

approaches to refine medical and other interventions for many of these disorders (8).

The findings in this report are subject to at least three limitations. First, for the majority of the disorders screened, the cumulative incidence was derived from screening results for the approximately 2 million live births that occurred in the four states during 2001–2006. Although this number of births is sufficient to provide accurate estimates for many of the disorders (as evidenced by the relatively narrow 95% confidence intervals), the results observed among the four states might not be representative of the entire U.S. population (9). The analysis did account for some of this variability, particularly for hemoglobinopathies (which vary substantially by race and ethnicity) by using race- and ethnicity-specific rates to calculate the expected number of births. However, this approach was not possible as a general strategy because of lack of sufficient numbers of cases by race and ethnicity and lack of race- and ethnicity-specific information for Massachusetts. Second, an assessment of the accuracy of the rates for the rare disorders will not be possible until additional, population-based newborn screening data become available. Nevertheless, even if the estimated rates for the rare disorders were inaccurate by a factor of twofold or threefold, they would have only a modest impact on the estimated number of children with disorders identified using the expanded newborn screening panel. Finally, this analysis was not able to account for variations in the screening and diagnostic protocols among states that might have affected state-specific prevalences and estimates of the total number of cases.

Newborn screening continues to be a critical public health program that ensures better health and developmental outcomes for newborns at high risk. The recent recommendation to expand newborn screening programs presents challenges in terms of 1) ensuring screening and follow-up for the many rare disorders and 2) facilitating the clinical care and management of complex and more common disorders (e.g., cystic fibrosis and hemoglobinopathies), which require different types of specialists and life-long clinical management.

Acknowledgment

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Thallium Poisoning from Eating Contaminated Cake – Iraq, 2008

Thallium is an odorless, tasteless, heavy metal formerly used in rodenticides and still used in some manufacturing processes (e.g., electronics, pharmaceuticals, and glass). Thallium also has been used for intentional poisonings (1). Acute thallium poisoning produces gastrointestinal symptoms and signs, such as vomiting and acute abdominal pain, in the first few hours after ingestion, and initially is indistinguishable from other causes of acute gastrointestinal toxicity. However, within several days of ingestion, acute thallium poisoning often produces neurologic symptoms, such as extreme pain and acute muscle weakness ascending from the lower extremities, consistent with heavy metal toxicity (2). On January 22, 2008, 10 of 12 members in two families in Baghdad, Iraq, developed gastrointestinal symptoms; four of those 10 persons subsequently died from acute thallium poisoning, and five developed neurologic symptoms but survived. The Jordan Field Epidemiology Training Program* investigated this cluster at the request of the World Health Organization (WHO) representative in Iraq. The preliminary investigation indicated this was an intentional poisoning, and law enforcement officials began a criminal investigation. Physicians who see the sudden onset of painful peripheral neuropathy and hair loss in patients should consider the possibility of thallium poisoning.

On January 22, 2008, 10 members of two families sought treatment at a health-care facility in Baghdad. All 10 of the ill patients were experiencing abdominal pain, vomiting, and dysphagia. Over the next 4 days, five of the patients developed

*The Jordan Field Epidemiology Training Program was begun in 1998 with funding by the U.S. Agency for International Development and support from CDC. It became independent in 2008 and operates as part of Jordan's Ministry of Health.

neurologic symptoms and signs of varying severity (i.e., pain, abnormal sensations, and weakness, especially in the lower limbs). On January 26, the treating physician submitted specimens from the patients and a sample of a cake, which all 10 had eaten, to the poison testing laboratory of the Iraq Ministry of Health in Baghdad. On January 27, the WHO representative in Iraq was notified that the laboratory had detected thallium qualitatively in patient specimens and the cake. One of the patients, a child aged 11 years, died on January 30.

On February 1, the nine surviving patients were evacuated to Amman, Jordan, to receive Prussian blue (ferric hexacyanoferrate) as an antidote for thallium poisoning, which was not available in Iraq. A second child, aged 2 years, died soon after arrival in Jordan, before therapy could begin. Prussian blue therapy was begun in the eight surviving patients 11 days after they had eaten the contaminated cake; however, two of the eight patients were already in coma with severe cerebral edema and subsequently died. Over the next 30 days, all six long-term survivors developed hair loss, and five of the six survivors developed muscle weakness and spasticity of the lower limbs, with differing severity.

An epidemiologic investigation was initiated on February 5, 2008. Investigators learned that the fathers of the two families (family A and family B) were board members of an Iraqi sporting club. The board held a routine meeting in the club's conference room in Baghdad at midday on January 21. The cake, divided into 10 pieces, was prepared by a local bakery and delivered to the board meeting as a gift from a former board member. However, the cake arrived late, after most board members had departed. The board members who remained (the fathers of two families) divided the cake and took the halves home to their families. No cake was eaten at the board meeting; the cake was eaten at both families' homes after the evening meal on January 21.

