目　次

＊　この米国食品事故対処事例集は、米国疾病管理予防センター（CDC）発行の「疫学週報（MMWR)」の中から、1997年以降発生した食品を媒介とした疾病の発生事例及び食品が原因と判明した事故事例について、事故の経緯及び行政（連邦政府、州政府、地方（市・郡）政府）の対応を中心に抄訳（仮訳）したものである。

また、本対処事例集の作成にあたっては、(財）国際医学情報センター（IMIC）のホームページ(http://www.imic.or.jp/)上で情報提供(毎週更新)されている「疫学週報 MMWR 抄訳版インデックス」(http://www.imic.or.jp/mmwr/sokuho.htm）に掲載された抄訳（仮訳）をIMICの承諾を得た上で引用・転載し、これに事故の経緯及び行政の対応等の情報を必要に応じて追加している。

なお、これらの事故事例のさらに詳しい内容については、抄訳の後に記載されているMMWRの原文もしくはCDCのMMWRのホームページ（http://www.cdc.gov/mmwr/）を参照下さい。

MMWR51 (31):684-686

タマリンドキャンディと民間療法薬による小児鉛中毒
カリフォルニア州，1999～2000 年

カリフォルニア州において、ルーチンスクリーニングで確認された非典型的な原因による鉛中毒小児 5 例（全例ラテンアメリカ系）を紹介する。

症例 1、2：1999 年 3 月に、ヒスパニック系の 4 歳男児とその 6 歳の姉が、カリフォルニア州の小児の衛生及び障害保護プログラム（California’s Child Health and Disability Prevention：CHDP）による定期検査で鉛中毒が特定された。男児の BLL（血中鉛濃度）は 88.0 g/dL、姉の BLL は 69.0 µg/dL であり、どちらもキレート療法を受けた。子供達の両親はメキシコの民間療法薬で鉛含有量の多い greta を使用していた。家の中にある陶器、塗料、埃からは高レベルの鉛は検出されなかったが、ファミリールームから集められた greta の粉末には 770,000 ppm の鉛が含まれ、家のミニブラインドの掃除用モップは鉛陽性であった。鉛に汚染されていることが後に確認された Dulmex ブランドの Bolirindo 棒付きキャンディを含む輸入品のキャンディが家の中で見つかった。

症例 3：2000 年 5 月に確認された 4 歳男児（CHDP により特定、BLL は 26 µg/dL）。家内から集められた輸入キャンディの包み紙から高レベルの鉛（16,000 ppm）が検出された。

症例 4：2000 年 6 月に確認された 2 歳男児（BLL は 26 µg/dL）。男児は、民間療法薬 greta と azarcon を与えられており、両親がメキシコで購入した様々なタマリンドフルーツキャンディをなめていた。これらの輸入キャンディのうち、Dulmex ブランドの Bolirindo 棒付きキャンディの棒と包み紙から高レベルの鉛（それぞれ 404 ppm、21,000 ppm）が検出された。米国食品医薬品局（FDA）によるその後のテストで、これらの製品の包み紙から高レベルの鉛が確認されたため、FDA とカリフォルニア州衛生局（CDHS）によって公衆衛生警告が発令された。

症例 5：2000 年 8 月に確認された 4 歳男児（カリフォルニア州のメディケイドプログラムにより特定、BLL は 22 µg/dL）。この男児の場合、メキシコ製キャンディをなめていたこと以外の鉛中毒の原因は見当たらなかった。

Editorial Note《編集注記》

これらの報告では、BLL のルーチンスクリーニングと、BLL が上昇している小児に対する経歴の聴取及び家の塗料や埃といった環境因子のサンプリングを含む徹底的なリスク評価を実施することの重要性を強調している。健康管理者は、キャンディを含む一部の食品の危険性を認識すべきであり、ラテンアメリカ系住民に対し一部のメキシコ製キャンディや民間療法薬が小児の鉛中毒の原因となる可能性があることを教育する必要がある。

なお、CDHS は他のタマリンドキャンディの鉛含有も特定している。さらに、FDA はタマリンドフルーツを含む食品の米国への輸入を禁止している。

August 9, 2002 / 51(31); 684-686

Childhood Lead Poisoning Associated with Tamarind Candy and Folk Remedies ---California, 1999--2000

Lead poisoning affects children adversely worldwide. In the United States, elevated blood lead levels (BLLs) (≥10 μg/dL) result primarily from exposure to lead-based paint or from associated lead-contaminated dust and soil; however, other sources of lead exposure, including folk remedies, Mexican terra cotta pottery, and certain imported candies, also have been associated with elevated BLLs in children (*1*). This report describes five cases in California of lead poisoning from atypical sources. Health-care providers should be aware of the potential hazards of certain food products, and community members should be educated about potential sources of lead poisoning for children.

Case Reports

Cases 1 and 2. In March 1999, two Hispanic children residing in Stanislaus County in the Central Valley, a boy aged 4 years and his sister aged 6 years, were identified during routine screening by California's Child Health and Disability Prevention (CHDP) Program. The boy had a BLL of 88.0 g/dL and the girl a BLL of 69.0 μg/dL. Both children underwent chelation therapy. Their parents had not traveled recently outside the United States but had used greta, a Mexican folk remedy (taken commonly for stomachache or intestinal illness) that usually contains high levels of lead. No pottery in the home tested positive for lead, and tests on paint and dust from their home did not indicate high lead levels. Greta powder collected from the family's home had 770,000 parts per million (ppm) of lead, and miniblinds on the windows of the home tested positive for lead by swab. Imported candies, including Dulmex-brand Bolirindo lollipops, which were identified later to be contaminated with lead, were found in the home.

