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GAMBLING WITH YOUR HEALTH

*Bacterial Contamination on
Casino Gaming Chips*



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ABOUT THE COVER



This month's cover article, "Gambling With Your Health: Bacterial Contamination on Casino Gaming Chips," explores the risk of exposure to infectious diseases, especially bacterial diseases, that are found on casino gaming chips. Never used and in-use chips were tested for bacterial and fungal contamination and the overall results were statistically significant for the presence of pathogenic contamination. The results show that chips can be carriers of organisms that can cause illness and highlights the need to create effective disease-prevention strategies for the safe handling and use of chips in casinos.

See page 8.

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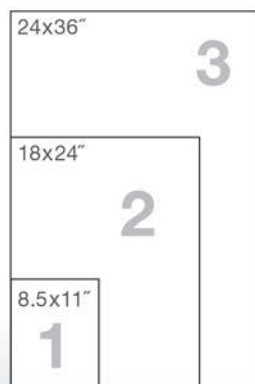
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► PRESIDENT'S MESSAGE



Vince Radke, MPH, RS,
CP-FS, DLAAS, CPH

Can You Hear Me?

The lesson for all of us is to listen to the people in our communities.

Back in the early 1980s, I was director of environmental health for the city of Stamford, Connecticut, and had a staff of eight sanitarians and a secretary. We did the typical work of an environmental health staff. We performed inspections of restaurants (all paper based with carbon copies), septic systems, private drinking water wells, solid waste, complaints, and foodborne and waterborne illness outbreaks. We also had programs for outdoor air monitoring, recreational water (beaches and swimming pools), and recreational shellfishing.

At that time, the U.S. Environmental Protection Agency (U.S. EPA) put out a request for proposals for grant money to local public health departments for noise reduction projects. We submitted our proposal and it was approved. Our noise reduction project had several components. Our environmental health staff worked with community leaders, faith-based groups, businesses, and schools to draft an ordinance for the mayor and city council to consider. After discussion, debate, and some amendments, the ordinance was approved. The ordinance established noise levels that were based on the science, medicine, and technology of the time for both stationary and mobile (e.g., trucks and cars) sources of noise. Our environmental health section handled the stationary sources of noise. The local police department handled the mobile sources of noise. We did not handle workplace noise issues. As part of the U.S. EPA grant, the health and police departments received noise meters, training, and consultation from subject matter experts from U.S.

EPA. Part of the training included the negative health impacts of noise.

The ordinance required the health department to investigate complaints of noise within the city limits of Stamford. Based on the complaint, we would monitor the noise levels over time. Sometimes we were required to monitor noise levels late at night. If necessary, we would issue warnings to those causing the noise. If after multiple warnings the noise level continued to be above the level established in the ordinance, a court summons was obtained and a fine could be assessed.

Noise is defined as unwanted sound. It has been well established by U.S. EPA, the World Health Organization, and other medical and health organizations that excessive noise causes serious harm to human health and interferes with people's daily lives. Noise, by some, is considered the most pervasive pollutant. Given the extent of its negative impact on health, noise is a very important hazard to monitor and control. Excessive noise can result in negative physiological and psychological effects on exposed individuals. The physiological effects include hearing loss, increased high blood pressure, stress, and

fatigue. The psychological effects can be loss of concentration, reduced performance, sleep disturbance, and depression. Excessive noise interferes with communication, including the difficulty in hearing a conversation, misunderstanding what is being said, or missing a warning signal.

The National Institute for Occupational Safety and Health (2019) states that in the U.S., hearing loss is the third most common chronic physical condition among adults. It is twice as prevalent as diabetes or cancer. About 11% of the working population has hearing difficulty and about 24% of the hearing difficulty among U.S. workers is attributed to occupational exposures.

Excessive noise leading to hearing loss also has a negative economic impact. Hearing loss not only contributes to lower productivity but also leads to lower income. Furthermore, there is the additional cost to provide health and other services for those with hearing loss. It is estimated that hearing loss cost \$297,000 over the lifetime of every affected person (Mohr et al., 2010). The national cost of initial hearing loss treatments is projected to multiply 6-fold between 2002 and 2030 from \$8.2 billion to \$51.4 billion (Stucky, Wolf, & Kuo, 2010).

As I mentioned at the beginning of this column, I applied for a U.S. EPA noise grant. I applied for the grant because the health department had received noise complaints from Stamford residents. The lesson for all of us is to listen to the people in our communities.

In 1974, a U.S. EPA report identified 70 decibels (dB) over 24 hours (75 dB over 8 hours) as the average exposure limit to environmental

noise. They identified levels of 55 dB outdoors and 45 dB indoors as the highest average levels of noise that will permit spoken conversation, sleeping, working, and recreation. These are average levels, not peak levels. Occasional higher noise levels should not cause noise-induced hearing loss if the 24 hours include a sufficient amount of quiet time for hearing recovery between high noise level exposures. These limits are not regulations but guidance. They give you, your community, and local and state governments the basic information to use in setting regulations. As I did with my community, you can work with individuals and organizations in your community to draft ordinances related to noise. Our noise ordinance in Stamford was related to complaints—noise annoyance rather than hearing hazard risks. Your ordinance might require warning

signs, the use of hearing protection, or lower noise levels between certain hours. In 2014, the Minneapolis city council passed an ordinance that required bars and clubs to offer free ear plugs to customers.

I will end with a few personal notes. I have some noise-induced hearing loss. I carry ear plugs with me and have a sound meter app on my smartphone. I look forward to hearing from all of you (at 45 dB or lower) at the National Environmental Health Association's 2019 Annual Educational Conference & Exhibition in Nashville, Tennessee, in just a few months on July 9–12 (see page 46 for information on the conference). 🎧

Vince _____
 President@neha.org

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Did You Know?

NEHA will host its Third Annual Hill Day in Washington, DC, on May 1. NEHA's board of directors will meet with elected officials and their staff to discuss the importance of environmental health. Stay tuned to www.neha.org for more information about this event!

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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.

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Gambling With Your Health: Bacterial Contamination on Casino Gaming Chips

Edward G. Mc Keown, PhD

Abstract The casino environment, consisting of employees and customers, can present a risk for exposure to infectious diseases, especially bacterial diseases that are found on casino gaming chips. The purpose of this study was to replicate a study from 2011 to determine bacterial microorganisms on casino chips. A total of 26 chips (13 used actively in a casino and 13 never used from a chip manufacturer) were used for the study. Bacteria and fungi development were found in statistically significant numbers ($p < .05$). Contamination was found on used versus unused chips based on the location being tested, namely the obverse (the side of the chip bearing the head or principal design), reverse, or edge of the chip—with overall results being statistically significant for the presence of pathogenic contaminants. This study also determined that the chips showed the presence of *E. coli* at statistically significant levels.

Introduction

According to Saldmann (2008), the list of illness-causing bacteria and viruses that can be spread through casual hand-to-hand or inanimate object-to-hand contact includes: *E. coli*, *Tatumella ptyseos*, *Serratia plymuthica*, *Citrobacter freundii*, *Proteus penneri*, *Erwinia*, and *Helicobacter pylori*. By coming into contact with objects that have been contaminated by individuals who are carriers of these illness-causing bacteria and viruses, these infectious diseases can be spread through casual human contact. Further, if bacteria or viruses are deposited on an object, (e.g., someone infected with human influenza sneezes without covering their mouth), then the infectious bacterial

organisms can live from several hours to up to 5 months on inanimate objects, depending on the environmental conditions (Brady, Fraser, Dunlop, Paterson-Brown, & Gibb, 2007; Kramer, Schwebke, & Kampf, 2006; Rutala, White, Gergen, & Weber, 2006; Saldmann, 2008).

In the medical and healthcare field, hand washing practices are determined by monitoring the bacterial levels located on objects such as keyboards and wireless communication devices (Brady et al., 2007; Rutala et al., 2006). The results of these studies show that despite continual use and cleaning, disinfectants were continually required to ensure that disease-causing microorganisms were controlled to safe levels (Brady et al., 2007;

Rutala et al., 2006). This vigilant approach is critical, especially in light of research that disease-causing viruses can remain on everyday surfaces such as door knobs, desk tops, and chairs—even after disinfectants have been used to sanitize the contaminated area (Terpstra et al., 2007).

One of the major barriers to effectively controlling the spread of infectious diseases is proper personal hygiene, particularly hand washing. The Centers for Disease Control and Prevention (CDC) has worked to create the Clean Hands Count (CHC) campaign in an effort to “create and support coordinated, sustained initiatives to significantly improve health and save lives through clean hands” (Centers for Disease Control and Prevention [CDC], 2018a). Research has shown that public restrooms are a source of bacterial and viral infection because of improper hand washing (Allwood, Jenkins, Paulus, Johnson, & Hedberg, 2004; Bakalar, 2005; Berry, Mitteer, & Fournier, 2014; de Kort & Velthuis, 2011; Guinan, McGuckin-Guinan, & Severeid, 1997; Oldfield, 2017). Further, if people are using public restrooms in a casino, then cross-contamination can occur on casino gaming chips, because studies have shown that on average, 35% of the U.S. population does not wash their hands after using the restroom (Altekruse, Yang, Timbo, & Angulo, 1999; Berry et al., 2014; Byrd-Bredbenner et al., 2007; “Did you wash your hands,” 1996; Fillion, Kukanich, Chapman, Hardigree, & Powell, 2011; Guinan et al., 1997).

It should be noted that even though 65% of the U.S. population has been found to

wash their hands after using the restroom, the duration of hand washing does not reach the recommended time to ensure that hands are effectively cleaned. Berry and coauthors (2014) found that the average time that individuals wash hands after using the restroom was 8.1 s, with the range being 0.52–57.7 s. The Food and Drug Administration recommends that when washing hands, you should: “(3) Rub together vigorously for at least 10 to 15 seconds while: (a) Paying particular attention to removing soil from underneath the fingernails during the cleaning procedure, and (b) Creating friction on the surfaces of the hands and arms or surrogate prosthetic devices for hands and arms, finger tips, and areas between the fingers” (U.S. Department of Health and Human Services, 2013, pp. 46–47).

Alternatively, CDC recommends that when washing hands, you should “rub your hands together vigorously for at least 15 seconds, covering all surfaces of the hands and fingers” (CDC, 2018b).

Casino employees and customers can be at risk for exposure to infectious diseases, especially bacterial diseases, through the handling of chips. A study by Mc Keown and coauthors (2011) was designed to determine if infectious bacteria were present on chips that were used by casino workers and customers by comparing the bacteria counts of these chips to new, never-used-before chips.

The purpose of this replication study was to determine to what degree the results obtained from the Mc Keown and coauthors’ 2011 study, where both bacteria and fungi were present in statistically significant numbers on both the unused (factory) and used (in use at casinos) chips, were due to happenstance or instead indicate a serious health issue. The secondary purpose of this study was to determine if *E. coli* or coliforms are among the illness-causing bacteria found on the chips being studied. The information gathered from this study will provide recommendations that can reduce and prevent infectious bacterial disease among casino workers and customers.

Methods

The protocol for this study closely follows that which was outlined in Mc Keown and coauthors (2011) with changes made to the

protocol outlined below. This study employs a case-control design to determine if infectious bacteria exist on chips. The in-use chips were purchased at a table game in the amount of \$100 in \$5 chips, resulting in a total of 20 chips being purchased for the study. The \$5 denomination was chosen as a chip that is available at the various table games and is actively in use in games with minimum bets ranging from \$1–\$25.

Then, 13 chips that have never been used in a casino were compared with 13 chips that had been in play at an undisclosed casino in the Midwest. It was determined that the number 13 was used in the original study because the primary investigator was self-funding the study and that was how many blood-agar Petri dishes could be purchased.

In this study, a total of 20 chips (\$5 denomination each) were collected from four different casinos, with 4 chips from a casino in the Gulf Coast and the other 15 chips (5 each) from three different casinos in Las Vegas, Nevada. Chips were randomly chosen in equal numbers from the four casinos until 13 chips had been tested. Each chip contains three sides (obverse or front, reverse or back, and side or rim), so a total of $n = 78$ tests were performed: 39 for the used chips, and 39 for the control group (never-used chips).

Obverse and reverse sides of the chips were determined based on the chip design and positioning of colored stripes in relation to wording and casino label. Chip labels closely oriented with the wording on the edge of the chip were considered the obverse side of the chip. In the Mc Keown and coauthors’ 2011 study, chips were randomly removed from sterilized plastic containers marked as either used or unused using sterilized forceps.

In this study, two biologists performed the tests and directly removed the chips from the zip-sealed plastic bags that they were collected in from the casinos. One biologist performed the tests on the used chips and a different biologist performed the tests on the new, unused chips. The biologists wore neoprene gloves while handling the chips for testing. Between the testing of each chip, the testing area and gloves were sterilized with an alcohol solution of 70% ethanol. Each chip was then swabbed for bacteria using 6-in. sterile cotton-tipped applicators that had been dipped into a sterile solution of elution fluid containing 1% tween and

0.3% lecithin (Gaonkar, Geraldo, Shintre, & Modak, 2006).

The obverse side of the chip surface area was swabbed first, followed by the reverse side, and finally the rim. To gauge the degree in which the process might generate unique findings, we reversed swabs 22–27 to determine if swabbing order affected the results of the study. Additionally, we introduced a different bottle of sterile elution fluid at swab number 49. Both bottles of sterile elution fluid were made at the same time and both sterile elution fluids were tested before and after the study was completed. These steps were taken to determine that the elution fluids were not contaminated.

Swabs were then directly streaked across numbered blood agar Petri dishes, with the number corresponding to the location of the chip being swabbed to determine reactionary issues based on microorganism growth. For this study, larger Petri dishes were inadvertently acquired, so lines were drawn to create three equal areas. Each area was labeled either with an O, R, or E to reference the obverse (front), reverse (back), or edge (side) of the chip. The Petri dishes were also labeled with an identifier indicating from which of the four casinos it originated.

Once all the Petri dishes had been swabbed, they were placed upside down (optimal growing condition) in a growth incubator set at 37 °C for 48 hr. After 48 hr, the Petri dishes were removed from the incubator and placed in a refrigerated cooling area until the results were analyzed. This protocol for growing bacteria from contaminated surfaces is standard procedure (Bykowski & Stevenson, 2008). At the end of the study, the purchased chips were used in other studies, then returned to the respective casinos and redeemed for the cash value.

Results

We used analysis of variance (ANOVA) to measure the bacterial growth comparisons between the control and casino-used chips. We used the statistical program Stata version 10.1, which is considered a powerful statistical analysis package, to perform these tests. A probability of $p < .05$ was used for determining significant differences between the control versus casino-used chips for bacterial growth. A total of 78 samples (39 from each set of control chips and casino-used chips)

offered enough statistical power (for $\alpha = .05$, $SD = 0.50$, $N = 78$; power = 0.865) to determine the statistical significance noted above.