Family A was composed of seven members (father, mother, and five children); family B was composed of five members

(father, mother, uncle, and two children). Ten cases of abdominal pain, vomiting, and dysphagia were identified among family members who consumed any portion of the cake on January 21. No other board members or their families reported illness, and no similar illnesses were found at the health facility in Baghdad or at nearby health facilities.

The overall attack rate was 83% (10 of 12 persons): six of seven persons in family A and four of five persons in family B. Four patients died (case-fatality rate = 40%; family-specific fatality rates = 33% [two of six] for family A and 50% [two of four] for family B) (Table). Food exposure histories were collected in Jordan through interviews with family members. Ten persons who ate portions of the cake on January 21 became ill; neither of the two persons who did not eat cake became ill (relative risk = undefined, $p=0.02$, Fisher exact test). However, one of the two had tasted the cake icing and tested positive for thallium in blood and urine specimens. Six (60%) of the ill patients were male; four (40%) were female. The median age of the patients was 12.5 years (range: 2–40 years). The median onset of illness was 24 hours after exposure (range: 12–72 hours) (Figure). An inverse relation was suggested between the amount of cake eaten and time to onset of symptoms. More rapid onset of illness occurred in persons who ate the most cake, and in adults. Of five patients who ate at least one piece[†] of cake, onset of illness was a median of 16 hours after exposure; of five patients who ate half a piece of cake or less, median onset of illness was 48 hours after exposure ($r = -0.56$, $p=0.09$, Pearson product-moment). Among the four patients aged ≥ 19 years, median onset of illness was 14 hours; among the six patients aged ≤ 14 years, median onset was 24 hours ($r = -0.58$, $p=0.08$, Pearson product-moment). Fatality was not significantly associated with sex, age, the amount of cake eaten, or the time to illness onset.

[†] "Piece" was not further defined; quantities were determined by subjective responses from family members.

TABLE. Number of persons who ate thallium-contaminated cake, became ill, and died, by amount of cake eaten* — Baghdad, Iraq, 2008

| Amount of cake eaten | No. exposed | Became ill | | Died | | Median incubation time (hrs) [†] | Median blood thallium ($\mu\text{g/L}$) [§] | Median 24-hr urine thallium ($\mu\text{g/L}$) |
|----------------------|-------------|------------|-------------|----------|-------------|---|--|---|
| | | No. | (%) | No. | (%) | | | |
| None | 1 | 0 | (0) | 0 | (0) | NA [¶] | NA | NA |
| Taste of icing | 1 | 0 | (0) | 0 | (0) | NA | 58 | 625 |
| 1/4 piece | 2 | 2 | (100) | 1 | (50) | 36 | 53 | 542 |
| 1/2 piece | 3 | 3 | (100) | 1 | (33) | 72 | 289 | 4,624 |
| 1 piece | 3 | 3 | (100) | 1 | (33) | 24 | 407 | 2,550 |
| 1 1/2 pieces | 2 | 2 | (100) | 1 | (50) | 14 | 808 | 7,549 |
| Total | 12 | 10 | (83) | 4 | (40) | 24 | 289 | 3,063 |

* Quantities were determined by subjective responses from family members; "piece" was not further defined.

[†] Correlation with amount of cake eaten, $r = -0.56$, $p=0.09$, Pearson product-moment.

[§] Correlation with amount of cake eaten, $r = 0.66$, $p=0.06$, Pearson product-moment.

[¶] Not applicable.

By 30 days after ingestion, eight (80%) patients had experienced hair loss, which had begun within 7 days after eating the cake, and five (50%) still had neurologic deficits (e.g., lower limb muscle weakness and spasticity, with differing severity). Quantitative thallium levels were determined from blood and urine specimens of nine patients on the 16th day after eating any portion of the cake. Thallium was detected in all nine patients; median blood thallium level was 289 $\mu\text{g/L}$ (range: 53–1,408 $\mu\text{g/L}$; reference level expected: <2 $\mu\text{g/L}$), and median calculated 24-hour urine excretion of thallium was 3,063 $\mu\text{g/L}$ (range: 542–12,556 $\mu\text{g/L}$; reference level expected: <5 $\mu\text{g/L}$) (3). Blood thallium levels were weakly correlated with the amount of cake reported eaten ($r = 0.66$, $p = 0.06$, Pearson product-moment). The father of family A, who did not become ill, but had tasted icing from the cake, had elevated blood and urine thallium levels.