Case 3. In May 2000, a Hispanic boy aged 4 years residing in Fresno County was identified during routine CHDP screening with a BLL of 26 μg/dL. His family had

moved to California recently from Oaxaca, Mexico, where they had used a ceramic bean pot and water jug regularly. An environmental investigation did not reveal high lead levels in dust, paint, or soil, but tests on imported candies collected from the home revealed a candy wrapper with a lead level of 16,000 ppm. The child's BLL had decreased to 13.2 μg/dL by February 2002.

Case 4. In June 2000, a Hispanic boy aged 2 years residing in Orange County was identified through routine screening as having a BLL of 26 μg/dL. The family's house was built in 1963 and had been renovated during early 2000. Tests on soil, paint, and dust in and around the child's home did not reveal high lead levels. The child had been given greta and azarcon (a folk remedy that usually contains substantial amounts of lead) and had eaten various imported tamarind fruit candies purchased routinely by his family in Mexico. High lead levels were found in one of the three brands of imported candies the child had eaten. A Dulmex–brand Bolirindo lollipop had levels of 404 ppm and 21,000 ppm of lead in the stick and wrapper, respectively, and 0.2 ppm and 0.3 ppm in the candy and seed, respectively. Subsequent tests by the Food and Drug Administration (FDA) confirmed high lead levels in the wrapper of this product, and a public health warning was issued by FDA and the California Department of Health Services (CDHS).

Case 5. In August 2000, a Hispanic boy aged 4 years residing in Los Angeles County was identified through routine screening by California's Medicaid program with a BLL of 22 μg/dL. When the child was tested at age 1 year, he had an acceptable BLL of 5 μg/dL. Family members reported that he had been eating Mexican candies regularly for 3 years but denied use of folk remedies and imported pottery. An environmental investigation of their apartment, which was built in 1986, did not reveal high lead levels. The child was born in the United States and had not traveled to Mexico, and investigators identified no other potential sources of lead other than the Mexican candies. The family was advised not to allow the child to eat Mexican candies. As of December 2001, the boy's BLL had decreased to 11 μg/dL.

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Editorial Note:

The findings in this report underscore the importance of routine screening for lead and of conducting a thorough risk assessment of children with elevated BLLs including taking a complete history and environmental sampling. Although household paint and resulting contaminated dust and soil are the most common sources of exposure, all sources of lead poisoning should be identified and removed.

Of approximately 1,000 cases of elevated BLLs among California children that were reported to CDHS during May 2001--January 2002, candy produced in Mexico was identified as a possible exposure source in approximately 150 cases. When children eat lead-contaminated candies, exposure can exceed FDA's provisional tolerable daily intake level (PTIL) for lead of 6 μg in a typical 30-g food serving. FDA's PTIL corresponds to a lead intake capable of elevating the BLLs of a small child by 1 μg/dL. In the cases described in this report, the wrappers often contained amounts of lead that could greatly exceed FDA's PTIL if the lead were to leach into the candy. In addition, a substantial quantity of the lead could be released into saliva by a child licking the wrapper. When conducting investigations of lead exposures, clinicians and health educators are encouraged to consider inquiring about these products, together with folk remedies and the use of imported pottery, as potential sources of lead poisoning.

Lead poisoning associated with tamarind candy has been reported previously (*2--5*). Although the lead content of the particular candies that the five children described in this report ate could not be measured because the candy had been eaten, substantial concentrations of lead were found in the wrappers in four cases. Because the candies are sticky and can adhere to the wrapper, the children might have ingested lead from the wrapper; in addition, other sources of lead exposure (e.g., greta consumption) were found. In the cases described in this report, the frequency of eating Mexican candies and the brands eaten were not always ascertained. An investigation is ongoing to determine which specific candy products are contaminated with lead. CDHS has identified lead in several other tamarind candies. In addition, FDA has embargoed food products containing tamarind fruit from entry into the United States because of filth from insects, rodents, and other pests.

These cases illustrate successful cooperation between FDA and state and local health departments to identify lead-contaminated products. Health-care providers

should be aware of the potential hazards of food products, including candy, when evaluating a child with an elevated BLL. In addition, increasing education efforts are needed to inform persons in Hispanic communities that certain Mexican candies, pottery, and folk remedies can be potential sources of lead poisoning for children (*6*). Additional information about childhood lead poisoning is available from CDHS at http://www.dhs.ca.gov/ps/deodc/childlead and from CDC at http://www.cdc.gov/nceh/lead/lead.htm.