First, the plates were examined to determine the results (Figure 1). We used microscopic examination to identify cellular morphology and reaction (Figure 2). The bacteria cultured from the unused (control) chips were morphologically similar throughout each plate (Table 1). Bacteria on the unused casino chips consisted of gram-positive bacillus (rodlike) populations on all plates analyzed (Table 2). According to the World Health Organization, *Corynebacteria*, *Propionibacteria*, and *Staphylococcus epidermidis* are common gram-positive bacteria that colonize human hands. Although gram-positive bacteria colonize the hands to a greater extent than gram-negative bacteria, a greater diversity of bacteria, fungi, and viruses are key features in the human hand microbiome compared to alternative sources of bacterial populations on inanimate objects (Cosseau et al., 2016; Wenzler, Fraidenburg, Scardina, & Danziger, 2016). Although outside the scope of this experimental design, the population of bacteria found on the unused chips might originate from the manufacturing and packaging process rather than direct human contact, thus explaining the low diversity of bacteria present on the surface of the chips.

The blood agar plates containing bacteria from the used chips displayed higher diversity of bacteria and fungi (Table 2). There were roughly 32% fungi and 68% bacteria on each plate. With the use of selective *E. coli* media and coliform media, we detected the presence of *E. coli*, a type of coliform and common food poisoning-related bacterium (Addis & Sisay, 2015). Plates 2, 4, 5, 6, 8, 11, and 12 contained both gram-positive and gram-negative bacillus and gram-negative cocci (spherical-like) bacteria. Furthermore, the identification of gram-negative cocci bacteria on plate 11 suggests the presence of genera *Neisseria*, *Moraxella*, or *Kingella*, which are causative agents for meningitis, sinusitis, and bronchopneumonia, and can be transmitted by genital-to-hand contamination (Wenzler et al., 2016; Zapka et al., 2011).

The presence of capsular and lipopolysaccharides increases pathogenicity and antiphagocytic qualities suitable for evading the human

FIGURE 1

Sample Blood Agar Plate

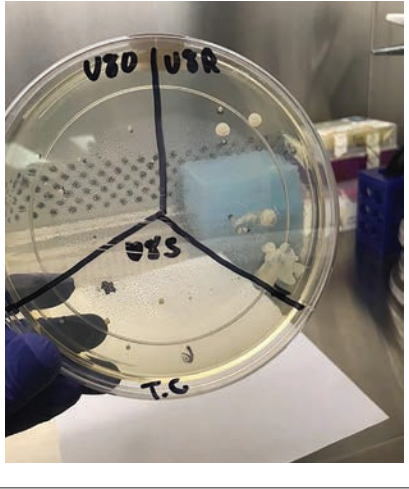
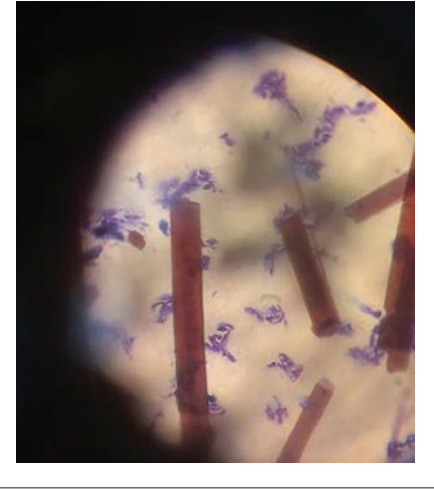


FIGURE 2

Microscopic Examination



immune system and can provide genetic diversity for increased multidrug-resistant populations (Arora, Devi, Chadha, & Malhotra, 2009). The differences in bacteria and morphology found is typical of fomites that have been in contact with a multitude of people.

Limitations of the study include genus and species identification of the diverse microbial communities present on used and unused chips using molecular identification, such as DNA sequencing, genomics, or proteomics. Additionally, swabbing might underestimate the total populations on the various surfaces of the chips, because swabbing does not access microbes embedded in the textured layers of the surface. The human hand influences the spread of disease, leaving and picking up microbes with each touch. With the use of standardized methods and increasingly larger studies, we will increase our understanding of the impact of casino chip sanitation on health outcomes.

Of the 78 tests completed, each test produced results that are considered usable for this study. We counted the number of bacteria or fungi colonies that grew in the agar Petri dish. For bacteria, the 78 usable results had a mean of 14.03 colonies and a standard deviation of 7.61 with a range of 1–33 colonies; alternatively, the fungi resulted in a mean of 1.44 colonies and a standard deviation of 1.92 with a range of 0–10 colonies. The *E. coli* test showed a mean of 2.1 colonies and a standard deviation of 3.74 with a range

of 0–19 colonies. The coliform test was negative for each case.

The ANOVA results [$F(1,76) = 43.56$, $p < .001$] indicated a statistically significant difference between the amount of bacteria found on used versus unused chips. According to the Bonferroni results, used chips have a higher mean score related to the number of bacteria found than that of unused chips, with a significance of $p < .01$. This study's measure of explained variation, however, shows that 36.43% of the variance in bacteria levels is explained by the differences between used and unused chips. Additionally, the fungi results were also statistically significant [$F(1,77) = 99.89$, $p < .001$], where 56.79% of the variance is explained by the difference between the used and unused chips. Finally, the *E. coli* results were also statistically significant [$F(1,77) = 92.22$, $p < .001$], where 54.82% of the variance is explained by the difference between the used and unused chips.

ANOVA was also performed to determine any differences in the swabbed areas (i.e., obverse, reverse, and edge). The bacteria, fungi, and *E. coli* found were not statistically significant for bacteria [$F(2,77) = 1.19$, $p > .05$], fungi [$F(2,77) = 0.68$, $p > .05$], or *E. coli* [$F(2,77) = 1.87$, $p > .05$]. The variance between the differences in the sections was 3.07% for bacteria, 1.77% for fungi, and 4.74% for *E. coli*.

Finally, the bacteria, fungi, and *E. coli* found on the obverse, reverse, and edge

TABLE 1

Control (Unused) Casino Gaming Chip Results

Chip #	Surface	Total # of Colonies	Size	Shape	Color	Margin	Elevation	Total # of <i>E. coli</i>	Total # of Coliforms	Gram Stain (+ or -)	Bacteria Morphology	Isolated Colonies (RNA Later)	# of Fungi
1	Obverse	11	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	33						0	0	+	Bacillus	1	0
	Edge	9						0	0	N/A	N/A	N/A	0
2	Obverse	15	SM	Round	Yellow, gray	Smooth	Raised	0	0	+	Bacillus	1	0
	Reverse	15						0	0	N/A	N/A	N/A	0
	Edge	16						0	0	N/A	N/A	N/A	0
3	Obverse	27	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	24						0	0	N/A	N/A	N/A	0
	Edge	11						0	0	N/A	N/A	N/A	0
4	Obverse	12	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	12						0	0	N/A	N/A	N/A	0
	Edge	10						0	0	N/A	N/A	N/A	0
5	Obverse	17	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	14						0	0	N/A	N/A	N/A	0
	Edge	24						0	0	N/A	N/A	N/A	0
6	Obverse	19	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	17						0	0	N/A	N/A	N/A	0
	Edge	29						0	0	N/A	N/A	N/A	0
7	Obverse	19	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	14						0	0	N/A	N/A	N/A	0
	Edge	23						0	0	N/A	N/A	N/A	0
8	Obverse	27	SM	Round	Yellow, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	22						0	0	N/A	N/A	N/A	0
	Edge	22						0	0	N/A	N/A	N/A	0
9	Obverse	23	SM	Round	White, gray	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	8						0	0	N/A	N/A	N/A	0
	Edge	17						0	0	N/A	N/A	N/A	0
10	Obverse	20	SM	Round	White, yellow	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	11						0	0	N/A	N/A	N/A	0
	Edge	19						0	0	N/A	N/A	N/A	0
11	Obverse	22	MD, SM	Round	White	Smooth	Raised	0	0	+	Bacillus	1	0
	Reverse	20						0	0	+	Bacillus	1	0
	Edge	17						0	0	N/A	N/A	N/A	0
12	Obverse	11	SM	Round	White	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	25						0	0	N/A	N/A	N/A	0
	Edge	10						0	0	N/A	N/A	N/A	0
13	Obverse	33	SM	Round	White	Smooth	Raised	0	0	N/A	N/A	N/A	0
	Reverse	29						0	0	N/A	N/A	N/A	0
	Edge	18						0	0	N/A	N/A	N/A	0

Note. We performed gram stain, bacterial morphology, isolated colonies, and fungi tests only on chips/petri dishes/colonies that were different. A lot of the colonies throughout the plates looked identical so we would isolate one of the colonies as a representation of the group. We isolated at least one colony out of all the colonies of the same group.

SM = small; MD = medium; N/A: not applicable.

TABLE 2

In-Use (Used) Casino Gaming Chip Results

Chip #	Surface	Total # of Colonies	Size	Shape	Color	Margin	Elevation	Total # of <i>E. coli</i>	Total # of Coliforms	Gram Stain (+ or -)	Bacteria Morphology	Isolated Colonies (RNA Later)	# of Fungi
1	Obverse	5	SM	Round	Yellow, gray	Smooth	Raised	15	0	N/A	N/A	N/A	0
	Reverse	10						5	0	N/A	N/A	N/A	3
	Edge	8						11	0	N/A	N/A	N/A	1
2	Obverse	20	LG, MD, SM	Round	White, yellow, gray	Smooth, rigid	Raised	14	0	+	Bacillus	1	5
	Reverse	8						6	0	N/A	N/A	N/A	1
	Edge	11						5	0	N/A	N/A	N/A	0
3	Obverse	19	MD, SM	Round	White, yellow	Smooth	Raised	4	0	N/A	N/A	N/A	4
	Reverse	27						1	0	N/A	N/A	N/A	4
	Edge	10						2	0	N/A	N/A	N/A	2
4	Obverse	16	LG, MD, SM	Round, rhizoid, filamentous	White	Smooth, rigid	Raised, flat	4	0	N/A	N/A	N/A	3
	Reverse	9						2	0	-	Bacillus	1	3
	Edge	9						6	0	N/A	N/A	N/A	2
5	Obverse	6	MD, SM	Round	Yellow, gray	Smooth	Raised	4	0	N/A	N/A	N/A	3
	Reverse	12						2	0	-	Bacillus	1	5
	Edge	9						5	0	N/A	N/A	N/A	4
6	Obverse	12	LG, MD, SM	Round	White, yellow, gray	Smooth, rigid	Raised	4	0	+	Bacillus	1	3
	Reverse	10						8	0	N/A	N/A	N/A	4
	Edge	10						2	0	N/A	N/A	N/A	4
7	Obverse	21	MD	Round	White, gray	Smooth	Raised	0	0	N/A	N/A	N/A	10
	Reverse	12						2	0	N/A	N/A	N/A	6
	Edge	8						3	0	N/A	N/A	N/A	3
8	Obverse	8	LG, MD, SM	Round, rhizoid	White, yellow, gray	Smooth, rigid	Raised, flat	19	0	N/A	N/A	N/A	2
	Reverse	8						2	0	N/A	N/A	N/A	4
	Edge	6						3	0	-	Bacillus	1	3
9	Obverse	11	MD, SM	Round	Yellow, gray	Smooth	Raised	7	0	N/A	N/A	N/A	3
	Reverse	10						3	0	N/A	N/A	N/A	2
	Edge	8						2	0	N/A	N/A	N/A	2
10	Obverse	5	MD, SM	Round	Yellow, gray	Smooth	Raised	1	0	N/A	N/A	N/A	1
	Reverse	2						1	0	N/A	N/A	N/A	2
	Edge	1						2	0	N/A	N/A	N/A	1
11	Obverse	12	MD, SM	Round	White, yellow, gray	Smooth	Raised	11	0	-	Cocci	1	4
	Reverse	9						0	0	N/A	N/A	N/A	3
	Edge	5						0	0	N/A	N/A	N/A	2
12	Obverse	12	LG, MD	Round, rhizoid	Yellow, gray	Smooth, rigid	Raised, flat	1	0	N/A	N/A	N/A	3
	Reverse	7						1	0	+	Bacillus	2	3
	Edge	7						1	0	+	Cocci	1	3
13	Obverse	1	MD, SM	Round	White, yellow, gray	Smooth	Raised	1	0	N/A	N/A	N/A	1
	Reverse	2						1	0	N/A	N/A	N/A	2
	Edge	3						3	0	N/A	N/A	N/A	1

Note. We performed gram stain, bacterial morphology, isolated colonies, and fungi tests only on chips/petri dishes/colonies that were different. A lot of the colonies throughout the plates looked identical so we would isolate one of the colonies as a representation of the group. We isolated at least one colony out of all the colonies of the same group.

SM = small; MD = medium; LG = large; N/A = not applicable.

($p < .001$) of the chips were statistically significant; however, the amount of explained variation for each test was low at 8.12%, 7.66%, and 6.95% for bacteria; 1.90%, 2.36%, and 1.38% for fungi; and 8.41%, 9.61%, and 6.24% for *E. coli*, respectively.

Discussion

As illustrated above, both bacteria and fungi were found in statistically significant amounts on used and unused chips. This finding aligns with the Mc Keown and coauthors (2011) study, which found:

“Further microscopic examination of the cell arrangements of the yellow colonies, found on plates 1, 4, 24, 28, 36, 43, 46, 49, 53, 56, 68, 71, and 77, were diplococcal and in tetrads, which means that this was most likely a hand bacterium known as *Micrococcus luteus* (Greenblatt et al., 2004). The fungus showed conclusively under a microscope to be a fungus; however, without expensive DNA sequencing it was not possible to determine which type. Moreover, the fungus resulted in complete hemolysis (rupture or destruction of red blood cells) within the agar Petri dish, also known as beta-hemolysis (β -hemolysis). This increased hemolysis suggested that the fungi were capable of being pathogenic.”

With the increased awareness of disease-causing microorganisms and the previous pandemics associated with influenza, these results show that chips can be carriers of organisms that can cause illness in susceptible populations (e.g., older people who tend to spend time at casinos, or infants/toddlers who find colorful chips laying around a hotel room or cruise ship stateroom).

An undercover investigation by *The Today Show* found just as many germs on the handle of a slot machine (373, well above the failure mark of 100) as on elevator buttons (370) (Rossen & Davis, 2015). The cleanliness of casino hotels and cruise ships are constantly being monitored by their respective health districts; unfortunately, the Vessel Sanitation Program 2011 Operations Manual created by CDC has no specific information regarding cleaning and sanitizing of the casino area. Every other area within a cruise ship is listed, with specific requirements and sanitation protocols—except for the casino (CDC, 2011; Cramer, Blanton, & Otto, 2008). Even the Southern Nevada Health District, which monitors hotels and casinos in the Las Vegas area, has only four items in a casino that are required to be cleaned and sanitized in an effort to control and prevent norovirus: “Casino cage counters, gaming chair backs, contact areas of gaming tables, and table game cup holders” (Southern Nevada Health District, 2007).

While this study was conducted using chips from four casinos compared with one casino in the study by Mc Keown and coauthors (2011), it only explored one specific denomination, specifically, the \$5 chip. Currently, there are hundreds of casinos around the world where chips are used and chips are available in multiple denominations, ranging from \$1–\$500 or higher; however, the \$5 chips are actively used in just about all casinos and are available in large quantities.

Conclusion

After testing for multiple types of pathogens on multiple chips from multiple casinos, tests are being conducted to determine the best method for cleaning and sanitizing

chips to ensure a healthy population, or if the chips should be redesigned to control for the ability to harbor these microorganisms. For example, we placed a chip in liquid bleach for 24 hours with no noticeable discoloration, in addition to placing a chip in an autoclave with no noticeable effects to the gaming chip. While these are two basic methods of sterilization, tests are being conducted on methods of sanitation that would be practical and usable within the casino industry. The eventual goal is to determine effective disease-prevention strategies for the safe handling and use of chips based on the presence of significant levels of infectious bacteria.

