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Acute Gastroenteritis on Cruise Ships — United States, 2008–2014

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From 1990 to 2004, the reported rates of diarrheal disease (three or more loose stools or a greater than normal frequency in a 24-hour period) on cruise ships decreased 2.4%, from 29.2 cases per 100,000 travel days to 28.5 cases (*1*,*2*). Increased rates of acute gastroenteritis illness (diarrhea or vomiting that is associated with loose stools, bloody stools, abdominal cramps, headache, muscle aches, or fever) occurred in years that novel strains of norovirus, the most common etiologic agent in cruise ship outbreaks, emerged (*3*). To determine recent rates of acute gastroenteritis on cruise ships, CDC analyzed combined data for the period 2008–2014 that were submitted by cruise ships sailing in U.S. jurisdiction (defined as passenger vessels carrying ≥13 passengers and within 15 days of arriving in the United States) (*4*). CDC also reviewed laboratory data to ascertain the causes of acute gastroenteritis outbreaks and examined trends over time. During the study period, the rates of acute gastroenteritis per 100,000 travel days decreased among passengers from 27.2 cases in 2008 to 22.3 in 2014. Rates for crew members remained essentially unchanged (21.3 cases in 2008 and 21.6 in 2014). However, the rate of acute gastroenteritis was significantly higher in 2012 than in 2011 or 2013 for both passengers and crew members, likely related to the emergence of a novel strain of norovirus, GII.4 Sydney (*5*). During 2008–2014, a total of 133 cruise ship acute gastroenteritis outbreaks were reported, 95 (71%) of which had specimens available for testing. Among these, 92 (97%) were caused by norovirus, and among 80 norovirus specimens for which a genotype was identified, 59 (73.8%) were GII.4 strains. Cruise ship travelers experiencing diarrhea or vomiting should report to the ship medical center promptly so that symptoms can be assessed, proper treatment provided, and control measures implemented.

According to U.S. Foreign Quarantine regulations, passenger vessels, including cruise ships, are required to report the number of persons meeting the diarrheal disease case definition to U.S. authorities at CDC's Vessel Sanitation Program (VSP) 24–36 hours before arriving in the United States from a foreign port, even if there are zero cases (*6*). Additional reports are required if VSP's alert threshold is reached (≥2% cumulative attack rate* among either passenger or crew populations) or an outbreak occurs (≥3% cumulative attack rate among either passenger or crew populations); outbreaks of diarrheal disease are posted on VSP's website (http://www.cdc.gov/nceh/vsp). In 2001, VSP and the cruise industry expanded the diarrheal illness case definition to include acute gastroenteritis to more thoroughly detect and respond to illnesses that cause diarrhea and vomiting (*4*).

Data for 2008–2014 were analyzed per ship and voyage, using the most recently submitted report. Only voyages of 3–21 days in duration were included in the analysis, because cruise-associated illnesses associated with voyages of <3 days are more likely to manifest after disembarkation and, among voyages longer than 21 days (such as world cruises), report data often are incomplete. Voyages were included if they carried ≥100 passengers, because small vessels can meet VSP's

* Cumulative attack rate refers to the attack rate for an entire voyage.

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outbreak threshold with a single case; however, 99% of vessels submitting reports carried >100 passengers. Rates of acute gastroenteritis illness for both passenger and crew populations were calculated as the number of persons ill per 100,000 travel days and assessed for seasonality. Frequency of outbreaks per 1,000 voyages and the number of outbreaks per 10 million travel days also were calculated. The trend for each of these rates during 2008–2014 was assessed by fitting a linear regression line, and the trend for the actual number of outbreaks during this period was evaluated by Cochran-Armitage trend test. The rates per 100,000 travel days for 2011 and 2012, and for 2012 and 2013 were compared separately using a z-test. Similarly, the numbers of outbreaks per 1,000 voyages and per 10 million travel days for the same pairs of years were compared using a t-test and z-test, respectively.

During 2008–2014, a total of 32,084 voyages required submission of a VSP report, ranging annually from 4,404 in 2012 to 4,808 in 2014 (Table); among these, 29,107 (90.7%) were voyages of 3–21 days and included >100 passengers. Among a total of 73,599,005 passengers who traveled during this period, 129,678 (0.18%) cases of acute gastroenteritis were reported; and among 28,281,361 crew members,† 43,132 (0.15%) cases were reported.

The rate of acute gastroenteritis per 100,000 travel days among passengers ranged from 20.9 in 2013 to 27.2 in 2008, and among crew members, from 19.3 in 2013 to 21.6 in 2014. The rate of illness demonstrated a decreasing but not statistically significant trend for either passengers (p = 0.16) or crew members (p = 0.96). However, the rates for passengers and crew members were significantly higher in 2012 than in 2011 (passengers: p<0.01; crew members: p = 0.02) and 2013

TABLE. Acute gastroenteritis illness among passengers and crew members on cruise ships — United States, 2008–2014

Outbreak characteristics	Year						
	2008	2009	2010	2011	2012*	2013	2014
Voyages							
Total no. of voyages	4,694	4,506	4,627	4,621	4,404	4,424	4,808
No. of voyages included in analysis	4,098	3,964	4,155	4,189	4,168	4,146	4,387
Passengers							
No. of acute gastroenteritis outbreaks	20	17	21	15	27†	17	15
No. of outbreaks per 1,000 voyages	4.4	4.0	3.8	3.3	6.5†	4.2	3.0
No. of outbreaks per 10 million travel days	2.9	2.5	2.7	1.9	3.3†	2.1	1.8
Rate of acute gastroenteritis per 100,000 travel days	27.2	22.6	26.4	22.7	25.8†	20.9	22.3
Crew members							
No. of acute gastroenteritis outbreaks	1	4	3	1	1	1	4
No. of outbreaks per 1,000 voyages	0.3	0.8	0.5	0.0	0.3	0.3	0.7
No. of outbreaks per 10 million travel days	0.4	1.5	1.0	0.3	0.3	0.3	1.2
Rate of acute gastroenteritis per 100,000 travel days	21.3	19.7	20.8	19.6	20.3	19.3	21.6

* A novel strain of norovirus (GII.4 Sydney) emerged in 2012.
† Statistically significant increases: 2012 compared with 2011 and 2013.

† Represents the sum of crew members for each voyage reported to VSP.

