R., Reid, S.W.J., Carmichael, S., & Taylor, D.J. (2000). Antimicrobial-resistance trends in bacterial isolates from companion-animal community practice in the UK. *Preventive Veterinary Medicine, 46*(4), 267–278.

Sáenz, Y., Brinas, L., Dominguez, E., Ruiz, J., Zarazaga, M., Vila, J., & Torres, C. (2004). Mechanisms of resistance in multiple-antibiotic-resistant *Escherichia coli* strains of human, animal, and food origins. *Antimicrobial Agents and Chemotherapy, 48*(10), 3996–4001.

Schwarz, S., Kehrenberg, C., & Walsh, T.R. (2001). Use of antimicrobial agents in veterinary medicine and food animal production. *International Journal of Antimicrobial Agents, 17*(6), 431–437.

Sidjabat, H.E., Townsend, K.M., Hanson, N.D., Bell, J.M., Stokes, H.W., Gobius, K.S., Moss, S.M., & Trott, D.J. (2006). Identification of *bla*(CMY-7) and associated plasmid-mediated resistance genes in multidrug-resistant *Escherichia coli* isolated from dogs at a veterinary teaching hospital in Australia. *Journal of Antimicrobial Chemotherapy, 57*(5), 840–848.

References

Simões, R.R., Poirel, L., Costa, P.M., & Nordmann, P. (2010). Seagulls and beaches as reservoirs for multidrug-resistant *Escherichi coli*. *Emerging Infectious Diseases, 16*(1), 110–112.

Srinivasan, V., Gillespie, B.E., Lewis, M.J., Nguyen, L.T., Headrick, S.I., Schukken, Y.H., & Oliver, S.P. (2007). Phenotypic and genotypic antimicrobial resistance patterns of *Escherichia coli* isolated from dairy cows with mastitis. *Veterinary Microbiology, 124*(3–4), 319–328.

van den Bogaard, A.E., & Stobberingh, E.E. (2000). Epidemiology of resistance to antibiotics: Links between animals and humans. *International Journal of Antimicrobial Agents, 14*(4), 327–335.

Williams, A.P., Avery, L.M., Killham, K., & Jones, D.L. (2005). Persistence of *Escherichia coli* O157 on farm surfaces under different environmental conditions. *Journal of Applied Microbiology, 98*(5), 1075–1083.

INTERNATIONAL PERSPECTIVES

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Prevalence of *Legionella* Strains in Cooling Towers and Legionellosis Cases in New Zealand

Although most of the information presented in the Journal *refers to situations within the United States, environmental health and protection know no boundaries. The* Journal *periodically runs International Perspectives to ensure that issues relevant to our international membership, representing over 20 countries worldwide, are addressed. Our goal is to raise diverse issues of interest to all our readers, irrespective of origin.*

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Abstract **Over 3,900 water samples from 688 cooling towers were tested for *Legionella* in 2008 in New Zealand. Of 80 (2.05% isolation rate) *Legionella* isolates, 10 (12.5%) were *L. pneumophila* serogroup 1; 10 (12.5%) were *L. anisa*; nine (11.2%) were *L. pneumophila* serogroup 8; and one (1.2%) was *L. longbeachae* serogroup 2. Forty-one (51.2%) *Legionella* isolates were *L. pneumophila* serogroups. Over 3,990 water samples from 606 cooling towers were tested for *Legionella* in 2009 in New Zealand. Of 51 (1.28% isolation rate) *Legionella* isolates, 18 (35.3%) were *L. pneumophila* serogroup 1, and 39 (76.4%) were other *L. pneumophila* serogroups.**

***L. pneumophila* serogroups were significantly associated with legionellosis cases in 2008 and 2009. *L. longbeachae* serogroups were equally significantly associated with legionellosis cases. This significant association of *L. longbeachae* with legionellosis particularly of *L. longbeachae* serogroup 1 is unique in that part of the world. The authors' study also showed that the aqueous environment of the cooling tower is not a natural habitat for pathogenic *L. longbeachae*. Regular monitoring and maintenance of cooling towers have prevented outbreaks of legionellosis.**

Introduction

The Australian Standard/New Zealand Standard (AS/NZS) 3666 Part 3:2000 provides a performance-based approach to cooling water systems maintenance in commercial buildings with respect to control of microorganisms, including *Legionella* species. The approach includes automatic regulated water treatment with monitoring, assessment, and control strategies to achieve a cooling water system that has a low-risk environment. With performance monitoring and control within cooling water systems as in a cooling tower, a representative sample of cooling water is taken at least once per month and assessed for the presence of *Legionella* species (in accordance with AS/NZS 3896:2008) and other heterotrophic microorganisms (in accordance with AS/NZS 4276.3.1:2007).

When *Legionellae* are detected (≥10 CFU/mL), a control strategy is immediately initiated. AS/NZS 3666 Part 3 recommends that cooling towers with *Legionella* counts 10–1,000 CFU/mL should be disinfected but those with counts ≥1,000 CFU/mL should be decontaminated to achieve a count of <10 CFU/mL. For the assessment of other heterotrophic microorganisms, a heterotrophic plate count (HPC) is carried out using the 35°C method. When the count is ≥100,000 CFU/mL, again a control strategy is immediately initiated. The HPC test is used to assess the efficiency of treatment with biocide and general cleanliness of the cooling water system.

Legionella bacteria cause the fatal pneumonia in Legionnaires' disease and they thrive in the warm temperatures of 30°C–40°C found in cooling towers. The presence of *Legionella* bacteria in cooling towers causing Legionnaires' disease has been reported (Brown et al., 1999; Fiore et al., 1998; Formica et al., 2000; Greig et al., 2004; Nguyen et al., 2006). The

TABLE **1**

Number of Cooling Tower Samples Tested From Regions in New Zealand in 2008–2009

Region	2008 # (%)	2009 # (%)
Auckland	1924 (35.2)	2013 (35.7)
Bay of Plenty	50 (0.9)	44 (0.8)
Canterbury	238 (4.4)	209 (3.7)
Hawkes Bay	248 (4.5)	318 (5.6)
Nelson	208 (3.8)	285 (5.1)
Northland	10 (0.2)	5 (0.1)
Palmerston North	0 (0.0)	55 (1.0)
Taranaki	187 (3.4)	241 (4.3)
Waikato	373 (6.8)	218 (3.9)
Wanganui	322 (5.9)	303 (5.4)
Wellington	1910 (34.9)	1950 (34.6)
Total	5470 (100.0)	5641 (100.0)

TABLE **2**

Number of Cooling Towers and Water Samples Tested Only for *Legionella* in 2008–2009

Range (#) of Water Samples Provided by Each Cooling Tower	# (%) of Cooling Towers Providing That Range of Water Samples in 2008	# (%) of Cooling Towers Providing That Range of Water Samples in 2009
1–4	389 (56.6)	289 (47.7)
5–8	68 (9.9)	58 (9.6)
9–11	91 (13.2)	89 (14.7)
≥12	140 (20.3)	170 (28.0)
Total	688 (100.0)	606 (100.0)

Note. Number of water samples tested for *Legionella* in 2008 and 2009 were 3,904 and 3,994, respectively.

first recorded outbreak of Legionnaires' disease in New Zealand was reported by Mitchell and co-authors (1991). In that outbreak, four confirmed cases of legionellosis and three probable cases occurred. The production and inhalation of *Legionella*-containing aerosols from cooling towers are responsible for legionellosis including the fatal form of pneumonia in Legionnaires' disease.

Our study examined the frequency and results of microbiological testing of cooling towers with reference to AS/NZS 3666 Part 3:2000. This is because a small number of cooling tower operators in New Zealand do not test concurrently for *Legionella* bacteria and HPC on a monthly basis. Financial cost is one probable reason, while another is perhaps the wrong assumption that a correlation may exist between *Legionella* and HPC level, which has been shown not to be the case (Miller & Kenepp, 1996; Reasoner, 2004).

The majority of legionellosis cases in New Zealand are sporadic, isolated, and community-acquired pneumonias, and although an effort is made to determine the source, it is frequently not identified (Lau & Harte, 2007). It is not known if contaminated cooling towers are contributing to this disease burden since it is not known what the prevalent *Legionella* species in cooling towers are. The predominant species responsible for disease in New Zealand are *L. pneumophila* and *L. longbeachae*. Although the *L. longbeachae* cases have invariably had an exposure to compost or potting mix, the source of *L. pneumophila* cases is often not identified (Cramp et al., 2010). Overseas studies also show that *L. pneumophila, L. longbeachae*, and *L. bozemanii* are major species responsible for community-acquired legionellosis (O'Connor et al., 2007; Yu et al., 2002).

Materials and Methods

Sampling

In 2008 and 2009, a total of 3,904 water samples (from 688 cooling towers) and 3,994 samples (from 606 cooling towers) were collected, respectively. These samples were tested for *Legionella* and HPC and they were collected from different geographical regions of the country (Table 1). Each water sample was couriered to the microbiology laboratory at Environmental Laboratory Service Ltd. and processed within 24 hours.

Clinical samples for laboratory diagnostic tests were sent to the *Legionella* Reference Laboratory at the Institute of Environmental Science and Research Ltd.

Microbiological Analysis

Legionella culture test of cooling tower water samples was based on the method in AS/NZS 3896:2008. Briefly, 0.1 mL of neat and acid-treated water sample was inoculated on buffered charcoal yeast extract (BCYE) and modified Wadowsky-Yee plates, incubated at 36°C and examined on days three and seven. Colonies suspected of being *Legionella* were subcultured on Columbia blood agar and BCYE agar with and without L-cysteine. Colonies growing on BCYE agar containing L-cysteine and not on the other agars were considered to be *Legionella* species and further tested using the Oxoid *Legionella* latex agglutination test to confirm as *Legionella*. The agglutination test allowed for separate identification of *L. pneumophila* serogroup 1, *L. pneumophila* serogroups 2–15, and

TABLE **3**

Comparative Prevalence of *Legionella* Strains in Cooling Towers Tested and From Laboratory-Diagnosed Cases of Legionellosis in 2008 and 2009

Legionella Species/ Serogroup	# (%) in 2008		# (%) in 2009		Total # (%) for 2008 and 2009	
	Cooling Tower Isolates	Laboratory-Diagnosed Cases	Cooling Tower Isolates	Laboratory-Diagnosed Cases	Cooling Tower Isolates	Laboratory-Diagnosed Cases
L. pneumophila sg 1	10 (12.5)	21 (27.6)	18 (35.3)	25 (32.0)	28 (21.4)	46 (29.9)
L. pneumophila sg 2	0	1 (1.3)	1 (2.0)	2 (2.6)	1 (0.8)	3 (1.9)
L. pneumophila sg 4	1 (1.2)	0	8 (15.7)	3 (3.8)	9 (6.8)	3 (1.9)
L. pneumophila sg 5	2 (2.5)	1 (1.3)	1 (2.0)	0	3 (2.3)	1 (0.6)
L. pneumophila sg 6	5 (6.3)	1 (1.3)	2 (3.9)	1 (1.3)	7 (5.3)	2 (1.2)
L. pneumophila sg 7	1 (1.2)	0	0	0	1 (0.8)	0
L. pneumophila sg 8	9 (11.3)	0	1 (2.0)	0	10 (7.6)	0
L. pneumophila sg 12	0	1 (1.3)	0	0	0	1 (0.6)
L. pneumophila sg 13	0	0	4 (7.8)	1 (1.3)	4 (3.0)	1 (0.6)
L. pneumophila sg 14	1 (1.2)	0	0	0	1 (0.8)	0
L. pneumophila sg 2–15	12 (15.0)	0	4 (7.8)	0	16 (12.2)	0
L. anisa	10 (12.5)	0	1 (2.0)	0	11 (8.4)	0
L. bozemanae sg 1	0	0	0	1	0	1 (0.6)
L. bozemanae sg unknown	1 (1.2)	2	0	0	1 (0.8)	2
L. dumoffii	0	5 (6.6)	0	5 (6.4)	0	10 (6.5)
L. feeleii sg 1	0	1 (1.3)	0	0	0	1 (0.6)
L. gormanii	0	2 (2.6)	0	2 (2.6)	0	4 (2.6)
L. longbeachae sg 1	0	19 (25.0)	0	23 (29.5)	0	42 (27.3)
L. longbeachae sg 2	1 (1.2)	1 (1.3)	0	4 (5.1)	1 (0.8)	5 (3.2)
L. longbeachae sg unknown	0	18 (23.7)	0	5 (6.4)	0	23 (14.9)
L. micdadei	0	1 (1.3)	0	2 (2.6)	0	3 (1.9)
L. rubrilucens	4 (5.0)	0	4 (7.8)	0	8 (6.1)	0
L. sainthelensi	1 (1.2)	0	2 (3.9)	1 (1.3)	3 (2.3)	1 (0.6)
Other *Legionella* species	22 (27.5)	2 (2.6)	5 (9.8)	3 (3.8)	27 (20.6)	5 (3.2)
Total	80 (100.0)	76 (100.0)	51 (100.0)	78 (100.0)	131 (100.0)	154 (100.0)

seven other *Legionella* species (*L. longbeachae* 1 and 2, *L. bozemanii* 1 and 2, *L. dumoffii, L. gormanii, L. jordanis, L. micdadei,* and *L. anisa*). Cooling tower *Legionella* isolates were further identified to species and serogroup level using direct fluorescent antibody test (DFAT) and by *mip* gene sequencing (Ratcliff, Lanser, Manning, & Heuzenroeder, 1998).

HPC using R2A agar (in accordance with AS/NZS 4276.3.1) was used to test the microbiological water quality in cooling towers in our study.

Legionella Typing by DFAT

Suspected *Legionella* isolates were further identified to species level using DFAT (Lau & Harte, 2007). Samples fixed to slides were stained with m-TECH fluorescein-conjugated anti-*Legionella* antibodies. *Legionella* bacteria stained as apple-green, rod-shaped coccibacilli or long rod-shaped bacilli.

Legionella Typing by Indirect Fluorescent Antibody Test (IFAT)

Clinical samples of acute phase and convalescent sera were performed to detect serum antibodies to heat-killed whole-cell antigens from *L. pneumophila* sg 1–15 and nine other species of *Legionella* including the two *L. longbeachae* serogroups. Antibodies to *Legionella* species were detected using fluorescein isothiocyanate conjugated sheep anti-human IgM, IgA, and IgG antibody. Patient sera were preabsorbed with a *Campylobacter*-soluble antigen prior to testing for block cross-reacting antibodies to some Gram-negative bacteria (Boswell, Marshall, & Kudesia, 1996). Laboratory-diagnosed cases were based on at least a fourfold rise in titer, or titers >512 in two or more serum samples, or a combination of a positive polymerase chain reaction (PCR) test and a high convalescent titer.

Molecular Tests PCR and DNA Sequence Analysis

When DNA in the PCR tests were isolated from clinical samples, the *Legionella* 16S rRNA gene (using an in-house method based on methods of Jonas and co-authors [1995] and van Der Zee and co-authors [2002]) or the *Legionella mip* gene (Ratcliff et al., 1998) were the gene targets. PCR was performed with forward and reverse primers in a thermal cycler for amplification. The PCR product

TABLE **4**

Comparison of *Legionella* Isolated From Cooling Tower Study and From Laboratory-Diagnosed Cases of Legionellosis in 2008 and 2009 by Region

Region	# of Cooling Tower Isolates in 2008; 2009	Rate (%) of *Legionella* Isolation From Cooling Towers in 2008; 2009	Laboratory-Diagnosed Cases of Legionellosis in 2008; 2009
Northland	0; 0	0.0; 0.0	0; 2
Auckland	14; 13	0.73; 0.65	15; 22
Waikato	7; 4	1.88; 1.83	15; 8
Bay of Plenty	0; 1	0.0; 2.0	10; 10
East Coast	0; 0	0.0; 0.0	0; 0
Taranaki	2; 2	1.07; 0.83	2; 1
Hawke's Bay	5; 0	2.02; 0.0	6; 5
Wanganui	0; 0	0.0; 0.0	2; 1
Wellington	46; 26	2.41; 1.33	5; 3
Wairarapa	0; 0	0.0; 0.0	0; 2
Nelson	0; 0	0.0; 0.0	2; 2
West Coast	0; 0	0.0; 0.0	1; 3
Canterbury	6; 5	2.52; 2.39	15; 15
Otago	0; 0	0.0; 0.0	2; 3
Southland	0; 0	0.0; 0.0	1; 1
Total	80; 51	–	76; 78

was analyzed by agarose gel electrophoresis and viewed with ethidium bromide staining. Absence of a ~730 base pair product indicates no *Legionella* being amplified. The *Legionella* 16S rRNA sequences were compared with those available through the European Bioinformatics Institute server (http://www.ebi.ac.uk/fasta33/nucleotide.html) using the Fasta3 alignment program. The *mip* gene sequences were compared with those available online at the UK Health Protection Agency's link (http://www.hpa-bioinfotools.org.uk/mip_ID.html).

Results

The high percentage of samples collected from Auckland (35.2%) and Wellington (34.9%) in 2008 and 35.7% and 34.6%, respectively, in 2009 reflected the higher number of cooling towers and population size of these cities on the North Island of New Zealand (Table 1). Based on the 2006 census (Statistics New Zealand, 2006) about 43% of New Zealand's total population is concentrated in the Auckland and Wellington regions, where commercial buildings with cooling towers are also concentrated.

A large number of cooling tower samples were submitted for *Legionella* and HPC tests for water quality from different geographical regions in New Zealand (Table 1).

Important features of a water-based induced draught cross-flow cooling tower are as follows. A fan system at the top of the cooling tower helps draw air into the tower to cool the heated water collected from the building. The air coming into the tower meets the heated water, which trickles down the fill during the cooling process. The drift eliminators help minimize the transmission of aerosols to the external environment. Aerosols that contain *Legionella* bacteria from contaminated water can escape through the fan system into the external environment because of the induced air flow system in the cooling tower.