As a result, this study shows that additional studies need to be performed, and are being performed, to determine precisely the amounts and types of microorganisms that can be found on chips. Due to limited funds, the variability of chip denominations and casinos was sacrificed. In addition, limited funds dictated the amount of testing that was performed. Continued studies on casino chips will include DNA profiling of the microorganisms in addition to testing for possible viral pathogens. 🚗

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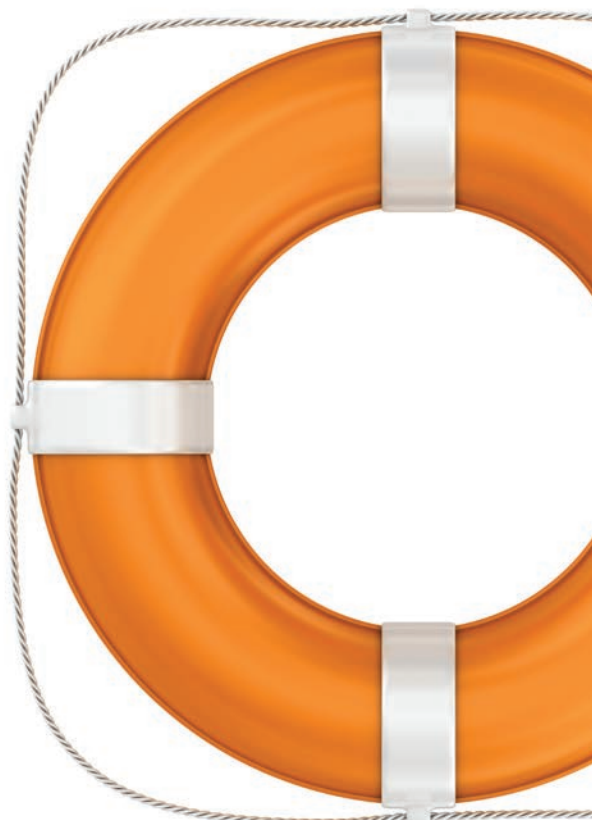


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Death From Unintentional Nonfire-Related Carbon Monoxide Poisoning in New York City During the Cold Season, 2005–2013

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Abstract Unintentional nonfire-related (UNFR) carbon monoxide (CO) poisoning is among the leading causes of unintentional poisoning deaths in the U.S. Our objective was to determine risk factors for UNFR CO poisoning deaths during the cold season in New York City (NYC). We examined data from death certificates and NYC Office of Medical Examiner records to describe decedent demographics, exposure circumstances, and CO sources during the cold months (October–April) between 2005–2013. Over the study period there were 32 UNFR CO deaths, with an average annual death rate of 0.4 per million people. Average annual cold-season death rates were higher among older adults (1.2 per million people ≥ 65 years) and men (0.8 per million men). The most common source of exposure was automobile engines ($n = 15$, 47%). The UNFR CO poisoning death rate in NYC is lower than the national average. Older adults and men are at greatest risk of death. Automobile exhaust is a significant and preventable source of exposure and should be emphasized in public health messaging and prevention efforts.

Introduction

Carbon monoxide (CO) is an odorless, colorless gas released during incomplete combustion of carbon, which is emitted from fuel-burning engines, including those in cars, boats, and generators. Unintentional nonfire-related (UNFR) CO poisonings are among the leading causes of unintentional poisoning deaths in the U.S. (Centers for Disease Control and Prevention [CDC], 2014).

Each year in the U.S., on average from 1999–2012, there were 438 UNFR CO deaths, with an average annual age-adjusted death rate of 1.46 per million persons, and a crude annual death rate of 1.43 per million persons (Sircar et al., 2015). Another study based on the National Vital Statistics System, the National Poison Data Center, hyperbaric oxygen treatment centers, and the National Electronic

Injury Surveillance System reported similar numbers of deaths (Iqbal, Clower, King, Bell, & Yip, 2012). In general, CO deaths are more common during winter months (Iqbal, Clower, et al., 2012; Sircar et al., 2015) and in rural areas (Sircar et al., 2015; Yoon, Macdonald, & Parrish, 1998). The most frequent place of exposure is the home (Sircar et al., 2015). Nationally, rates of nonfatal UNFR CO-related emergency department visits and hospitalizations are highest in the winter season. Most of these CO exposures occurred in homes (Iqbal, Law, Clower, Yip, & Elixhauser, 2012). Between 1979–1988, 57% of UNFR CO deaths in the U.S. were due to automobile exhaust (Cobb & Etzel, 1991), the majority (83%) of which were associated with stationary automobiles. In addition, many studies report that death from UNFR CO poisoning is more

common among men and older adults (CDC, 2007; Cobb & Etzel, 1991; Sircar et al., 2015).

Although there are similarities in decedent characteristics, sources of CO and exposure circumstances for UNFR CO deaths can vary by geography. In Florida, CO poisoning deaths from 1999–2007 occurred most commonly in the home and most decedents were exposed to CO from vehicle exhaust (Harduar-Morano & Watkins, 2011). In Oklahoma, however, most UNFR deaths from 1994–2003 were related to furnaces and home heating (Bowles & Mallonee, 2007).

We reviewed death certificate data and medical examiner records for CO decedents in New York City (NYC) to determine CO source, risk factors, and exposure circumstances during cold-season months, when CO poisoning occurs most frequently. To our knowledge, this study is the first in-depth examination of recent CO deaths in NYC.

Methods

CO deaths were defined by the Centers for Disease Control and Prevention's (CDC) National Environmental Public Health Tracking Network case criteria in effect in 2013 as those with an International Classification of Diseases 10th Revision (ICD-10) code of T58 ("Toxic effect of carbon monoxide") as a contributing or underlying cause (CDC, 2018). Intentional deaths were defined as those with ICD-10 codes X6-Y09, Y35, or Y36 in any field and were excluded. Fire-related deaths were defined as ICD-10 codes in the range of X0 in any field and were also excluded. Nonfire-related deaths were defined as ICD-10 codes X47 in any field and were included.

Decedents with an unknown cause were defined as those having no fire or nonfire codes, or having both fire and nonfire codes,

and were also included. Partially de-identified electronic death certificate data, including medical examiner numbers, were obtained from the NYC Department of Health and Mental Hygiene (DOHMH) Bureau of Vital Statistics for the cold-season months (October 1–April 30) during the years 2005–2013.

In NYC, the medical examiner investigates all deaths suspected of being due to an external cause, including CO poisoning. A research agreement with the Office of Chief Medical Examiner (OCME) allowed us to query OCME records for CO cases identified in Bureau of Vital Statistics data. Research assistants abstracted relevant data from medical examiner records into a Microsoft Access database and analyzed the data in SAS version 9.2. We also reviewed OCME documents to ensure the death met the case definition of being nonfire related and unintentional, and for exposure occurring in one of the five NYC boroughs.

We used death certificate data to determine race/ethnicity, sex, age, borough of residence, and educational levels. When data were missing from the death certificate but available in OCME data, we used the OCME data. By reviewing OCME documents, we determined source of exposure, presence and functioning of a CO alarm, whether the death occurred during a power outage, and whether the decedent was homeless or suspected to be homeless.

We noted alcohol abuse as chronic when either the death certificate or the OCME investigation documents mentioned current or past alcoholism. Evidence of alcohol use at the time of death was considered positive when there was a report of intoxication around the time of death or blood ethanol level was $>0.05\%$ on the toxicology report. A threshold of 0.05% was chosen because it is the legal blood alcohol limit for operating a motor vehicle in New York State. Comorbid or contributing medical conditions were determined from the autopsy report or death certificate, as well as by report in any investigation documents.

Average annual cold-month death rates were calculated for borough of residence, sex, race/ethnicity, and age group. Denominator data were based on the 2010 U.S. Census of NYC (New York City Department of Health and Mental Hygiene, 2015). We did not generate a death rate by borough of occurrence (as opposed to residence) because we had no appropriate

denominator. We defined an annual cold-season rate as the total number of deaths over the study period divided by the number of study years (9 years), expressed per 1 million people. A year-round rate was calculated and age adjusted to the U.S. standard age population for year 2000 using the direct standardization method (Klein & Schoenborn, 2001). Source of exposure was defined as the appliance or device that generated the CO. Place of exposure was defined as the physical location where the decedent was injured or found.

For sex, age, race, and borough of decedent residence, we used SAS software to generate a chi-square likelihood ratio test to test the null hypothesis that death rates were independent of category. Because counts in all categories were low, we assumed a Poisson distribution. A p -value of $<.05$ was considered statistically significant. If the null hypothesis was not rejected, no further statistical tests were performed. If the omnibus test was statistically significant, a reference category was set and a Poisson statistic was generated to compare rates within a variable.

The NYC DOHMH Institutional Review Board reviewed this study and determined that it was exempt research.

Results

Over the 9-year study period, 36 decedents were identified as meeting the UNFR CO death case definition in Bureau of Vital Statistics data. After reviewing OCME records, four records were excluded from the analysis and included one case of exposure outside NYC, two classified as unknown cause that were determined to be fire-related after review of OCME records, and one that was not CO-related. One decedent had a legal residence outside of NYC, but was visiting a family member for a long-term stay in the Bronx; therefore, we included this case as residing in the Bronx for the analysis. There were only five UNFR CO deaths during warm months over the study period (data not shown) that were not included in this analysis, bringing the total year, age-adjusted rate to 0.49 per million.

The overall cold-season death rate was 0.4 per million. Deaths occurred in each year, although there was variation in counts by year, with an annual average of 3.5 deaths. Of the 32 UNFR CO deaths during the cold season, 27 (84%) were men and five (16%) were women (Table 1). The average annual death

rate was higher among men than women (0.8 versus 0.1 deaths per million, $p < .001$).

The UNFR CO cold-season death rate increased with age. Approximately two thirds of deaths occurred among adults ≥ 45 years. Adults 45–65 years had a death rate of 0.6 deaths per million people, while adults ≥ 65 years had a rate of 1.2 per million people.

Deaths were reported in all NYC boroughs. Queens was the borough with the highest number of deaths ($n = 15$, 47%). There was no statistically significant difference in rates of cold-season death by borough of residence, although Staten Island ($n = 3$, 9%) had the highest rate of death (0.7 deaths per million residents).

The most common source of UNFR CO exposure in NYC was automobile emission (47%, Table 2), with decedents exposed in automobiles in enclosed garages, in homes with automobiles left running in attached garages, or in automobiles outdoors with or without mention of exhaust systems blocked by snow.

Automobiles were also the most common location of exposure (47%, Table 2), most often outside but also in enclosed residential garages. Of the 15 decedents found in an automobile, the source of exposure for 11 decedents was vehicle exhaust, including four vehicles running in attached garages and four vehicles with exhaust systems blocked by snow. The remaining four were exposed to nonvehicle sources running inside the vehicle, including a contained coal fire for warmth, generators, and a lawn mower.

The second most common location of exposure was the home (44%, Table 2). Home exposures resulted from furnaces, ovens, space heaters, and generators, as well as four cases where decedents died in homes after exposure to exhaust from vehicles left running in attached garages. There was one case in which a decedent was thought to be exposed at home, but there was no report of the specific source and so this case was reported as “household exposure.”

The presence or absence of a CO alarm was infrequently noted in the death scene investigation reports for indoor exposures (Table 2). Two decedents were indoors and had no CO alarm. Five decedents were noted as having an alarm in place. Of those with an alarm, in three cases the alarm was noted to be non-operational: two decedents who died in the same incident had a CO alarm that was dis-

connected from the batteries, and the other decedent did not have batteries in the alarm. Two decedents, who died in the same incident and were both hard of hearing, had a CO alarm but it was not clear from the records whether it had sounded or was operational. It was noted, however, that a neighbor's CO alarm had sounded.

Overall, six decedents were homeless (Table 2), five of whom were exposed in vehicles and one in a place of business. There were also seven (22%) work-related deaths, of which three were due to generator emissions. Only two cases were known to involve a utility outage. In one case, the power was turned off due to unpaid bills. In the other case, the reason for the outage was not recorded.

The most common comorbid medical condition was cardiovascular disease ($n = 16$, 50%, Table 3), defined as either atherosclerotic disease, history of stroke, heart failure, history of myocardial infarction, pacemaker present, or hypertensive cardiomyopathy. There was evidence of alcohol use at the time of death in 16% of cases and 25% of decedents had a history of alcohol abuse. Nearly 40% percent of decedents had a history of either drug or alcohol abuse, or had evidence of drug or alcohol use at time of death. Respiratory disease and diabetes were noted in several cases.

The environmental CO level was noted in only seven indoor exposure cases. Some records commented on CO detection but did not report a value and some records noted that doors and windows had been opened prior to fire rescue taking a measurement. Excluding a case in which the CO measurement was zero, the mean environmental CO measured was 372 ppm with a standard deviation of 168 ppm (Table 4). In all, 28 of the 32 decedents (88%) had a recorded carboxyhemoglobin blood level (Table 4). The mean carboxyhemoglobin level for those in whom it was measured was 61% with a standard deviation of 14%.

Discussion

In NYC, between 2005–2013, the average annual cold-month death rate was 0.4 deaths per million people based on OCME data (Table 1). Similar to other areas, death rates were higher among older adults and men. Automobile exhaust was the most common source of exposure. While infrequent, these deaths occur each year and are preventable.

TABLE 1

Demographics of Unintentional Nonfire-Related Carbon Monoxide Poisoning Deaths, New York City, 2005–2013 (October–April)

Demographic	<i>n</i>	%	Average Annual Cold Month Rate per 1,000,000	<i>p</i> -Value
Sex				<.001 ^b
Female	5	16	0.1	ref
Male	27	84	0.8	<.001
Age (years)				.001 ^b
0–17	1	3	0.1	ref
18–24	2	6	0.3	.250
25–44	8	25	0.3	.106
45–64	10	31	0.6	.038
≥65	11	34	1.2	.004
Race				.541 ^b
White (non-Hispanic)	11	34	0.4	
Black (non-Hispanic)	10	31	0.6	
Hispanic	9	28	0.4	
Asian	2	6	0.2	
Borough of death				
Bronx	5	16		
Brooklyn	8	25		
Manhattan	2	6		
Queens	15	47		
Staten Island	2	6		
Borough of residence ^a				.106 ^b
Bronx	7	22	0.6	
Brooklyn	7	22	0.3	
Manhattan	2	6	0.1	
Queens	13	41	0.6	
Staten Island	3	9	0.7	
Education				
Less than high school	5	16		
High school graduate or GED	15	47		
Some college credit, no degree	3	9		
College or higher	5	16		
Unknown	4	13		
Total	32	100	0.4	

^aFrom Bureau of Vital Statistics data. One decedent lived outside of New York City but was staying long term with family in the Bronx and was counted as a resident of the Bronx.

^bLog likelihood ratio chi-squared test.