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MMWR47 (48):1041-1043, 1998

輸入キャンディ及び食品着色料に関連して発生した鉛中毒事例 カリフォルニア州及びミシガン州

小児における鉛中毒の多くは、家庭の壁や窓の下枠等に用いられた鉛素材の塗料が劣化して発生した埃に起因することが多いが、それ以外の要因であっても過度の鉛暴露があれば中毒が発生する可能性がある（BLL（血中鉛濃度）≧10 μg/dL）。

本報告では、カリフォルニア及びミシガン両州でそれぞれ発生した、海外から持ち込まれた食品あるいは食品着色料を原因とする2例の中毒例につき紹介した。

例1:1993年に発生したカリフォルニア州在住の6才男児の中毒事例(BLLは59 μg/dL)で、その後の調査の積み重ねにより、1997年にその原因がメキシコで製造されたタマリンド（マメ科の植物）のキャンディジャム（1993年以降、米国内への輸入が禁止されている）が原因と判明した事例である(カリフォルニア州衛生局は1998年4月3日に消費者に対し、これらの製品を摂取しないようにという警告を発令し、さらに米国食品医薬品局（FDA）はこれらの製品が将来、米国に輸入されることがないよう行政処置を行った）。

例2：1997年ミシガン州在住の3才の子供の中毒事例（BLLは27 μg/dL）で、その後の調査の結果、1998年4月にイラク産のスパイス（lozeena：オレンジ色粉末で、米や肉の着色に使用、鉛含有量7.8～8.9%）が原因と判明した事例である（税関職員は旅行者によってlozeenaがイラクから米国に持ち込まれた可能性について報告を受けた。また、オークランド郡衛生局は、当該地域の212名に対し、鉛中毒に関する検査を実施したが、高いBLLは確認されなかった）。

Editorial Note《編集注記》

高いBLLが認められたため、カリフォルニア州は1993年にタマリンドキャンディ製品を禁輸したが、当該製品は未だカリフォルニアのエスニック・マーケット、スワップ・ミーツ（不良品交換会）、旅の行商人により販売されている。また、当該製品はメキシコ旅行者によって少量ではあるが、頻繁に国内に持ち込まれる。FDAは6歳未満の小児につい

て、一日の鉛摂取許容量を 6 μg とし、鉛曝露による神経及び行動への微少な悪影響を回避するよう提唱している。

December 11, 1998 / 47(48); 1041-1043

Lead Poisoning Associated with Imported Candy and Powdered Food Coloring -- California and Michigan

Although the most common source of pediatric lead poisoning is dust within the home that contains deteriorated lead-based paint from walls and windowsills, other less common sources (1-3) can result in excess exposure among children (i.e., blood lead levels {BLLs} greater than or equal to 10 ug/dL). This report describes two cases of pediatric lead poisoning associated with eating imported candy and food stuffs and underscores the importance of thorough history-taking to identify unusual sources of lead exposure. Case 1

In 1993, a 6-year-old boy in California was identified by routine screening during a well-child examination as having a BLL of 59 ug/dL. During 1993-1997, he underwent chelation therapy seven times to reduce his BLL. His five siblings, ranging in age from 11 to 17 years, also were tested within 9 months of their brother and had BLLs of 35-46 ug/dL; the mother had a BLL of 26 ug/dL. In 1995, two cousins, aged 3 and 7 years, were identified with BLLs of 50 ug/dL and 57 ug/dL, respectively. In addition, a ninth child (a niece of the index case patient) was born in 1996 and had a BLL of 26 ug/dL at age 1 year.

No potential source of exposure was identified for the children and mother. However, on review of serial BLLs, elevations coincided with the return of the maternal aunt from visits to Mexico.

In 1997, repeated questioning of family members revealed that the aunt had transported in her personal baggage tamarindo candy jam products, produced in Mexico and restricted from importation into the United States since 1993, and had given it to the children. Although the family had been cautioned about the ingestion of ethnic remedies, they were unaware of the potential dangers of ingesting candy packaged in ceramic jars from Mexico.

No product was available from the family for analysis. The California Department of Health Services issued a health alert on April 3, 1998, warning consumers to avoid eating these products. In addition, the Food and Drug Administration (FDA) initiated administrative actions to prevent future importation of these products into the United States (4). Case 2

In May 1997, a 3-year-old boy in Michigan had a BLL of 27 ug/dL. His 2-year-old brother had a BLL of 36 ug/dL. Subsequently, their home was cleaned professionally with a trisodium phosphate solution and a high-efficiency particulate air (HEPA) filter vacuum; interior dust samples were found negative for lead. Despite extensive history-taking and several environmental investigations of both the home and the father's workplace, no source of lead was determined.

By January 1998, the two brothers and both parents had BLLs of 50 ug/dL to 60 ug/dL. The brothers' BLLs increased after chelation therapy. In April 1998, samples of household spices were analyzed; no significant lead levels were found in any spice except lozeena, a bright orange powder used by Iraqis to color rice and meat, which contained 7.8%-8.9% lead.

Nine of 18 extended family members subsequently tested had elevated BLLs ranging from 25 ug/dL to 84 ug/dL. Elevated BLLs were found only among maternal relatives who had eaten food prepared with a single supply of lozeena. The lozeena had been purchased in Iraq and brought into the United States by the maternal grandmother. The contaminated lozeena was removed from the affected households, and the family was encouraged to destroy any frozen foods made with this supply of lozeena.

Customs officials were notified about the possibility of travelers bringing contaminated lozeena into the United States from Iraq. Educational materials were translated into Arabic, and health alerts were sent to local physicians. The Oakland County Health Department screened 212 persons in the community for lead, and no other elevated BLLs were identified.