In 2008 a total of 3,904 water samples from 688 cooling towers were tested for *Legionella,* while in 2009 the number was 3,994 water samples from 606 cooling towers (Table 2). The AS/NZS 3666 Part 3:2000 proposes a monthly water sample of a cooling tower for microbiological (*Legionella* and/or HPC) testing. In our study, however, only 39.4% of cooling towers in 2008 and 44.7% of cooling towers in 2009 provided 12 or more samples each for microbiological tests per year.

In our study only 20.3% of cooling towers in 2008 and 28.0% of cooling towers in 2009 provided the recommended 12 or more samples each for *Legionella* tests per year (Table 2).

L. pneumophila sg 1 predominated as the causal *Legionella* strain for laboratory-diagnosed cases of legionellosis in 2008 and 2009 at 27.6% and 32.0%, respectively (Public Health Surveillance, 2008, 2009) (Table 3); additionally, 5.2% and 9.0% of legionellosis cases were due to *L. pneumophila* sg 2–15 in 2008 and 2009, respectively. The predominant strain isolated from cooling towers was also *L. pneumophila* sg 1 at 12.5% in 2008 and at 35.3% in 2009 (Table 3).

Seventy-six and 78 laboratory cases of legionellosis were diagnosed in 2008 and 2009, respectively (Table 3). These represented a similar rate of 1.8 notifiable cases per 100,000 population in 2008 and 2009. Four and two deaths occurred from legionellosis in 2008 and 2009, respectively (Public Health Surveillance, 2008, 2009). The laboratory-diagnosed cases of legionellosis were from sporadic community-acquired cases with no reported outbreaks identified.

L. longbeachae strains were responsible for 50.0% and 41.0% of legionellosis cases in 2008 and 2009, respectively (Table 3). *L. longbeachae* strains, however, were noncolonizers of cooling towers tested in our study except for one isolate (1.2%) in 2008 (Table 3).

Table 4 compares the prevalence of *Legionella* strains isolated from cooling towers and those from laboratory-diagnosed cases of legionellosis by regions in 2008 and 2009. The rate of *Legionella* isolation from cooling towers in our study varied from zero to 2.52%. In 2008 and 2009 the reported rate of laboratory-diagnosed legionellosis cases was similar at 1.8 per 100,000 population (Public Health Surveillance, 2008, 2009).

Table 5 shows that the majority of laboratory-diagnosed legionellosis cases were in the warmer months of the southern hemisphere's summer (26.1%) and spring (31.2%) with

TABLE **5**

Number and Percentage of Laboratory-Diagnosed Cases of Legionellosis by Quarter (Q) in 2004–2008

Legionella Type	# in Q1 (Jan–Mar)					# in Q2 (Apr–Jun)					# in Q3 (Jul–Sep)				
	'04	'05	'06	'07	'08	'04	'05	'06	'07	'08	'04	'05	'06	'07	'08
L. pneumophila sg 1	4	3	10	5	5	–	11	5	9	9	5	21	–	1	1
L. pneumophila sg 2–15	4	4	1	2	1	5	–	2	2	1	2	3	8	2	–
Subtotal *L. pneumophila* serogroups	8	7	11	7	6	5	11	7	11	10	7	24	8	3	1
L. longbeachae spp.	8	6	5	8	8	4	1	5	5	7	1	7	1	2	5
Other *Legionella* spp.	6	6	1	4	–	6	1	1	4	2	3	1	3	1	4
Subtotal non-*L. pneumophila* species	14	12	6	12	8	12	2	6	9	9	4	3	5	3	9
Total all *Legionellae*	22	19	17	19	14	17	13	13	20	19	11	27	13	6	10

	# in Q4 (Oct–Dec)					# (%) for 2004–2008				
	'04	'05	'06	'07	'08	Q1	Q2	Q3	Q4	Total for Q1–Q4
L. pneumophila sg 1	4	4	6	4	6	27 (23.9)	34 (30.1)	28 (24.8)	24 (21.2)	113 (100.0)
L. pneumophila sg 2–15	2	1	1	4	1	12	10	15	9	46
Subtotal *L. pneumophila* serogroups	6	5	7	8	7	39 (24.5)	44 (27.2)	43 (27.0)	33 (20.8)	159 (100.0)
L. longbeachae spp.	10	15	1	13	18	35 (27.1)	22 (17.1)	16 (12.4)	57 (43.4)	130 (100.0)
Other *Legionella* spp.	3	1	4	3	5	17	14	12	16	59
Subtotal non-*L. pneumophila* species	15	16	5	17	23	52 (31.1)	38 (22.8)	24 (14.9)	76 (31.7)	190 (100.0)
Total all *Legionellae*	21	21	12	25	30	91 (26.1)	82 (23.5)	67 (19.2)	109 (31.2)	349 (100.0)

Note. January–March is summer; April–June is autumn; July–September is winter; and October–December is spring.

the fewest cases during the winter months (19.2%). Table 6 also showed that *Legionella* bacteria were least isolated in winter (11.4%) but most isolated in the warmer months of summer (50.4%) and less in autumn (23.7%).

Discussion

As the presence of *Legionella* bacteria in cooling towers can lead to the transmission of Legionnaires'-disease-causing *Legionella* bacteria, it is critical that regular monitoring for *Legionellae* on a monthly basis is maintained. This is one of the performance-based recommendations of AS/NZS 3666 Part 3. The regular monthly testing regime, however, has not been adopted for many cooling towers as shown in Table 2, where only 20.3% and 28% of all cooling towers tested in 2008 and 2009, respectively, submitted a monthly water sample for *Legionella* isolation test. Of concern is that a high percentage of cooling towers in the study, 56.6% in 2008 and 47.7% in 2009, each submitted only 1–4 samples for *Legionella* culture test. The recommendation is to test monthly for *Legionella.*

In New Zealand sporadic cases have occurred of Legionnaires' disease associated with contaminated cooling towers (Allan, 2005; Mitchell et al., 1991). In the report by Mitchell and co-authors (1991), Legionnaires' disease in four confirmed cases was due to a contaminated new cooling tower that had never been treated with chemicals including biocides in a new office building. In the second report by Allan (2005) and also recorded in the New Zealand Public Health Surveillance Report (2005), an outbreak of 19 cases of Legionnaires' disease with three deaths was traced to a cooling tower of a biochemical plant. Both of these reported outbreaks occurred in the southern city of Christchurch in the Canterbury region. These two outbreaks clearly indicate the potential risks of contamination of cooling towers with *Legionella* bacteria if no structured system exists to manage potential risks.

The finding that 51.2% and 76.5% of cooling tower isolates from cooling towers tested in 2008 and 2009, respectively, were from potentially pathogenic *L. pneumophila* serogroups (Table 3) showed that such towers are potential sources for the transmission of such pathogenic *Legionella* strains. Clearly the contaminated cooling towers in our study were potential reservoirs of infection and may be responsible for some legionellosis cases in the community without being recognized. The transmission of *Legionella*-contaminated aerosols from cooling towers has been reported to occur over large distances of more than 3 km (Addiss et al., 1989). Therefore, in

TABLE **6**

Number and Percentage of *Legionella* Isolates From Cooling Towers by Quarter (Q) in 2008 and 2009

Isolate	Q1 (Jan–Mar)		Q2 (Apr–Jun)		Q3 (Jul–Sep)		Q4 (Oct–Dec)		# (%) for 2008 and 2009				
	'08	'09	'08	'09	'08	'09	'08	'09	Q1	Q2	Q3	Q4	Total for Q1–Q4
L. pneumophila sg 1	7	8	–	5	–	2	3	3	15 (53.6)	5 (17.9)	2 (7.1)	6 (21.4)	28 (100.0)
L. pneumophila sg 2–15	16	10	7	8	4	2	4	–	26	15	6	4	51
Subtotal *L. pneumophila* serogroups	23	18	7	13	4	4	7	3	41 (51.9)	20 (25.3)	8 (10.1)	10 (12.7)	79 (100.0)
L. anisa	4	–	3	–	1	–	2	1	–	–	–	–	–
L. bozemanae	–	–	1	–	–	–	–	–	–	–	–	–	–
L. longbeachae sg 2	–	–	1	–	–	–	–	–	–	1	–	–	1
L. rubrilucens	–	3	2	1	–	–	2	–	–	–	–	–	–
L. sainthelensi	–	–	–	2	1	–	–	–	–	–	–	–	–
Other *Legionella* spp.	14	4	1		4	1	3	1	–	–	–	–	–
Subtotal non-*L. pneumophila* species	18	7	8	3	6	1	7	2	25 (48.1)	11 (21.1)	7 (13.5)	9 (17.3)	52 (100.0)
Total all *Legionellae*	41	25	15	16	10	5	14	5	66 (50.4)	31 (23.7)	15 (11.4)	19 (14.5)	131 (100.0)

Note. January–March is summer; April–June is autumn; July–September is winter; and October–December is spring.

the case of contaminated cooling towers in New Zealand, the potential exists for a major outbreak where the towers are close to heavily populated areas. Fortunately, during 2008 and 2009 only sporadic community-acquired cases of legionellosis occurred with no outbreaks identified.

L. longbeachae strains were responsible for 50.0% and 41.0% of legionellosis cases in 2008 and 2009, respectively (Table 3). This is a unique epidemiological observation for legionellosis in this part of the world. Table 5 also showed that the majority of *L. longbeachae* cases occurred during the warmer summer and spring months when more agricultural and horticultural activities occur, with associated exposure to soils and composts/potting mixes. *L. longbeachae* strains, however, were insignificant colonizers of cooling towers tested, with only one isolate (1.2%) in 2008 and 0.0% in 2009 (Table 3). This would indicate that *L. longbeachae* are not natural inhabitants in the aqueous environment but prefer the solid medium of potting mixes, composts, and soils. This is supported in recent comparative and functional genomics research by Cazalet and coauthors (2009), in which they found that *L. longbeachae* does not code for flagella but encodes a capsule. The absence of flagella production may explain why *L. longbeachae* prefers a soil environment to an aqueous environment like a cooling tower. This is particularly relevant in New Zealand, where an extensive agricultural economy and common use of potting mixes and composts by horticultural enthusiasts are present.

Conclusion

Potentially pathogenic *L. pneumophila* serogroups 1–15 are significant *Legionella* strains that colonized New Zealand cooling towers in 2008–2009. The pathogenicity of *L. pneumophila* serogroups 1–15 is reflected in the number of legionellosis cases attributed to these strains in 2008 and 2009. Although high rates of legionellosis cases occurred as a consequence of *L. longbeachae* infections in New Zealand in 2008–2009, this *Legionella* species is not a natural colonizer of cooling towers as shown in our study, except for a single isolate in 2008. Therefore, it is most probable that legionellosis cases due to *L. longbeachae* in New Zealand would have come from inhaling contaminated aerosols or dust particles of potting mixes, composts, and soil material and not contaminated aerosol from cooling towers.

The microbiological monitoring and assessment of cooling towers in New Zealand need to be undertaken with more regularity in accordance with AS/NZS 3666 Part 3 in order to better control the presence of *Legionella* bacteria and the outbreak of Legionnaires' disease. It is of utmost importance from a public health safety perspective as determination of health risks from cooling towers is not reliable if infrequent *Legionella* tests are done (Bentham, 2000).

To minimize the risk of Legionnaires' disease as a consequence of contaminated cooling towers, a structured system to manage potential risks and a register of every water-based cooling tower in operation in a region must exist. Such a register will be very important in the event of a Legionnaires' disease outbreak as it facilitates the search for the reservoir of infection in the area.

To prevent the transmission of pathogenic *L. longbeachae*, the use of gloves, face masks, a well-ventilated work area, washing of hands after operation, and damping down potting mixes before use in confined areas are important safety measures.

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References

Addiss, D.G., Davis, J.P., La Venture, M., Wand, P.H., Hutchinson, M.A., & Kinney, R.M. (1989). Community-acquired Legionnaires' disease associated with a cooling tower: Evidence for longer distance transport of *L. pneumophila*. *American Journal of Epidemiology, 130*(3), 557–568.

Allan, V. (2005). *CDHB releases water cooling tower results*. Canterbury District Health Board [Press release]. Retrieved from http://www.q-net.net.au/~legion/Legionnaires_Disease_New_Zealand.htm

Australian/New Zealand Standard AS/NZS 3666 Part 3:2000. (2000). Air handling and water systems of buildings—microbiological control. Part 3: Performance-based maintenance of cooling tower systems. Sydney: Standards Australia International Ltd. and Wellington: Standards New Zealand.

Australian/New Zealand Standard AS/NZS 4276.3.1:2007. (2007). Water microbiology—heterotrophic colony count methods—pour plate method using yeast extract agar. Sydney: Standards Australia International Ltd and Wellington: Standards New Zealand.

Australian/New Zealand Standard AS/NZS 3896:2008. (2008). Waters—examination for *Legionella* spp. including *Legionella pneumophila*. Sydney: Standards Australia International Ltd and Wellington: Standards New Zealand.

Bentham, R.H. (2000). Routine sampling and the control of *Legionella* spp. in cooling tower water systems. *Current Microbiology, 41*(4), 271–275.

Boswell, T.C., Marshall, L.E., & Kudesia, G. (1996). False-positive titres in routine clinical serology testing detected by absorption with *Campylobacter*: Implications for the serological diagnosis of Legionnaires' disease. *Journal of Infection, 32*(1), 23–26.

Brown, C.M., Nuorti, J.P., Breiman, R.F., Hathcock, A.L., Fields, B.S., Lipman, H.B., Llewellyn, G.C., Hofmann, J., & Cetron, M. (1999). A community outbreak of Legionnaires' disease linked to hospital cooling towers: An epidemiological method to calculate dose of exposure. *International Journal of Epidemiology, 28*(2), 353–359.

Cazalet, C., Gomez-Valero, L., Rusniok, C., Lomma, M., Dervins-Ravault, D., Newton, H.J., Sansom, F.M., Jarraud, S., Zidane, N., Ma, L., Bouchier, C., Etienne, J., Hartland E.L., & Buchrieser, C. (2010). Analysis of the *Legionella longbeachae* genome and transcriptome uncovers unique strategies to cause Legionnaires' disease. *PloS Genetics, 6*(2), e1000851.

Cramp, G.J., Harte, D., Douglas, N.M., Graham, F., Schousboe, M., & Sykes, K. (2010). An outbreak of Pontiac fever due to *Legionella longbeachae* serogroup 2 found in potting mix in a horticultural nursery in New Zealand. *Epidemiology and Infection, 138*(1), 15–20.

Fiore, A.E., Nuorti, J.P., Levine, O.S., Marx, A., Weltman, A.C., Yeager, S., Benson, R.F., Pruckler, J., Edelstein, P.H., Greer, P., Zaki, S.R., Fields, B.S., & Butler, J.C. (1998). Epidemic Legionnaires' disease two decades later: Old sources, new diagnostic methods. *Clinical Infectious Diseases*, *26*(2), 426–433.

Formica, N., Tallis, G., Zwolak, B., Carnie, J., Beers, M., Hogg, G., Norbert, R., & Yates, M. (2000). Legionnaires' disease outbreak: Victoria's largest identified outbreak. *Communicable Diseases Intelligence, 24*(7), 199–202.

Greig, J.E., Carnie, J.A., Tallis, G.F., Norbert, J.R., Tan, A.G., Gordon, I.R., Zwolak, B., Leydon, J.A., Guest, C.S., & Hart, W.G. (2004). An outbreak of Legionnaires' disease at the Melbourne Aquarium, April 2000: Investigation and case-control studies. *Medical Journal of Australia, 180*(11), 566–572.

Jonas, D., Rosenbaum, A., Weyrich, S., & Bhakdi, S. (1995). Enzyme-linked immunoassay for detection of PCR-amplified DNA of *Legionellae* in bronchoalveolar fluid. *Journal of Clinical Microbiology, 33*(55), 1247–1252.

Lau, R., & Harte, D. (2007). The presence of *Legionella* bacteria in public water features. *Environmental Health,* 7(2), 45–51.

Miller, R.D., & Kenepp, K.A. (1996). *Legionella* in cooling towers: Use of *Legionella*-total bacteria ratios (pp. 99–107). In M. Muilenberg & H. Burge (Eds.), *Aerobiology.* Boca Raton, FL: CRC Press.

Mitchell, P., Chereshsky, A., Haskell, A.J., & Brieseman, M.A. (1991). Legionellosis in New Zealand: First recorded outbreak. *New Zealand Medical Journal, 104*(915), 275–276.

New Zealand Public Health Surveillance Report. (2005). Legionellosis and environmental *Legionella*. Wellington: Institute of Environmental Science and Research and Ministry of Health. Retrieved from http://www.surv.esr.cri.nz/surveillance/NZPHSR.php?we_objectID=845

Nguyen, T.M.N., Ilef, D., Jarraud, S., Rouil, L., Campese, C., Che, D., Haeghebaert, S., Ganiayre, F., Marcel, F., Etiene, J., & Desenclos, J.C. (2006). A community-wide outbreak of Legionnaires' disease

References

linked to industrial cooling towers—how far can contaminated aerosols spread? *Journal of Infectious Diseases, 193*(1), 102–111.

O'Connor, B.A., Carman, J., Eckert, K., Tucker, G., Givney, R., & Cameron, S. (2007). Does using potting mix make you sick? Results from a *Legionella longbeachae* case-control study in South Australia. *Epidemiology and Infection, 135*(1), 34–39.