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(passengers: p<0.01; crew members: p<0.01). Monthly rates during 2008–2014 were higher during October–April (Figure 1).

The number of annual acute gastroenteritis outbreaks among passengers ranged from 15 in 2011 and 2014, to 27 in 2012 (3.0–6.5 outbreaks per 1,000 voyages), and among crew members, ranged from one in 2008, 2011, 2012, and 2013 to four in 2009 and 2014 (0–0.8 outbreaks per 1,000 voyages) (Table); these differences exhibited a decreasing, although not statistically significant, linear trend (passengers: p = 0.89; crew members: p = 0.98). All but one of the crew outbreaks occurred concurrently with a passenger outbreak.

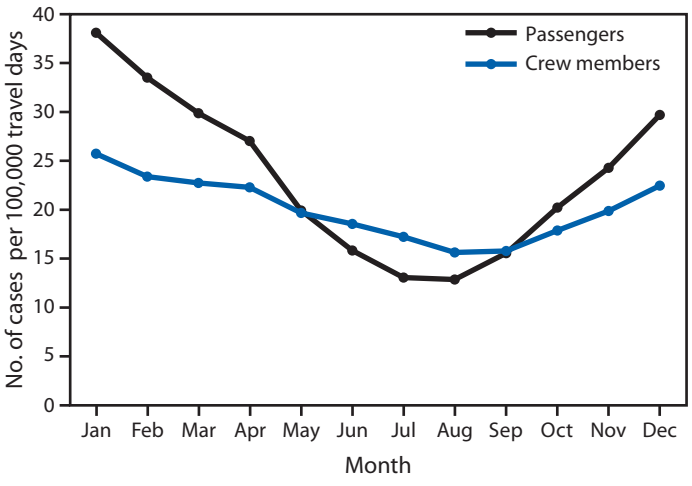
The number of outbreaks per 10 million travel days varied by year, ranging from 1.8 (2014) to 3.3 (2012) for passengers and from 0.3 (2013) to 1.5 (2009) for crew members. Although the raw number of outbreaks and rates per 10 million travel days decreased over time, the differences were not statistically significant (passengers: p = 0.52 and 0.29, respectively; crew members: p = 0.96 and 0.89, respectively). However, the rate increase among passengers in 2012 was statistically significant compared with 2011 (p = 0.04) but not compared with 2013 (p = 0.06). The rate of acute gastroenteritis outbreaks among crew members in 2012 was not statistically significantly different compared with either 2011 (p = 0.50) or 2013 (p = 0.49).

Of the 29,107 voyages, 133 (0.5%) had an outbreak. Among the 95 (71%) outbreaks for which clinical specimens were available for testing, viruses were identified as the only causative agent in 87 (92%) outbreaks, bacterial agents in three (3%), both viral and bacterial agents in four (4%), and viral and parasitic agents in one (1%) outbreak. All of the viral gastroenteritis outbreaks were caused by norovirus, including four that were caused by more than one strain. The bacterial agents implicated in the cruise ship outbreaks that affected passengers were enterotoxigenic *Escherichia coli* (six outbreaks), *Shigella sonnei* (one), and *Campylobacter jejuni* (one). One crew-only outbreak was caused by *Clostridium perfringens*. In addition, the parasite *Cyclospora cayetanensis* caused one outbreak (Figure 2). There were 129,678 passenger acute gastroenteritis cases during the study period. Only 19,273 (14.9%) of these cases were part of an outbreak (133 outbreaks on 29,107 voyages), and 13,568 (70.4%) cases were part of an outbreak in which the causative agent was laboratory-confirmed norovirus. Similarly, there were 43,132 crew member acute gastroenteritis cases of which only 1,984 (4.6%) were part of an outbreak, and 1,343 (67.7%) were part of an outbreak caused by norovirus.

Discussion

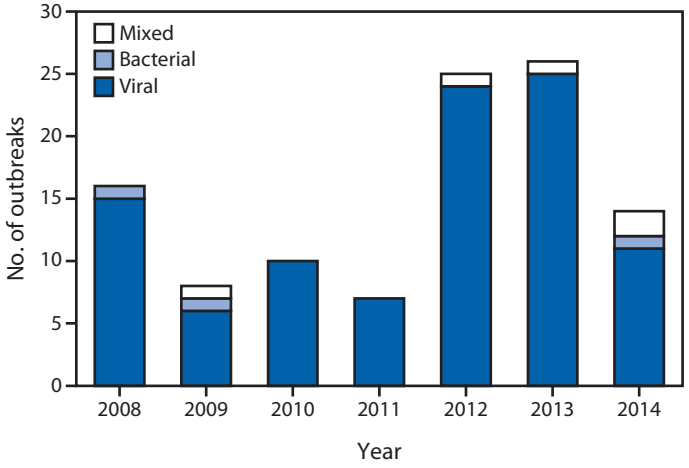
Approximately 73.5 million passengers sailed on voyages that required a VSP report during 2008–2014. During that period, 172,810 passengers and crew members met VSP's case definition for acute gastroenteritis, accounting for 0.18% of passengers and 0.15% of crew members (outbreak and non-outbreak illnesses combined). Among cruise ship outbreaks with clinical specimens tested, 92% were caused by norovirus, with enterotoxigenic *E. coli* the second most common etiologic agent. Noroviruses are highly transmissible, can spread easily, especially in environments where persons live in close quarters such as long-term care facilities or dormitories, and can remain infectious on environmental surfaces for long periods of time (*7,8*). Good hand hygiene is vital to preventing outbreaks of acute gastroenteritis, including on cruise ships. This is best accomplished by washing hands with soap and water because it allows for the mechanical removal of the virus from the hands

FIGURE 1. Monthly rates of acute gastroenteritis cases on cruise ships, by patient type — Vessel Sanitation Program, United States, 2008–2014*



* Data combined for the period 2008–2014, and incidence calculated by month.

FIGURE 2. Number of acute gastroenteritis outbreaks* on cruise ships, by year and causative agent type — Vessel Sanitation Program, United States, 2008–2014



* Five acute gastroenteritis outbreaks on cruise ships had more than one causative agent.

(*7*,*9*). Alcohol-based hand sanitizer use alone has shown limited efficacy, but can be used in conjunction with handwashing with soap and water (*7*).