TABLE 2

Source of Carbon Monoxide (CO) and Environmental Risk Factors Among Decedents (n = 32), New York City, 2005–2013 (October–April)

Variable	n	%
Source of exposure		
Automobile	15	47
Generator	5	16
Oven	4	13
Space heater	3	9
Charcoal fire	1	3
Furnace	1	3
Gas hot water heater	1	3
Unspecified household exposure	1	3
Lawn mower	1	3
Place of exposure		
Automobile	15	47
In residential garage ^a	4	
Snow-related ^b	4	
Other outdoors ^c	7	
Home ^d	14	44
Business ^e	3	9
Power outage		
Yes	2	6
No	3	9
Unknown/NA	27	84
Homeless		
Yes	6	19
No	25	78
Unknown	1	3
CO alarm present		
Yes	5	16
Not operational	3	
Unknown if operational	2	
No	2	6
Unknown/NA	25	78
Work-related		
Yes	7	22
No	25	78

NA = not applicable

^aIncludes four decedents found in automobiles in home garages.^bAll decedents were in an automobile and involved snow obscuring the automobile exhaust system.^cSources of CO for four decedents found in an automobile included one lawn mower, two generators, and one charcoal fire.^dIncludes four decedents who died in homes with attached garages where automobiles were the source of exposure.^eIncludes one store, one shed at a construction site, and one trailer at a construction site.

The UNFR CO death rate in NYC appears to be lower than national and regional rates. The cold-season death rate in NYC was 0.4 deaths per million. The year-round age-adjusted death rate was 0.49 per million, lower than the year-round age-adjusted Northeast death rate of 0.91 deaths per million and the national rate of 1.46 deaths per million from 1999–2012 (Sircar et al., 2015).

The higher CO poisoning death rates in men that we observed in NYC are consistent with studies in other jurisdictions (Iqbal, Clower et al., 2012). One national study found that nonfatal CO exposures and emergency department visits were more common among women, whereas men were more likely to experience death (Iqbal, Clower, et al., 2012). It is possible that men might be more vulnerable to death because they are more likely to abuse alcohol and substances (Merikangas & McClair, 2012), which could decrease the likelihood that they will recognize early symptoms and remove themselves from the exposure. Men might also have more acute high-level exposure because they could be more likely to work with tools or appliances that emit CO in an occupational setting (CDC, 2007; Iqbal, Clower, et al., 2012). In our study, all work-related exposures (n = 7) were among men.

In this study, the risk of fatal UNFR CO poisoning increased with age, also consistent with other studies (CDC, 2007; Sircar et al., 2015). This trend might be because older people are 1) less likely to experience symptoms or 2) unable to recognize early symptoms of CO poisoning, which can be nonspecific (Muo & Gambert, 2015). An increasing prevalence with age of medical comorbidity, especially cardiovascular disease, could make older adults more prone to the effects of CO on the heart. Finally, older adults could be more likely to live in social isolation, resulting in a longer CO exposure and lower likelihood of being found before death.

Coronary artery disease was the clinical condition most associated with death by UNFR CO poisoning. We considered a decedent as having coronary artery disease if it was present on autopsy. Autopsy dissection of coronary arteries showing atherosclerosis, however, might not indicate clinically apparent heart disease during life. Young subjects with no clinical diagnosis of cardiovascular disease might be diagnosed as having atherosclerosis on imaging and autopsy (Tuzcu

et al., 2001). Both clinical and subclinical coronary artery disease can increase risk of death from CO poisoning because it makes the victim more susceptible to the effects of hypoxia, as well as to CO toxicity directly affecting the myocardium. In addition, coronary artery disease can predispose exposed people to arrhythmia and other cardiac complications of CO poisoning (Lippi, Rastelli, Meschi, Borghi, & Cervellin, 2012).

Housing stock, transportation, weather, and employment likely all contributed to differences in rates of death between jurisdictions. From 1999–2012, NYC victims of fatal UNFR CO poisoning were somewhat less likely to be exposed at home (44%) than deaths nationally (54%) (Sircar et al., 2015) or in Florida (77%) (Harduar-Morano & Watkins, 2011). In November 2004, NYC passed a law mandating CO detectors in dwellings, which include apartment buildings, single-family homes, and multifamily homes. Our study period occurred after this law was instituted, so it is possible that we were seeing fewer deaths in homes due to CO alarm warnings. A previous study of incidence of CO poisoning in NYC before and after the CO detector law found nonsignificant decreases in hospitalizations and deaths from UNFR CO poisoning after the law; however, the relatively short study period and small number of identified deaths might have limited our ability to detect a significant change (Wheeler-Martin et al., 2015).

In our study, the most common source of CO poisoning was automobile emissions (47%). This result is consistent with national studies that have also found vehicle emissions to be the most common exposure source, most frequently via stationary vehicles (Cobb & Etzel, 1991; Sircar et al., 2015). Somewhat lower rates of residential death in NYC might be due, in part, to the fact that most people in NYC live in apartment buildings without interior garages or with commercial garages that are staffed.

In NYC, the highest death rates, although not statistically different, were seen in Queens and Staten Island. These boroughs have the highest density of cars (New York Office of Information Technology Services, 2014) and the highest percentage of residential lots with single family or row houses (New York City Department of City Planning, 2012), which are more likely to have garages in close prox-

TABLE 3
Carbon Monoxide Decedent Medical Conditions and Contributing Factors, New York City, 2005–2013 (October–April)

Variable	<i>n</i>	%
Cardiovascular disease	16	50
Respiratory disease	5	16
Diabetes	3	9
Recorded history of illegal substance or opioid use	4	13
Chronic alcohol abuse		
Yes	8	25
No	10	31
Unknown	14	44
Evidence of alcohol use at time of death ^a	5	16
History of illegal substance, opioid, chronic alcohol abuse, or alcohol use at time of death	12	38
Total	32	100

^aBy report or positive toxicology ethanol level of >0.05% measured in blood.

imity to living space. Single-family dwellings also have less stringent requirements related to proof of installation and maintenance of CO alarms (New York City Department of Housing Preservation and Development, 2013).

Nationally, 3% of UNFR CO deaths from 1999–2012 were work related (Sircar et al., 2015). There was a higher proportion of occupational exposures (16%) in NYC, which likely is the result of a lower number of non-occupational deaths, rather than an indication of unusually unsafe work places. The possibility of unsafe work environments, however, should be considered in prevention efforts. Three of the deaths took place in sheds or trailers that were heated with a generator in the winter: all three decedents were guarding goods or construction sites overnight. As such, the most common work-related deaths occurred outside in areas (such as vehicles and worksites) that may not be covered by the current CO alarm rule.

Only 16% of decedents were noted to have evidence of alcohol use around the time of death. Of UNFR CO decedents in New Mexico from 1980–1995 who were tested for the presence of alcohol, 42% had a blood alcohol concentration of >0.01% (Yoon et al., 1998). A study in California reported 33% of UNFR CO deaths involved alcohol as documented in

medical examiner report (Girman, Chang, Hayward, & Liu, 1998). Nearly 40% of decedents in our study, however, had a history of either drug or alcohol use or had evidence of drug or alcohol use at time of death, indicating that substance use and abuse can also be a significant risk factor for UNFR CO deaths in NYC.

There are some limitations to this study. We examined deaths occurring only in cold-season months, so risk factors specific to summer deaths are not captured in this analysis. Most UNFR CO deaths in NYC and the Northeast, however, occur during cold months; over the entire study period, there were five UNFR CO deaths that occurred during warm months. In addition, our case definition included two cases with an IC-10 code (Y17) denoting an event of undetermined intent. Some CO deaths might also go undetected (Varon & Marik, 2002). CO exposure, for instance, can trigger an acute coronary event and be unrecognized as a CO-related death (Sward, Sethuraman, Wong, & Rosenthal, 2016).

This analysis took place at the decedent rather than the incident level. In our data, there were three poisoning events that resulted in six deaths. The method we used, however, indirectly takes into account the most concerning CO source, because those

TABLE 4

Decedent Carboxyhemoglobin and Environmental Carbon Monoxide Levels (n = 32), New York City, 2005–2013 (October–April)

Variable	n	Mean	SD	Minimum	Maximum
Carboxyhemoglobin blood level (%)	28	61	14	35	83
Environmental carbon monoxide level (ppm)	6	372	168	200	642

sources that caused more deaths were, in effect, overcounted.

A strength of this study is the use of medical examiner investigation data to describe the source of CO and the context in which the deceased was exposed, which is information generally not available from death certificates. In addition to CO detector regulations, one of the strategies for prevention of CO exposure is public health messaging to educate the public and at-risk populations about how to protect themselves and avoid exposure. The results of this study can help ensure that public health messaging appropriately emphasizes current risks in NYC. While continued focus on educating the public about the importance of functioning home CO detectors, safe use of home heating and other equipment, and proper use of generators (particularly during emergencies) remains appropriate, the results of this study demonstrate that vehicle exhaust is a major source of exposure in NYC.

The dangers of running vehicles in garages attached to homes or in enclosed spaces need to be emphasized in public health messaging. Many decedents were older men and mes-

saging should also be directed at that population, as well as those who care for older people generally. Given the higher risks of CO exposure in the winter, CO safety messaging should continue to be integrated into seasonal and emergency winter and extreme cold safety messaging. For instance, the risks of exposure to vehicle exhaust resulting from tailpipes blocked by snow should be highlighted in public education materials that are disseminated during the cold season.

In addition, there should be further study regarding the extent to which car exhaust is the cause of CO poisoning in warm or cold months throughout the country. If vehicle exhaust is a major contributor to CO death, engineering solutions such as alternative alarm systems for vehicle CO emissions that can perform well in that environment could be explored.

Conclusion

In NYC, the UNFR CO death rate is lower than national rates, with the majority of exposures due to car exhaust. Preventive measures such as ensuring home CO detectors, maintaining home heating equipment, and proper use of home heating and cooking

equipment, as well as reducing CO in consumer products such as generators continue to be relevant. Our study shows that the dangers of automobile emissions—including automobiles in attached garages—as a source of fatal CO poisoning at home are important in NYC and should be emphasized in public health messaging. 🚗

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- | | | | |
|------|------|------|-------|
| 1. c | 4. b | 7. b | 10. a |
| 2. d | 5. b | 8. a | 11. a |
| 3. a | 6. d | 9. c | 12. b |

→ Quiz deadline: August 1, 2019

1. Each year in the U.S., on average from 1999–2012, there were __ unintentional nonfire-related (UNFR) carbon monoxide (CO) deaths.
 - a. 438
 - b. 538
 - c. 638
 - d. 738
2. In general, CO deaths are more common during __ months.
 - a. spring
 - b. summer
 - c. fall
 - d. winter
3. This study reviewed __ to identify CO decedents in New York City (NYC).
 - a. death certificate data
 - b. medical examiner records
 - c. emergency department records
 - d. a and b
 - e. all the above
4. Over the 9-year study period, __ decedents were identified as meeting the UNFR CO death case definition.
 - a. 33
 - b. 36
 - c. 39
 - d. 42
5. There were __ UNFR CO deaths during warm months over the study period that were not included in this study.
 - a. three
 - b. four
 - c. five
 - d. six
6. Of the UNFR CO deaths identified during the study period during the cold season, __ were male and __ were female.
 - a. 16%; 84%
 - b. 33%; 67%
 - c. 67%; 33%
 - d. 84%; 16%
7. Approximately __ of UNFR CO deaths in NYC occurred among adults ≥45 years.
 - a. one quarter
 - b. one third
 - c. one half
 - d. two thirds
8. __ were the most common location of UNFR CO exposure in NYC.
 - a. Homes
 - b. Automobiles
 - c. Businesses
9. The presence or absence of a CO alarm was frequently noted in the death scene investigation reports from indoor exposures.
 - a. True.
 - b. False.
10. The most common comorbid medical condition was
 - a. cardiovascular disease.
 - b. diabetes.
 - c. respiratory disease.
 - d. none of the above.
11. In all, __ of the decedents had a recorded carboxyhemoglobin blood level.
 - a. 32%
 - b. 50%
 - c. 64%
 - d. 88%
12. In NYC, the UNFR CO death rate is __ national rates.
 - a. lower than
 - b. the same as
 - c. higher than

Detecting Styrene With Spectral Fluorescence Signature Analysis

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Abstract The large global production of plastics and their presence everywhere in society and the environment have created a need for assessing chemical hazards and risks associated with plastic products. Plastics from polystyrene can release potentially toxic products (including styrene), particularly when heated. In this study we used a Fluo-Imager Analyser with software for spectral fluorescence signature (SFS) analysis. The objective of this study was to evaluate and compare the amount of styrene released into food and beverages by using SFS on a Fluo-Imager Analyser. Our results showed that concentrations of released styrene were in the range of 1.45–9.95 µg/L for hot water and 0.10–2.78 µg/L for room temperature water. The results indicate that this fluorescence diagnostic method is an effective tool for analysis of styrene released into food and beverages from polystyrene containers and cups, and could be useful in further investigations of styrene toxicity.

Introduction

Plastic is widely used in everyday life due to its continuous production in the last 60 years. In the 8-year period from 2008–2016, production of plastic products has sharply increased 37%, from 245 million metric tons in 2008 to 335 million metric tons in 2016 (Lithner, Larsson, & Dave, 2011; Plastics Europe, 2017). One of the most frequent uses of plastics is in packaging, storing, and serving food and beverages. Plastic products, in all stages of their life cycle, occupy almost every aspect of our lives and we tend to neglect their potential harmful impacts to our health and the health of our environment (Lithner, Damberg, Dave, & Larsson, 2009).

The most important organic substances that can be released from plastic consumer prod-

ucts are styrene, 1,3-butadiene, melamine, formaldehyde, acrylamide, di-2-ethylhexyl phthalate, di-2-ethylhexyl adipate, vinyl chloride, and bisphenol A (Durusoy & Karababa, 2011). These substances are endocrine disrupting and have carcinogenic effects (Durusoy & Karababa, 2011). The aforementioned substances can be released into food or beverages depending on the chemical characteristics of the plastic or food/beverage; temperature during packing, storing, and processing; UV exposure; and time of storage (Durusoy & Karababa, 2011). The risk of the release of these substances is increased in several ways: during the contact with fatty/oily or acidic food/drink, by heating the food in plastic containers, by drinking hot drinks from plastic cups, by using old and damaged plastic, or

using some surfactants (Durusoy & Karababa, 2011). Keeping this information in mind, we have to be aware of the need for continuous hazard and risk assessment related to plastic products, especially for new consumer products (Lithner, Nordensvan, & Dave, 2012).

Currently there are many types of plastic materials on the market but the most frequently used are products made of polystyrene, especially in the fast food industry (delivering and serving hot meals and drinks) (Plastics Europe, 2017). Styrene, as a basic compound of polystyrene, can adversely affect human health in many ways due to its tendency to accumulate in tissue. According to the Agency for Toxic Substances and Disease Registry (ATSDR), styrene was detected in adipose tissues and blood (ATSDR, 2018). Prolonged exposure to small amounts of styrene can have neurotoxic (bad mood, insomnia, nervousness), hematologic (reduced platelet count and hemoglobin), cytogenetic (chromosomal abnormalities and lymphatic), and carcinogenic effects (Dowty, Laseter, & Storer, 1976). There is also evidence that styrene is hepatotoxic and pneumotoxic (Chung, Shen, Jiang, Yuan, & Zheng, 2012; Chung, Yuan, Liu, & Zheng, 2006), it decreases reproduction capacity (Chamkhia, Sakly, & Ben Rhouma, 2006), it is ototoxic (Lawton, Hoffmann, & Triebig, 2006; Morata & Campo, 2002; Nies, 2012), and it can cause a slowdown in growth and development (Durusoy & Karababa, 2011).