Public Health Surveillance. (2008). *Annual surveillance report for 2008*. Wellington, New Zealand: Institute of Environmental Science and Research and Ministry of Health. Retrieved from http://www.surv.esr.cri.nz/surveillance/annual_surveillance.php?we_objectID=1987

Public Health Surveillance. (2009). *Annual surveillance report for 2009*. Wellington, New Zealand: Institute of Environmental Science and Research and Ministry of Health. Retrieved from http://www.surv.esr.cri.nz/surveillance/annual_surveillance.php?we_objectID=2311

Ratcliff, R.M., Lanser, J.A., Manning, P.A., & Heuzenroeder, M.W. (1998). Sequence-based classification scheme for the genus *Legionella* targeting the *mip* gene. *Journal of Clinical Microbiology, 36*, 1560–1567.

Reasoner, D.J. (2004). Heterotrophic plate count methodology in the United States. *Internal Journal of Food Microbiology, 92*(3), 307–315.

Statistics New Zealand. (2006). *2006 census of population and dwellings*. Wellington, New Zealand: Author. Retrieved from http://www.stats.govt.nz/browse_for_stats/population/census_counts/2006-census_HOTPProvisionalCounts.aspx

van Der Zee, A., Verbakel, H., de Jong, C., Pot, R., Bergmans, A., & Peeters, M. (2002). Novel PCR-probe assay for detection of and discrimination between *Legionella pneumophila* and other *Legionella* species in clinical samples. *Journal of Clinical Microbiology, 40*(3), 1124–1125.

Yu, V.L., Plouffe, J.F., Pastoris, M.C., Stout, J.E., Schousboe, M., Widmer, A., Summersgill, J., File, T., Heath, C.M., Paterson, D.L., & Chereshsky, A. (2002). Distribution of *Legionella* species and serogroups isolated by culture in patients with sporadic community-acquired legionellosis: An international collaborative survey. *Journal of Infectious Diseases, 186*(1), 127–128.

SPECIAL REPORT

State Public Health Laboratory Biomonitoring Programs: Implementation and Early Accomplishments

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Abstract **In 2009, the Centers for Disease Control and Prevention funded three state-based public health laboratory biomonitoring programs. These programs are the foundation for a National Biomonitoring Plan that consists of a larger network of state and local biomonitoring programs. To understand the utility of these programs and plan for the larger network, interviews were conducted with the program officials. The goal was to gather information on the challenges, successes, and lessons learned in program launch and implementation. Representatives of all programs participated. Projects range from statewide efforts to focused community investigations. Each program focuses on specific analytes including metals, pesticides, and other organics. Main accomplishments reported include development of laboratory and field capacity as well as generation of analytical results. Common challenges reported were laboratory setup and operation, sample collection and logistics, and staff recruitment. Respondents made specific recommendations for improving effectiveness of the current programs as well as ways to advance the National Biomonitoring Plan.**

Introduction and Background

Biomonitoring is one direct method for measuring human exposure to environmental contaminants (Committee on Human Biomonitoring for Environmental Toxicants, 2006). Just as monitoring contaminants in the ambient environment informed pollution prevention policies over the past decades, biomonitoring serves as a powerful tool to shape environmental public health (Association of Public Health Laboratories [APHL], 2009a). Examples of environmental health policy successes supported by biomonitoring include declines in blood lead levels following the ban of leaded gasoline and declines in cotinine levels in nonsmokers following a smoking ban (Annest et al., 1983; Centers for Disease Control and Prevention [CDC], 2007). Biomonitoring is often used to calm fears related to potential exposures (Teeguarden et al., 2011) or to effectively target limited environmental remediation funding (APHL, 2009b).

Biomonitoring remains indispensible as the field of environmental public health advances toward answering emerging questions concerning: exposures across the lifespan (Woodruff, Zota, & Schwartz, 2011); health impacts of chemical mixtures (Payne-Sturges, Cohen, Castorina, Axelrad, & Woodruff, 2009); and assessment of cumulative risks including multiple chemicals in addition to population variability and susceptibility (Ryan, Burke, Cohen Hubal, Cura, & McKone, 2007).

The environmental health profile of the National Health and Nutrition Examination Survey (NHANES) now includes more than 200 chemicals and the resulting data establish population-based reference ranges for each chemical as well as fostering a body of descriptive and analytical epidemiological research (CDC, 2009; Crinnion, 2010; Hightower, O'Hare, & Hernandez, 2006; Navas-Acien et al., 2009). While NHANES is an essential national resource, it has important limitations: little information exists on sources of exposure, and it provides no data at the state and local levels. Lacking data on location and exposure sources, NHANES cannot inform exposure reduction actions that might be taken by state or local health officials.

The Association of Public Health Laboratories (APHL) developed the National Biomonitoring Plan (NBP) to fill this information gap by improving state and local capacity for biomonitoring (APHL, 2009a). The goal entails a coordinated national approach to addressing public health issues related to chemical exposures: one which will enable benchmarking and comparisons across studies.

In addition to supporting APHL, the Centers for Disease Control and Prevention (CDC) have supported the development of state ca-

pacity for biomonitoring through a cooperative agreement program, initially awarded to three states: California, New York, and Washington. The cooperative agreements were initiated in 2009 and will provide up to five years of funding with an annual renewal. The three state public health laboratory-based pilot projects are working to understand state and local exposure conditions; take action to reduce those exposures; and lead the way toward the NBP. Each program is briefly described below.

- Biomonitoring California is lead by the California Department of Public Health (CDPH) working jointly with the Office of Environmental Health and Hazard Assessment and the Department of Toxic Substance Control (DTSC) in California's Environmental Protection Agency. Two laboratories are affiliated with the biomonitoring program: the CDPH Environmental Health Laboratory and the DTSC Environmental Chemistry Laboratory. Biomonitoring California operates under a legislative mandate.
- The New York State (NYS) biomonitoring program is part of the Wadsworth Center, the NYS Department of Health's (NYSDOH's) public health laboratory. The biomonitoring program collaborates with the New York City Department of Health and Mental Hygiene and NYSDOH Center for Environmental Health.
- The biomonitoring program staff in Washington work in two divisions of the state health department: the public health laboratory and office of epidemiology. The biomonitoring staff collaborate with toxicologists in the Division of Environmental Health and the department's information technology specialists.

To document the early utility of these programs and plan for expanding the state-based biomonitoring effort, APHL in collaboration with CDC contracted with the Johns Hopkins Bloomberg School of Public Health to interview the state biomonitoring program officials. The goal of the interviews was to gather information on the challenges, successes, and lessons learned in program launch and implementation. This article presents the state-of-the-practice related to biomonitoring for nonfunded states, i.e., the successes and challenges of the early work of the funded programs to inform other states planning programs or building biomonitoring capacity.

Approach and Methods

Interview Development and Conduct

Johns Hopkins investigators worked collaboratively with APHL and CDC. The interview questions were based on the elements of the original request for applications, the state-specific proposals, and the recommendations of the National Research Council report on Human Biomonitoring for Environmental Chemicals (Committee on Human Biomonitoring for Environmental Toxicants, 2006). Interview topics included budget and funding, projects, public health impact, accomplishments, ways to improve effectiveness, and lessons learned.

Interviews were scheduled via e-mail and were conducted by telephone in February and March 2011 (during the second year of funding). Interviews were completed for respondents from each state biomonitoring program. In total five interviews were conducted with six respondents. Two calls were with principal investigators (PI), two calls were with laboratory directors (LD), and one call was with both PI and LD responding. Three respondents were from California, two respondents were from New York State, and one respondent was from Washington. Response was voluntary and some respondents did not complete all questions. Interview notes were transcribed and provided to respondents to check for completeness and accuracy prior to compiling the data.

Data Compilation and Analysis

The answers to each question were summarized by call/interview. One question involved ranking of program and technical priorities. Ranks for each priority item were averaged over the number of respondents for that item. Items that were tied in average rank are presented together. The results below summarize the range of responses and identifies common responses and themes.

Results

Applying the Science

While CDC's National Exposure Report provides a snapshot of overall population exposure, the state biomonitoring programs are going beyond reference ranges to ascertain site- and population-specific investigations, as summarized in Table 1. Each state has developed capacity for a variety of analytes including metals, pesticides, and other organics.

The diversity of ongoing projects in California results from that state's legislative mandate for population biomonitoring. Biomonitoring California partners with organizations to identify populations or groups for biomonitoring projects. Projects underway aim to characterize statewide population exposures as well as exposures within several special populations such as mothers and infants, firefighters, adolescent girls, and specific exposed communities. For example, a completed project in California assessed perchlorate exposure in an Imperial Valley community. The project was a collaboration with the Environmental Public Health Tracking (EPHT) program and CDC (English et al., 2011).

New York State projects include analyses of archived samples from the New York City Health and Nutrition Examination Survey, a community project, and a firefighter study. The Washington State program launched its statewide evaluation of population exposure to arsenic and arsenic species, selected pesticides, and organics.

While a great deal of work occurred in the first year of the cooperative agreements, a great deal of work remains. The data need to be analyzed, communicated, and maybe even used to drive interventions or policies. Expected benefits of these programs according to the six respondents included the following:

- shaping or evaluating policies such as chemical use regulations or worker protection;
- understanding actual exposures in the state by creating a baseline for future reference;
- assisting local health departments to address exposures or lack thereof;
- intervening on adverse exposure conditions to improve health; and
- bringing attention to special populations, e.g., related to environmental justice, mothers and infants, and selected occupational groups.

Accomplishments and New Partnerships

Reported accomplishments comprised development of laboratory and field capacity. One respondent emphasized the rapid speed of program implementation, given that each program had some analyses completed during the first year of funding as outlined above.

Each biomonitoring program reported working with their respective state EPHT programs. In one state, the EPHT program provided funding for water sampling to iden-

tify sources of exposure; in the other states the biomonitoring and EPHT programs were collaborating on community projects. Other types of partners included local health departments, other state health departments, and universities.

Challenges and Priorities

Common challenges reported by the three programs were acquiring instrumentation and developing capacity, laboratory operations (e.g., methods validation and obtaining standards), field operations, and staff recruitment. Regarding the latter, two PIs and one LD mentioned that it can be difficult to find people with the necessary skills, particularly laboratory staff, field workers, and communication staff required for a successful biomonitoring program. In addition, program development and implementation are time intensive, a challenge that highlights the accomplishments of these programs in completing some analyses in the first year of funding.

Several other scientific or technical challenges were reported by individual respondents:

- accessing samples and getting the right population and type of sample,
- interpreting results and establishing reference ranges,
- validating methods and finding comparable data, and
- lack of a coordinated proficiency testing program.

Program Priorities

Funding ranked first among program priorities in both the short term (about a year) and long term (up to five years). Participants from each program indicated use of additional money on top of the CDC cooperative agreement funding in the first year. In one state the counterterrorism program provided equipment and training and the EPHT program provided funding for some analyses. In another state a fee assessed on industries supports biomonitoring. Other sources of funding include other federal agency grants and cooperative agreements (National Institutes of Health and the Agency for Toxic Substances and Disease Registry).

Beyond funding concerns, short-term program priorities in rank order were partnerships and personnel, infrastructure and support for policy change, and organizational restructuring. In the longer term, program priorities were personnel, partnerships, and support for policy change and organizational restructuring.

Quality assurance and quality control (QA/QC) ranked high among technical priorities in both the short- and long term. Short-term technical needs beyond QA/QC were (in descending order) communicating and training, study design issues, and technical expertise and equipment. In the longer-term, study design issues (population selection and sampling) were tied with QA/QC for top rank. Other longer-term technical needs were (in descending order) technical expertise and equipment, communicating, and training.

TABLE **1**

Projects and Project Status by State Public Health Laboratory Biomonitoring Program

State	Project (Status as of March 2011)	Target Analytes[a]
California	• Access and utility of biospecimens from California's prenatal and newborn screening programs (under development) • Exposure Assessment of Cohort of Young Girls' Nutrition, Environment, and Transitions (ongoing) • Firefighter study (ongoing) • Kaiser biobank repository (under development) • Maternal and Infant Environmental Exposure Project (ongoing) • Organophosphate exposure in Tulare County (ongoing) • Perchlorate exposure in Imperial Valley (Complete)	• Target analytes vary by project and can include metals, PFOS, PBDE, PAHs, PCBs, phenols, organophosphate, and pyrethroid pesticides
New York	• Exposure to uranium and depleted uranium in residents near and workers of national lead industries (ongoing) • Firefighter study (ongoing) • Analysis of New York City's Community Health and Nutrition Examination Survey archived samples (ongoing) » Trace metals analyses (complete)	• Target analytes vary by project and can include metals, cyanide, and various organics
Washington	• Washington Environmental Biomonitoring Survey (ongoing) » Analyses of metals, total arsenic (complete) » Drinking water sample collection (complete)	• Target analytes are arsenic and arsenic species, organophosphate, and pyrethroid pesticide metabolites • Various organics analyses are also planned but procedures are currently under development

[a]PFOS = perfluorooctane sulfonate; PBDE = polybrominated diphenyl ethers; PAHs = polycyclic aromatic hydrocarbons; PCBs = polychlorinated biphenyls.

Improving Effectiveness and Lessons Learned

Improving Effectiveness

Ideas offered to improve effectiveness were varied. Coordination both within and between state biomonitoring programs and with CDC was mentioned by two respondents. Within programs, project coordination and communication between the laboratorians, epidemiologists, and environmental health staff were seen as a key to good project management and overall program effectiveness. Establishing communication with other state biomonitoring programs was suggested as a way to share experiences that could improve program operations. These respondents also would like to have improved

coordination with CDC on technology transfer, e.g., anticipating differences in types of equipment and training for specific equipment. From the laboratory perspective, improved effectiveness can be achieved via enhanced capacity such as sample preparation automation, formal processes for analytical method development, proficiency testing programs, and availability of reference standards. The other responses for improving effectiveness were making links to environmental monitoring programs and data and developing a basic guide for establishing field programs.

Lessons Learned During Implementation

In reflecting on their experiences, the biomonitoring program respondents had very practical advice regarding implementation and planning for additional state-based biomonitoring programs. The advice focused on resources, program and project coordination, and proficiency testing. Regarding resources, the responding PIs cautioned that careful planning to match resources to program scope and mission is required. In addition to staff with scientific expertise, having program and project coordinators was reported by two PIs as critical to day-to-day operations and overall program success. Thinking ahead to a broader network of state, local and national biomonitoring programs as the NBP is implemented, two LDs and one PI noted that a proficiency testing program would be needed to ensure comparability across programs.

Discussion

Limitations

With only three programs currently funded by CDC, this interview project is limited to describing the range of program activities and approaches to program implementation at the time of the interviews (February–March 2011). Work is underway for many projects but final results are not available; policy impacts are not known and comparisons across states are not possible at this time.

Of five interviews conducted, three were with staff of Biomonitoring California and only one interview each was with staff from New York State and Washington. Having a majority of respondents from one program has the potential to bias the responses toward the particulars of that program. The many commonalities of responses across all three states, however, suggest that program operations were largely similar in years 1–2 of funding and each respondent has contributed valuable insights for this work.

Common Themes and Priorities

Many common responses occurred across the survey topics, organized here as related to program implementation, communication and translation, and scientific issues.

Each program was making efficient use of resources within the primary agency as well as across sister agencies, and drawing upon expertise already available. For example, each program had ongoing collaborations with EPHT programs and reported channeling communications efforts through epidemiology or environmental health staff. The frequently voiced concern about funding is natural; biomonitoring requires advanced instrumentation and trained laboratory personnel as well as expertise in epidemiology, toxicology, and communication.

Common themes under the category of scientific issues were the challenges of laboratory setup and operation such as difficulties obtaining reference standards. Respondents from each program identified QA/QC and proficiency testing as priorities. Comparability of data across states will become important in the full implementation of APHL's NBP.

Program priorities described above remained consistent across state and types of respondent as well as in terms of short- and long-term rankings. The high priority of funding and personnel are likely due to the resource intensity of biomonitoring from equipment and staff training to field work and communication. Partnerships established in each program appear to be a key to implementation. The lower priority for policy change and organizational restructuring may result from the early stage of the program work; projects are underway but results are not final so no current policy initiatives exist as a result of biomonitoring. Programs just got up and running, so organizational changes may not be needed until later years.

Technical priorities described above were somewhat variable across state and types of respondent as well as in terms of short- and long-term rankings. QA/QC ranked high both in short- and long terms. One difference was that communication and training ranked higher in the short term than long term. This may reflect the newness of these programs and priorities of capacity building for trained laboratory and communications personnel. Study design issues ranked higher in the long term. This is perhaps because in the early phases programs pick the "low-hanging fruit" with relatively straightforward designs and respondents likely expected investigations to become more complex and larger in scale over time. Technical expertise and equipment issues also ranked higher in the long term, possibly reflecting anticipation of staff turnover and increased maintenance or replacement costs of equipment.

Conclusions and Recommendations

It is clear from the results that currently funded states highly value their developing biomonitoring capabilities as a tool to protect populations and effectively direct environmental health protection efforts. The interest in biomonitoring among nonfunded states also remains high, as 30 states competed for funding in response to CDC's last request for applications (CDC, 2012). The work presented here can serve as a guide for new programs to assist in identifying and addressing program implementation challenges early on in program development.

The interview findings indicate that the biomonitoring programs currently funded by CDC remain strong and well positioned to protect public health. Each program reported progress in program capacity and implementation, with some analyses completed in the first year of work. Each program worked efficiently and creatively to develop and launch projects. Each program prioritized quality to maximize public health impacts.

Concerns about too little and inconsistent funding were uppermost in the minds of the program leads. To their credit, they built internal and external partnerships to obtain funding or garner in-kind resources in order to achieve program goals. This strategy has been successful to date but is likely not sustainable in the long term since programs that have been allied with biomonitoring (e.g., the counterterrorism program) are also subject to budget cuts.

In a number of other areas, however, key strategies emerged from the interviews that will enhance capacity and impacts of the current programs and that will help move towards a network of local, state, and national biomonitoring resources.

To ensure public health impacts of the current biomonitoring programs, two respondents identified the need to understand sources of exposure through environmental sampling within biomonitoring studies

and making links to existing environmental monitoring data. Biomonitoring data in the absence of exposure source information is inadequate to develop public health activities.