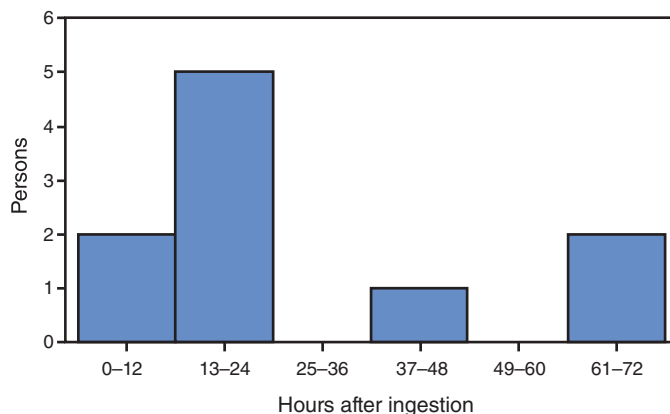
Reported by: Z Al-Masbhadani, A Al-Fatlawy, K Abu Nawas, Jordan Field Epidemiology Training Program; M Al-Nsour, B Hijawi, A Belbeisi, Ministry of Health, Hashemite Kingdom of Jordan; R Sharqawi, I Juma', S Hamaid, E Al-Saqa, F Al-Amouri, S Hameedi, S Sbeitan, L Mohammed, Jordan Specialty Hospital; M Jaghbeer, Univ of Jordan. N Al-Gasseer, O Mekki, B Ghanem, Office of WHO Representative in Iraq; A Adel Mohsin, Inspector General in Iraq; H Badar Musa, A Saloom, A Al-Alai, Baghdad Medical City. S Thomas, A Vale, T Sheehan, S Bradberry, UK National Poisons Information Svc. R Gerber, Div of Global Public Health Capacity Development, Coordinating Office for Global Health, CDC.

Editorial Note: When ingested, thallium is a systemic poison that can produce multiple organ toxicity involving the gastrointestinal, neurologic, and cardiovascular systems (2). Among the distinctive effects of thallium poisoning are hair loss and painful, usually ascending, peripheral neuropathy (e.g., extreme pain, paresthesia, and weakness in distal extremities). In 1973, WHO recommended that thallium sulfate use as a rodenticide be discontinued because of its toxicity (4), and use in the United States for this purpose has been banned since 1975 (5). Approximately 60%–70% of thallium production is used in the electronics industry, with the remainder being used in manufacturing pharmaceuticals and glass.

Prussian blue, a pigment discovered in the 1700s, acts as a sequestering agent for certain heavy metal ions and as an antidote to thallium poisoning. In 2003, the U.S. Food and Drug Administration approved the use of Prussian blue in 500 mg capsules as safe and effective for treatment of known or suspected internal contamination with thallium (radioactive or nonradioactive) or radioactive cesium.

Deliberate contamination of food during production and preparation is rare (6,7), but instances of intentional thallium poisoning have been reported (1). This report describes one of the largest known clusters of thallium poisoning (8–10). Initial clinical findings in this report (i.e., gastrointestinal [100%] and

FIGURE. Hours to onset of symptoms among 10 persons who ate thallium-contaminated cake — Baghdad, Iraq, 2008



neurologic symptoms [50%]) are similar to findings reported from previous clusters (gastrointestinal symptoms ranged from 11%–100% in previous clusters and neurologic symptoms ranged from 50%–100%). Although, the attack rate in this report (83%) is similar to those of previous clusters (71%–100%), the case-fatality rate in this report (40%) is higher than in previous clusters (0%–20%). Differences in clinical findings and case-fatality rates might be related to dosing and timing of ingestion, vehicles used (e.g., soft drinks, marzipan candy, and coffee), or formulation of the poisons (one cluster included both arsenic and thallium). The progression of signs and symptoms in this report are similar to those of previous clusters.

Multiple government agencies and private sector health-care providers assisted and worked with each other, within and between countries, during the response to this incident. Such coordination and cooperation is critical for immediate, effective response to such events, whether they arise from unintentional or intentional circumstances. The sudden appearance of the characteristic signs and symptoms of hair loss and painful peripheral neuropathy in patients should prompt clinical consideration of thallium poisoning. Because of historical precedents, investigation should include assessment for criminal intent.

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Notice to Readers

National Child Passenger Safety Week – September 21–27, 2008

In 2006, in the United States, 462 children aged ≤ 4 years died and approximately 45,000 were treated in emergency departments because of injuries sustained in motor-vehicle crashes (1,2). This year, National Child Passenger Safety Week, September 21–27, 2008, will focus on the importance of the correct installation and use of child safety seats.

The use of child safety seats has been found to reduce the risk for death in a crash by 71% for infants and by 54% for toddlers (i.e., children aged 1–4 years) (3). Child safety seat use is mandatory in every state in the United States and in the District of Columbia, although the age at which children can transition to adult safety belts varies by state.