Previous research has shown that styrene and other aromatic organic compounds are continuously secreted from polystyrene plastics used in the food and beverage industry (Choi, Jitsunari, Asakawa, & Lee, 2005; Gelbke et al., 2014; Gennari, Albrizio, &

TABLE 1

Polystyrene Sample Descriptions

Sample #	Characterization Description (Volume)
1	Transparent sample cup with red screw top (200 mL)
2	Yellow drinking cup (200 mL)
3	Brown drinking cup (150 mL)
4	White drinking cup (200 mL)
5	Transparent green drinking cup (200 mL)
6	Transparent drinking cup (330 mL)
7	Transparent drinking cup (50 mL)
8	Transparent drinking cup (40 mL)
9	Styrofoam food container (350 mL)
10	Transparent container for serving food (500 mL)

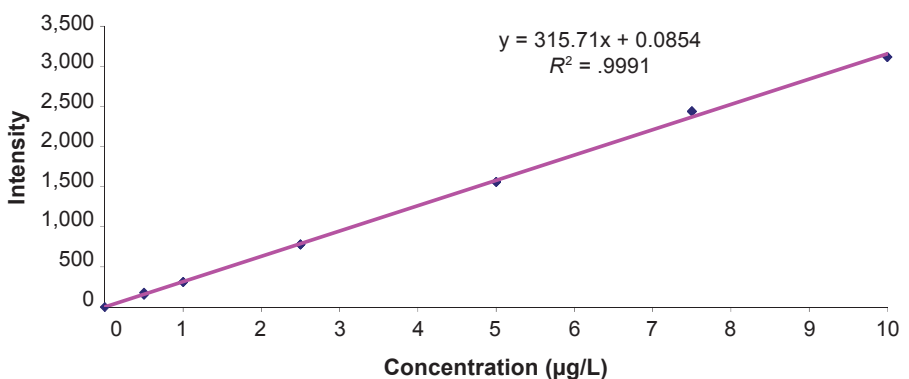
TABLE 2

Repeated Analysis on the Same Real Sample Using Spectral Fluorescence Signature Method

Measurement	Styrene Concentration ($\mu\text{g/L}$)
1	4.20
2	4.16
3	4.18
4	4.19
5	4.15
6	4.17
7	4.22
8	4.21
9	4.24
10	4.26
Average	4.20
SD	0.035
Relative SD (%)	0.84

Monteiro, 2012; Genualdi, Nyman, & Begley, 2014; Linssen, Janssens, Reitsma, & Roozen, 1991; Miller, Newhook, & Poole, 1994; Paraskevopoulou, Achilias, & Paraskevopoulou, 2012). Styrene has been reported in yogurt,

FIGURE 1

Instrumentation Calibration Curve for Styrene for Spectral Fluorescence Signature Analysis

cream, salad, soft cheese, margarine, hot and cold beverages, fresh and cooked meat, candied fruit, and fast food packed in polystyrene (Miller et al., 1994).

The standard method for detection and quantification of those compounds is gas chromatography with mass spectrometry (GC-MS) (Garrigós, Marín, Cantó, & Sánchez, 2004; Kusch & Knupp, 2002). This analytical method is considered to be the gold standard but it is quite expensive, time-consuming, and requires the use of harmful solvents during sample preparation. Therefore, for this feature, we attempted a new way to detect styrene: spectral fluorescence signature (SFS). SFS is widely used in research on phytoplankton (Babichenko, Leeben, Poryvkina, van der Wagt, & de Vos, 2000; Kaitala, Babichenko, Poryvkina, & Leeben, 1994) and dissolved organic matter (Babichenko, Kaitala, Leeben, Poryvkina, & Sepälä, 1999). Fluorescence spectrometry is a standardized method (ASTM International, 2012) with great potential, as recognized by the International Council for the Exploration of the Sea (Ariese, Beyer, Jonsson, Porte, & Krahn, 2005).

According to the available literature, SFS has not been used for the determination of styrene released from plastic consumer products. In previous studies, SFS was validated for the determination of organic compounds using GC-MS as the gold standard; the matching of the finding was >99% (Ferretto et al., 2014; Poryvkina, & Babichenko, 2010). There

is also evidence that SFS is comparable with GC-MS in interlaboratory proficiency testing (i.e., produced satisfactory z-scores) (Kammann et al., 2013). As a fast and inexpensive method that does not require the use of harmful solvents, SFS could be of great value in the area of styrene detection and quantification.

With these considerations, we tried to demonstrate the value of SFS in the determination of styrene as a product of polystyrene's release.

Methods**Materials**

We looked at plastic consumer products made of polystyrene (cups for serving hot and cold beverages, containers for the delivery of food, and containers for storing food and drinks). We purchased from a local market Styrofoam food containers and food-grade, rigid, and open polystyrene cups that are commonly used for drinking of water, tea, and coffee. Based on most common use, we chose 10 different types of polystyrene cups and containers (Table 1). With each type of cup and container, we measured 10 parallel samples with room temperature water and 10 parallel samples with hot water (temperature of approximately 80 °C).

The release of styrene in each of 20 parallel (10 hot water, 10 cold water) plastic products was monitored without exposure to UV radiation after 5, 15, and 30 min and 24 hr, so we analyzed a total of 1,000 samples. Results are expressed in $\mu\text{g/L}$. We used Milli-Q ultra-

TABLE 3

Reproducibility of Standard Solutions Using the Spectral Fluorescence Signature Method

Replicate #	Styrene Concentration (µg/L)		
	0.5 µg/L Standard	1.0 µg/L Standard	5.0 µg/L Standard
1	0.520	1.020	5.01
2	0.510	1.000	4.99
3	0.480	0.960	5.02
4	0.500	0.980	4.95
5	0.490	1.040	4.98
6	0.530	1.020	4.96
Average	0.505	1.003	4.99
SD	0.019	0.029	0.027
Relative SD (%)	3.71	2.93	0.55

pure water. As the standard, we used styrene of purity >99.5%, gas-chromatography grade (Sigma-Aldrich). For the control sample, we used laboratory glass beakers made of borosilicate glass.

Measurements of Spectral Fluorescence Signature

Measurements of SFS typical of styrene were done on the Fluo-Imager Analyser M53 together with its respective control software and additionally cooled detector (Scalar Analytical B.V.) according to the ASTM International (2012) method. Benefits of Fluo-Imager are 1) no prior sample preparation and 2) it does not require the use of organic solvents like some other methods and therefore does not endanger the human environment or human health.

The Fluo-Imager screens SFS of water samples. SFS is a matrix of the fluorescence intensity in coordinates of excitation and emission wavelengths, and can be viewed on a computer monitor as a filled contour plot of equal fluorescence intensity where the excitation wavelength corresponds to the y-axis and the emission one to the x-axis. Colors (from blue to yellow and finally white) indicate the growth of fluorescence intensity. The absolute maximum of fluorescence signal is marked by a black dot. Through the measurement cycle the excitation wavelength is scanned from 240–360 nm and fluorescence is registered in spectral range from 265–585

nm. Different organic compounds in water have different topography of SFS. In leachates from plastic consumer products made of polystyrene, only styrene was detected.

In this study, the method was validated and the sensitivity level of the instrument was determined with a certified reference standard styrene (Sigma-Aldrich) to prove that this method was precise and linear for determination of styrene released from polystyrene consumer products.

Results

Validation of Method

The method of determining the spectral fluorescence fingerprint was validated with a certified reference standard of styrene (Sigma-Aldrich) according to ASTM International (2012) on a Fluo-Imager. Validation of the method included linearity, repeatability of sample measurement, reproducibility, detection limit, and quantification limit. Six solutions containing a standard with concentrations between 0.5–10.0 µg/L were prepared to study linear range (Figure 1). The squared correlation coefficients (R^2) was .9991.

The real water sample was measured 10 times and an average value was calculated so we could assess the repeatability of the method. The results are shown in Table 2.

For reproducibility, we prepared three concentrations of reference material (Merck) and took measurements to gauge accuracy and

TABLE 4

Instrumentation Minimum Detection Level (MDL) and Minimum Quantification Level (MQL) for Spectral Fluorescence Signature Analysis Using a 0.5 µg/L Standard Solution

Replicate #	Styrene Concentration (µg/L)
1	0.50
2	0.51
3	0.51
4	0.49
5	0.50
6	0.51
7	0.51
8	0.49
9	0.51
10	0.49
Average	0.502
SD	0.009
Relative SD (%)	1.83
MDL	0.03
MQL	0.10

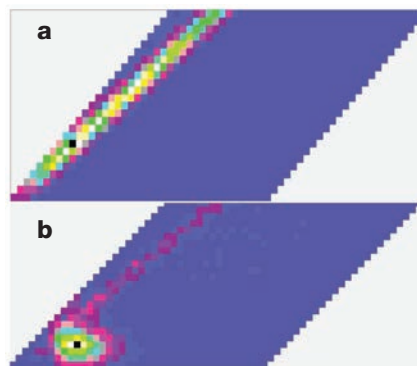
relative standard deviation (RSD). The reproducibility was studied by analyzing six measurements of each prepared concentrations of reagent water spiked at three different concentration levels. For the purpose of this method, an RSD of ≤10% was acceptable and Table 3 shows that all values are below 10%.

Taking into account the linearity ranges, we prepared concentrations at the lower values of the linear range. We analyzed 10 replicate solutions of 0.5 µg/L of styrene and determined the SD. We calculated the values of minimum detection level (MDL) and minimum quantification level (MQL) using the formulae ($3 \times SD$) and ($10 \times SD$), respectively (United States Pharmacopeial Convention, 2002); values are presented in Table 4.

Our results showed that this method for measurement of styrene in water samples and in leached samples of plastic products of polystyrene by fluorescence spectroscopy is linear (from 0–50 µg/L) and is satisfactory for this purpose.

FIGURE 2

Spectral Fluorescence Signatures for a Clean Water Sample (a) and a Water Sample With 10 µg/L Styrene (b)



Results of Measurements of Spectral Fluorescence Signature

SFS analysis of water samples measured with a Fluo-Imager generated color plots that represent the levels of fluorescence intensity (from blue to white) in coordinates of excitation and emission wavelength. Maximal SFS intensity is marked by black dots (Figure 2).

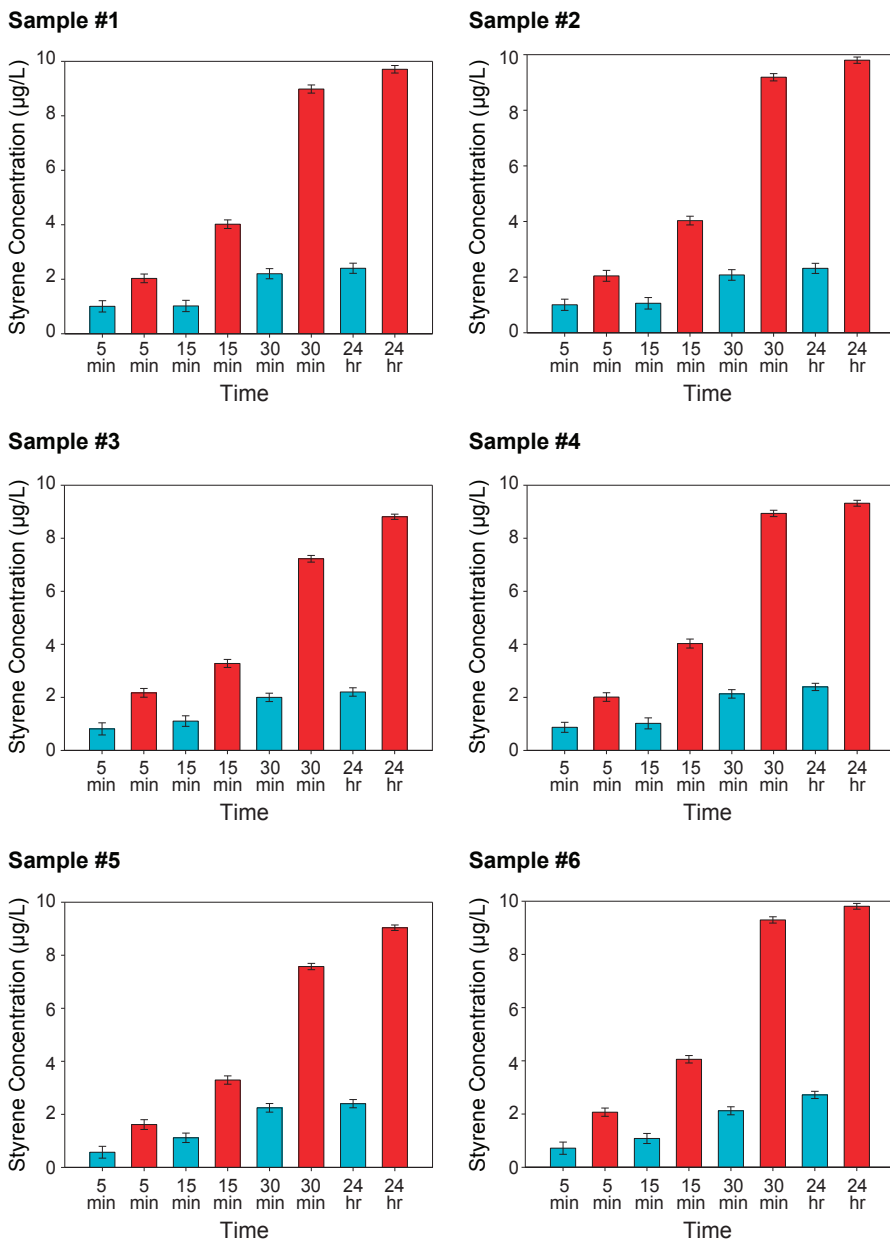
Movement of styrene concentrations measured in samples by SFS analysis is presented in Figure 3. Concentrations were measured in hot water (around 80 °C) and in room temperature water. Concentrations for hot water ranged from 1.45–9.95 µg/L and 0.10–2.78 µg/L for room temperature water (Figure 3).

Discussion

The use of various types of plastic, including polystyrene, is growing. Knowing that plastic releases toxic substances into food and drinks, it is necessary to develop methods that will enable us to quickly and in a cost-effective manner determine concentrations of these harmful substances in food and drinks. Acute toxicity is something we do not expect to occur, but we cannot neglect public health with regard to plastic components that can leach into drinks and food. Time of exposure with plastic leachates is more important than the amount of toxic substances leached. The most vulnerable time of human exposure is during the growth phase from birth to the end of puberty (Ahmad & Bajahlan, 2007).

FIGURE 3

Sample Concentrations of Styrene Released From Plastic Products Using the Spectral Fluorescence Signature Analysis



continued on page 28

In this study we wanted to check the possibility of using the SFS method for determination of the chemical compound styrene, as SFS is a fast and inexpensive method. In our review of the literature, we did not find a method this quick and simple that measured such low styrene concentrations as the SFS

method. We focused on SFS because it does not require the preparation of water samples and therefore it is fast and does not require much time to get the result.