Several ideas offered will advance the NBP. These included

- establishing communication links between states with biomonitoring programs,
- preparing to support more advanced study designs,
- facilitating laboratory operations (e.g., obtaining standards),
- developing infrastructure to support analysis of metabolites, and
- developing a proficiency testing program.

At the national level biomonitoring has been transformative in informing policy priorities, spurred tremendous advances in exposure science, and is the cornerstone of such initiatives as the National Children's Study. While current funding levels and continued slow economic growth raise serious questions about program sustainability, it is clear that at the state level biomonitoring can achieve its full promise as a public health tool in responding to specific community exposure conditions and concerns, informing more focused and effective environmental policies, and ultimately evaluating policy impacts in the long term.

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References

Annest, J.L., Pirkle, J.L., Makuc, D., Neese, J.W., Bayse, D.D., & Kovar, M.G. (1983). Chronological trend in blood lead levels between 1976 and 1980. *New England Journal of Medicine*, *308*(23), 1373–1377.

Association of Public Health Laboratories. (2009a). *National biomonitoring plan: Five-year plan*. Retrieved from www.aphl.org/aphlprograms/eh/Documents/National_Biomonitoring_Plan_For_Public_Health_Laboratories.pdf

Association of Public Health Laboratories. (2009b). *Moving forward: Biomonitoring stories from the states*. Retrieved from http://www.aphl.org/AboutAPHL/publications/Documents/EH_2009Oct_Moving-Forward-Biomonitoring-Stories-from-the-States.pdf

Centers for Disease Control and Prevention. (2007). Reduced secondhand smoke exposure after implementation of a comprehensive statewide smoking ban—New York, June 26, 2003–June 30, 2004. *Morbidity and Mortality Weekly Report, 56*(28), 705–708.

Centers for Disease Control and Prevention. (2009). *Fourth national report on human exposure to environmental chemicals*. Retrieved from www.cdc.gov/exposurereport/

Centers for Disease Control and Prevention. (2012). *National biomonitoring program: State grant activities*. Retrieved from www.cdc.gov/biomonitoring/state_grants.html

Committee on Human Biomonitoring for Environmental Toxicants. (2006). *Human biomonitoring for environmental toxicants*. Retrieved from www.nap.edu/openbook.php?record_id=11700&page=R1

Crinnion, W.J. (2010). The CDC fourth national report on human exposure to environmental chemicals: What it tells us about our toxic burden and how it assists environmental medicine physicians. *Alternative Medicine Review, 15*(2), 101–109.

English, P., Blount, B., Wong, M., Copan, L., Olmedo, L., Patton, S., Haas, R., Atencio, R., Xu, J., & Valentin-Blasini, L. (2011). Direct measurement of perchlorate exposure biomarkers in a highly exposed population: A pilot study. *PloS One, 6*(3), e17015.

Hightower, J.M., O'Hare, A., & Hernandez, G.T. (2006). Blood mercury reporting in NHANES: Identifying Asian, Pacific Islander, Native American, and multiracial groups. *Environmental Health Perspectives, 114*(2), 173–175.

Navas-Acien, A., Tellez-Plaza, M., Guallar, E., Muntner, P., Silbergeld, E., Jaar, B., & Weaver, V. (2009). Blood cadmium and lead and chronic kidney disease in U.S. adults: A joint analysis. *American Journal of Epidemiology, 170*(9), 1156–1164.

Payne-Sturges, D., Cohen, J., Castorina, R., Axelrad, D.A., & Woodruff, T.J. (2009). Evaluating cumulative organophosphorus pesticide body burden of children: A national case study. *Environmental Science and Technology, 43*(20), 7924–7930.

Ryan, P.B., Burke, T.A., Cohen Hubal, E.A., Cura, J.J., & McKone, T.E. (2007). Using biomarkers to inform cumulative risk assessment. *Environmental Health Perspectives*, *115*(5), 833–840.

Teeguarden, J.G., Calafat, A.M., Ye, X., Doerge, D.R., Churchwell, M.I., Gunawan, R., & Graham, M. (2011). Twenty-four hour human urine and serum profiles of bisphenol A during high dietary exposure. *Toxicological Sciences, 123*(1), 48–57.

Woodruff, T.J., Zota, A.R., & Schwartz, J.M. (2011). Environmental chemicals in pregnant women in the United States: NHANES 2003–2004. *Environmental Health Perspectives*, *119*(6), 878–885.

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INTERNATIONAL PERSPECTIVES

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Health and Safety Inspection of Hairdressing and Nail Salons by Local Authority Environmental Health Practitioners

Although most of the information presented in the Journal *refers to situations within the United States, environmental health and protection know no boundaries. The* Journal *periodically runs International Perspectives to ensure that issues relevant to our international membership, representing over 20 countries worldwide, are addressed. Our goal is to raise diverse issues of interest to all our readers, irrespective of origin.*

Joanne Harris-Roberts
Jo Bowen
Jade Sumner
David Fishwick
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Abstract **The objective of the study described in this article was to provide environmental health practitioners (EHPs) with an evaluation of the levels of understanding of, and compliance with, health and safety legislation in hairdressing and nail salons. EHPs carried out a series of inspections of 205 salons in a large British city, consisting of a site assessment and an assessment of employee knowledge of relevant regulations, including those relating to control of exposure to hazardous substances.**

Two-fifths of senior salon employees understood Control of Substances Hazardous to Health (COSHH) assessments and could provide evidence of their completion. Most employees had been trained and made aware of the health hazards associated with carrying out their work and took suitable precautions to protect themselves and their clients.

The results suggest that senior employees within the salons sampled, have knowledge of the risks to health and have been taking measures to control these risks. Initiatives such as the Health and Safety Executive's (in collaboration with local authorities and the hairdressing industry) "Bad Hand Day?" campaign and sector-specific COSHH essentials guidance help raise awareness levels and aim to support good control practice in salons.

Introduction

Hairdressing and nail salons are expanding small-business sectors, and a number of reports in the literature highlight health concerns associated with working in these environments. During nail enhancement work, technicians are potentially exposed to a number of substances, such as solvents, lacquers, acrylic polymers (including ethyl methacrylate [EMA]), adhesives, and dust. Studies have highlighted respiratory symptoms and musculoskeletal and skin problems in this population (Harris-Roberts et al., 2011; Roelofs, Azaroff, Holcroft, Nguyen, & Doan, 2008; Spencer, Estill, McCammon, Mickelson, & Johnston, 1997). Hairdressing work has been associated with occupational asthma (Slater et al., 2000), rhinitis, and occupational contact dermatitis (Ferrari, Moscato, & Imbriani, 2005; Khumalo, Jessop, & Ehrlich, 2006; Moscato et al., 2005; Moscato & Galdi, 2006), and a recent study reported musculoskeletal, skin, and respiratory symptoms in hairdressers (Bradshaw, Harris-Roberts, Bowen, Rahman, & Fishwick, 2011). In November 2006, the Health and Safety Executive (HSE; the regulator for work-related health and safety in Great Britain), in collaboration with local authorities and the hairdressing industry, launched the "Bad Hand Day?" campaign to raise awareness of work-related dermatitis in the hairdressing industry (www.badhandday.HSE.gov.uk).

Given the potential health concerns highlighted in the literature, the "Bad Hand Day?" campaign, and a perception from local authority environmental health practitioners (EHPs) that the application of control measures in this industry is varied, hairdressing and nail salons were highlighted by a local authority for a planned inspection campaign.

The objective of our study was to analyze the information gained from this series of inspections to provide EHPs with a baseline evaluation of the current levels of understanding of, and compliance with, health and safety legislation in hairdressing and nail salons as well as good control practice in this sector.

TABLE **1**

General Questions About the Control Of Substances Hazardous to Health (COSHH) Regulations and Health and Safety Training

Question	Hairdressers			Nail Salons			Hairdressing and Nail Services		
	Yes	No	N/A	Yes	No	N/A	Yes	No	N/A
Does the business keep a list of the hairdressing and beauty products that they use and record which of these products are potentially hazardous to human health?	57/115 49.6%	58/115 50.4%	0/115 0%	9/31 29.0%	22/31 71.0%	0/31 0%	19/47 40.4%	28/47 59.6%	0/47 0%
Does the business keep any written procedures on how to control exposure, etc.?	35/111 31.5%	76/111 68.5%	0/111 0%	6/33 18.2%	27/33 81.8%	0/33 0%	13/47 27.7%	34/47 72.3%	0/47 0%
Do they consider the possible associated health risks when buying a new hairdressing/nail salon product?	65/122 53.3%	34/122 27.9%	23/122 18.8%	22/32 68.8%	8/32 25.0%	2/32 6.3%	29/43 67.4%	13/43 30.2%	1/43 2.3%
Does anyone check that the employees follow health and safety procedures and that they implement their health and safety training?	89/110 80.9%	21/110 19.1%	0/110 0%	19/28 67.9%	9/28 32.1%	0/28 0%	35/44 79.5%	9/44 20.5%	0/44 0%

Methods

Health and Safety Inspection

Study salons were selected for inspection from a database held by the local EHPs. As part of the usual inspection process, EHPs undertook an initial walk-through survey to assess products being used, treatments provided, premises, staffing, the health and safety risks, and provision of any control measures.

In addition, duty holders were asked whether risk assessments had been carried out and how these had been carried out. EHPs also requested to see any relevant health and safety documentation.

EHPs were also asked to collect where possible specific data, including knowledge of appropriate regulations by senior employees (collected during general inspection discussions with senior employees), awareness of sector-specific Control of Substances Hazardous to Health (COSHH) essentials guidance, glove use, and "Risk Control Indicator" data in line with Health and Safety Executive/Local Authority Enforcement Liaison Committee (HELA) general risk control indicators (www.hse.gov.uk/foi/internalops/fod/inspect/rcisummary.htm). Completed inspection checklists were returned to the study team for subsequent analysis.

Salon Inspections and Checklist Returns

Data from 205 of 380 anticipated inspections of hairdressing and nail salons over an eight-month study period were returned to the study team for analysis. One hundred twenty-two (59.5%) of these sites were hairdressers, 36 (17.6%) were nail salons, and 47 (22.9%) were salons providing both hairdressing and nail services.

Statistical Analysis

Descriptive statistical analysis was performed using SPSS software version 10. A number of inspection checklists were returned with incomplete data. Descriptive statistical analysis was calculated using the total number of responses for each question and Pearson Chi squared analysis was used to identify differences between salon types and to determine any significant associations between variables.

Results

General Requirements: COSHH Regulations and Health and Safety Training

Overall, hairdressing and nail salons did not keep written records of health and safety assessments and procedures. Less than half (44.0% [85/193]) kept lists of products used (and recorded which of the products were potentially hazardous to health) and only 28.3% (54/191) had written procedures covering exposure control. Additionally, only 38.6% (76/197) of senior employees understood COSHH assessments and could provide evidence of assessments completed. From the inspectors' perspectives, however, 88% (161/183) of the senior employees in the salons sampled appeared to understand the main risks to employee health associated with the use of hairdressing and beauty products. The majority of salons (79.7% [153/192]) had evidence of steps in place to control the risk to health from hazardous products used by employees and 58.9% (116/197) considered the possible associated health risks when buying new hairdressing/nail salon products. The majority of salons generally stored and mixed beauty products (83.0% [166/200]) and disposed of used and unused products/chemicals in an appropriate fashion (84.8% [168/198]). In most of the salons inspected (88.9% [168/189]), the EHPs considered that employees had been trained and made aware of the health hazards associated with carrying out their work. Furthermore, in a similar number of salons (89.0% [162/182]) the employees took suitable and sufficient precautions to protect themselves and clients.

Although the intent of our study was not to compare hairdressing and nail salons, various descriptive differences between salon type are shown in Table 1 and salon type appeared to influence certain outcome measures. For example, hairdressing salons were more likely to keep listings of products and record which were potentially hazardous to health in comparison to nail salons (49.6% vs. 29.0%, $p = .041$). Nail salons noted a significantly greater use of antibacterial hand gels and sanitizers (61.3% vs. 31.2%, $p = .002$), although this may reflect differences in the techniques used between the differing salon types.

TABLE **2**

General Skin Care and Glove Use

Question	Hairdressers			Nail Salons			Hairdressing and Nail Services		
	Yes	No	N/A	Yes	No	N/A	Yes	No	N/A
Are gloves provided by the business for all work involving products/chemicals and wet work?	103/121 85.1%	16/121 13.2%	2/121 1.7%	23/32 71.9%	9/32 28.1%	0/32 0%	43/46 93.5%	3/46 6.5%	0/46 0%
Are gloves provided on a personal and single-use only basis?	72/78 92.3%	5/78 6.4%	1/78 1.3%	16/16 100%	0/16 0%	0/16 0%	33/33 100%	0/33 0%	0/33 0%
Is there evidence that these gloves are actually being used (e.g., evidence of them in the waste bin)?	77/101 76.2%	23/101 22.8%	1/101 1.0%	16/23 69.6%	6/23 26.1%	1/23 4.3%	30/40 75.0%	8/40 20.0%	2/40 5.0%
Does management provide instructions/guidance on how to put on and remove gloves without contaminating the hands?	49/102 48.0%	51/102 50.0%	2/102 2.0%	7/23 30.4%	15/23 65.2%	1/23 4.3%	17/42 40.5%	25/42 59.5%	0/42 0%
What type of glove is used?	Latex: 75.0% (39/52) Vinyl: 19.2% (10/52) Polythene: 3.8% (2/52) Nitrile: 1.9% (1/52)			Latex: 90.9% (10/11) Vinyl: 9.1% (1/11)			Latex: 78.3% (18/23) Vinyl: 17.4% (4/23) Nitrile: 4.3% (1/23)		

General Skin Care and Glove Use

Most salons (99.0% [195/197]) provided hot and cold running water facilities for employees, with 98.0% (196/200) also providing hand cleaning products. Additionally, most of the salons (94.9% [188/198]) provided good-quality soft clean towels in the wash area and 88.8% (174/196) provided skin creams for employee use.

Table 2 contains details specifically of glove use within salons. While the recorded provision of gloves was commonplace, inspectors found visual evidence in only 75% (123/164) of salons providing gloves that the gloves were actually used (for example, used gloves in the waste bin). Less than half of the salons provided instructions or guidance on how to put on and remove gloves without contaminating the hands, and over three-quarters of salons provided latex gloves. Information regarding nature and type of glove, e.g., single use or powdered, was not provided, however.

Use of Products and Tools in Salons Providing Nail Services

Table 3 details the approaches taken by salons carrying out some form of nail work. It is clear that while general hygiene measures were well attended to (for example, treatment tables were wiped clean between services in 92.1% of salons), the provision of specific pieces of equipment designed to reduce operator and client exposure were less commonplace. For example, only 16%

TABLE **3**

Use of Products and Tools in Salons Providing Nail Services

Question	Nail Salons			Hairdressing and Nail Services		
	Yes	No	N/A	Yes	No	N/A
Are treatment tables wiped clean between clients?	30/34 88.2%	3/34 8.8%	1/34 2.9%	40/42 95.2%	2/42 4.8%	0/42 0%
Do you use makeup brushes for dusting down equipment?	17/34 50%	17/34 50%	0/34 0%	11/40 27.5%	29/40 72.5%	0/40 0%
Are single-use, sterile instruments used whenever possible?	21/31 67.7%	8/31 25.8%	2/31 6.5%	30/40 75.0%	10/40 25%	0/40 0%
Is nondisposable equipment effectively cleaned, disinfected, and/or sterilized between clients?	28/32 87.5%	4/32 12.5%	0/32 0%	33/38 86.8%	4/38 10.5%	1/38 2.6%
Does the business use an autoclave?	6/33 18.2%	26/33 78.8%	1/33 3.0%	6/42 14.3%	36/42 85.7%	0/42 0%
Do employees wear a disposable dust mask when carrying out nail services?	21/33 63.6%	12/33 36.4%	0/33 0%	18/38 47.4%	19/38 50.0%	1/38 2.6%
Are containers that aren't being used kept closed thus reducing exposure?	33/33 100%	0/33 0%	0/33 0%	32/40 80.0%	4/40 10.0%	4/40 10.0%
Are ventilated treatment tables used that vent to the outside?	6/32 18.8%	26/32 81.3%	0/32 0%	5/38 13.2%	30/38 78.9%	3/38 7.9%
Are electric drills used?	17/33 51.5%	15/33 45.5%	1/33 3.0%	9/38 23.7%	27/38 71.1%	2/38 5.3%
If yes, is use restricted to filing artificial nails only (not natural nails)?	14/15 93.3%	1/15 6.7%	0/15 0%	9/9 100%	0/9 0%	0/9 0%
What type of nail extension system is used?	UV nails: 14.8% (4/27) Powder/acrylic: 40.7% (11/27) All: 37.0% (10/27) N/A: 7.4% (2/27)			UV nails: 26.1% (6/23) Powder/acrylic: 17.4% (4/23) Wraps: 4.3% (1/23) All: 30.4% (7/23) N/A: 21.7% (5/23)		