The enterotoxigenic *E. coli* outbreaks all occurred outside the United States on ships sailing back to the United States after visiting Central or South America. Overall, 14,911 passenger and crew acute gastroenteritis cases were associated with norovirus outbreaks during 2008–2014; these accounted for only 0.01% of the estimated 140 million norovirus cases in the United States during that period (*3*). Monthly rates of acute gastroenteritis on cruise ships were higher during October–April for all years of the study period, with the highest rates of illness occurring during 2012, when a novel strain of norovirus was identified (GII.4 Sydney) (*5*). The overall seasonality and higher levels of illness in 2012 were similar to non-cruise ship U.S. acute gastroenteritis outbreak data, which showed higher rates of illness during November–April each year, and more norovirus illnesses during years when a novel strain of the virus was identified (*3*).

Rates of acute gastroenteritis and the number of acute gastroenteritis outbreaks were consistently lower among crew members than passengers, likely for multiple reasons. First, there are strict reporting and isolation requirements for crew members who experience acute gastroenteritis (*4*). Crew members who do not report symptoms of diarrhea or vomiting face discipline, which can include employment termination, because the risk they pose either through food handling or passenger interactions can lead to the spread of acute gastroenteritis. Additionally, because good hand hygiene is known to limit the spread of norovirus (*9*), hand washing stations are required at the entrances to all crew eating areas, and crew members are monitored in their use. Hand washing stations also are required to be located in food handling areas such that "no employee must walk more than 8 meters (26 feet) to reach a station" (*10*). As an additional food safety measure, crew members are not allowed bare hand contact with ready to eat foods (*4*). Finally, cruise lines have worked diligently to remove reporting barriers for passengers and crew members and encourage immediate reporting of any diarrhea or vomiting for medical assessment, treatment, and monitoring.

The findings in this report are subject to at least four limitations. First, voyages that did not include a foreign port or did not exceed VSP's alert or outbreak thresholds were not required to have a VSP report submitted, so the results might not reflect all voyages that occurred in U.S. waters. Second, VSP reports are only required when sailing from a foreign port to a U.S. port or when the cumulative incidence of acute gastroenteritis in either passenger or crew populations exceeds VSP's alert or outbreak thresholds; ships sailing between U.S. ports with a cumulative incidence lower than VSP's thresholds are not required to submit a report. Therefore, these data might not reflect the final case count at the end of the voyage. Third, case counts reported by ships and included in this study include only those persons who had symptoms while on the cruise ship and reported their symptoms to a crew member. The number of persons who experienced symptoms of acute gastroenteritis but did not report them is not known; thus, total case counts are likely underreported. Finally, in 2011, VSP included the phrase "or what is above normal for the individual" to the definition of diarrhea to align with federal regulation and the World Health Organization definition of diarrhea. This addition might have affected the number of persons who met the case definition because the definition now requires an assessment of "normal" rather than simply noting a frequency of ≥3 episodes in 24 hours.

Summary

What is already known on this topic?

From 1990 to 2004, the reported rates of diarrheal disease (three or more loose stools or a greater than normal amount in a 24-hour period) on cruise ships decreased 2.4%, from 29.2 cases per 100,000 travel days to 28.5 cases. In 2001, the Vessel Sanitation Program and the cruise industry expanded the diarrheal illness case definition to include acute gastroenteritis (diarrhea, or vomiting that is associated with loose stools, bloody stools, abdominal cramps, headache, muscle aches, or fever). The most common causative agent has been norovirus.

What is added by this report?

From 2008 to 2014, the rate of acute gastroenteritis on cruise ships decreased among passengers from 27.2 cases per 100,000 travel days in 2008 to 22.3 in 2014, while the rate among crew members was essentially unchanged. The rate among both passengers and crew members was higher in 2012 compared with the preceding and following years, likely because of the emergence of a new norovirus strain. Among 73,599,005 passengers on cruise ships during 2008–2014, a total of 129,678 (0.18%) cases of acute gastroenteritis were reported during outbreak and nonoutbreak voyages; among 28,281,361 crew members, 43,132 (0.15%) cases were reported. Only a small proportion of those cases were part of a norovirus outbreak.

What are the implications for public health practice?

Cases of acute gastroenteritis illness on cruise ships are relatively infrequent. Norovirus, the most common causative agent of outbreaks, accounted for 14,911 cases among passengers and crew members during 2008–2014, 0.01% of the estimated number of norovirus cases in the United States during the study period. To further reduce acute gastroenteritis on cruise ships, travelers should practice good hand hygiene, especially after using the toilet and before touching the face or eating; persons experiencing diarrhea or vomiting should promptly report their illness for proper assessment, treatment, and monitoring.

The number and severity of cruise ship outbreaks of acute gastroenteritis varied during the study period, but were lower than rates reported during 2001–2004 (*2*). Collaborative efforts with the cruise industry have allowed VSP to provide more rapid support to cruise lines and ships experiencing higher than expected levels of acute gastroenteritis. Fewer and less severe outbreaks are likely the result of earlier detection of acute gastroenteritis, along with cruise industry efforts to identify and control outbreaks by developing and implementing required Outbreak Prevention and Response Plans (*4*), using processes and chemical disinfectants known to be effective against a norovirus surrogate and proactively seeking strategies to limit acute gastroenteritis spread, using the most currently available evidence.