Our results show that the styrene quantification limit for this method is 0.10 µg/L and that the method is linear in the range of 0.5–10.0

µg/L. For comparison, the linearity of head-space gas chromatography with flame ionization detection (HS-GC-FID) was 5.0–750.0 µg/L (Hansson & Hakkarainen, 2006) and for online solid-phase extraction liquid chromatography with diode array detector (SPE-LC-DAD) linearity was 10.0–1000.0 µg/L (Saim, Osman, Sabian, Zubir, & Ibrahim, 2012).

During our research, we established that RSD for the SFS method ranged from 0.84–3.7% depending on the measured concentrations. This finding is similar to other methods: for gas chromatography, the RSD had been found to be 3% (Hansson & Hakkarainen, 2006) and in the range of 2.4–9.3% (Lin, Song, Fang, Wu, & Wang, 2017), while liquid chromatography has been found to be 5.3% (Gennari et al., 2012) and in the ranges of 2.1–3.3% (Saim et al., 2012) and 0.1–0.3% (Moradi, Kiarostami, & Amini, 2017).

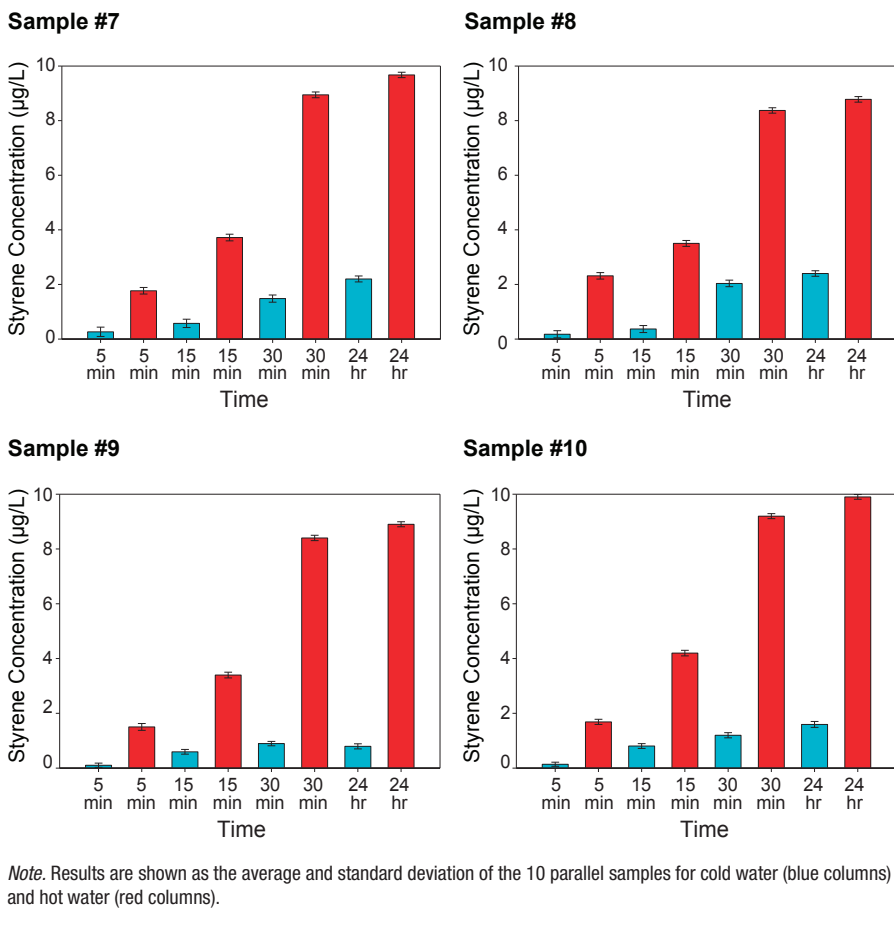
As for our measured results, the range in cups and food containers for hot water depending on the time of exposure was 1.45–9.95 µg/L and for room temperature water was 0.10–2.78 µg/L. Results obtained in previous studies (Ahmad & Bajahlan, 2007) ranged up to 29.5 µg/L, higher than the World Health Organization's (2008) guideline value limit of 20 µg/L. During our research, it was noticeable that time exposure plays a significant role in amount of released styrene. Regarding the temperature of water used in our experiments, there was also a significant difference between the results, in that we found higher concentrations in samples for which we had used hot water; this finding correlates with previous research (Ahmad & Bajahlan, 2007; Withey, 1976). Different results of styrene measured in different cups and food containers can be caused by different manufacturing processes for polystyrene cups and food containers. Other researchers have observed this difference (European Chemicals Bureau, 2002). Other components can vary from industry to industry and differ based on production process; analyses have found ethyl benzene (<0.1%), isopropyl benzene, toluene, benzene, p-xylene, and 2-phenylpropene, but these and other identified aromatic compounds were detected in much smaller amounts than styrene (Ahmad & Bajahlan, 2007), so we have analyzed only styrene.

Conclusion

This study proposed a simple, low-cost, and rapid method with easy operation for deter-

FIGURE 3 continued from page 27

Sample Concentrations of Styrene Released From Plastic Products Using the Spectral Fluorescence Signature Analysis



mination of styrene in water samples by SFS detection, which is available to most research laboratories. Compared with the previously published methods, the proposed SFS method shows adequately low limits of detection and quantification, good repeatability, and low consumption of solvent and sample volumes. Therefore, the presented method can be considered as a routine laboratory method for analysis of styrene in aqueous samples.

The SFS method is suitable for routine analyses, but the use of GC-MS would be advisable to confirm identification of compounds and get further quantitative information if such information is needed. Regarding given results, this method is suitable for measuring styrene and we believe that it is necessary to further develop this method in order

to detect harmful compounds in the samples, thus protecting the health of people who use plastic daily.

Considering the toxic characteristic of styrene and its leaching into water and other products, polystyrene material should be avoided for food packaging; furthermore, rigid polystyrene and foam cups should not be used for hot drinks. It is also recommended that a public awareness program about avoiding the use of Styrofoam cups for hot drinks be launched.

Some of the limitations of this SFS method are that if some of the water samples are polluted by a large amount of organic matter, disturbance and misalignments of the measured parameter can occur. We feel that we can ignore this limitation, however, because

we used pure polystyrene plastics and only distilled water, which has a spectrum that is characterized by a hardly visible fluorescence organic matter band. Also, our results might be limited in that we used samples randomly from store shelves, so it could be that we did not cover all possible types of polysty-

rene plastic. The release of toxic substances largely depends on the technological process in the production of polystyrene plastic products. Further research should be conducted to analyze other types of plastics using the SFS method, which has the advantages of high sensitivity and rapid identification. 🐞

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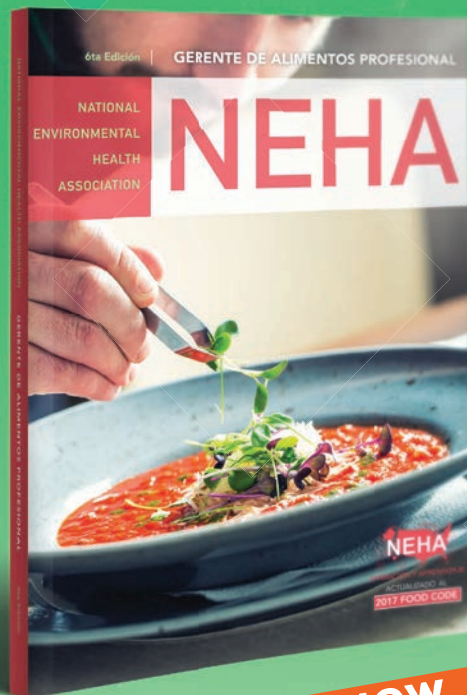


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Good Pool Chemistry Keeps Swimming Healthy and Safe

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 this column on environmental health services from the Centers for Disease Control and Prevention (CDC) in every issue of the *Journal*.

In these columns, authors from CDC's Water, Food, and Environmental Health Services Branch, as well as guest authors, will share insights and information about environmental health programs, trends, issues, and resources. The conclusions in these columns are those of the author(s) and do not necessarily represent the official position of CDC.

Michele Hlavsa is chief of the Healthy Swimming Program in CDC's National Center for Emerging Zoonotic and Infectious Diseases (NCEZID). CDR Joseph Laco serves as an environmental health officer at CDC's National Center for Environmental Health. Vincent Hill is chief of the Waterborne Disease Prevention Branch in CDC's NCEZID.

Many pool chemicals are used to protect the health and safety of swimmers and aquatics staff. For example, to help prevent outbreaks of infectious diseases, chlorine or bromine is added as a barrier to pathogen transmission. Muriatic (hydrochloric) acid is added to maintain pH at 7.2–7.8, taking into account disinfectant efficacy, swimmers, and equipment. Clarifiers are added to maximize water clarity, which enable lifeguards and others to identify distressed swimmers underwater and help prevent drownings.

Pool Chemical Injuries

While pool chemicals help pool owners and operators maintain healthy and safe water

conditions, chemical handling mistakes can lead to serious injuries. National Electronic Injury Surveillance System (NEISS) data tell us pool chemical injuries annually lead to an estimated 3,000–5,000 U.S. emergency department (ED) visits. Almost half of ED patients are less than 18 years. Poisoning due to inhalation or ingestion and dermatitis/conjunctivitis are the leading injury diagnoses (Centers for Disease Control and Prevention, 2009, 2011; Hlavsa, Robinson, Collier, & Beach, 2014).

As you would expect, the injuries typically occur during the summer swim season (Memorial Day weekend to Labor Day). NEISS injury reports indicate that injuries can be caused by an individual pool chemical or the mixing of

incompatible pool chemicals (e.g., in a bucket). Chlorine and acid are a powerful disinfection combination when each is diluted before they are mixed together; however, mixing concentrated chlorine and acid generates toxic chlorine gas. NEISS injury reports also indicate handling pool chemicals without using personal protective equipment, particularly when opening containers, and not securing pool chemicals away from children can lead to pool chemical injuries. The Agency for Toxic Substances and Disease Registry's Hazardous Substances Emergency Events Surveillance (now called the National Toxic Substance Incidents Program) data indicate human error is the leading factor that contributes to releases of pool chemicals (Anderson, Welles, Drew, & Orr, 2014).

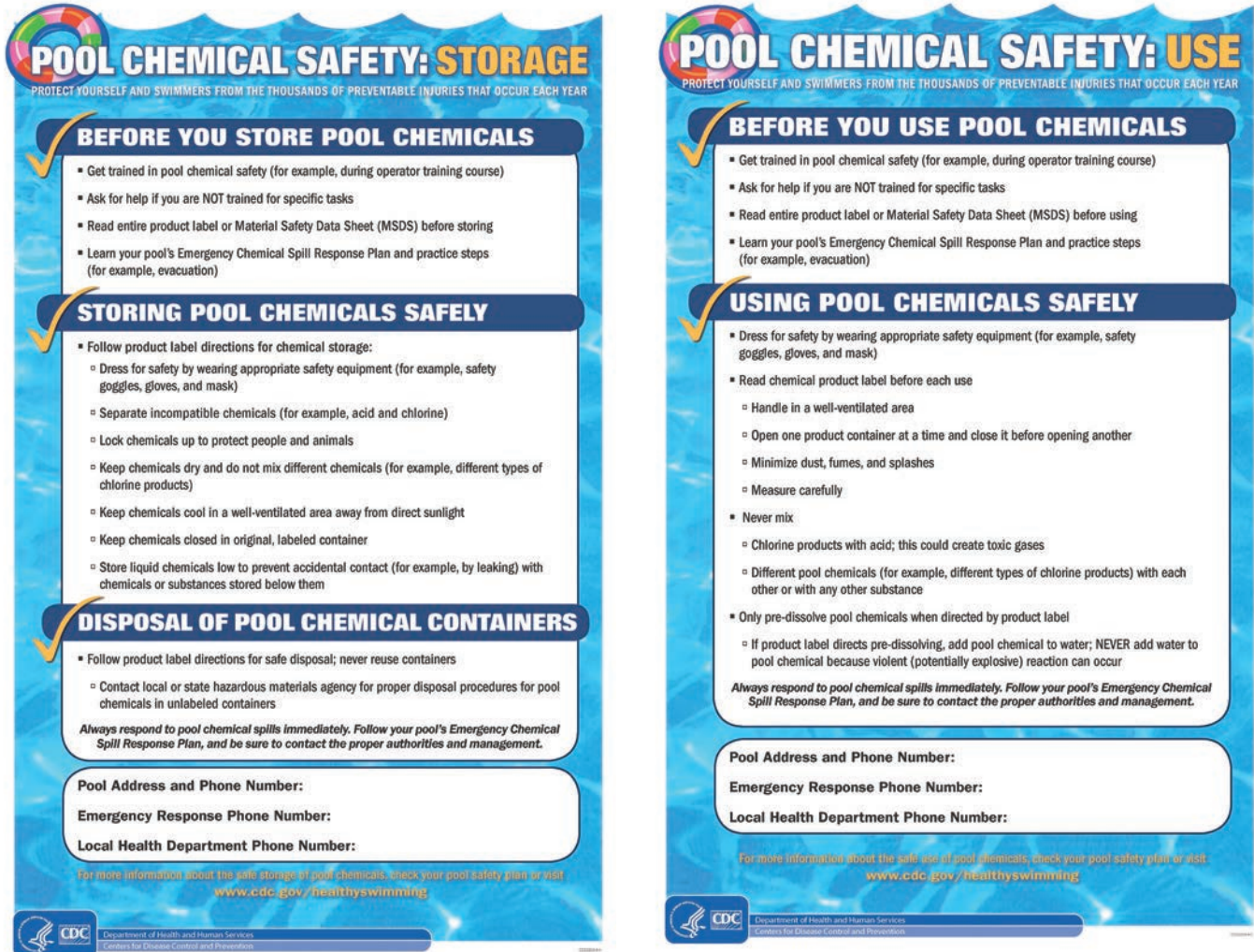
NEISS records are on individual injured patients and the described pool chemical injuries typically lead to one individual visiting the ED, which isn't always the scenario with pool chemical injuries. One toxic chlorine gas event can affect scores of swimmers and aquatics staff (Hlavsa et al, 2018; Wilken et al, 2017). U.S. national outbreak data indicate toxic chlorine gas events can occur if there is no or low water flow in the recirculation system while the chemical feed system simultaneously continues to run. This combination of events allows concentrated chlorine and acid to mix and the generated toxic chlorine gas to build up in the recirculation system. The toxic chlorine gas is released through the inlets and into the pool when normal water flow is restored within the recirculation system.

Preventing Pool Chemical Injuries

Fortunately, pool chemical injuries are preventable through education, engineering,

FIGURE 1

Free Laminated Pool Chemical Safety Poster Available in English and Spanish



To order, visit www.cdc.gov/pubs/cdcInfoOnDemand.aspx?ProgramID=93.

and enforcement. To minimize the risk of these injuries, pool chemical safety training (Figure 1) should be included in operator training and provided to any aquatics staff involved in storing or handling pool chemicals. Additionally, preventing unauthorized access to chemical storage spaces, exhausting air from these spaces at rates that help protect occupant health and safety, and providing eyewash stations in these spaces can minimize the risk of pool chemical injuries or at least their severity. To specifically minimize risk of toxic chlorine gas events, the chemical (chlorine and acid) feed should be deac-

tivated if there is no or low water flow in the recirculation system.