TABLE **4**

General Risk Control Indicators

Indicator	Hairdressers Compliance	Nail Salons Compliance	Hairdressing and Nail Services Compliance
Management systems: Effective organization and arrangements including adequate Control Of Substances Hazardous to Health assessment, provision of information, instruction, training, and supervision; evidence of management commitment and arrangements for review	Full: 5.2% (6/116) Broad: 35.3% (41/116) Some: 47.4% (55/116) Limited/no: 12.1% (14/116)	Full: 13.3% (4/30) Broad: 20.0% (6/30) Some: 50.0% (15/30) Limited/no: 16.7% (5/30)	Full: 5.1% (2/39) Broad: 33.3% (13/39) Some: 48.7% (19/39) Limited/no: 12.8% (5/39)
Control strategy: Substitution considered and effected where possible; adequate engineering controls provided, used, maintained, examined, and tested at suitable intervals; suitable personal protective equipment (PPE) provided, worn and stored correctly, suitably cleaned, and well maintained; appropriate instruction and training in proper use of engineering controls and PPE	Full: 6.9% (8/116) Broad: 47.4% (55/116) Some: 32.8% (38/116) Limited/no: 12.9% (15/116)	Full: 13.3% (4/30) Broad: 30.0% (9/30) Some: 46.7% (14/30) Limited/no: 10.0% (3/30)	Full: 0% (0/39) Broad: 51.3% (20/39) Some: 43.6% (17/39) Limited/no: 5.1% (2/39)
Health surveillance: A competent person has considered the need for health surveillance and provides it for everyone at risk and it is repeated as necessary; health records are kept; reportable cases of occupational ill health are reported under the Reporting of Injuries, Diseases, and Dangerous Occurrences Regulations	Full: 1.9% (2/107) Broad: 23.4% (25/107) Some: 52.3% (56/107) Limited/no: 22.4% (24/107)	Full: 3.6% (1/28) Broad: 28.6% (8/28) Some: 39.3% (11/28) Limited/no: 28.6% (8/28)	Full: 2.6% (1/38) Broad: 26.3% (10/38) Some: 42.1% (16/38) Limited/no: 28.9% (11/38)
Management of risk: Management enthusiastic and competent, has identified the main risks and knows the relevant health and safety standards for each one; the necessary measures have been put in place and checks are made to see they are used properly; evidence of effective self-regulation	Full: 6.9% (8/116) Broad: 53.4% (62/116) Some: 35.3% (41/116) Limited/no: 4.3% (5/116)	Full: 10.0% (3/30) Broad: 26.7% (8/30) Some: 53.3% (16/30) Limited/no: 10.0% (3/30)	Full: 5.1% (2/39) Broad: 48.7% (19/39) Some: 41.0% (16/39) Limited/no: 5.1% (2/39)
Working environment: Workplace is well lit, well ventilated, tidy, and clean (if inspected, good welfare facilities)	Full: 39.7% (46/116) Broad: 50.0% (58/116) Some: 7.8% (9/116) Limited/no: 2.6% (3/116)	Full: 53.3% (16/30) Broad: 30.0% (9/30) Some: 16.7% (5/30)	Full: 51.3% (20/39) Broad: 38.5% (15/39) Some: 7.7% (3/39) Limited/no: 2.6% (1/39)

of salons used an autoclave and 15.7% used ventilated tables. In salons providing artificial nail treatments, 34% (17/50) used a combination of UV, powder/acrylic, and wraps, 30% (15/50) used powder/acrylic systems only, 20% (10/50) used UV systems only, and 2% (1/50) used wraps only.

Of those salons that used UV or powder/acrylic excluding wrap systems, approximately half (48.8% [20/41]) reported that they did not contain either ethyl methacrylate (EMA) or methyl methacrylate monomer (MMA) ingredients; the remaining salons used systems thought to contain EMA 48.8% (20/41) and MMA (1/41).

Influence of Prior Training

In terms of the benefits of previous training at any salon, those salons who had previously trained their employees and made them aware of health hazards associated with their work more often took suitable and sufficient precautions to protect themselves and their clients (91.9%) in comparison to salons without previous training (61.1%, $p < .001$). Similarly, salons with training more commonly considered the possible health risks associated with buying new hairdressing or nail products (71.1%) in comparison to those salons without previous training (37.5%, $p = .006$).

Inspectors more often found visual evidence that the gloves were actually used in salons that provided instructions or guidance on how to put on and remove gloves (80.9%) in comparison to salons with no provision of instructions for glove use (66.7%, $p = .043$).

HELA General Risk Control Indicators

Table 4 details salon "Risk Control Indicators." Just over a third (38.9% [72/185]) of salons either had full or broad compliance in their management systems (which will have covered adequate COSHH assessment, provision of health and safety information, employees' training, and management commitment). Just over half (51.9% [96/185]) had full or broad compliance in their strategy to control exposures.

Approximately one quarter of salons (27.2% [47/173]) had full or broad compliance with health surveillance requirements, which included consideration of the need for health surveillance and appropriate provision if required. Just over half of all salons (55.1%, [102/185]) were considered to have full or broad compliance with "Management of Risk," which includes the identification of hazards and associated risks and the implementation of the necessary steps to control the risks to health.

Discussion

Our study is the first to report baseline EHP evaluation of the current levels of understanding of, and compliance with, health and safety legislation in hairdressing and nail salons as well as good control practice in this sector.

As cosmetic products are subject to COSHH (Health and Safety Executive [HSE], 2002), employers are required to carry out a risk assess-

ment under these regulations. Furthermore, if employers have five or more employees, a record must be kept of the main findings of the COSHH assessment, either in writing or in electronic form. The CHIP regulations (Chemical Hazard Information & Packaging for Supply, 2005), however, do not apply to cosmetics, so suppliers of many products used in these salons would not be required by law to provide a material safety data sheet. Under these circumstances, the duty holder or enforcement officers (for example, local authorities or trading standards) may obtain further information from the supplier directly. In addition, various regional differences exist. For example, nail salons and beauty salons (but not hairdressers) in London must hold a special treatment license. Although a system for registration (but not licensing) of some special treatments outside London exists, this process does not apply specifically to manicure treatments.

Our study found a variable level of practice in comparison to what would be regarded as ideal. While it was clear that EHPs thought that most of the senior employees in the hairdressing and nail salons understood the main risks to employee health associated with the use of hairdressing/beauty products, documented evidence of this information was not generally available. Indeed, less than two-fifths of salons inspected were found to be compliant with these requirements under COSHH. Reassuringly, however, a positive association existed between those salons where most employees had been trained and made aware of the health hazards associated with carrying out their work and 1) taking suitable and sufficient precautions to protect themselves and their clients and 2) considering the possible health risks associated with buying new hairdressing or nail products. This association corresponds with a recent literature review that found a positive effect of training on occupational health and safety knowledge, attitudes, and behaviors (Robson et al., 2011).

With regard to glove usage, COSHH essentials guidance (HSE, 2005, 2006), Habia Dermatitis and Glove Use Guidance (Habia, 2007), and HSE's "Bad Hand Day?" campaign advises that protective gloves are provided for certain nail services and hairdressing activities. Generally, the inspected salons were compliant with this guidance, as over four-fifths of salons provided single-use gloves for all work involving products/chemicals or wet work. Only approximately half of the salons, however, provided instructions or guidance on how to put on and remove gloves without hand contamination. Interestingly, inspectors more often found visual evidence that the gloves were actually used in those salons that provided instructions or guidance on how to put on and remove gloves.

Over three quarters of the salons who provided gloves provided latex gloves for their employees, although the "Bad Hand Day?" campaign and COSHH essentials SR11 and SR13 advocate the use of nonlatex gloves. Indeed, the SR13 for nail salons states, "If you must use latex gloves, use only 'low protein, powder-free' gloves." As this finding was not anticipated prior to the EHP inspection, the checklist did not inquire if the latex gloves in use were powdered, low protein, or powder free.

It also raises the possibility that hairdressers, nail salons, and beauty salons could be using powdered latex gloves, although further investigation would be required to clarify this. Joint initiatives such as the "Bad Hand Day?" campaign, inspection initiatives, and COSHH essentials, however, have helped to raise the levels of awareness of risks of dermatitis among hairdressers and nail technicians and aim to support good control practice in salons.

COSHH essentials SR13 describes good practice and suitable equipment to control nuisance odors and dusts. In particular, the guidance suggests that sterile single-use instruments are provided for use whenever possible, precluding the need for an autoclave. Approximately two-thirds of the salons inspected used single-use sterile instruments whenever possible. If nondisposable equipment was used, however, over four-fifths of salons were reported to have effectively cleaned, disinfected, or sterilized equipment in between clients. In addition, SR13 guidance suggests that caps and lids are "put back on containers straight away"; most salons were found to be compliant with this practice, so reducing the risk of exposure. Similarly, a "good standard" of ventilation is recommended, and an extractor hood or downdraft table is suggested. Our study, however, only identified approximately one in 10 salons using ventilated tables for nail manicuring.

SR13 guidance also suggests that dust masks are not acceptable as a control measure, yet in approximately half of the salons inspected, employees wore a disposable dust mask when carrying out nail services. Also, half of the nail salons visited used makeup brushes to dust down equipment, which could increase, rather than reduce, the quantity of airborne dust.

Both EMA and MMA have traditionally been used in acrylic-based nail extension systems. The use of MMA was banned, however, in around 23 U.S. states in 1999 (Beauty for Nails, 2006). Although MMA is not banned in the UK, its use is in decline. This is thought to be due to good working practices, which has resulted in it being largely replaced by EMA.

Of those salons inspected that used UV or acrylic/powder nail extension systems, approximately half were reported not to contain either EMA or MMA. This is surprising as EMA is the monomer commonly used in these systems.

Regarding the five HELA general risk control indicators, a lack of compliance was observed under "Management Systems" in two-thirds of the salons inspected. This suggested they did not have adequate COSHH assessment, provision of information, employee training, and management commitment. This lack of compliance in management systems may have contributed to the fact that only just over half of the salons had full or broad compliance in their strategy to control exposure or management of risk. This information suggests that further advice and awareness-raising initiatives could be directed at this industry (including suppliers of products) to improve the knowledge and implementation of COSHH and ensure an adequate assessment and control of the risks to health.

HSE's "Bad Hand Day?" campaign provided guidance and instructions on how to prevent dermatitis by the correct use of gloves, washing, drying, and moisturizing the hands and by encouraging employees to check their hands for early signs of dermatitis. While focused specifically on hairdressers, this type of approach may have had impact on other related workers such as those in nail salons. Indeed, most of the salons inspected complied with HSE dermatitis prevention advice and provided hot and cold running water facilities, hand-cleaning products, good-quality soft clean towels, and skin creams for employees.

Despite our study noting areas of good practice and knowledge of appropriate risks posed by beauty products, significant scope remains for further improvement in these

areas. In particular, the findings of our study would support the development of a practical tool to assist risk assessment by illustrating the principles upon which COSHH is based and to encourage written documentation of any risk assessment findings in order to develop a more systematic approach to health and safety management in this sector.

Conclusion

Our study, the first to report baseline EHP evaluation of the current levels of understanding of, and compliance with, health and safety legislation in hairdressing and nail salons, identified a variable level of practice in comparison to what would be regarded as ideal. Most of the senior employees in the hairdressing and nail salons understood the main risks to employee health associated with the use of hairdressing/beauty products, although documentation to support these issues was generally less easy to identify. Most employees had been trained and made aware of the health hazards associated with carrying out their work and took suitable and sufficient precautions to protect themselves and their clients. Various differences between salon type were evident and prior training of employees appeared to have certain benefits. Health issues in this sector are important, and their assessment is important to integrate practically into the risk assessment process.

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References

Beauty for Nails. (2006). *MMA the safety issue for nails*. Retrieved from http://www.creativenailplace.com/Products/mma.htm

Bradshaw, L., Harris-Roberts, J., Bowen, J., Rahman, S., & Fishwick, D. (2011). Self-reported work-related symptoms in hairdressers. *Occupational Medicine, 61*(5), 328–334.

Chemical Hazard Information and Packaging for Supply Regulations, SI2002 No. 1689 (2002).

Ferrari, M., Moscato, G., & Imbriani, M. (2005). Allergic cutaneous diseases in hairdressers. *La Medicina del Lavoro, 96*(2), 102–118.

Habia. (2007). *Dermatitis and glove use for hairdressers*. Retrieved from http://www.habia.org/uploads/Dermatitis%20Booklet.pdf

Harris-Roberts, J., Bowen, J., Sumner, J., Stocks-Greaves, M., Bradshaw, L., Fishwick, D., & Barber, C.M. Work-related symptoms in nail salon technicians. *Occupational Medicine, 61*(5), 335–340.

Health and Safety Executive. (2002). *Working with substances hazardous to health: What you need to know about COSHH*. Retrieved from http://www.hse.gov.uk/pubns/indg136.pdf

Health and Safety Executive. (2005). *SR11 COSHH essentials, hairdressing*. Retrieved from http://www.hse.gov.uk/pubns/guidance/sr11.pdf

Health and Safety Executive. (2006). *SR13 COSHH essentials, nail salons*. Retrieved from http://www.hse.gov.uk/pubns/guidance/sr13.pdf

Khumalo, N.P., Jessop, S., & Ehrlich, R. (2006). Prevalence of cutaneous adverse effects of hairdressing: A systematic review. *Archives of Dermatology, 142*(3), 377–383.

Moscato, G., & Galdi, E. (2006). Asthma and hairdressers. *Current Opinion in Allergy and Clinical Immunology, 6*(2), 91–95.

Moscato, G., Pignatti, P., Yacoub, M.R., Romano, C., Spezia, S., & Perfetti, L. (2005). Occupational asthma and occupational rhinitis in hairdressers. *Chest, 128*(5), 3590–3598.

Robson, L.S., Stephenson, C.M., Schulte, P.A., Amick, B.C., Irvin, E.L., Eggerth, D.E., Chan, S., Bielecky, A.R., Wang, A.M., Heidotting, T.L., Peters, R.H., Clarke, J.A., Cullen, K., Rotunda, C.J., & Grubb, P.L. (2011). A systematic review of the effectiveness of occupational health and safety training. *Scandinavian Journal of Work, Environment & Health,* doi: 10.5271/sjweh.3259. [Epub ahead of print].

Roelofs, C., Azaroff, L.S., Holcroft, C., Nguyen, H., & Doan, T. (2008). Results from a community-based occupational health survey of Vietnamese-American nail salon workers. *Journal of Immigrant and Minority Health, 10*(4), 353–361.

Slater, T., Bradshaw, L., Fishwick, D., Cheng, S., Kimbell-Dunn, M., Erkinjuntti-Pekkanen, R., Douwes, J., & Pearce, N. (2000). Occupational respiratory symptoms in New Zealand hairdressers. *Occupational Medicine, 50*(8), 586–590.

Spencer, A.B., Estill, C.F., McCammon, J.B., Mickelsen, R.L., & Johnston, O.E. (1997). Control of ethyl methacrylate exposures during the application of artificial fingernails. *American Industrial Hygiene Association Journal, 58*(3), 214–218.

Did You Know?

Besides bacterial infections, nail fungus and ringworm infections can be transmitted to nail salon patrons during their visits.

Source: WebMD Health News, www.webmd.com/healthy-beauty/news/20010504/nail-salon-health-hazards

DIRECT FROM CDC ENVIRONMENTAL HEALTH SERVICES BRANCH

Elaine Curtiss, MEd

Kick Off 2013 With Exciting Workforce Development Opportunities

Editor's Note: **NEHA strives to provide up-to-date and relevant information on environmental health and to build partnerships in the profession. In pursuit of these goals, we feature a column from the Environmental Health Services Branch (EHSB) of the Centers for Disease Control and Prevention (CDC) in every issue of the *Journal*.**

In this column, EHSB and guest authors from across CDC will highlight a variety of concerns, opportunities, challenges, and successes that we all share in environmental public health. EHSB's objective is to strengthen the role of state, local, and national environmental health programs and professionals to anticipate, identify, and respond to adverse environmental exposures and the consequences of these exposures for human health. The services being developed through EHSB include access to topical, relevant, and scientific information; consultation; and assistance to environmental health specialists, sanitarians, and environmental health professionals and practitioners.

The conclusions in this article are those of the author(s) and do not necessarily represent the views of the CDC.

Elaine Curtiss is a former middle school teacher currently serving as a technical writer on the Model Aquatic Health Code and other projects within EHSB.

On behalf of the Centers for Disease Control and Prevention's Environmental Health Services Branch (EHSB), Happy New Year! January marks a new beginning—prompting people all over the world to make resolutions to improve their health, get organized, or learn a skill they've always wanted to master. Many professionals also start their progression by earning advanced educational degrees, working toward promotions, or improving or updating workplace systems. As an environmental public health professional, you already know that learning and developing do not end once you accept your diploma. Therefore, EHSB has developed several excellent learning opportunities—both online and in traditional classroom settings—to help you enhance your effect on environmental health and make 2013 the best year yet!

Environmental Public Health Online Courses

Whether you are a student preparing for certification/registration or a public health veteran needing continuing education credit, our environmental public health online courses are an excellent way to strengthen your practical environmental public health knowledge. This online, on-demand package of e-learning modules is a comprehensive and affordable workforce development resource for all environmental public health professionals. *Better yet, NEHA will award Registered Environmental Health Specialist/Registered Sanitarian continuing education credit for each successfully completed module.* Module topics run the gamut from general environmental health and food protection to vector control, drinking water, wastewater, and radiation exposures—certainly something for every environmental public health professional.

Environmental Assessments for Foodborne-Illness Outbreaks

Foodborne-illness outbreaks happen way too often, which means environmental public health professionals must be prepared. Therefore, knowledge and skills needed for outbreak investigations must be acquired. EHSB, through its Environmental Health Specialists Network (EHS-Net), is collaborating with its grantees, the Food and Drug Administration and U.S. Department of Agriculture, to improve this knowledge and skill set. These partners are developing another free e-learning course to allow users to conduct virtual foodborne-illness outbreak environmental assessments (FIOEAs). This self-paced, interactive course—presented in the context

of a simulated virtual classroom—will teach foundational skills required to conduct a FIOEA. This course will include an overview of FIOEAs, introduce techniques to ensure effective interviewing, critical thinking skills, and demonstrate the importance of consulting with colleagues in epidemiology and in laboratory sciences. In essence, this training will model the collaboration and teamwork that characterizes a cohesive outbreak-investigation team. When implemented, this virtual training can help environmental public health professionals hone their abilities to conduct FIOEAs and prevent future outbreaks. Stay tuned for the release of this exciting and innovative new training expected early this year.