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References

1. Cramer EH, Gu DX, Durbin RE; Vessel Sanitation Program Environmental Health Inspection Team. Diarrheal disease on cruise ships, 1990–2000: the impact of environmental health programs. Am J Prev Med 2003;24:227–33. http://dx.doi.org/10.1016/S0749-3797(02)00644-X.
2. Cramer EH, Blanton CJ, Blanton LH, et al.; Vessel Sanitation Program Environmental Health Inspection Team. Epidemiology of gastroenteritis on cruise ships, 2001–2004. Am J Prev Med 2006;30:252–7. http://dx.doi.org/10.1016/j.amepre.2005.10.027.
3. Hall AJ, Lopman BA, Payne DC, et al. Norovirus disease in the United States. Emerg Infect Dis 2013;19:1198–205. http://dx.doi.org/10.3201/eid1908.130465.
4. Vessel Sanitation Program. 2011 operations manual. Atlanta, GA: US Department of Health and Human Services, CDC, Vessel Sanitation Program; 2011. http://www.cdc.gov/nceh/vsp/operationsmanual/opsmanual2011.pdf.
5. Leshem E, Wikswo M, Barclay L, et al. Effects and clinical significance of GII.4 Sydney norovirus, United States, 2012–2013. Emerg Infect Dis 2013;19:1231–8. http://dx.doi.org/10.3201/eid1908.130458.
6. Foreign quarantine. 42 C.F.R. Part 71. Washington DC: US Government Publishing Office; 2010. https://www.gpo.gov/fdsys/granule/CFR-2010-title42-vol1/CFR-2010-title42-vol1-part71/content-detail.html.
7. Hall A, Vinje J, Lopman B, et al. Updated norovirus outbreak management and disease prevention guidelines. MMWR Recomm Rep 2011;60(No. RR-03).
8. Zheng DP, Widdowson MA, Glass RI, Vinjé J. Molecular epidemiology of genogroup II-genotype 4 noroviruses in the United States between 1994 and 2006. J Clin Microbiol 2010;48:168–77. http://dx.doi.org/10.1128/JCM.01622-09.
9. Wikswo ME, Cortes J, Hall AJ, et al. Disease transmission and passenger behaviors during a high morbidity norovirus outbreak on a cruise ship, January 2009. Clin Infect Dis 2011;52:1116–22. http://dx.doi.org/10.1093/cid/cir144.
10. Vessel Sanitation Program. 2011 construction guidelines. Atlanta, GA: US Department of Health and Human Services, CDC, Vessel Sanitation Program; 2011. http://www.cdc.gov/nceh/vsp/constructionguidelines/constructionguidelines2011.pdf.

Sudden Deaths Among Oil and Gas Extraction Workers Resulting from Oxygen Deficiency and Inhalation of Hydrocarbon Gases and Vapors — United States, January 2010–March 2015

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In 2013, an occupational medicine physician from the University of California, San Francisco, contacted CDC's National Institute for Occupational Safety and Health (NIOSH), and the Occupational Safety and Health Administration (OSHA) about two oil and gas extraction worker deaths in the western United States. The suspected cause of these deaths was exposure to hydrocarbon gases and vapors (HGVs) and oxygen (O_2)-deficient atmospheres after opening the hatches of hydrocarbon storage tanks. The physician and experts from NIOSH and OSHA reviewed available fatality reports from January 2010 to March 2015, and identified seven additional deaths with similar characteristics (nine total deaths). Recommendations were made to industry and regulators regarding the hazards associated with opening hatches of tanks, and controls to reduce or eliminate the potential for HGV exposure were proposed. Health care professionals who treat or evaluate oil and gas workers need to be aware that workers might report symptoms of exposure to high concentrations of HGVs and possible O_2 deficiency; employers and workers need to be aware of this hazard and know how to limit exposure. Medical examiners investigating the death of oil and gas workers who open tank hatches should consider the contribution of O_2 deficiency and HGV exposure.

Workers at oil and gas well sites often manually gauge the level of fluid or collect a sample from storage tanks containing process fluids. These workers climb to the top of the tanks, open a "thief" hatch (a closable aperture on atmospheric tanks, used to sample the tank contents) (Figure), and either place a device into the hatch to measure the fluid level or lower a "thief" sampler (a hollow tube) into the tank to collect liquid samples. In 2013, an occupational medicine physician from the University of California, San Francisco, received a report of a 2012 oil and gas worker fatality in North Dakota; that state's medical examiner attributed death to the inhalation of petroleum hydrocarbons. The male worker, aged 21 years, was gauging crude oil production tanks on the well site, at night and alone. A coworker found the victim unconscious near the open hatch. Colleagues initiated cardiopulmonary resuscitation, and the worker was transported to the hospital where he was pronounced dead approximately 2 hours later. An autopsy found no obvious signs of traumatic injury. Toxicology testing identified detectable quantities of low–molecular weight hydrocarbons (propane and butane), and evidence of heavier molecular weight hydrocarbons. No indication of exposure to hydrogen sulfide (H_2S) was identified. Initially, the death was attributed to cardiovascular disease and later to hydrocarbons. The occupational medicine physician subsequently identified a second worker who died from a sudden cardiac event in 2010 while performing tank gauging; H_2S was excluded as a factor. The physician contacted NIOSH and OSHA about these two deaths.

To identify other oil and gas extraction worker fatalities associated with exposure to HGVs, the physician and experts from NIOSH and OSHA reviewed media reports, OSHA case files, and the NIOSH Fatalities in Oil and Gas database. Cases were defined as nontraumatic oil and gas extraction worker deaths occurring during January 2010–March 2015, in which the workers were 1) performing tank gauging, sampling, or fluid transfer activities at oil and gas well sites; 2) working in proximity to a known and concentrated source of HGVs (e.g., an open hatch); 3) not working in a confined space; and 4) not exposed to H_2S, fires, or explosions. All available information on identified fatalities was reviewed, including OSHA investigations, coroner and toxicology reports, gas monitor data, and exposure assessment data.

Nine deaths, occurring from January 2010 to March 2105, were identified (Table); six of the deaths occurred during 2014. Three deaths occurred in Colorado, three in North Dakota, and one each in Montana, Oklahoma, and Texas. The median age of workers was 51 years (range = 20–63 years), and all were male. All of the victims were working alone at the time of the incidents and were found collapsed on a tank or catwalk, or at the base of the catwalk stairs. In at least five cases, the hatch was open when the worker was found. Five of the fatalities occurred during the collection of a fluid sample, and four occurred during tank gauging. Toxicologic data on HGVs were not consistently collected during autopsy, but petroleum hydrocarbon vapors were noted as a cause of death for three workers.

Only one of the nine workers was known to have been provided a respirator, but fit-testing had not occurred, and the air-purifying respirator was not suitable for high concentrations of HGVs or O_2 deficiency. The exposure assessment conducted by OSHA following the 2010 case found O_2 concentrations as low as 11% at 1 foot above the open thief hatch (O_2 concentrations

FIGURE. An oil field worker manually gauges the level of process fluid in a fixed production oil tank* — United States

Photo/Todd Jordan, Occupational Safety and Health Administration.