These examples of preventive education and engineering measures are recommended in the Model Aquatic Health Code (MAHC, www.cdc.gov/mahc). The MAHC's overarching objective is to prevent illness and injuries associated with public treated recreational water venues (i.e., pools, hot tubs/spas, and water playgrounds), which it does through providing recommendations based on the latest science or best practices. State and local jurisdictions, depending on their individual needs, can voluntarily adopt all or part of the

MAHC. Because the MAHC provides prevention recommendations in its chapters on design and construction, operation and maintenance, and policy and management, recommendations to prevent a specific illness or injury can appear in multiple MAHC chapters.

State and local environmental health colleagues have reported that it can be difficult to find all the relevant MAHC code and supporting annex rationale language. In response, the Centers for Disease Control and Prevention is developing Mini-MAHCs. Mini-MAHCs are concise documents that aggregate MAHC code and annex language

on a specific public health issue. One Mini-MAHC addresses general pool chemical safety (Preventing Pool Chemical Injuries), while another specifically addresses toxic chlorine gas events (Preventing Toxic Chlorine Gas Events, www.cdc.gov/mahc/editions/current.html). All Mini-MAHCs reference content from the 2018 MAHC, 3rd Edition.

Maximizing the positive public health impact of pool chemicals calls for minimizing the risk of pool chemical injuries. State and local environmental health practitioners are on the frontline of prevention through educating pool operators about pool chemical safety, inspecting on pool code elements that minimize the risk of pool chemical injuries, investigating pool chemical injuries to identify their root cause(s), and informing the development of optimized measures to prevent future events. Without state and local environmental health practitioners, we cannot have healthy and safe swimming.

For more information on preventing pool chemical injuries, visit www.cdc.gov/healthy

water/swimming/aquatics-professionals/preventing-pool-chemical-events.html. 🐼

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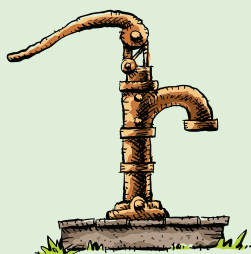
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▶ INTEGRATING PUBLIC HEALTH IN LAND REUSE AND REDEVELOPMENT

Editor's Note: The National Environmental Health Association is publishing a three-part series that highlights collaboration and partnerships with the Agency for Toxic Substances and Disease Registry (ATSDR) and redevelopment stakeholders to promote environmental health and land reuse as environmental and public health practices. This series will serve as a guide for identifying new and existing resources that can be adopted at the local environmental health level to safely reuse environmentally impacted land to improve community health outcomes. The conclusions in this series are those of the author(s) and do not necessarily represent the official position of the Centers for Disease Control and Prevention and ATSDR.

Part 2: Assessing Local Health Agency Capacity to Integrate Environmental Health and Land Reuse Work

Laurel Berman, PhD
Agency for Toxic Substances
and Disease Registry

Stephanie DeFlorio-Barker, MPH, PhD
U.S. Environmental Protection Agency

Sandra Whitehead, PhD
National Environmental
Health Association

Abstract Many local health departments (LHDs) across the country coordinate with their service areas on environmental health or land reuse. The Brownfields & Reuse Opportunity Working Group (BROWN) is a multipartner land reuse stakeholder network that includes member representatives from state and local health agencies, federal agencies, environmental consultants, environmental health professionals, and academia. In 2015, BROWN provided input on five Environmental Health Resources Self Learning Modules (Epidemiology, Risk Assessment, Risk Communications, Land Reuse Sites, and Toxicology) that the Agency for Toxic Substances and Disease Registry (ATSDR) was developing. ATSDR created the educational modules as resources and self-study guides to increase LHD capacity to respond to environmental issues. Following input from BROWN members on the modules, the National Environmental Health Association independently developed a short survey to identify baseline capacity of environmental professionals, primarily LHD professionals, to address environmental health and land reuse issues. The survey results of 93 LHD personnel indicated variation in the level of education among LHD employees and how often specific environmental health and land reuse services were requested. A subset of three LHD respondents also provided input into the learning modules.

Introduction Through the Brownfields & Reuse Opportunity Working Network (BROWN) (www.atsdr.cdc.gov/sites/brownfields/stakeholders.html), a brownfields and land reuse collaboration, the U.S. Environmental Pro-

tection Agency (U.S. EPA), Agency for Toxic Substances and Disease Registry (ATSDR), and National Environmental Health Association (NEHA) engage in public health-focused land reuse and brownfields redevelopment. We broadly use the terms brownfields and

land reuse sites to represent properties that are potentially contaminated and might be reused (www.atsdr.cdc.gov/sites/brownfields). One key goal of our collaboration and the objective in publishing this column is to ensure that health agencies, particularly local health departments (LHDs), are prepared for engagement in brownfields and land reuse sites. Health agency engagement in land reuse can lead to healthy redevelopment that can revive not only the economy and environment but also reduce health disparities through built environment improvements.

While federal and state environmental agencies are primarily responsible for brownfields and land reuse, state or local health agencies are often engaged, particularly if there are community concerns about potential contamination and exposures. In the 2013 *National Profile of Local Health Departments*, the National Association of County and City Health Officials surveyed 2,000 LHDs in the U.S. to describe their infrastructure and practice (National Association of County and City Health Officials, 2014). About 83% of the LHDs reported environmental health partnerships with community organizations, of which 31% reported land use involvement. In addition, LHDs engaged in pollution prevention (22%), hazardous materials response (17%), air pollution (16%), hazardous waste disposal (15%), and policy or advocacy activities in community-level urban design and land use policies to encourage physical activity (26%). All of these activities can be

TABLE 1

National Environmental Health Association Local Health Department Survey Questions

#	Question
1	Do you work at a local health department (LHD)?
2	What is the size of your service area in terms of population?
3	How would you describe your service area (urban, rural, suburban, territorial, other)?
4	How many employees work at your health department?
5	Do you work on environmental health and land reuse issues?
6	What is your title?
7	Do people ask for your help regarding environmental health AND land reuse/brownfields issues?
8	If yes to question 7: Who requests your services? How often are you consulted? What services do you perform?
9	If no to question 7: Is there someone else in your agency to whom these questions are referred?
10	Do you perform any of the following services: risk communication, risk assessment, toxicology assessments, epidemiology assessments, and consultations on land reuse/brownfields or hazardous waste sites?
11	Have you had any training in the following: risk communication, risk assessment, toxicology, epidemiology, and land reuse/brownfields or hazardous waste sites? [A range of training levels from no formal training through advanced graduate degrees was included.]
12	If free, self-study, online training was available in the following topics, do you think it could increase your skills: all topics, risk communication, risk assessment, toxicology, epidemiology, and land reuse/brownfields or hazardous waste sites?
13	If you had an opportunity to test proposed online training in risk communication, risk assessment, epidemiology, toxicology, and land reuse/brownfields or hazardous waste sites, would you participate?
14	If you are willing to participate in a focus group to test proposed online training in risk communication, risk assessment, epidemiology, toxicology, and/or land reuse/brownfields or hazardous waste sites, please provide your contact information [name, company, city, state, e-mail address, and phone number].

necessary in brownfield and land reuse site assessment and redevelopment.

BROWN is a multisector land reuse collaboration that provides free consultation to communities with concerns about contaminated properties. BROWN includes representatives from ATSDR, U.S. EPA, other federal agencies, NEHA, state and local health agencies, environmental consultants, and academia. One key goal of BROWN is to support environmental health education that is geared towards health agencies. For example, BROWN provided input into ATSDR's Environmental Health Resource Self Learning Modules—Epidemiology, Land Reuse Sites, Risk Assessment, Risk Communications, and Toxicology—that are available at www.atsdr.cdc.gov/sites/brownfields/for_health_agencies.html#LearningModules. This collaboration ultimately resulted in a survey developed by NEHA to assess LHD staff skills in environmental health and to gauge the effectiveness and potential impact of the modules. In another collaboration, we created a 3-part short video series, Engaging Health Departments in Brownfields/Land Reuse Redevelopment, that highlights ways that health agencies can promote and build capacity to become involved in land reuse/brownfields work (www.atsdr.cdc.gov/sites/brownfields/videos.html).

Methods
In June 2015, ATSDR held an in-person focused discussion with BROWN members to collect input on the Environmental Health

Methods

Resources Self Learning Modules so that they would be specifically useful to increase LHD capacity in land reuse work. Subsequently, from May to June 2016, NEHA independently conducted an online survey among its members, who are primarily from LHDs, as a first step to identify a baseline capacity of local environmental health professionals to address environmental health and land reuse issues. NEHA surveyed its members through its membership database. As a support organization, NEHA is nonfederal and is not subject to federal human subjects and Office of Management and Budget regulations. All NEHA members who participate in the survey did so voluntarily. The 14-question survey took less than 5 minutes to complete (Table 1). Through its collaboration in BROWN, NEHA included a survey question to establish a subset of LHD respondents who would be willing to participate in a brief second survey conducted by NEHA (data not included here) to test the draft learning modules.

LHDs can have jurisdiction over several types of service areas and as such, NEHA evaluated service areas as rural, urban, suburban, or mixed to see if differences existed in terms of the types of services requested, education level, or interest in online training by service area. NEHA also examined the proportion of respondents typically asked to perform risk communication, risk assessment, toxicology, epidemiology, and land reuse/brownfields work according to the level of education. NEHA shared the summary results of the survey with U.S. EPA and ATSDR. No individuals were identified in the summarized survey results. To obtain a more refined statistical analysis, U.S. EPA further assessed the summary survey results using Stata version 13.

Results
Brownfields & Reuse Opportunity Working Network Input Into Environmental Health Resources Self Learning Modules
BROWN members made several suggestions to improve the learning modules, including providing case examples in some of the modules, emphasizing that the modules were not intended to replace formal training, and ensuring that all five modules were consistent in format and utilized plain language.

Results

Brownfields & Reuse Opportunity Working Network Input Into Environmental Health Resources Self Learning Modules

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Results of the National Environmental Health Association's Local Health Department Survey

The NEHA survey had 109 respondents, of which 93 (85%) indicated that they currently worked in a health department and 85 (91%) responded to survey questions pertaining to their health department (Table 2). The majority of respondents (68%) indicated that they worked in relatively small LHDs (1–24 employees). Approximately 53% of the survey participants indicated servicing a rural area, 20% a suburban area, and 19% a mixture of rural, urban, suburban, or territorial areas.

The level of education or the type of activities requested did not differ among different service areas (see supplemental tables at www.neha.org/jeh/supplemental). The level of education, however, varied with how often services were requested of LHDs in risk communication, risk assessment, toxicology, epidemiology, and land reuse/brownfields (Figure 1). Of the respondents who performed tasks within the five areas, a range of 5–75% of LHD employees had no formal training. Only 4 of 85 respondents performed toxicology tasks, of which 75% had no formal training. Among LHD respondents who indicated working on land reuse/brownfields issues, almost 75% indicated having either no formal education (e.g., college-level classes) or only continuing education courses related to land reuse/brownfields.

On the last question of the survey, 29 respondents indicated interest in testing the draft ATSDR Environmental Health Resource Self Learning Modules. NEHA randomly contacted a subset of nine individuals and three provided feedback. Their feedback indicated that the learning modules ranged from somewhat to very useful and would be useful for increasing their capacity in environmental health and land reuse.

Discussion

ATSDR updated the Environmental Health Resource Self Learning Modules to reflect the changes suggested by BROWN members. The survey conducted by NEHA provided valuable insight as to the capacity and education attained among those working on important community environmental health issues. The survey results indicated mixed levels of training completed by LHD staff in five different areas of environmental health

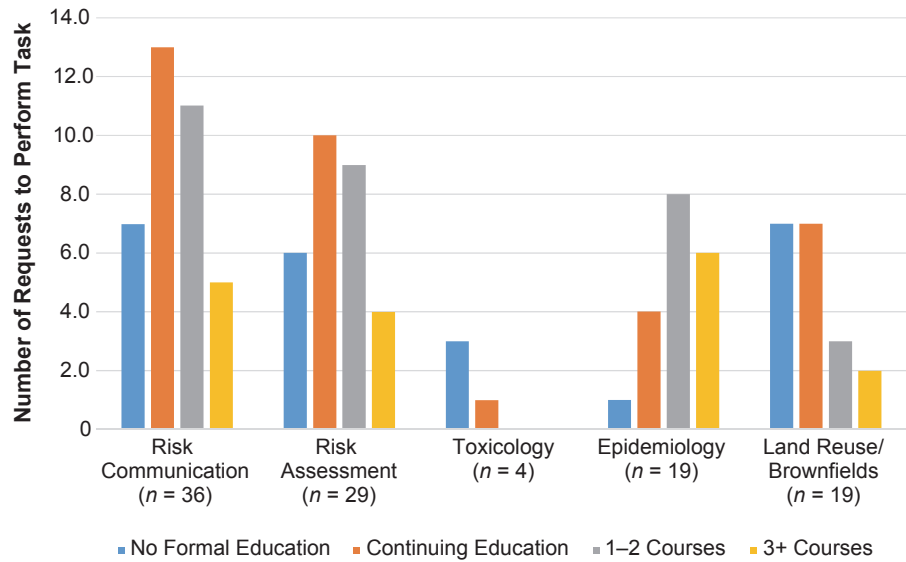
TABLE 2

National Environmental Health Association Local Health Department Survey Participant Characteristics (n = 85)

Characteristic	# (%)
Number of health department employees	
1–24	58 (68.2)
25–99	13 (15.3)
100–249	6 (7.1)
250–499	6 (7.1)
500–999	1 (1.2)
≥1,000	1 (1.2)
Type of service area	
Rural	45 (52.9)
Suburban	17 (20.0)
Rural/urban	7 (8.2)
Urban	6 (7.1)
Rural/urban/suburban	5 (5.9)
Rural/suburban	3 (3.5)
Rural/urban/suburban/territorial	1 (1.2)
Population of health department service area	
0–4,999	3 (3.5)
5,000–24,999	33 (38.8)
25,000–99,999	27 (31.2)
100,000–499,999	15 (17.7)
500,000–999,999	2 (2.35)
≥1,000,000	5 (5.9)
Job title	
Director of public health	26 (30.6)
Sanitarian	11 (12.9)
Health officer	9 (10.6)
Director of environmental health	6 (7.1)
Environmental supervisor	6 (7.1)
Health educator	4 (4.7)
Health equity/planning/policy manager	4 (4.7)
Public health nurse	4 (4.7)
Engineer	3 (3.5)
Environmental health scientist	3 (3.5)
Dietician/nutritionist	2 (2.4)
Health programs coordinator	2 (2.4)
Program/section manager	2 (2.4)
Administrative	1 (1.2)
Food inspector	1 (1.2)
No title	1 (1.2)

FIGURE 1

Summary of Education Level and Request Frequency of Health Department Employees to Perform Tasks in Five Specific Environmental Health Areas



(Figure 1). Feedback on the use of the learning modules, while limited to only three LHD survey respondents, was positive and indicated that the modules were useful for providing knowledge about an unfamiliar topic and giving LHD personnel confidence to increase their skills in specific environmental health topics pertaining to land reuse.