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Editorial Note:

Because lead poisoning in children can result from multiple sources, successful case management requires a systematic review of all potential sources of lead exposure. This review includes thorough history-taking and home inspection to prevent further lead exposure or clinical lead poisoning and to avoid increased lead absorption should chelation therapy be required.

When a child's BLLs are persistently elevated and case-management efforts fail to identify a source, screening other members of the index household for blood lead should be considered. Detecting excess lead exposure in more than one family member of the same household can be important to directing the investigation toward a shared source of exposure. Blood from other household contacts, extended family, or visitors that may regularly share this exposure source also should be screened for lead.

Several commercial retail lots of the tamarindo jellied fruit candy were embargoed by California in 1993 because of high lead levels in the product. The tamarindo products still are being sold in California through ethnic markets, swap meets, and itinerant vendors. Persons frequently bring these products into the United States in small quantities while traveling from Mexico. These products can be found under the brand names Margarita-brand Tamarindo Pulpa (with and without chili), Licona-imported Tamarindo, Picarindo-brand jellied tamarindo candy, and Jarrita Chonita-brand jellied tejocote candy with chili. All four fruit-derived products are packaged in stoneware or terra cotta ceramic jars. The lead-based glazing applied to the jars appears to be the major source of lead in these products. Improperly fired lead-glazed pottery is a well-known source of food adulteration (1, 3, 5). Candied jam in green jars had the highest lead levels. Both tamarindo and tejocote fruits are acidic, which increases lead leaching. However, some jams from plastic-lined jars contain substantial amounts of lead and may have been contaminated with lead from another source. Chili, an ingredient in some of these products, can be contaminated by lead through the practice of air-drying or fuel-assisted drying in Mexico, where leaded gasoline is used

as fuel (R. Jacobs, PhD, FDA, San Francisco District Office, personal communication, 1998).

FDA recommends a 6-ug per day tolerable limit for dietary intake of lead for children aged less than 6 years to prevent the more subtle adverse neurologic and behavioral effects of lead exposure (6). A typical serving of 60 g of the tejocote product could expose a child to 6.7-1956.0 ug of lead; the same serving of the tamarindo products would provide 11.4-36.0 ug of lead.

Spices occasionally have been implicated as lead sources in other countries (T. Venkatesh, St. Johns Medical College, Bangalore, India, personal communication, 1998). Lead is sometimes added to certain ethnic foods or food supplements to impart a yellow or orange color or a sweet taste or to increase weight (7).

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MMWR52 (33): 788-791

ヨルガオ中毒の疑い
オハイオ州，2002 年

2002 年 10 月 11 日～11 月 20 日にかけて、オハイオ州アクロン市及びクリーブランド市の青少年 14 名（年齢中央値 17(12～19)歳、男 12 女 2）が、チョウセンアサガオ（D. inoxia）中毒を発症した。

本レポートでは、「ヨルガオ（moonflowers）」として一般に知られている様々な植物が引き起こす事故事例について議論し、植物をレクリエーション的に使用する際の潜在的な毒性について啓蒙の必要性を強調するものである。

シンシナティ市の薬品・中毒情報センター（Drug and Poison Information Center: DPIC）が報告を受け、治療のアドバイスをした。全例共、種を食べたか、種を使ったお茶を飲んだ直後に発症し救急診療部（ED）を受診した。瞳孔散大、頻拍、幻覚及び尿貯留などの抗コリン作用性症状を呈し、24～48 時間持続した後、支持療法とベンゾジアゼピン投与により回復した。原因究明の結果、患者の両親より ED に提出された植物の写真及び臨床例からチョウセンアサガオ（D. inoxia）が原因であり、DPIC の毒物学者もこれに同意した。

また、ヨルガオ汚染またはヨルガオに関する報告については、DPIC のデータベース（DPIC の 2002 年の未発表データ）には、2000～2001 年の間にアクロン市及びクリーブランド市地域では発見されていない。チョウセンアサガオ（D. stamonium : D. inoxia に似ており一般に乱用されている）中毒については、同じ期間では実質的に増加していなかった。

Editorial Note《編集注記》

ヨルガオについては麻薬取締局（U.S. Drug Enforcement Agency's）のリストに載っていないが、アクロン市及びクリーブランド市地域では、地域法の強制措置として不正な使用のために（莢入りの）種子を販売することは禁止されている。

チョウセンアサガオは、スコポラミン、ヒヨスチアミンを主成分とし、抗コリン作用性毒性を示す。症状は、通常摂取から 60 分以内に発現し、24～48 時間持続する。症状とし

て中枢神経系では錯乱、不穏、不安、幻覚、痙攣、昏睡、末梢系では粘膜乾燥、口渇、顔面紅潮、かすみ目、高熱、尿貯留、腸運動の低下などが挙げられている。治療法として支持療法、腸内洗浄、ベンゾジアゼピン、フィゾスチグミンの投与などがある。

August 22, 2003 / 52(33); 788-791

Suspected Moonflower Intoxication --- Ohio, 2002

During October 11--November 20, 2002, the Cincinnati Drug and Poison Information Center (DPIC) received notification of and offered treatment advice for 14 adolescents in the Akron/Cleveland, Ohio, area who became ill after intentional exposure to toxic seeds that DPIC identified as *Datura inoxia* (Figure). All became ill shortly after eating the seeds or drinking tea brewed using the seeds. All patients recovered fully after treatment. This report summarizes these cases, discusses the characteristics of the various plants known commonly as "moonflowers," and underscores the need for awareness of the potential toxicity from recreational use of a plant.