Environmental Health Training in Emergency Response

One thing none of us hopes will happen this year is a natural disaster or other public health emergency; however, as recent history has shown, hurricanes, tornadoes, floods, and other disasters continue to happen. Since we cannot prevent such disasters, we must be prepared for them in the most innovative and comprehensive way possible. That is why EHSB's Environmental Health Training in Emergency Response (EHTER) course was developed. This popular four-day basic-awareness-level course provides environmental public health professionals with basic knowledge, skills, and resources to prepare for environmental health problems caused by emergency situations and disasters—including food safety, water/wastewater issues, shelter assessment/sanitation, vector control/pest management, responder safety, solid waste/hazardous materials, and radiation. Hundreds of environmental public health professionals throughout the U.S. are better prepared for emergencies because they have been trained through this EHSB course.

Integrated Pest Management

Along with natural disasters and public health emergencies, nobody wants to think about disease-carrying insects and rodents. If left unchecked, these pests can become far more than a nuisance; they can quickly cause illness and an outbreak. EHSB's "Biology and Control of Vectors and Public Health Pests: The Importance of Integrated Pest Management" (IPM) course presents a comprehensive, systems-based approach to pest management. IPM advocates the use of the safest and most effective, economical, and sustainable methods to control these pests. Available both online and as in-person training, this course teaches IPM methods that will help reduce the risk not only from the pests but also from the overuse or inappropriate use of hazardous chemical products to control them. This course teaches users how to routinely inspect and monitor indoor and outdoor areas to identify the presence of vectors and pests and whether conditions are conducive to infestations. This course will also help the environmental public health professional to identify the type or species of pest and establish effective control methods. In addition, this course will show how to monitor and evaluate applied measures to determine their success and the next course of action.

EHSB is dedicated to serving you, the environmental public health professional, and we hope that you will take advantage of the learning opportunities provided. We appreciate your hard work and dedication to protect our nation's communities by continuing to learn and develop as environmental public health professionals. So, here's to YOU and your growth, success, and health! Happy 2013!

For more information about the training opportunities discussed here, see

- Environmental Public Health Online Courses: www.cdc.gov/nceh/ehs/eLearn/EPHOC.htm
- Foodborne Illness Outbreak Environmental Assessment Training: www.cdc.gov/nceh/ehs/eLearn/EA_FIO/index.htm
- Environmental Health Training in Emergency Response: cdp.dhs.gov/schedules/program/s.html
- Integrated Pest Management: www.cdc.gov/nceh/ehs/eLearn/IPM.htm

For additional information on other training opportunities from EHSB, see

- www.cdc.gov/nceh/ehs/eLearn
- www.cdc.gov/nceh/ehs/Workforce_Development/training.htm

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DEMYSTIFYING THE FUTURE

Thomas Frey

City of the Future: Part One

Editor's Note: **Significant and fast-paced change is occurring across society in general and our profession in particular. With so much confusion in the air, NEHA is looking for a way to help our profession better understand what the future is likely to look like. The clearer our sense for the future is, the more able we are to both understand and take advantage of trends working their way through virtually every aspect of our lives today. To help us see what these trends are and where they appear to be taking us, NEHA has made arrangements to publish the critical thinking of the highly regarded futurist, Thomas Frey.**

The opinions expressed in this column are solely that of the author and do not in any way reflect the policies and positions of NEHA and the *Journal of Environmental Health*.

Thomas Frey is Google's top-rated futurist speaker and the executive director of the DaVinci Institute®. At the Institute, he has developed original research studies enabling him to speak on unusual topics, translating trends into unique opportunities. Frey continually pushes the envelope of understanding, creating fascinating images of the world to come. His talks on futurist topics have captivated people ranging from high-level government officials to executives in Fortune 500 companies. He has also authored the book *Communicating with the Future*. Frey is a powerful visionary who is revolutionizing our thinking about the future.

Great communities are founded on great ideas. At the same time, our most admired communities become a magnet, attracting the brightest minds. The relational effect is clear: bright minds make a community great, and great communities attract bright minds.

In the future, communities will be designed around ways to stimulate new ideas using such things as creative environments, imagination sparkers, and inspirational architecture.

They will also be designed around new ways for people to meet people. Future communities will be judged by their vibrancy, their interconnectedness, and their fluid structures for causing positive human collisions.

Yet, at the same time, there is a diminishing value to physical proximity. In the middle of this relationship, with all things proximate, is where we can begin to comprehend how cities are changing, and how they will continue to change in the future.

The Diminishing Value of Proximity

To understand the city of the future, we must first understand the importance of proximity. As we try to uncover the driving forces of change, we need to look at the changing dynamic of our personal relationship with the physical community we find ourselves in.

In the past, we decided where to live, work, and conduct business based on how close we were to key assets. Very often that decision was based on things like

1. income streams,
2. goods and services,
3. schools,
4. friends and family,
5. activity centers,
6. weather,
7. crime rate,
8. airport,
9. cultural opportunities, and
10. recreation opportunities.

However, our need for physical proximity is changing.

We live in an increasingly interconnected society and, at the same time, an increasingly mobile society. Our ability to communicate instantly on a thousand different levels with people all over the world, coupled with an increasingly efficient travel network to drive or fly great distances causes us to place lesser importance on the community around us.

With more and more people figuring out ways to work from their homes, either through remote employment, project-based work, or home-based or Internet-based businesses, their relationship with their physical community changes.

Eight Dimensions of Human Connectedness

When thinking about the city of the future, jobs and income streams are not the only consideration. But that is where we will begin.

The city of the future will form around eight dimensions of human connectedness, the interface created between people and their surrounding community. A well-connected community will be a vibrant community where ideas are exchanged, energies are exchanged, and people become extremely loyal to the networks that connect them to the rest of the world. While it is now easy to communicate with people all over the world, we can only physically interact with people and places locally.

Human connectedness involves much more than just communication. And it's not just about business life, family life, or what we do for entertainment. It is all of that and much more.

For the purposes of this discussion, we will look at human connectedness from eight different perspectives, with the final part looking at the role of city government and how it is adapting within these parameters.

Every dimension of human connectedness is an information sphere that requires its own unique user interface.

There are many ways to look at the human interface, but for the purposes of this analysis, our dimensions of human connectedness have been divided into the following eight categories:

1. education and learning;
2. money and income;
3. culture, events, and entertainment;
4. health and fitness;
5. goods and services;
6. sports and recreation;
7. love and relationships; and
8. government.

Before going into each of these areas in more detail, one of the key drivers is our move into an increasingly fluid society.

Flowing to Areas of Least Resistance

With transportation becoming easier, making us a more mobile society, and with cell phones and the Internet speeding up our digital communications, our cities are becoming a much more fluid environment.

Much like water that flows downhill using the path of least resistance, businesses and social structures have begun to move from areas we find less appealing to areas that are more appealing.

Using this line of thinking, we can envision many decision points where transitions are starting to occur.

- With so many obstacles in our paths, we tend to take the route with the least number of gatekeepers.
- As the cost of owning and operating a physical business continues to climb, many have begun to migrate their business operations into the digital world.
- Too many laws will force businesses to move some of their operation underground, or at least under the radar.

We buy where it is easy, we eat where it is convenient, and we relax where it is comfortable.

It is easy to dismiss all this as general laziness. But it gets to a far more fundamental motivation driving human behavior—respect.

We patronize places that respect our time, respect our needs, and respect our status.

We all set our minimum quality standards, and once those have been met we look for other attributes such as convenience, friendliness, and speed. When we hear words like "complicated," "arduous," or "painful" we tend to run the other direction.

We spend our time, our attention, and our money where it is most respected.

Money and Income

Unbeknownst to most, the 8,000 pound gorilla hovering in the background of our economy is the shifting population base. Any fluctuation in the number of consumers changes the demand side of the supply and demand equation.

The 1900s were a very fertile century where the earth's population grew from 1.6 billion people to 6.4 billion within 100 years. Never before in history had the human population exploded like this, and we all became conditioned to think there would be a never-ending supply of young people, and a never-ending supply of demand for real estate.

But a strange thing happened along the way. As doom and gloom predictions started painting scary scenarios of an overpopulated earth where food shortages threatened the very existence of humanity, the full impact of birth control technology, invented in the 1960s, began to take effect.

Today, the population in the U.S. has begun to level off, while at the same time nearly all of Europe and major parts of Asia are in serious decline. Since people create the economy, the lack of people creates just the opposite. This drop in demand will manifest itself in many areas, including a drop in the demand for real estate, as well as other goods and services.

The Coming Free Agent Workforce: According to Daniel Pink, author of *Free Agent Nation*, "In the past, the standard working arrangement was that employees gave loyalty and the organization gave security. However, that bargain is now kaput."

Analyst Christopher Dwyer of Aberdeen Group estimates freelance workers already make up 20% of the labor force, a figure that will rise to 25% as early as next year.

Steve Armstrong, operations manager for Kelly Services, believes the expanding use of contract workers is at least partly fueled by some Americans who see more flexibility, and even security, in freelancing. He sees young workers who saw their parents lose jobs in the past couple of years take on more of a free-agent mentality.

Since September, the number of workers taking temp jobs has risen by 404,000, making up 68% of the 593,000 jobs added by private employers, according to the Bureau of Labor Statistics. At the same time, many laid-off workers who have been unable to land a permanent job have transitioned over to independent contractors and consultants.

The Coming Surge of Entrepreneurship: At the same time that free agents are becoming a business-of-one, we are seeing a much larger wave of entrepreneurial activity.

Traditionally it could be predicted that for every 100 people who join the ranks of the jobless, seven would attempt to start their own business. Some find business niches, others invent, and still others find a better way to do something markets are already craving. Ingenuity and daring often are the catalysts for setting business and commerce in motion. Nothing stimulates the entrepreneurial mind like the lure of cash, but this time there is little to be found.

However, entrepreneurs are not ones to accept "no" for an answer. The online digital world is an engine that requires little startup capital. Profitability for a new online business can often be achieved even for those with little or no money. For this reason there is little wonder that more and more talent is shifting

away from physical products toward the online marketplace.

Losing the War of Electrons: Cities don't realize it but they are losing the war of electrons. Cities operate in the physical world and electrons represent everything in the digital world.

Starting a business in the physical world requires permits, inspections, licensing, tax collection, and monitoring. Constant monitoring. Time delays can often drag on for months. Cities have acted as the perennial gatekeeper and their relationship with the business community is often described as adversarial.

However, in the digital world there are few gatekeepers, and the time it takes to be up and operating is measured in minutes and hours, not months and years. Yes, there may be a requirement for licensing and tax collection, but it tends to be far less painful.

People have a choice, and when they weigh their options between a physical business or a digital one, most often the digital option comes out on top.

As the business world shifts away from bricks and mortar, cities are left with little to claim as their own, and with a declining sale tax base, very little revenue to pay for the services the residents have long come to expect.

Business Colonies: In the future, businesses will operate in a far more fluid manner with talent and projects converging for short periods of time. In much the same way the movie industry works, where a single movie project will attract camera people, script writers, lighting and sound people, actors, and make-up artists, future business projects will attract various skills for temporary assignment. Once the project is complete, team members will disband and form around other projects.

Operating as a free agent often involves a number of challenges that not all are equipped to handle. As a support mechanism for their growing numbers, business colonies will begin to form around such diverse industrial sectors as photonics, nanotech, biotech, IT niches, and many more.

Often times the colonies will be formed to support large corporate players in a specific industry. As an example, companies like Sony, Microsoft, and Nintendo could easily spawn gamer colonies as a way to drive the development of new games for their consoles.

Over the next few years, experimental colonies will proliferate, testing a variety of operational and support systems. Free agents who join as members of the colony will be attracted by the prospects of steady project flow. Project leads will be drawn to the available talent pools. And host cities will be most interested in generating jobs and employment for their constituencies.

Future of Economic Development: The current efforts most communities are now using to attract companies to relocate into their city will need to be rethought.

As the number of telecommuters increases, there are few assurances that the relocation of a company will bring many of its people along with it. Yes, many of the old school employers still require all of their workers to show up in the office every day, but even those who are most determined to resist this trend are showing signs of softening.

According to the Telework Research Network, 40% of U.S. employees hold jobs that that could be done at home (50 million), and half of all U.S. businesses are home based (16 million).

Digging deeper into the statistics, 40% of federal employees are eligible but only 17% do so. Similarly, 36% of private sector employees are eligible but only 16% do so. This indicates vast room for improving both the telecommuting interface and for the numbers to increase.

Alternatively, instead of focusing on the whole company, economic development professionals may want to focus on recruiting key individuals who have the option to live anywhere. Many people who telecommute bring with them two essential ingredients—a budget and hiring authority. In the future, any person who can choose where they want to live and comes with a budget and hiring authority will become a prime target in the new age of economic development.

Next month's column—City of the Future: Part Two.

Interested in sharing your thoughts? Go to www.FuturistSpeaker.com.

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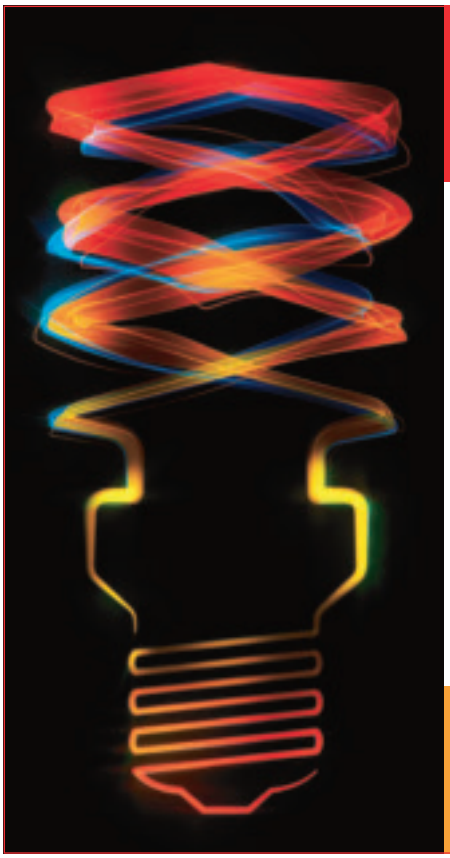

2013 Environmental Health Innovation Award

NEW This new award has been established by NEHA's board of directors to recognize a NEHA member or organization for creating a new idea, practice, or product that has had a positive impact on environmental health and the quality of life. Innovative change that promotes or improves environmental health protection is the foundation of this award.

Environmental health professionals face the dilemma of finding and implementing new ways of doing business without sacrificing the quality of their environmental health programs. This annual award recognizes those who have made an innovative contribution to the field, as well as encourages others to search for creative solutions. Take this opportunity to submit a nomination to highlight the innovations being put into practice in the field of environmental health!

Nominations are due in the NEHA office by March 15, 2013.

For more information, please visit **www.neha.org/about/awardinfo.html.** Nomination materials can be obtained by e-mailing Terry Osner at **tosner@neha.org**.

NEW 2013 Educational Contribution Award

This new award has been established by NEHA's board of directors to recognize NEHA members, teams, or organizations for an outstanding educational contribution within the field of environmental health. This award provides a pathway for NEHA members and environmental health agencies to share creative methods and tools to educate one another and the public about environmental health principles and practices. Don't miss this opportunity to submit a nomination to highlight the great works of your colleagues!

Nominations are due in the NEHA office by March 15, 2013.

For more information, please visit **www.neha.org/about/awardinfo.html**. Nomination materials can be obtained by e-mailing Terry Osner at **tosner@neha.org**.

CAREER OPPORTUNITIES

Food Safety Inspector

Everclean Services is the leader in the restaurant inspections market. We offer opportunities throughout the country. We currently have openings for professionals to conduct Q.A. audits of restaurants.

Alaska	McAllen, TX	Roger, AR
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Fort Lauderdale, FL	Oklahoma City, OK	Virginia Beach, VA
Indianapolis, IN	Omaha, NE	Wichita, KS
Lincoln, NE	Pittsburgh, PA	
Little Rock, AR	Richmond, VA	

Past or current food safety inspecting is required.
Interested applicants can send their resume to: Bill Flynn at Fax: 818-865-0465. E-mail: bflynn@evercleanservices.com.

EH CALENDAR

UPCOMING NEHA CONFERENCES

July 9–11, 2013: Hyatt Regency Crystal City at Reagan National Airport, Washington, DC, Area. For more information, visit www.neha2013aec.org.

NEHA AFFILIATE AND REGIONAL LISTINGS

Arizona
March 21, 2013: AZEHA Spring Conference, sponsored by the Arizona Environmental Health Association. For more information, visit www.azeha.org.

California
April 1–4, 2013: 62nd Annual Educational Symposium, sponsored by the California Environmental Health Association. For more information, visit www.ceha.org.

Idaho
March 13–14, 2013: IEHA Annual Education Conference, sponsored by the Idaho Environmental Health Association. For more information, visit www.ieha.wildapricot.org.

Michigan
March 20–22, 2013: MEHA Annual Educational Conference, sponsored by the Michigan Environmental Health Association. For more information, visit www.meha.net/aec/.

Minnesota
January 31, 2013: MEHA Winter Conference, sponsored by the Minnesota Environmental Health Association. For more information, visit www.mehaonline.org/events.

Nevada
January 30–31, 2013: 2013 Southwest Environmental Health Conference, hosted by the Arizona County Directors of Environmental Health Services Association. For more information, visit www.southwestconference.net.

Ohio
April 23–24, 2013: 2013 Spring Annual Education Conference, hosted by the Ohio Environmental Health Association. For more information, visit www.ohioeha.org/AnnualEducationalConference.aspx.

Washington
May 6–7, 2013: 2013 Educational Conference, hosted by the Washington State Environmental Health Association. For more information, visit www.wseha.org/workshops.html.

Wisconsin
March 12, 2013: WEHA Spring Educational Conference, hosted by the Wisconsin Environmental Health Association. For more information, visit www.weha.net.