* 1) The worker peers down an open hatch of the oil tank. 2) In some regions, workers gauge oil tanks by opening tank hatches, visually observing liquid levels, and then manually measuring liquid oil levels. 3) As commonly designed, fixed oil tanks often are interconnected for both liquid and vapor, allowing contents to equalize over multiple tanks. Equalization of tanks can result in a high volume of off-gassed vapors when a tank hatch is opened. 4) The windsock is a visual indicator for the worker to stay positioned upwind while gauging.

in ambient air = 21%). In addition, HGV concentrations were in excess of the lower-explosive limit (minimum concentration of a gas necessary to support its combustion in air), suggesting exposures high enough (>10,000 parts per million [ppm]) to cause acute central nervous system symptoms. In case number seven, the worker wore a data-logging, continuous multi gas monitor as a regular work practice. Three weeks before the fatal event, he was examined in an emergency department after experiencing altered consciousness while gauging a tank. Gas monitor data during this event revealed a 5-minute interval, concurrent with his symptoms, when O_2 concentrations were in the range of 10% to 15% and flammable HGVs exceeded the lower-explosive limit. On the day of his death, the gas monitor again indicated that the lower-explosive limit had been exceeded, with O_2 concentrations as low as 7%.

Discussion

During January 2010–March 2015, at least nine deaths of oil and gas workers occurred in the United States, with exposure to HGVs a confirmed or suspected factor. Oil and gas extraction is a high-risk industry, with overall occupational fatality rates seven times the national average (*1*). Although safety hazards in the industry are well-known, few published reports address chemical exposures and acute occupational illness associated with oil and gas extraction. Recent exposure assessments have identified that opening thief hatches and manual gauging or sampling from hydrocarbon-containing tanks, outdoors in nonconfined spaces, is widely practiced and poses substantial and potentially lethal hazards to workers (*2–4*). These hazards include sudden exposure to high concentrations (>100,000 ppm) of low–molecular weight HGVs, accompanied by displacement of air, resulting in O_2 deficiency. Inhaled O_2 concentrations of <15% can significantly impair central nervous system function, and concentrations of <10% can result in loss of consciousness and possible death within seconds to minutes (*5*). Low O_2 blood levels (hypoxemia) can exacerbate cardiac ischemia and increase the release of epinephrine (adrenalin). High concentrations (i.e., 50,000 ppm to ≥100,000 ppm) of low–molecular weight hydrocarbons, particularly butane, have been shown in animal studies and human reports to sensitize the heart to epinephrine-induced ventricular fibrillation, a lethal cardiac arrhythmia (*6–8*). The simultaneous exposure to high levels of low–molecular weight HGVs and a low O_2 atmosphere above an open tank hatch poses a risk for sudden cardiac death. Preexisting coronary artery disease can exacerbate that risk. In addition, high levels of low–molecular weight HGVs can exert anesthetic effects that contribute to central nervous system depression (*9*). The exposure-assessment samples also showed concentrations of propane, butane, pentane, and 2-methylbutane exceeding 100% of the lower-explosive limit (*3*). Concentrations of explosive gases in excess of 10% of the lower-explosive limit are considered immediately dangerous to life or health. Because of the nine identified fatalities, the exposure-assessment findings, and the potential mechanism for sudden cardiac death, OSHA, NIOSH and multiple industry stakeholders collaboratively issued a hazard alert on tank gauging at oil and gas well sites (*10*). In addition, the Bureau of Land Management has proposed changes to current federal

TABLE. Sudden deaths caused by inhalation of hydrocarbon gases and vapors and oxygen deficiency among oil and gas extraction workers — United States, January 2010–March 2015

Worker	Year of death	Age (yrs)	State	Job title	Job task	Location/position of decedent when found	Time of day found	Coroner's stated cause of death
1	2010	30	Montana	Crew worker	Gauging	Slumped over on catwalk	3:00 a.m.	Hypertensive and atherosclerotic cardiovascular disease
2	2012	21	North Dakota	Flow tester	Gauging	On catwalk next to open hatch	12:30 a.m.	Hydrocarbon poisoning due to inhalation of petroleum vapors
3	2013	39	North Dakota	Truck driver	Collecting sample	On knees, slumped over catwalk railing in front of open hatch	10:20 a.m.	Sudden cardiac arrhythmia (primary), morbid obesity and arteriosclerotic heart disease (contributory)
4	2014	57	Oklahoma	Truck driver	Collecting sample	Slumped over on catwalk next to tank	10:12 a.m. (time of death)	Undetermined (no autopsy performed)
5	2014	51	Colorado	Truck driver	Collecting sample	Hanging from guardrail, hooked by clothing	10:39 a.m. (time of death)	Sudden cardiac death due to ischemic heart disease
6	2014	57	Colorado	Truck driver	Collecting sample	Collapsed over open hatch	10:30 a.m.	Atherosclerotic cardiovascular disease
7	2014	59	Colorado	Truck driver	Collecting sample	Collapsed over open hatch	1:40 p.m.	Toxic gas inhalation and oxygen displacement by volatile hydrocarbons (primary), atherosclerotic cardiovascular disease
8	2014	63	Texas	Tank gauger	Gauging	At bottom of catwalk stairs	4:14 a.m.	Arteriosclerotic and hypertensive cardiovascular disease
9	2014	20	North Dakota	Flow tester	Gauging	Face down over open hatch	5:00 a.m.	Cardiac arrhythmia, with cardiac hypertrophy, coronary artery hypogenesis, obesity and petroleum hydrocarbon vapors

regulations* that replace outdated technology and practices with remote tank gauging technologies, reducing or eliminating the need for manual tank gauging.

Health professionals need to recognize the signs and symptoms of exposure to high concentrations of HGVs and possible O_2-deficient atmospheres in oil and gas workers. Health and safety professionals need to recognize and act on nonfatal warning signs and symptoms, such as dizziness, confusion, immobility, and collapse in oil and gas workers who might have been exposed to high concentrations of HGVs and to O_2-deficient atmospheres. As required by OSHA regulations, employers should reduce or eliminate the hazard; this can include practices that allow for alternative fluid sample collection points, remote monitoring of fluid levels, proper use of gas monitors, respiratory protection meeting OSHA requirements, and worker training. Employers also need to ensure that workers do not work alone where they might have risks for exposures to high concentrations of hydrocarbons and low-O_2 environments. Having automated external defibrillators available at worksites is also important. Medical examiners and coroners investigating workplace fatalities need to be aware of the possibility that exposure to high concentrations of HGVs and O_2-deficient atmospheres can result in sudden cardiac death in oil and gas extraction workers. Analysis of antemortem or postmortem blood for documentation of HGV exposure is available from clinical toxicology laboratories.