Of the 14 patients, 12 (86%) were male; median age was 17 years (range: 12--19 years). All 14 patients reported to the emergency department (ED) with anticholinergic signs and symptoms, including dilated pupils, tachycardia, hallucinations, and urinary retention. Signs and symptoms typically lasted 24--48 hours, and the illness resolved with supportive care and benzodiazepine administration. No long-term effects were documented.

On November 5, a local newspaper described some of the cases of "toxic seed" exposure. Use of the common name moonflower had led to some confusion about which of the several moonflower plants were involved in these exposures. Parents of several adolescents who ingested these seeds as a group reported that the seeds were from a moonflower plant, specifically *D. inoxia*, and noted that this plant was cultivated widely and available in local garden stores. On the basis of clinical presentations and a photograph taken of a plant submitted to the ED by one of the parents, a toxicologist at DPIC agreed that *D. inoxia* was the source of these illnesses.

No reports of moonflower exposure or moonflower information calls in the Akron/Cleveland area during 2000--2001 were found in the DPIC database (DPIC, unpublished data, 2002). Calls about poisonings with *D. stramonium*, a commonly abused plant related to *D. inoxia*, did not increase substantially during the same period.

Reported by: *R Goetz, PharmD, E Siegel, PharmD, J Scaglione, PharmD, Cincinnati Drug and Poison Information Center, Ohio. M Belson, MD, M Patel, MD, Div of Environmental Hazards and Health Effects, National Center for Environmental Health, CDC.*

Editorial Note:

Moonflower is not on the U.S. Drug Enforcement Agency's list of controlled substances, but local law enforcement measures in the Akron/Cleveland area prohibit selling seedpods for illicit use. The cluster of moonflower exposures reported to DPIC might represent a new form of substance abuse in the Akron/Cleveland area. The illicit use of this plant might be related to the increasing knowledge of moonflower's hallucinogenic properties combined with the local availability of this plant.

Plants with large fragrant flowers that bloom at dusk are referred to as moonflowers. Poisindex® lists two species as moonflower: *Ipomoea muricata* (purple moonflower) and *I. alba* (white moonflower) (*1*). Ingestion of *I. muricata* might cause hallucinations and cholinergic effects such as diaphoresis, salivation, lacrimation, and diarrhea. Neither hallucinations nor other anticholinergic effects occur with *I. alba* poisoning (*1*).

The clinical features of cases reported to DPIC are most consistent with the anticholinergic properties of *Datura* species. Scopolamine and hyoscyamine, both of which are major constituents of *Datura* species, are most concentrated in the seeds and can cause anticholinergic poisoning in exposed persons.

Symptoms of *Datura* toxicity occur typically within 60 minutes after ingestion and continue for 24--48 hours. Ingestion of *Datura* manifests as a classic anticholinergic syndrome comprising central and peripheral signs and symptoms. Central toxic effects include confusion, agitation, anxiety, hallucinations, seizures, and coma. Peripheral toxic effects include dry mucous membranes, thirst, flushed face, blurred vision, hyperthermia, urinary retention, and decreased gut motility (*2*). Treatment consists of supportive care, gastrointestinal decontamination (e.g., activated charcoal), benzodiazepines as needed for agitation, and, in severe cases, physostigmine, the antidote for anticholinergic poisoning (*3*).

D. inoxia is a plant with large white flowers that blooms at dusk; it has a bushy growth habit with up to 200 seeds borne in pods with closely spaced thorns (*4*). *D. inoxia* is

related to another commonly abused plant, *D. stramonium* (jimson weed) (*5--7*). *D. stramonium* has clinical features of toxicity similar to *D. inoxia* (*8--10*). The plant features described by the parents of the exposed adolescents are consistent with *D. inoxia* but not *D. stramonium* or the other moonflower plants.

This report highlights four important points. First, the clinical effects of recreational use of a plant might vary drastically from the desired effects. Adolescents and parents should be aware of the potential toxicity from recreational use of a plant and the need for medical attention if an exposure occurs. Second, gardening practices in a community might provide novel opportunities for experimenting with intoxicating substances. Because *D. inoxia* is used as an ornamental plant in the Akron/Cleveland area, local garden suppliers should discuss the potential toxicity of the plant at the time of purchase. Third, because toxicity differs for various plants of this type, use of the common name moonflower can be misleading clinically and might complicate identification of some species. Finally, poison-control centers can detect new trends in drug abuse or poisonings and provide information that local and state health departments can use to inform the public. In Ohio, an early-warning network is designed to release timely alerts to inform schools, health-care providers, and the public statewide about emerging drug-abuse trends and poisonings (*10*).