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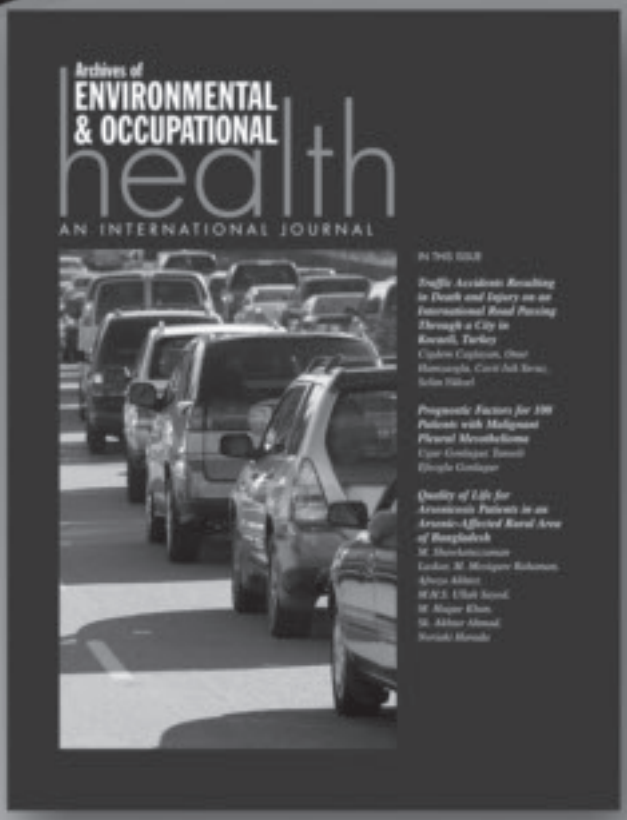

For more than 90 years, **Archives of Environmental & Occupational Health** has provided objective documentation of the effects of environmental agents on human, and in some cases, animal populations. This noted journal consolidates the latest research from such varying fields as epidemiology, toxicology, biostatistics, and biochemistry.

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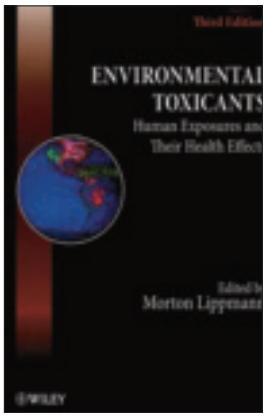
Environmental Toxicants: Human Exposures and Their Health Effects (Third Edition)

Edited by Morton Lippmann (2009)

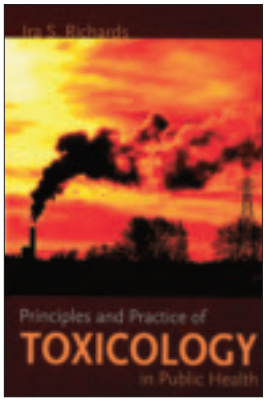


The third edition of this book has been thoroughly updated and revised with the latest findings on the effects of human exposure in nonoccupational settings to chemical agents and physical factors. It offers the most current information on performing and analyzing the results of risk assessments for exposed individuals and populations. In addition to examining individual toxicants, the book explores broader social and scientific issues such as individual and community risk, environmental engineering for risk reduction, pulmonary medicine, and lessons learned from the industrial sector.

1,167 pages / Hardback / Catalog #1076
Member: $184 / Nonmember: $194

Principles and Practice of Toxicology in Public Health

Ira S. Richards (2008)

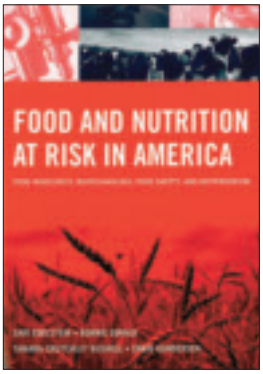


In four sections, this book offers an introduction to the field of toxicology, as well as the basics of toxicology principles, systemic toxicity, and toxicology practice. It offers thorough coverage of the basic principles of toxicology without being too technical or specialized. The text uses reader-friendly language making it accessible to professions from a variety of backgrounds including environmental health, industrial hygiene, engineering, and more. Finally, it includes a section on the application of toxicology in the field.

464 pages / Paperback / Catalog #800
Member: $85 / Nonmember: $89

Food Nutrition at Risk in America: Food Insecurity, Biotechnology, Food Safety, and Bioterrorism

Sari Edelstein, Bonnie Gerald, Tamara Crutchley Bushell, and Craig Gunderson (2009)

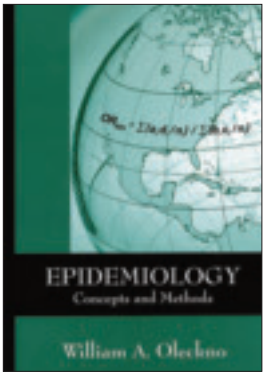


This book addresses the major food and nutrition issues of our time. Each section covers the latest threats to our nation's food systems, such as international and unintentional contamination of the food supply, food insecurity issues within our borders, and the effect of crop manipulation on human health. This groundbreaking and thought-provoking text offers readers the opportunity to consider the current status of pressing food safety issues, as well as the types of assistance and policies needed in the future to ensure the health and welfare of Americans.

315 pages / Paperback / Catalog #1085
Member: $89 / Nonmember: $99

Epidemiology: Concepts and Methods

William A. Oleckno (2008)



Comprehensive in its coverage, this text is a full-scale, pedagogically rich introduction to fundamental ideas and procedures in epidemiology. It covers the major concepts, principles, methods, and applications of both conventional and modern epidemiology using clear language and frequent examples to illustrate important points and facilitate understanding. While the author provides thorough treatment of the more customary aspects of conventional and modern epidemiology, he also introduces several important design and analytical issues that are only rarely approached in fundamental epidemiology textbooks. Concepts as diverse as competing risks, maturation, futility, and the prevalence and bias effects in the context of screening are just a few examples of the broad range of concepts covered in this text.

649 pages / Paperback / Catalog #763
Member: $51 / Nonmember: $54

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JEH QUIZ

FEATURED ARTICLE QUIZ #4

Fish Consumption Patterns and Mercury Exposure Levels Among Women of Childbearing Age in Duval County, Florida

Available to those holding an Individual NEHA membership only, the *JEH* Quiz, offered six times per calendar year through the *Journal of Environmental Health,* is a convenient tool for self-assessment and an easily accessible means to accumulate continuing-education (CE) credits toward maintaining your NEHA credentials.

1. Read the featured article carefully.
2. Select the correct answer to each *JEH* Quiz question.
3. a) Complete the online quiz at www.neha.org (click on "Continuing Education"),
   b) Fax the quiz to (303) 691-9490, or
   c) Mail the completed quiz to
   JEH Quiz, NEHA
   720 S. Colorado Blvd., Suite 1000-N
   Denver, CO 80246.
   Be sure to include your name and membership number!
4. One CE credit will be applied to your account with an effective date of January 1, 2013 (first day of issue).
5. Check your continuing education account online at www.neha.org.
6. You're on your way to earning CE hours!

Quiz Registration

Name

NEHA member number

Home phone

Work phone

E-mail

→ Quiz deadline: April 1, 2013

1. The U.S. Environmental Protection Agency human health criterion for fish mercury concentration is
   a. 0.3 parts per million.
   b. 1 part per million.
   c. 3 parts per million.
   d. 30 parts per million.
2. Mercury exposure is influenced by
   a. sources of local pollution.
   b. population demographics.
   c. commonly eaten fish species.
   d. cultural fish consumption habits.
   e. all of the above.
3. The greatest source of mercury in the environment is
   a. naturally occurring.
   b. medical waste pollution.
   c. industrial pollution.
   d. all of the above.
   e. none of the above.
4. Coal-burning power plants are the largest contributors of mercury air pollution in the U.S. accounting for over __ of total domestic human-made mercury emissions.
   a. 30%
   b. 50%
   c. 70%
   d. 90%
5. Florida receives much of its mercury deposition from domestic sources.
   a. True.
   b. False.
6. Which of the following is not considered a "high-risk" fish?
   a. Shellfish.
   b. King mackerel.
   c. Swordfish.
   d. Chilean sea bass.
   e. All are considered a "high-risk" fish.
7. Among the study group, __ of women reported eating a high-risk fish species in the past 60 days.
   a. 14.4%
   b. 25.2%
   c. 30.8%
   d. 31.4%
8. The Food and Drug Administration's (FDA's) recommendation regarding fish consumption for women and young children is
   a. two fish meals per month.
   b. four fish meals per month.
   c. eight fish meals per month.
   d. 12 fish meals per month.
9. The mean monthly fish consumption for the total study population was __ FDA's recommended monthly consumption.
   a. higher than
   b. the same as
   c. less than
10. The study showed that women with the __ household income had the __ mercury levels.
    a. lowest; lowest
    b. lowest; highest
    c. highest; lowest
    d. highest; highest
11. Knowledge of mercury and its related fish consumption advisories __ age and education.
    a. decreased with
    b. remained the same regardless of
    c. increased with
12. Studies have shown that commercially caught fish may have higher mercury levels than fish caught from local contaminated waterways.
    a. True.
    b. False.

JEH Quiz #2 Answers
October 2012

1. e	4. a	7. a	10. b
2. b	5. c	8. b	11. c
3. d	6. c	9. d	12. b

Association of Environmental Health Academic Programs *Announces*

The 2013 AEHAP/NCEH Student Research Competition

for undergraduate and graduate students enrolled in an EHAC-accredited program or an environmental health program that is an institutional member of AEHAP

Win A $500 AWARD

and up to $1,000 in travel expenses

Students will be selected to present a 20-minute platform presentation at the National Environmental Health Association's Annual Educational Conference & Exhibition in Washington, DC, July 9–11, 2013.

Entries must be submitted by April 8, 2013, to
Dr. David Gilkey
Colorado State University
146 EH Building
Fort Collins, CO 80523-1681
E-mail: dgilkey@colostate.edu

For additional information and research submission guidelines, please visit www.aehap.org.

AEHAP gratefully acknowledges the support of the National Center for Environmental Health, U.S. Centers for Disease Control and Prevention, for this competition.

Opportunity for Students

From EHAC-Accredited Environmental Health Degree Programs to Win a $3,500 PAID INTERNSHIP

The Association of Environmental Health Academic Programs (AEHAP), in partnership with NSF International, is offering a paid internship project to students from National Environmental Health Science and Protection Accreditation Council (EHAC)-accredited programs. The NSF International Scholarship Program is a great opportunity for an undergraduate student to gain valuable experience in the environmental health field. The NSF Scholar will be selected by AEHAP and will spend 8–10 weeks (March–May 2013) working on a research project identified by NSF International.

Project Description
The applicant shall work with a professor from their degree program who will serve as a mentor/supervisor and agree to providing a host location from which to do the research. The research project involves administering a survey of the 50 states to determine how they have responded to the 2009 FDA *Model Food Code*. This project is a continuation of a research project started by the 2009 NSF Scholar.

Application deadline: January 18, 2013

For more details and information on how to apply please go to www.aehap.org/resources/student-resources/aehap-scholarships/nsf-paid-summer-internship-opportunity-for-students

For more information, contact info@aehap.org or call 206-522-5272.

NSF

AEHAP
ASSOCIATION OF ENVIRONMENTAL HEALTH ACADEMIC PROGRAMS

SUPPORT THE NEHA ENDOWMENT FOUNDATION

The NEHA Endowment Foundation was established to enable NEHA to do more for the environmental health profession than its annual budget might allow. Special projects and programs supported by the foundation will be carried out for the sole purpose of advancing the profession and its practitioners.

Individuals who have contributed to the foundation are listed below by club category. These listings are based on what people have actually donated to the foundation—not what they have pledged. Names will be published under the appropriate category for one year; additional contributions will move individuals to a different category in the following year(s). For each of the categories, there are a number of ways NEHA recognizes and thanks contributors to the foundation. If you are interested in contributing to the Endowment Foundation, please fill out the pledge card or call NEHA at 303.756.9090.

Thank you.

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I maintain that these perspectives collectively point us to different universes than the one I see unfolding before us. By some magic, in these alternative universes, the laws of physics, economics, and politics make it possible to turn back clocks, create wealth from deficits, give people expensive public services for no cost, and impose public policies on large segments of the population who oppose them. That's not the universe that I live in and understand.

The universe that I see is one that is being reshaped by budget deficits and problematic revenue streams, the automation of routine intellectual work (that is displacing large segments of the middle class including many who practice in environmental health), the globalization of the production and distribution of goods and services, the rise of e-commerce, the emergence of "big data" that draws increasing volumes of information from sensors and simulations (which will eliminate the need for many of the inspections currently being done by professions like ours), and the changing nature of employment (as work groups move from formalized structures into increasingly virtual—and less expensive—work environments). And these are only a few of the winds that are blowing us into the future.

Yes, we have been battered by an economic event that has no equal since the Great Depression. And yes, that particular event was caused by several specific factors such as the bursting of a real estate bubble and the overnight disappearance of billions of dollars of personal equity, the collapse of our financial system, the disappearance of credit, and a failure in regulatory oversight. But the full story of what is currently happening out there extends well beyond these oft-cited reasons for the recession. We risk not seeing these other factors at play when we simply (and dismissively) wave our hands and blame "the economy" (whatever that is) for our problems. We also take out of our hands the power to do something about both our situation and more importantly, our future, when we obliquely define the problem as "the economy." In order to see these other factors and appreciate them for what they are, let's spend a moment talking about one of the fundamental arcs of human history.

From our beginnings, humankind has been driven by an unrelenting quest to improve our lots in life. To put it another way, we've spent an entire history figuring out how to do more for less. This force has been particularly strong in market economies where efficiencies and productivity gains tend to get rewarded with financial gain.

In the exciting book *Abundance*, the authors (Peter Diamandis and Steven Kotler) talk about how in the 1800s, going from Boston to Chicago took two weeks and a month's earnings. Today it takes two hours and a day's wage. According to the Cato Institute, just a decade ago, it took 70 employees to sort 35,000 letters in an hour. Today it takes two. Other such examples of finding ways to get more for less and improve the quality of our lives in the process are literally countless. The examples are countless because this is the way society has worked since we first learned to walk.

Matt Ridley is a zoologist who was taught and trained at Oxford. He studies human evolution and behavior. He has a most interesting definition of prosperity. He defines it simply as "saved time." The less time it takes us to do something, the more time we have to do what we want, which is prosperity. (And since time is money, the more time we save, the more money we have.)

In short and like it or not, we live in a universe that drives itself to ***continuously*** find ways to do more for less. When one considers the growing capabilities of automation and the employment implications of a global economy and then realizes that they are but manifestations of this quest to find ways to do more for less, it can't be surprising when we hear people like Tom Frey warn us that significant numbers of midlevel routine professional jobs will increasingly be replaced by computer software and machines. And in this universe, no amount of protesting is going to decelerate this primal force. Moreover and especially with the budgetary pressures that are bearing down on governments all over the world today, it's not just in the private sector that we will see changes of this nature play out. Indeed, when government finance officers, city managers, and county administrators talk about new normal, this is precisely what they are talking about.

Not convinced?

Through our new Center for Priority Based Budgeting program, we now have empirical evidence as to what is going on "out there" in community after community in America. Reams of data can be simplified and summarized in one simple story.

For decades, the sacred cow of local government has been public safety (often defined as police and fire). In the past, when cutting became necessary, local officials were quick to confer immunity on these programs. No more!

In this era of new normals, some fascinating things are happening.

The New York Times recently reported that Sacramento's police department has been cut by a whopping 30%+ since 2008. Camden, New Jersey, just closed its police department altogether, ceding police control to the county. Our Center program is learning that many other communities are courageously unbundling police and fire programs and are eliminating those police and fire activities that don't directly impact public safety.

If communities are cutting these sacred cows—and they are in legions—who in the world could possibly believe that other public service professions such as ours aren't also vulnerable to cuts? And yet, in those alternative universes that many of our leaders and professional societies fixate on, we are led to believe that if we make enough noise and send enough letters to policy makers, our funding levels will increase as if revenues can be created based on decibels and postage stamps. That's neither the way I think our particular universe works nor the way that I would want it to work, if I had the power to make it so. If our profession's future simply depended on an ever-present supply of funding, where would the pressure for innovation and accomplishing more (for less) come from? Where would the excitement of discovering new and better ways for doing our work come from? And where would the drive to improve our skill sets and even learn skills more appropriate for 2020 come from?

And even if we have the chance to do so, would we really want our profession to become known for being separated from that arc of human history that drives humanity to advance through discoveries that enable us to do more for less?

So what is the point of all this?

A favorite quote of mine is that there is no future, there are only futures. Accordingly, we are standing here today looking out upon a

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LETTERS TO THE EDITOR

Toward a National Biomonitoring System

Dear Editor:
As one of the authors of "State Public Health Laboratory Biomonitoring Programs: Implementation and Early Accomplishments," which appears in this issue, I wanted to provide some updates to accompany the article.

As noted, concerns about human exposure to chemicals in our environment continue to increase. Many believe that "biomonitoring" remains one of the best ways to address related questions. The current design of the Centers for Disease Control and Prevention's (CDC's) National Report on Human Exposure to Environmental Chemicals, however, does not allow calculation of exposure estimates on a state-by-state or city-by-city basis. That is why CDC funds states to do their own biomonitoring, and why the Association of Public Health Laboratories (APHL) calls for a National Biomonitoring Network (APHL, 2009).

Since Dr. Fox interviewed the three states funded by CDC to do biomonitoring (in February and March 2011), both the states and APHL made significant advances.