As awareness of brownfields and land reuse sites increases, opportunities to engage LHDs increases. LHD staff, with their proximity to communities, can ensure the safe reuse of land and assessment of potential exposures to contaminants associated with brownfields and land reuse sites. Through BROWN, ATSDR, U.S. EPA, NEHA, and other partners

intend to collaborate with other stakeholders to continue to help build capacity of LHDs to engage in environmental health and land reuse work.

One outcome is a newly developed ATSDR-NEHA Environmental Health and Land Reuse (EHLR) Certificate Program that will be completed in late spring 2019 and will subsequently be available as free training for environmental professionals, such as those in LHDs, to further increase their understanding of and skills in environmental health and land reuse. Participants who successfully complete the training will be eligible for continuing education credits from ATSDR and a Certificate of Completion in EHLR issued by NEHA. Ultimately, we hope all the tools and resources geared towards educating LHDs in environmental health and land reuse lead to increased abilities to perform a range of environmental health services and improved overall public health in local communities. 🚗

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Reference

National Association of County and City Health Officials. (2014). *2013 national profile of local health departments*. Washington, DC: Author.



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July 12–15, 2021: NEHA 2021 Annual Educational Conference & Exhibition, Spokane, WA.

NEHA AFFILIATE AND REGIONAL LISTINGS

Alabama

October 16–18, 2019: Annual Conference, hosted by the Alabama Environmental Health Association, Lake Eufaula, AL. For more information, visit www.aeha-online.com.

Colorado

September 17–20, 2019: Annual Education Conference, hosted by the Colorado Environmental Health Association, Keystone, CO. For more information, visit www.cehawe.com.

Florida

July 30–August 2, 2019: Annual Education Meeting, hosted by the Florida Environmental Health Association, Howey in the Hills, FL. For more information, visit www.feha.org/events.

Georgia

June 12–14, 2019: Annual Education Conference, hosted by the Georgia Environmental Health Association, Stone Mountain, GA. For more information, visit www.geha-online.org.

Illinois

April 30–May 1, 2019: IEHA Central Chapter Annual Educational Conference, hosted by the Central Chapter of the Illinois Environmental Health Association, Normal, IL. For more information, visit <http://ieha.coffeecup.com/calendar.html>.

Minnesota

May 9–10, 2019: Spring Conference, hosted by the Minnesota Environmental Health Association, Deerwood, MN. For more information, visit <https://mehaonline.org>.

Montana

September 17–18, 2019: 2019 MPHA/MEHA Conference, hosted by the Montana Public Health and Environmental Health Associations, Bozeman, MT. For more information, visit www.mehawe.org.

Nebraska

September 25–26, 2019: NEHA Region 4 Fall Conference, hosted by the Nebraska Environmental Health Association, Omaha, NE. For more information, visit www.nebraskaneha.com/region4conference.html.

North Carolina

May 14, 2019: Spring Educational Conference, hosted by the North Carolina Public Health Association, Raleigh, NC. For more information, visit <https://ncpha.memberclicks.net>.

Texas

October 14–18, 2019: 64th Annual Educational Conference, hosted by the Texas Environmental Health Association, Austin, TX. For more information, visit www.myteha.org.

Utah

May 8–10, 2019: Spring Conference, hosted by the Utah Environmental Health Association, Cedar City, UT. For more information, visit www.ueha.org/events.html.

Washington


May 6–8, 2019: 67th Annual Educational Conference, hosted by the Washington State Environmental Health Association, Yakima, WA. For more information, visit www.wseha.org.

Wisconsin

October 16–18, 2019: Annual Educational Conference, hosted by the Wisconsin Environmental Health Association, Elkhart Lake, WI. For more information, visit www.weha.net.

TOPICAL LISTING

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September 11–13, 2019: *Legionella* Conference 2019, presented by NSF International and the National Environmental Health Association, Los Angeles, CA. For more information, visit www.legionellaconference.org. 

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Healthy and Safe Swimming Week is May 20–26. This year's theme is "Pool Chemistry for Healthy and Safe Swimming." The week highlights the roles that swimmers, parents of young swimmers, aquatics and beach staff, residential pool owners, and public health officials play in preventing disease outbreaks, drowning, and pool chemical injuries. Learn more at www.cdc.gov/healthywater/observances/hss-week.

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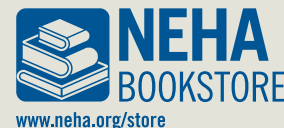
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Edited by David L. Heymann, MD (2015)



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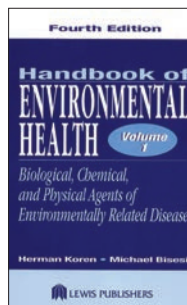
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Handbook of Environmental Health, Volume 1: Biological, Chemical, and Physical Agents of Environmentally Related Disease (4th Edition)

Herman Koren and Michael Bisesi (2003)



A must for the reference library of anyone in the environmental health profession, this book focuses on factors that are generally associated with the internal environment. It was written by experts in the field and copublished with NEHA. A variety of environmental issues are covered such as food safety, food technology, insect and rodent control, indoor air quality, hospital environment, home environment, injury control, pesticides, industrial hygiene,

instrumentation, and much more. Environmental issues, energy, practical microbiology and chemistry, risk assessment, emerging infectious diseases, laws, toxicology, epidemiology, human physiology, and the effects of the environment on humans are also covered. Study reference for NEHA's REHS/RS credential exam.

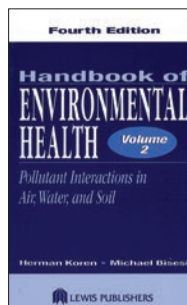
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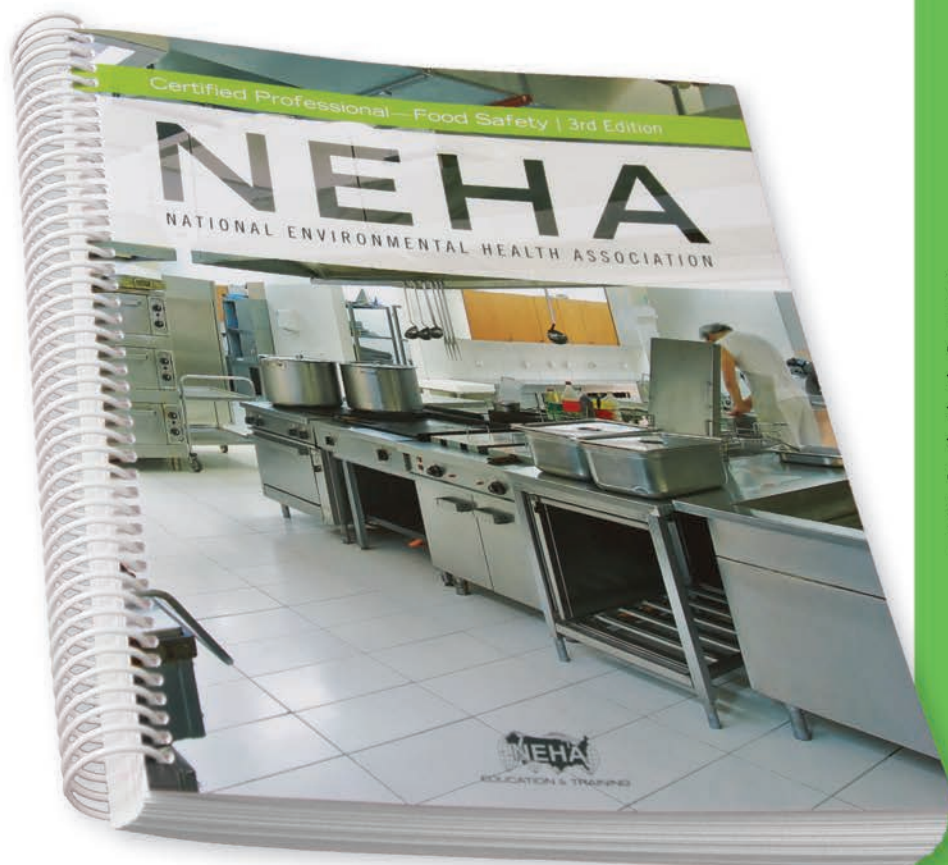
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*Creating a Voice to Protect the Nation
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Robert Kadlec, MS, MTM&H, MD
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CLOSING SESSION

*New Threats, New Techniques,
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Grayson C. Brown, PhD
Executive Director,
Puerto Rico Vector Control



GRAND SESSION KICKOFF

*A Profession United?
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FOOD SAFETY

A variety of food safety topics will be offered including retail and home restaurants, cannabis, and food safety and defense.

Be sure to check out the food safety sessions hosted by the National Center for Environmental Health including: *Improving Restaurant Food Safety; Critical Contributions; and Environmental Health Data That Inform Foodborne Illness Outbreak Prevention.*

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Learn the latest hot topic issues such as *Fentanyl Contamination, Exposure, Detection, Risk, and Decontamination; Weeding Out Public Health Risks of Cannabis; NEHA LeadHERship Sessions; Per- and Polyfluoroalkyl Substances (PFAS)—Opportunities and Challenges; and Food Freedom vs. Food Safety: AB 626, California's Newest Law.*

Other informative topics include Climate & Health, Data & Technology, Emergency Preparedness & Response, General Environmental Health, Healthy Communities, Infectious & Vectorborne Diseases, Special Populations, Water, and Workforce & Leadership.



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DirectTalk

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directors in large urban centers and influential jurisdictions. These respected professionals are NEHA members. I contend there are three attributes that make us natural leaders.

First, many of us have cultivated political savvy as a function of our jobs. We weave, bob, and broker conversations among the regulated community and elected officials. We understand the concerns of families and the public at large.

Second, we literally speak the language of our local constituents. How else could we effectively communicate expectations around compliance and best practice? In fact, I float the proposition that Darwinian forces, once applied, result in environmental health directors that excel in communication. We know how to work with our constituents and understand what motivates them.

Third, we spend most of our time in our business and regulated communities, not in an office. Most of us are detailed oriented. We generally have strong science educations. We know where the environmental risk factors exist in our regions. Who else would you want in the room if resource and personnel decisions need to be made to protect and promote the public's health? I'd want an informed, educated, and experienced leader.

Ecological Fallacy #3: Environmental Health Is Not Part of Emergency Preparedness and Response

Ladies and gentlemen, I hear this sentiment frequently, particularly in federal government circles. For example, environmental health is not specifically mentioned in the existing version of the Pandemic and All-Hazards Preparedness Act. Currently there is a notable absence of a Public Health Emergency Preparedness environmental health capability. It would seem at first blush from the national



The National Environmental Health Association's executive director in an underground coal mine, circa 1991. Photo courtesy of David Dyjack.

perspective that there is no role for us. Of course, this is patently untrue at the local level.

Who makes decisions about reoccupancy of smoke-damaged homes adjacent to wildfires? Who assists 20% of the public who use decentralized water sources after localized floods? When is the day care facility safe to reoccupy after a roof leak or sewage line break? Who better understands risk factors for norovirus in temporary shelters? The environmental health profession is an intimate and central player in emergency preparedness and response at the local level.

Ecological Fallacy #4: We Have the Data

On the face of it, environmental health professionals have plenty of data. In fact, a large fraction of public health data is environmentally oriented. For those of you in local and state government, you are familiar with Public Health Accreditation Board requirements. According to reputable sources, our profession generates much of the data used in Public Health Accreditation Board accreditation efforts.

Citizen scientists are quickly overtaking our monopoly of data collection and reporting. Websites and apps where citizen scientists report everything from ticks to foodborne illness are becoming increasingly common. Environmental health data are no longer solely within our control. Contemporary public health data are increasingly asymmetrical, dynamic, continuous, and reported immediately. I attended two excellent presentations earlier this week at the Oregon Environmental Health Association's conference. Both were focused on shoreline and aquatic health and safety, and both highlighted the important role of citizens in promoting our collective health and safety.

There are many urban legends and ecological fallacies about our profession that merit our attention. When everyone accepts the prevailing wisdom, no one thinks very much. Don't let that happen to you. 🐼

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Did You Know?

This year you can strengthen NEHA by participating in the Be a Beacon for NEHA Membership campaign! A growing NEHA means greater prominence for environmental health, more resources and support for members, and a larger community to tap into for support, collaboration, and friendship. Learn more about the campaign at www.neha.org/nehabeacon.

► **DirectTalk** MUSINGS FROM THE 10TH FLOOR

David Dyjack, DrPH, CIH

Brooks Brothers suits. Seven-fold Italian silk ties. Merino wool jackets and skirts. Jimmy Choo shoes. Association executive director events are predominantly a display of status.

Most, but not all, of my colleagues at these events are professional association executives. They lead their professions, often very effectively. In most cases, however, they are not part of the profession. Medical associations might not be led by a physician. Engineering associations might not be led by a professional engineer. This occurrence does not mean these executives are less committed to their members. On the contrary, they can be very committed, but yet are not part of the profession. If you were to drop in on one of these events, you would likely draw a conclusion about association executive directors. They are professional chief executive officers and not subject matter experts. Now, consider the National Environmental Health Association's (NEHA) executive director, yours truly.

I have worked in environmental health and safety for over 30 years. Along the way I have personally collected hundreds of lead samples from firing ranges, paint, water, and soil. I spent years conducting indoor air quality assessments in hospitals, schools, and day care centers. I've assessed carbon monoxide exposure to U.S. Secret Service personnel, measured solvent exposure in refinery workers, and wrote an oil spill health and safety plan for the North Atlantic and Gulf of Mexico. I share this past work with you to illustrate an ecological fallacy. What might be generally true about executive directors

Ecological Fallacies

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(i.e., they are not subject matter experts), might not be true at the individual level. An ecological fallacy occurs when group data are used to draw conclusions about individuals.

Ecological fallacies are abundant in our profession. I'm going to use this column to shine a light on a few prominent ones and I'm confident you have a few of your own. Let's start with one that is certain to perplex many of you.

Ecological Fallacy #1: There Is a Public Health Workforce Crisis

There is no workforce crisis. There is, on the other hand, a case to be made that there is a leadership and human resource management crisis. It was 2006 when Dr. Linda Rosenstock, the dean of the University of California, Los Angeles School of Public Health, first introduced this idea in my professional sphere. The general notion was that our country was on the leading edge of a national wave of Nixon-era professional retirements

and that the workforce would soon lose its intellectual capital. The argument had merit.

Fast forward 13 years and we remain mired in some derivative of the same conversation. At the same time, the National Center for Education Statistics reports that the second largest undergraduate enrollment in the U.S. is health and health services, coming in at around 228,000 individuals. While not all will choose public health or environmental health, that is a sizeable student population. At the graduate level, health professions represent the third largest student cohort behind business and education.

There is plenty of skilled and educated talent waiting to ascend into leadership in the public and private sectors. We hypothesize the very long hiring processes (particularly in the governmental sector), coupled with noncompetitive salaries and not a paucity of potentially qualified applicants, to be the crisis. Existing leaders at the local level should make it a priority to address salary and hiring bottlenecks and watch this issue self-resolve.

Ecological Fallacy #2: There Are Limited Opportunities for Leadership in Environmental Health

Approximately 15–20% of all local county and city health officials are registered environmental health professionals and/or registered sanitarians. I acknowledge that a large fraction of those leaders resides in rural and frontier communities; however, I personally know public health and healthcare agency

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