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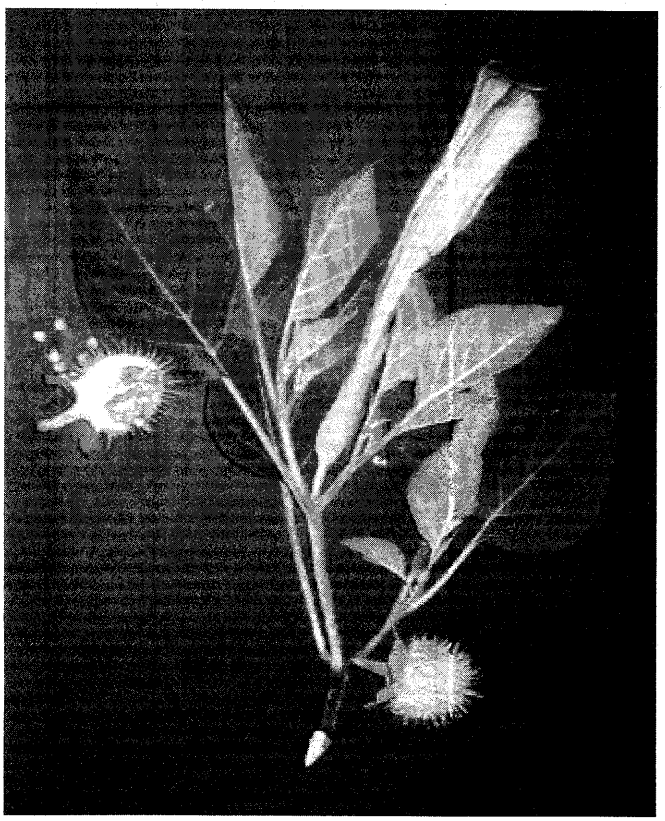
FIGURE. *Datura inoxia*, one of several plants known commonly as "moonflowers"



Photo/R Goetz, Cincinnati Drug and Poison Information Center

MMWR51 (47):1065- 1067

カワカワ含有製品による肝毒性の可能性
米国、ドイツ、スイス，1999～2002 年

1999 年以来、ドイツ、スイス及び米国においてカワカワ（kava kava または Piper methysticum）を含む栄養補助食品が関与していると考えられる重症の肝毒性が報告され、これら 5 症例の報告を受けて米国食品医薬品局（FDA）は 2002 年 3 月 25 日に消費者に対する注意書きを発表し、米国で起こった類似症例に関する調査を完了した。FDA は消費者及び健康管理の提供者に対し、カワカワが含まれている製品の使用に関するリスクについての通告を継続する。

例 1：米国では、2001 年 5 月、45 歳の健康な女性がカワカワ含有栄養補助食品の服用を始めてから約 8 週後に悪心と脱力感を訴えた。パッケージにはカワカワエキス（根）、標準 30%カバラクトン（75mg）、ホップ（球果）、ジャーマンカモミール（頭状花）、トケイソウ（花及び果実）、ゼラチン、自然野菜繊維を成分とし、服用量は 1 日 3 回と記載されていた。この女性は 1 日 2 回 1 錠ずつ服用し、他に薬物や栄養補助食品は摂取しておらず、アルコール摂取も年 1－2 回程度であった。胃酸逆流症状に対して処方されたラベプラゾールを 4 日間服用し、カワカワ含有製品の服用は中止したが、数日後に黄疸と肝炎により入院した。肝生検により亜急性（subfulminant）肝壊死が認められ、2001 年 7 月に肝移植を行った。

例 2：2000 年 12 月に、14 歳の女児が悪心、嘔吐、食欲減退、体重低下及び疲労感を訴え、急性肝炎により入院した。この女児は 8 月下旬から 12 月中旬にかけて、2 種類のカワカワ含有製品（成分は不明）を表示通りに 1 日 2 カプセル、約 44 日間及び 7 日間服用しており、アルコール及び薬物摂取はなかった。入院時の肝機能検査では ALT:4,076 U/L、AST:3,355 U/L、γ-GTP:148 U/L、総ビリルビン:16.2mg/dl、アンモニア:17mg/dl、プロトロンビン時間:29.4sec と異常値を示し、肝生検では活動性劇症肝炎を呈したため、同所性肝移植を行った。両症例共、日常生活機能を回復している。ヨーロッパでカワカワ含有製品に関連する肝不全により肝移植を受けた症例は 8 例（ドイツ 6 例、スイス 2 例、男性 2 例（32 および 50 歳）、女性 6 例（22－61 歳）、カワカワ摂取期間は 8 週～12 ヶ月、摂取

量は 60～240mg/日）で、インフルエンザ様症状や黄疸を含む多様な症状を呈し、全例劇症肝炎に至った。医療従事者は、肝傷害のみられる患者に対し、栄養補助食品及びハーブ製品の使用歴について質問することを検討すべきである。

Editorial Note《編集注記》

FDA は、カワカワを含む製品の使用に関連する肝障害のリスクについて、消費者及び医療従事者に忠告している。

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Hepatic Toxicity Possibly Associated with Kava-Containing Products --- United States, Germany, and Switzerland, 1999--2002

Since 1999, health-care professionals in Germany, Switzerland, and the United States have reported the occurrence of severe hepatic toxicity possibly associated with the consumption of products containing kava (i.e., kava kava or *Piper methysticum*). A total of 11 patients who used kava products had liver failure and underwent subsequent liver transplantation (*1--7*). On March 25, 2002, in response to five such case reports (four in Europe and one in the United States), the Food and Drug Administration (FDA) issued a consumer advisory (*8*) and subsequently completed an investigation already underway of a similar U.S. case. This report presents the investigation of the two U.S. cases of liver failure associated with kava-containing dietary supplement products and summarizes the European cases. FDA continues to advise consumers and health-care providers about the potential risk associated with the use of kava-containing products.