In the first national probability sample of correct child safety seat use, the National Highway Traffic Safety Administration (NHTSA) reported in 2006 that 28% of infants aged < 1 year were not placed in rear-facing seats, and 44% of children who weighed 20–40 pounds were not in forward-facing child seats, as recommended by NHTSA (4). An estimated 73% of child safety seats are incorrectly installed or misused (5). The most common errors are loose harness straps and loose or improper attachment of the child safety seat to the vehicle using the seat-belt or LATCH (i.e., lower anchors and tethers for children) system (5,6). Incorrect installation or use reduces child safety seat effectiveness.

Information about National Child Passenger Safety Week activities and child passenger safety is available from NHTSA at <http://www.nhtsa.dot.gov> and from CDC at <http://www.cdc.gov/ncipc/factsheets/childpas.htm>.

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Errata: Vol. 57, No. 33

In Vol. 57, No. 33 (August 22, 2008), in “Final 2007 Reports of Nationally Notifiable Infectious Diseases,” errors occurred in Table 2, “Reported cases of notifiable diseases, by geographic division and area — United States, 2007.” On page 908, under “Lyme disease,” the number of cases for the following areas and states should read, **United States, 27,444; New England, 7,786; New Hampshire, 896; E.N. Central, 2,102; Michigan, 51; Wisconsin, 1,814; W.N. Central, 1,398; Iowa, 123; Kansas, 8; Minnesota, 1,238; Nebraska, 7; E.S. Central, 51; Alabama, 13; Mississippi, 1; Tennessee, 31; W.S. Central, 91; Arkansas, 1; Mountain, 45; Arizona, 2; Idaho, 9; Montana, 4; Wyoming, 3; Pacific, 103; Oregon, 6.**

Errata: Vol. 55, No. 53

In Vol. 55, No. 53 (March 21, 2008, for 2006), “Summary of Notifiable Diseases —United States, 2006,” an error occurred in Table 8, “Reported cases of notifiable diseases — United States, 1999–2006.” On page 76, under “Botulism, total (including wound and unspecified),” the total for 2006 should read **165**.



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Federal Air Travel Restrictions for Public Health Purposes — United States, June 2007–May 2008

Persons with communicable diseases who travel on commercial aircraft can pose a risk for infection to the traveling public (1,2). In June 2007, federal agencies developed a public health Do Not Board (DNB) list, enabling domestic and international public health officials to request that persons with communicable diseases who meet specific criteria and pose a serious threat to the public be restricted from boarding commercial aircraft departing from or arriving in the United States. The public health DNB list is managed by CDC and the U.S. Department of Homeland Security (DHS). To describe the experience with the public health DNB list since its inception, CDC analyzed data from June 2007 to May 2008. This report summarizes the results of that analysis, which indicated that CDC received requests for inclusion of 42 persons on the public health DNB list, all with suspected or confirmed pulmonary tuberculosis (TB). From the requests, 33 (79%) persons were included on the list. The public health DNB list enables public health officials to prevent travel on commercial aircraft by persons who pose a risk for infection to other travelers. State and local health departments in the United States and other countries should be aware of this new public health tool.

The public health DNB list is intended to supplement local public health measures when they are deemed insufficient to prevent persons who are contagious from boarding commercial aircraft. Use of the list is limited to diseases that would pose a serious health threat to fellow air travelers. The list is authorized under the Aviation and Transportation Security Act of 2001* and is managed jointly by DHS and CDC; however, DHS defers to CDC regarding public health decisions and actions.

To request that a person be placed on the public health DNB list, state or local public health officials contact the

CDC Quarantine Station for their region†; health-care providers make requests by contacting their state or local public health departments, and foreign and U.S. government agencies contact the Director's Emergency Operations Center (DEOC) at CDC in Atlanta. To include someone on the list, CDC must determine that the person 1) likely is contagious with a communicable disease that would constitute a serious public health threat should the person be permitted to board a flight; 2) is unaware of or likely to be nonadherent with public health recommendations, including treatment; and 3) likely will attempt to board a commercial aircraft. Once a person is placed on the list, airlines are instructed not to issue a boarding pass to the person for any commercial domestic flight or for any commercial international flight arriving in or departing from the United States. The public health DNB list does not apply to other means of transportation (e.g., buses or trains). Governments of foreign countries are notified when their citizens or persons residing in their countries are placed on the list.

Multiple criteria are used to decide whether a person with a communicable disease constituting a serious public health threat should be placed on the public health DNB list and when a person can be removed. For persons with suspected

† Available at http://www.cdc.gov/ncidod/dq/resources/quarantine_station_contact_list.pdf.

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*49 USC § 114 (f) and (h).