* https://www.federalregister.gov/articles/2015/09/30/2015-24008/onshore-oil-and-gas-operations-federal-and-indian-oil-and-gas-leases-measurement-of-oil.

Acknowledgments

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Summary

What is already known on this topic?

Oil and gas extraction workers experience high rates of traumatic work-related fatalities. Tank gauging and sampling activities can expose workers to high concentrations of hydrocarbon gases and vapors (HGVs), in some cases at levels immediately dangerous to life or health.

What is added by this report?

Exposure to high concentrations of HGVs and oxygen-deficient atmospheres during manual tank gauging and sampling can pose a risk for sudden cardiac death. Although the first two deaths described in this series were not immediately recognized as work-related, the occurrence of seven additional deaths under similar circumstances suggests that HGV exposure during manual tank gauging and sampling can be life-threatening.

What are the implications for public health practice?

Health care professionals need to be aware of the risks to oil and gas extraction workers related to exposure to high concentrations of HGVs and to oxygen deficiency. Medical examiners and coroners investigating worksite fatalities need to be aware that these exposures can result in sudden cardiac death and include appropriate toxicology analyses in their investigation. A thorough worksite assessment is warranted if any workers exhibit signs or symptoms of HGV exposure or oxygen deficiency. Implementation of measures to reduce or eliminate HGV exposures is important, including practices that allow for alternative fluid sample collection points, remote monitoring of fluid levels, proper use of gas monitors, respiratory protection meeting the requirements of the Occupational Safety and Health Administration, and worker training.

References

1. Mason KL, Retzer KD, Hill R, Lincoln JM. Occupational fatalities during the oil and gas boom—United States, 2003–2013. MMWR Morb Mortal Wkly Rep 2015;64:551–4.
2. Esswein EJ, Snawder J, King B, Breitenstein M, Alexander-Scott M, Kiefer M. Evaluation of some potential chemical exposure risks during flowback operations in unconventional oil and gas extraction: preliminary results. J Occup Environ Hyg 2014;11:D174–84. http://dx.doi.org/10.1080/15459624.2014.933960.
3. Jordan T. Hydrocarbon exposures during tank gauging and sampling operations [presentation]. NIOSH National Occupational Research Agenda Oil and Gas Sector Council Meeting, March 19, 2015. http://www.nationalstepsnetwork.com/docs_tank_gauging/NORA_Oil_and_Gas_Council_Meeting_March2015.pdf.
4. King B, Esswein E, Retzer K, et al. UPDATE: reports of worker fatalities during manual tank gauging and sampling in the oil and gas extraction industry. In: NIOSH Science Blog. Washington DC: National Institute for Occupational Safety and Health. http://blogs.cdc.gov/niosh-science-blog/2015/04/10/flowback-3/.
5. Miller TM, Mazur PO. Oxygen deficiency hazards associated with liquefied gas systems: derivation of a program of controls. Am Ind Hyg Assoc J 1984;45:293–8. http://dx.doi.org/10.1080/15298668491399811.
6. Chenoweth MB. Ventricular fibrillation induced by hydrocarbons and epinephrine. J Ind Hyg Toxicol 1946;28:151–8.
7. Reinhardt CF, Azar A, Maxfield ME, Smith PE Jr, Mullin LS. Cardiac arrhythmias and aerosol "sniffing". Arch Environ Health 1971;22:265–79. http://dx.doi.org/10.1080/00039896.1971.10665840.
8. Fuke C, Miyazaki T, Arao T, et al. A fatal case considered to be due to cardiac arrhythmia associated with butane inhalation. Leg Med (Tokyo) 2002;4:134–8. http://dx.doi.org/10.1016/S1344-6223(02)00004-4.
9. Drummond I. Light hydrocarbon gases: a narcotic, asphyxiant, or flammable hazard? Appl Occup Environ Hyg 1993;8:120–5. http://dx.doi.org/10.1080/1047322X.1993.10388128.
10. Occupational Health and Safety Administration National Steps Alliance. Tank hazard alert: gauging, thieving, fluid handling. How to recognize and avoid hazards [poster]. Washington, DC: U.S. Department of Labor, Occupational Health and Safety Administration; 2015. http://www.nationalstepsnetwork.org/docs_tank_gauging/TankHazardInfographicFinal04_22_15.pdf.

Notes from the Field

Subacute Sclerosing Panencephalitis Death — Oregon, 2015

Juventila Liko, MD[1]; Judith A. Guzman-Cottrill, DO[2]; Paul R. Cieslak, MD[1]

In 2015, the Oregon Health Authority was notified of the death of a boy with subacute sclerosing panencephalitis (SSPE), a rare and fatal complication of measles. The patient, aged 14 years, had reportedly been vaccinated against measles in the Philippines at age 8 months. However, the patient contracted measles at age 1 year while still in the Philippines. He had been well until 2012, when his neurodegenerative symptoms began. After the diagnosis of SSPE was made, the patient remained in home hospice care until his death. Investigators from the Oregon Health Authority and the Oregon Health and Science University reviewed the patient's medical records and interviewed the parents. Vaccination against measles can prevent not only acute measles and its complications, but also SSPE.

Investigators learned that, in 2012, at age 11 years, the boy, who was previously healthy and developmentally normal, had been admitted to a tertiary care children's hospital in Oregon with severe, progressive encephalopathy. Before the onset of his neurologic illness, the patient had been a straight-A, fifth-grade student who played soccer and basketball. The symptoms began approximately 4 months before the hospital admission, when the patient began to struggle with homework, drop utensils, and doze off during meals, eventually progressing to falling asleep while walking. During the subsequent month, his mother reported that he was less alert and sometimes seemed confused. He experienced myoclonic jerks and involuntary hand and arm movements, which became increasingly frequent, and his coordination deteriorated. He missed 3 weeks of school and required a home tutor. His appetite decreased, and he lost 12 pounds but remained playful and interactive.