Case Reports

Case 1. In May 2001, a previously healthy woman aged 45 years reported the onset of nausea and weakness approximately 8 weeks after beginning use of a kava-containing dietary supplement that listed on the package label, "Kava kava extract (root), standardized to 30% kavalactones (75 mg), hops (strobiles), German chamomile (flower head), passion flower (flower and fruit), gelatin, and natural vegetable fiber." The patient reported taking one tablet twice daily, which was less than the package label recommendation of one tablet three times daily. The patient reported no concomitant medication or dietary supplement use and rare alcohol ingestion (one to two drinks a year). The patient was initially prescribed rabeprazole for acid reflux symptoms, and this drug was taken for 4 days. In addition, the patient discontinued use of the kava-containing supplement. Several days later, the patient was hospitalized with

jaundice and hepatitis. Liver biopsy demonstrated subfulminant hepatic necrosis. Autoimmune and infectious hepatitis tests were negative. Liver transplantation was performed in July 2001, and the patient resumed daily activities following recovery from the procedure.

Case 2. In December 2000, a previously healthy girl aged 14 years reported the onset of nausea, vomiting, decreased appetite, weight loss, and fatigue. One week later, the patient had scleral icterus and was hospitalized with acute hepatitis. During late August to mid-December 2000, the patient reportedly used two kava-containing products. One product was taken intermittently in accordance with package directions (two capsules once daily). The patient estimated that she used the product on approximately 44 days during this period. The patient reported taking the second product in accordance with package directions (two capsules once daily) for 7 consecutive days at the beginning of the 4-month period. Because the product labels were unavailable, other product ingredients were unknown. The patient reported no use of alcohol or medications other than occasional ibuprofen. At the time of hospitalization, the patient's liver-function tests were markedly abnormal (alanine aminotransferase: 4,076 U/L, aspartate aminotransfease: 3,355 U/L, gamma-glutamyltransferase: 148 U/L, total bilirubin: 16.2 mg/dL, ammonia: 17 mg/dL, and prothrombin time: 29.4 seconds) (*5*). Tests for human immunodeficiency virus (HIV), cytomegalovirus, Epstein-Barr virus, Wilson's disease, a-antitrypsin deficiency, antinuclear antibodies, and hepatitis A, B, C, and E were negative. Initial liver biopsy revealed active fulminant hepatitis with extensive centrilobular necrosis, approximately 25% hepatocellular viability, and mixed inflammatory infiltrates consisting of lymphocytes, histiocytes, scattered eosinophils, and occasional neutrophils. No viral cytopathic changes were identified, and immunohistochemical stains for hepatitis B surface and core antigens were negative. The patient underwent successful orthotopic liver transplantation. Pathological examination of the native liver revealed active fulminant hepatitis with total hepatocyte necrosis and extensive parenchymal infiltration by lymphocytes, histocytes, and occasional eosinophils (*5*). The patient resumed daily activities following recovery from the procedure.

Summary of European Case Reports

Eight hepatic transplant cases following hepatic failure associated with the use of kava-containing products have been reported in Europe (six in Germany and two in Switzerland). Two male patients aged 32 and 50 years and six females aged 22--61

years required liver transplants after using kava-containing products. The duration of kava use ranged from 8 weeks to 12 months. The products were used at doses ranging from 60 mg to 240 mg per day. Seven patients used kava prepared either by ethanol or acetone extraction methods; one patient used an unspecified type of kava-containing product. The patients had varying symptoms, including influenza-like symptoms and jaundice. Each patient's condition worsened and progressed to fulminant hepatic failure. Four of these cases have been reported in medical literature (*1*--*4*). Additional information about these cases is available from the German regulatory authority, the Federal Institute for Drugs and Medical Devices, Bonn, Germany, at http://www.bfarm.de. A ninth European transplant case was reported directly to FDA's MedWatch System by a U.S. pharmaceutical manufacturer.

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Editorial Note:

Kava is a botanical product derived from the rhizome and roots of *Piper methysticum*, a shrub indigenous to the South Pacific. In the United States, kava-containing products are sold as dietary supplements and marketed for the treatment of anxiety, occasional insomnia, premenstrual syndrome, and stress. These supplements often are in the form of raw plant material or concentrated extracts, which are obtained by using either acetone or ethanol extraction or cryoprecipitation. Preparations marketed for human consumption contain a mixture of components collectively known as kava pyrones (i.e., kavalactones). Kava-containing products might differ based on the absolute amount of kava pyrones present and on the relative distribution of kava pyrones. Several countries, including Germany, Switzerland, Canada, Australia, and France, have restricted the sale of kava-containing products based on the occurrence of hepatic adverse events and the documented hepatic toxicity following rechallenge with a kava-containing product (*9*). FDA research suggests that <1% of the severe adverse events that occur with the use of dietary supplements are reported to FDA (*10*).