California

"Upholstery in airplanes, hotels, nursing homes, hospitals, and even prisons is required to be fire resistant, but outside of California, there is no flammability standard for upholstered furniture sold for use in the home (National Association of State Fire Marshals, 2006)." This serves as one very simplified reason (of many) why Californians experience disproportionate exposures to chemicals appropriate for biomonitoring, particularly flame retardants (i.e., polybrominated diphenyl ethers [PBDEs]). Other potentially important exposures include agricultural pesticides and mercury in fish. Biomonitoring California conducts several collaborative studies investigating these and other exposures in specific subpopulations.

The Biomonitoring Exposures Study is one collaboration the program initiated. Biomonitoring California and Kaiser Permanente Northern California's (KPNC) Division of Research work together to measure chemical exposures in a representative sample of adult KPNC members in the state's central valley. Analysis focuses on environmental chemicals or their metabolites, including PBDEs, organophosphate and pyrethroid pesticides, mercury and other metals, phthalates, and environmental phenols (such as bisphenol A [BPA]).

New York

An idyllic-looking, all-American town, Colonie holds the title of most-populous suburb of Albany. Regularly, the town ranks among the safest places to live in the U.S. (Wikipedia, 2012). It's the soil that may spoil this town's reputation.

Texas-based NL Industries operated in Colonie for 24 years until a state Supreme Court closed the factory for illegal uranium emissions. Before then, NL used depleted uranium and thorium to make armor-piercing munitions. During its operation, these and other potentially toxic materials released from exhaust stacks spread to site buildings, the plant's grounds, and 56 nearby properties.

The New York State Department of Health began work to determine total uranium concentrations in the urine of former plant workers and other individuals who lived or worked nearby, as well as uranium isotope ratios in a subset of workers and residents as a "fingerprint" of depleted uranium exposure.

Washington

Compounds of arsenic (referred to as the *Poison of Kings* and the *King of Poisons* [Vahidnia, Van Der Voet, & De Wolff, 2007]) once comprised the most widely applied pesticides in Washington State. In addition, past emissions from metal-refining contaminated Washington soil with arsenic (Washington State Department of Health, 2012). This arsenic binds strongly to soil and remains near the surface of the land for hundreds of years.

Washington's Environmental Biomonitoring Survey collected urine from a random sampling of the general population for both total and speciated arsenic. Other chemicals of concern include trace elements, metabolites of pyrethrin-based pesticides (pyrethroids), as well as organophosphate pesticides (OPs), phthalates, and BPA. At the end of Year 3, the team completed trace elements, total arsenic, and speciated arsenic for over 1,500 specimens. The laboratory is more than halfway through the pyrethroid/OP study and is gearing up for phthalates and BPA.

Results indicate that indeed, Washington's residents have higher levels of arsenic in their urine compared to the general U.S. population. Since that study, the local health department advised those with the highest risk on lowering their arsenic exposure, while the Washington Non-Infectious Conditions Epidemiology office is examining disease data for correlations.

Association of Public Health Laboratories

APHL's work toward building a national biomonitoring system envisions a coordinated approach to the design and development of exposure studies, more effective use of limited resources, higher-quality data, improved environmental policy and practice, data sharing, and eventually a healthier population.

The recently published APHL *Guidance for Laboratory Biomonitoring Programs* (APHL, 2012) represents one way to assure a more efficient and effective system among state and national laboratory partners by supporting high-quality standardized laboratory practice. The Council of State and Territorial Epidemiologists recently completed a companion document for epidemiologists.

In addition, APHL recently launched a Biomonitoring Database (APHL, 2011) to increase efficiency and collaboration among members as well as with academia, toxicologists, epidemiologists, community advocates, and environmental health practitioners. Using this tool, member laboratories compile biomonitoring capability information, including analytes, matrices and instrumentation,

LETTERS TO THE EDITOR

as well as methods, collaboration opportunities, and discussions. Partners can search the database based on biomonitoring needs and reach out to labs meeting their criteria.

Conclusion

Funded states, APHL, and CDC continue progressing toward a national biomonitoring system. Despite no additional funding and significant barriers, major advancements continue. Biomonitoring remains an important environmental health tool that, while underutilized, continues to be identified as crucial to advancing the field and meeting community environmental health needs (National Conversation Leadership Council, 2011; National Research Council of the National Academies, 2006).

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References

Association of Public Health Laboratories. (2009). *National biomonitoring network: Five-year plan.* Retrieved from http://www.aphl.org/aphlprograms/eh/Pages/nationalbioplan.aspx

Association of Public Health Laboratories. (2011). *Biomonitoring database.* Retrieved from http://www.aphl.org/aphlprograms/eh/biomonitoringdatabase/pages/default.aspx

Association of Public Health Laboratories, Biomonitoring Subcommittee. (2012). *Guidance for laboratory biomonitoring programs.* Retrieved from http://www.aphl.org/AboutAPHL/publications/Documents/EH_2012_Guidance-for-Laboratory-Biomonitoring-Programs.pdf

National Association of State Fire Marshals. (2006). *National furniture flammability standard: Frequently asked questions.* Retrieved from http://www.firemarshals.org/pdf/Oct30pressconf_FAQ.pdf

National Conversation Leadership Council. (2011). *Addressing public health and chemical exposures: An action agenda.* Retrieved from http://www.nationalconversation.us/

National Research Council of the National Academies. (2006). *Human biomonitoring for environmental chemicals.* Washington, DC: National Academies Press.

Vahidnia, A., Van Der Voet, G.B., & De Wolff, F.A. (2007). Arsenic neurotoxicity—a review. *Human & Experimental Toxicology, 26*(10), 823–832.

Washington State Department of Health. (2012). *Arsenic.* Retrieved from http://www.doh.wa.gov/CommunityandEnvironment/Contaminants/Arsenic.aspx

Wikipedia. (2012). *Colonie, New York.* Retrieved from http://en.wikipedia.org/wiki/Colonie,_New_York

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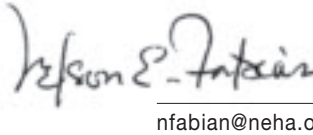
host of different futures and universes. (Some physicists argue that every future possibility opens up a new universe.) Some of these universes hold the promise of environmental health becoming a much more daring and exciting profession. Others, unfortunately, point toward downward slides into oblivion at worst or marginal relevance at best.

NEHA members deserve to know that your association has not been using its resources to wage protracted battles against forces that cannot be defeated. Rather, NEHA has been searching for ways to understand and master these forces, all for the purpose of opening up opportunities for you and for the field of environmental health at large. We seek to invigorate the profession with new ideas, skills (the subject of my next column), and even responsibilities.

This is why we have teamed up with Decade Software as we seek to push IT sophistication and capability across this profession. This is why we continue to push the limits with our educational program at the Annual Educational Conference & Exhibition. This is why we're exploring partnerships with other professions as we seek to form multidisciplined teams to tackle problems that are multifaceted in nature. And this is why we have been so energized over leading the profession into new areas of practice that include sustainability, health effects of global climate change, healthy communities, and the built environment.

The bottom line is that we see our mission as being tied up in an all-out effort to find a universe within which both you and our profession can be breathtakingly successful. If we land in any other universe, we will have failed both you and our mission.

And that's as unacceptable as living in a universe without Peyton Manning quarterbacking our Broncos here in Denver!



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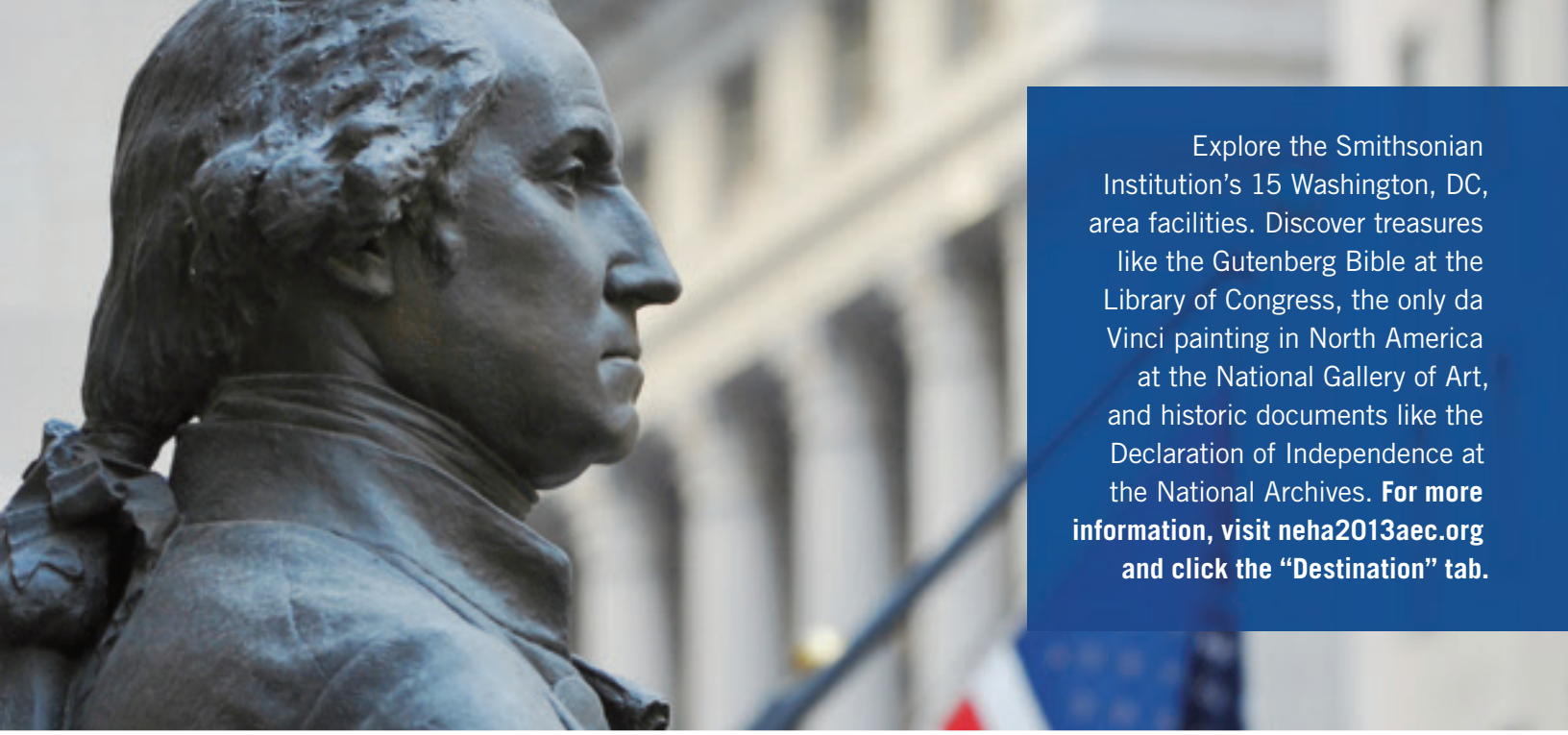

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REASONS WHY

Attending the NEHA AEC Is a Wise Investment for You and Your Organization

Difficult times make it more important than ever that you NOT miss the skills, knowledge, and expertise that can be derived from the NEHA AEC, which can help you and your organization build for a better tomorrow.

1. The NEHA AEC is a unique opportunity for you to gain the skills, knowledge, and expertise needed to help solve your environmental health organization's daily and strategic challenges, and to make recommendations to help improve your bottom-line results.
2. NEHA's AEC is the most comprehensive training and education investment your organization can make all year.
3. Your attendance at the NEHA AEC is a solid investment in your organization that will result in immediate and longer-term benefits.
4. You can earn Continuing Education (CE) credit to maintain your professional credential(s).
5. NEHA provides a return on the investment made for you to attend the AEC.

Need additional reasons why you should attend?

Check out the videos on neha2013aec.org to hear what other environmental health professionals are saying about the NEHA AEC.

Experience Washington, DC! Though the NEHA 2013 AEC venue is technically in Arlington, Virginia, you will be just a few short minutes away from all that Washington, DC, has to offer. Enjoy access to fascinating, FREE attractions and historic sights. **For more information, visit neha2013aec.org and click the "Destination" tab.**

KEYNOTE SPEAKER

NEHA is honored to announce Dr. Graham Allison as the keynote speaker at the 2013 AEC. Dr. Allison will speak to the topic, "What Do the Cuban Missile Crisis and Environmental Health Have in Common?"

This is a unique opportunity to hear from an expert with experience at the highest level of government discuss policy and share lessons learned in decision making. Register today for the 2013 AEC so you don't miss this opportunity!

Dr. Allison has served as Special Advisor to the Secretary of Defense under President Reagan and as Assistant Secretary of Defense for Policy and Plans under President Clinton, where he coordinated Department of Defense strategy and policy towards Russia, Ukraine, and other states of the former Soviet Union. During his keynote presentation at the NEHA 2013 AEC, Dr. Allison will talk about decision making in the most extreme of circumstances where literally the fate of the planet hangs in balance. The insights that he has learned about decision making will be shared to benefit each and every environmental health professional who is involved daily in decisions regarding politics, policies, finances, technology, human resources, legal considerations, liabilities, and of course, environmental health!

Dr. Allison has the sole distinction of having twice been awarded the Department of Defense's highest civilian award, the Distinguished Public Service Medal. In addition, he is the author of *Essence of Decision: Explaining the Cuban Missile Crisis*, an all-time bestseller, and *Nuclear Terrorism: The Ultimate Preventable Catastrophe*, which was selected by the New York Times as one of the "100 most notable books of 2004."

Additional biographical information about Dr. Allison is posted on neha2013aec.org

The keynote speaker is sponsored by NSF International.

NEHA 2013 AEC

PRELIMINARY SCHEDULE

You can find detailed session and event information on neha2013aec.org beginning in January.

Sunday // July 7	Monday // July 8	Tuesday // July 9	Wednesday // July 10	Thursday // July 11
Pre-Conference Workshops	Pre-Conference Workshops	1st Time Attendee Workshop	Town Hall Assembly	Educational Sessions
Credential Review Courses	Credential Review Courses	Educational Sessions	Exhibition Open	Networking Luncheon
	Credential Exams	Awards Ceremony & Keynote Address	Poster Session	President's Banquet
	Golf Tournament	Exhibition Grand Opening & Party	Silent Auction	
	Community Volunteer Event		Student Research Presentations	
	Annual UL Event		Educational Sessions	

REGISTER TODAY!

Registration information is available at neha2013aec.org. For personal assistance, contact Customer Service toll free at 866.956.2258 (303.756.9090 local), extension 0.

	Member	Non-Member
Full Conference Registration	$565	$725
One Day Registration	$305	$355
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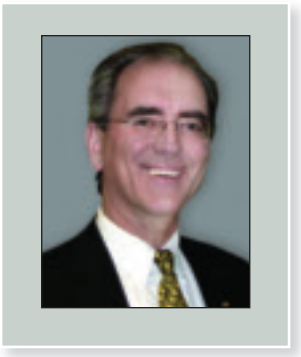
MANAGING EDITOR'S DESK



Nelson Fabian, MS

Parallel Universes

Many serious physicists believe that reality encompasses an infinite number of parallel universes. As of late, I'm thinking that maybe I've become aware of some of them. Allow me to explain.

If you've read even a handful of my columns over the past several years, you've probably noticed my growing obsession with the future. It fascinates and excites me to think of the future possibilities for environmental health.

This obsession also explains why I brought the highly acclaimed futurist, Tom Frey, into the pages of our *Journal*. I am hoping that his column serves to bring the future to life for many of our readers. The more familiar we can get with the future, the easier it becomes to imagine what the future of environmental health can be. Developing an intuition for the possibilities before us helps us to better understand how our journey into the future could and should unfold.

For our upcoming trip into the future, it's important to know where we're starting from. If we get our starting point wrong, we're likely to build our trip on any number of incorrect assumptions. And depending on the assumptions we hold, trips into any number of different universes are possible. My column will argue that our starting point needs to aim us at a universe that allows for a rich and dynamic professional practice. I will also point out that other starting blocks aim us at other universes where environmental health fares much more poorly. Which universe we end up in depends to an extraordinary extent on our starting point and the assumptions that define it. So let's start there.

Any understanding of our profession's "present" must necessarily include the sobering realization that environmental health is one of many professions that is today being rocked by economic convulsions that themselves are being driven by technology, automation, the emergence of an integrated global economy, the puncture of the real estate bubble and the resulting consequences of severe revenue miscalculations on the part of many communities, geopolitics, and even new models for how companies and governments can work (to name a few!). But blaming our plight on the economy (as most commentaries invariably tend to do) takes us to that same mushy and useless place that troubled couples often find themselves in when they blame the equally amorphous generalization of "poor communication" as the reason for their troubles. "The economy" or "poor communication" doesn't give us anything to grab ahold of and really work with.

And in this universe, no amount of protesting is going to decelerate this primal force.

If our condition is seen as only a function of the economy, then we're pretty much reduced to ***hoping*** that better days will come because none of us have any real power to change today's economic circumstances. And as people smarter than me have written, "hope is not a strategy" for accomplishing anything.

If we are to have any chance of actually doing something that will benefit our cause and profession as it journeys into the future, we need to understand the present in terms of the forces that are causing the economic stresses that we see and feel (and that have led to downsizings, layoffs, and restructurings in our profession and many others). To understand the real drivers of change is to understand how we can adapt, adjust, and even ride these forces in ways that enable us to improve environmental health. The alternative is to simply stand aside and let the fate of our profession be defined by where we crash land after the storm. At least for NEHA, that's not an acceptable option.

Unfortunately, I hear very little about how public and environmental health might successfully ride these forces into the future. To the contrary, many of our leaders seem more inclined to engage in conversations that are hopeless (the economy has changed and we are victims of it and there's nothing we can do about it), self-pitying (we are victims of forces beyond our control and it isn't fair), fruitless (we need to win the political battle and find a way to get our funding increased [ain't gonna happen folks]), or fantasy (we had a good thing going in 1982 and we have to find a way to rebuild the system to what it looked like then).

continued on page 119

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