A pediatric neurologist was consulted. No family history of neurologic disease was reported. The initial evaluation included a lumbar puncture and magnetic resonance imaging of the brain, both of which were unremarkable. An electroencephalogram (EEG) was abnormal, with frequent, high-amplitude bifrontal slowing, a nonspecific finding. Despite extensive evaluation, the cause of the neurologic degeneration was not identified.

During the following month, the patient's cognitive and motor skills declined further and included the onset of repetitive behaviors, as well as inability to sit still, frequent falling, and asking seemingly meaningless questions. He became aggressive and could no longer be tutored. During the month before his hospital admission, he began to shuffle and walk on his toes; he eventually refused to walk. He cried continuously, became increasingly aggressive, and began sleeping for longer periods. Although he was responsive at that time, his speech became difficult to understand; eventually he could say only a few words. A few days before hospital admission, he experienced worsening spasticity and rapid decline in mental status; he became incontinent and was unable to eat or drink. He did not fix on or follow objects, and he no longer appeared to recognize his family members' faces or voices.

Upon admission to the hospital in 2012, he had abnormal movements of the arms and legs, was unresponsive to questions, and unable to follow commands. He withdrew to touch and pain but evidenced spasticity and marked rigidity. All immunologic studies were normal. The EEG during this admission showed moderate, diffuse background slowing and disorganization, with multiple spikes and sharp waves, characteristic of SSPE. His serum measles IgG level was markedly elevated at >11.00 index value (IV) (positive ≥1.10 IV), and his cerebrospinal fluid (CSF) measles IgG level was >10.00 IV (positive >0.89 IV). Serum measles IgM was negative. The CSF measles IgG was confirmed at CDC's measles virus laboratory (titer = 1:40,960), and a diagnosis of SSPE was made. Because no specific therapy was available, the patient was discharged after 14 days and died in home hospice care 43 months later, in 2015.

The patient's clinical characteristics, typical EEG pattern, and elevated CSF measles antibody level are all consistent with SSPE (*1*,*2*), a progressive neurodegenerative disease associated with persistent measles virus infection in the central nervous system caused by aberrant viral gene expression.* The clinical time course and features of SSPE are highly variable, and its initial symptoms can be subtle.† The disease typically develops 7–10 years after infection with measles virus (*1*).

The patient had documentation of receipt of 1 dose of measles vaccine at age 8 months in the Philippines, although the patient contracted measles at age 1 year. Whereas nothing is known about the storage conditions or potency of the vaccine administered in the Philippines, vaccination during early infancy has been associated with lower seroresponses than the seroresponses of children vaccinated later, related to interference by circulating maternal antibody (*3*). Two doses of measles-containing vaccine are routinely recommended to ensure protection against measles (*4*).

* http://www.cdc.gov/vaccines/pubs/pinkbook/downloads/table-of-contents.pdf.
† http://indianpediatrics.net/apr1998/337.pdf.

Analysis of SSPE among persons who had measles during the 1989–1991 U.S. measles resurgence indicated an incidence of 4–11 SSPE cases per 100,000 measles cases, approximately 10 times higher than earlier estimates (*1*). Specimens for detection of viral RNA and genotyping were not available for this patient, but studies have shown that measles vaccine strains do not cause SSPE (*1*,*5*,*6*).

SSPE is a rare, long-term complication of measles. Widespread use of measles vaccine[§] has been associated with the near disappearance of SSPE in the United States. This case underscores the importance of maintaining high population immunity, through routine administration of 2 doses of measles-containing vaccine to all eligible children. The first dose should be administered at age ≥12 months, and the second dose at age 4–6 years. Infants aged 6–11 months who are traveling abroad should receive 1 dose of measles, mumps, and rubella (MMR) vaccine. Infants who receive MMR vaccine before age 12 months should be considered potentially susceptible to all three diseases and should be revaccinated with 2 doses of MMR vaccine, the first dose administered when the child is aged 12–15 months (12 months if the child remains in an area where disease risk is high) and the second dose at least 28 days later.

[§] Measles, mumps, and rubella (MMR) and measles, mumps, rubella, and varicella (MMRV) vaccines are the only measles-containing vaccines available in the United States.

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References

1. Bellini WJ, Rota JS, Lowe LE, et al. Subacute sclerosing panencephalitis: more cases of this fatal disease are prevented by measles immunization than was previously recognized. J Infect Dis 2005;192:1686–93. http://dx.doi.org/10.1086/497169.
2. Maldonado YA. Rubeola virus (measles and subacute sclerosing panencephalitis) [Chapter 227]. In: Long SS, ed. Principles and Practice of Pediatric Infectious Diseases. 3rd ed. Philadelphia, PA: Elsevier Churchill Livingstone; 2008:1120–6.
3. Redd SC, King GE, Heath JL, Forghani B, Bellini WJ, Markowitz LE. Comparison of vaccination with measles-mumps-rubella vaccine at 9, 12, and 15 months of age. J Infect Dis 2004;189(Suppl 1):S116–22. http://dx.doi.org/10.1086/378691.
4. McLean HQ, Fiebelkorn AP, Temte JL, Wallace GS. Prevention of measles, rubella, congenital rubella syndrome, and mumps, 2013: summary recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep 2013;62(No. RR-04).
5. Kühne M, Brown DW, Jin L. Genetic variability of measles virus in acute and persistent infections. Infect Genet Evol 2006;6:269–76. http://dx.doi.org/10.1016/j.meegid.2005.08.003.
6. Miki K, Komase K, Mgone CS, et al. Molecular analysis of measles virus genome derived from SSPE and acute measles patients in Papua, New Guinea. J Med Virol 2002;68:105–12. http://dx.doi.org/10.1002/jmv.10176.

Announcement

Glaucoma Awareness Month — January 2016

January is Glaucoma Awareness Month, a time to encourage persons at risk for glaucoma to schedule a comprehensive, dilated eye exam to detect and manage glaucoma. CDC's Vision Health Initiative in the Division of Diabetes Translation has partnered with the National Eye Institute's National Eye Health Education Program to educate the public and raise awareness about glaucoma, its risk factors, and its prevention.

Glaucoma affects the optic nerve in the back of the eye, and is one of the leading causes of preventable blindness (*1*). Glaucoma has no warning signs, and approximately 50% of persons with glaucoma are unaware that they have the disease; therefore, a comprehensive, dilated eye examination is important for early detection and timely treatment (*2*). In 2010, approximately 2.7 million persons in the United States aged ≥40 years had glaucoma; by 2050, this number is projected to increase to 5.5 million persons (*3*). Groups at high risk include non-Hispanic blacks aged >40 years, Hispanics, Asians, persons aged ≥60 years, and those who have diabetes or a family history of glaucoma (*4*). Glaucoma also is associated with an increased risk for falls, fall-related injuries, depression, and reduced overall health and quality of life (*5*–*7*).

In addition to regular comprehensive, dilated eye examinations to detect and treat glaucoma, innovative community-based interventions are proving successful in reaching populations at high risk (*8*). Additional information about activities to promote early detection and treatment of glaucoma is available (http://www.cdc.gov/visionhealth, https://nei.nih.gov/nehep/programs/glaucoma).

References

1. CDC. Enhancing public health surveillance of visual impairment and eye health in the United States. Atlanta, GA: US Department of Health and Human Services, CDC; 2012. http://www.cdc.gov/visionhealth/pdf/surveillance_background.pdf.
2. American Academy of Ophthalmology Preferred Practice Patterns Committee. Comprehensive adult medical eye evaluation. San Francisco, CA: American Academy of Ophthalmology; 2010.
3. America PB. The future of vision: forecasting the prevalence and costs of vision problems. Chicago, IL: Prevent Blindness America; 2014. http://forecasting.preventblindness.org/disease-projections/visual-impairment-and-blindness.
4. Coleman AL, Miglior S. Risk factors for glaucoma onset and progression. Surv Ophthalmol 2008;53(Suppl1):S3–10. http://dx.doi.org/10.1016/j.survophthal.2008.08.006.
5. Ramulu PY, van Landingham SW, Massof RW, Chan ES, Ferrucci L, Friedman DS. Fear of falling and visual field loss from glaucoma. Ophthalmology 2012;119:1352–8. http://dx.doi.org/10.1016/j.ophtha.2012.01.037.
6. Agorastos A, Skevas C, Matthaei M, et al. Depression, anxiety, and disturbed sleep in glaucoma. J Neuropsychiatry Clin Neurosci 2013;25:205–13. http://dx.doi.org/10.1176/appi.neuropsych.12020030.
7. Goldberg I, Clement CI, Chiang TH, et al. Assessing quality of life in patients with glaucoma using the Glaucoma Quality of Life-15 (GQL-15) questionnaire. J Glaucoma 2009;18:6–12. http://dx.doi.org/10.1097/IJG.0b013e3181752c83.
8. Owsley C, Rhodes LA, McGwin G Jr, et al. Eye Care Quality and Accessibility Improvement in the Community (EQUALITY) for adults at risk for glaucoma: study rationale and design. Int J Equity Health 2015;14:135. http://dx.doi.org/10.1186/s12939-015-0213-8.

Erratum

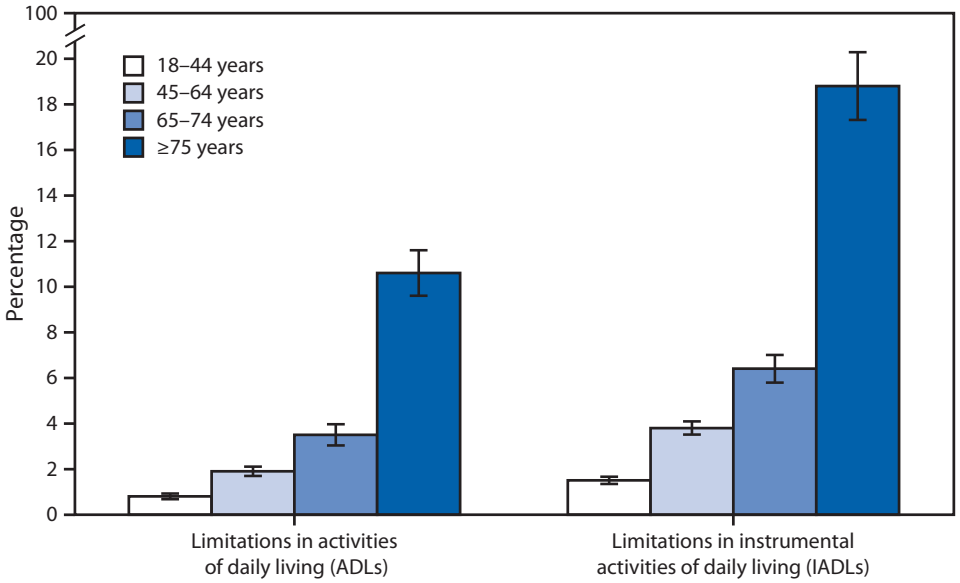
Vol. 64, Nos. 50 & 51

On page 1388, in the QuickStats "Birth Rates Among Females Aged 15–19 Years, by Race/Ethnicity* — National Vital Statistics System, United States,† 1991 and 2014," the x-axis label on the figure should have read "**No. of live births per 1,000 females aged 15–19 yrs.**"

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage of Adults with Activity Limitations, by Age Group and Type of Limitation* — National Health Interview Survey,† United States, 2014



* Percentages shown with 95% confidence intervals. Limitations in ADLs are based on response to the question, "Because of a physical, mental, or emotional problem, does [person] need the help of other persons with personal care needs, such as eating, bathing, dressing, or getting around inside this home?" Respondents were asked to answer regarding themselves and other family members living in the same household. Limitations in IADLs are based on response to the question, "Because of a physical, mental, or emotional problem, does [person] need the help of other persons in handling routine needs, such as everyday household chores, doing necessary business, shopping, or getting around for other purposes?" Respondents were asked to answer regarding themselves and other family members living in the same household.

† Estimates are based on household interviews of a sample of the civilian noninstitutionalized U.S. population and are derived from the National Health Interview Survey Family Core component. Unknowns were excluded from the denominators when calculating percentages.

In 2014, the percentages of adults aged ≥18 years with limitations in activities of daily living (ADLs) and limitations in instrumental activities of daily living (IADLs) increased with age. Adults aged ≥75 years were most likely to require the help of another person when performing ADLs (10.6%) and IADLs (18.8%).

Source: National Health Interview Survey, 2014 data (http://www.cdc.gov/nchs/nhis.htm).

Reported by: Patricia F. Adams; Michael E. Martinez, MPH, MHSA, bmd7@cdc.gov, 301-458-4758.

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