FDA has advised consumers and health-care providers about the potential risk for hepatic toxicity associated with the use of kava-containing products (*7*). Additional caution by persons who have pre-existing liver disease or are at risk for liver disease might be warranted. Health-care providers should consider questioning patients with evidence of hepatic injury about the use of dietary supplements and herbal products. Adverse events associated with the use of any dietary supplement should be reported to FDA's MedWatch Program, telephone 800-332-1088, or http://www.fda.gov/medwatch.

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MMWR51 (19):414-416

最新情報：フロリダフグによる神経疾患
2002 年

2002 年 5 月 15 日時点で、フロリダ州タイタスビル市近海で捕獲されたフグによる神経症状の発症が合計 13 例（それらは 2002 年 1 月 1 日から 4 月 25 日に発生）報告されている。13 例のうち 9 例はフロリダ州の各地の中毒情報センターを通して、4 例は各病院の救急科による監視と各郡保健局の食品媒介疾病の報告記録を通じて認知された。

主症状は、刺痛またはしびれ感で口唇部（13 名）、顔面（8 名）、腕（10 名）、足（7 名）指先（1 名）であり、運動失調（2 名）、筋脱力感（1 名）、悪心（6 名）、嘔吐（4 名）といった症例も報告されている。症状はフグを食べた後、30 分から約 8 時間（中央値 2 時間）に発現し、10 時間から 45 日間（平均 6.6 日、中央値 24 時間）持続した。救急外来（ED）を受診した 11 名中 5 名が入院したが全員回復している。食べた量と重症度には相関は認められず、同じようにフグを食べても症状の発現を認めない例もあった。

米国食品医薬品局（FDA）食品安全応用栄養センター（CFSAN）がフロリダ州魚類・野生生物保護委員会（Fish and Wildlife Conservation Commission : FWC）の協力を得て、フロリダ州の近海数ヶ所からフグ 25 匹を捕獲した。ニューヨーク市クイーンズ区にある食品医薬品局北東地域研究室（FDA's Northeast Regional Laboratory）による検査の結果、タイタスビル埠頭付近のインディアン川の河口域で捕獲したフグからサキシトキシンが検出された（2～53 μg/g）。発症例は、インディアン川の河口域で捕獲されたフグを食したものと考えられ、FWC は 4 月 25 日、ボルシア、ブリヴァード、インディアンリバー、セント・ルーシー郡内でのフグの捕獲を禁止した。捕獲は 7 月中旬まで禁止とされている。

現在、フロリダ州全域でフグのサンプリング調査が進行中であるが、インディアン川及び南バナナ川水域にしかサキシトキシンを持っているフグはいないことが判明した。通常、サキシトキシン中毒は軟体動物（mollusk）に関連することから、CFSAN とフロリダ州農業・消費者サービス局（Department of Agriculture and Consumer Services）は、インディアン川の潟（がた）周辺の 5 地点（タイタスビル市地域を含む）に所在する養殖リース地（aquaculture lease site）からホンビノスガイ（hard-shell clam）約 100 個のサンプル

を採取した。さらに別の二つの地点（フロリダ半島西岸、タイタスビル市は東岸に所在）からのホンビノスガイが対照サンプルとして採取された。全サンプルがサキシトキシンに陰性反応を示した。

フグは約 100 種類が知られ、フロリダでは 9 種が生息している。北インディアン川では南洋フグが増えているが、今まで中毒を起こした報告はない。今後、釣り人に対して注意を呼びかけ、フグ中毒に関する情報を提供し、重症の場合には救急外来（ED）を受診するよう周知徹底する必要があると思われる。

May 17, 2002 / 51(19); 414-6

Update: Neurologic Illness Associated with Eating Florida Pufferfish, 2002

As of May 15, 2002, a total of 13 presumptive cases of saxitoxin poisoning were reported in Florida residents who ate pufferfish caught in waters near Titusville, Florida. Five cases were reported in April (*1*), and eight cases were identified through increased surveillance by Florida poison control centers, hospital emergency departments (EDs), and county health departments. This report updates the investigation of these cases.

All 13 cases occurred during January 1--April 25, 2002. Nine were identified through Florida poison control centers; four were identified by active surveillance of hospital EDs and health department foodborne illness complaint logs.

Investigators defined a case as tingling or numbness in the mouth and/or lips in a person who had eaten Florida pufferfish. All ill persons reported at least one of the following symptoms after a meal that included pufferfish: tingling or numbness in the mouth or lips (13 persons), face (eight), arms (10), legs (seven), and fingertips (one). In initial reports, two patients reported ataxia, and one reported muscle weakness. Some ill persons experienced nausea (six) and vomiting (four) before presenting to a hospital ED. Symptom onset occurred 30 minutes to approximately 8 hours after ingestion of fish (median: 2 hours). Duration of illness ranged from 10 hours to 45 days (mean: 6.6 days; median 24 hours). Eleven persons were treated in an ED, and five were admitted to the hospital. Some patients received intravenous fluids. All cases resolved.

Severity of illness was not associated with amount of pufferfish eaten, and nine meal partners who also ate the pufferfish did not become ill. Testing of approximately 25 pufferfish collected from Florida waters by the Food and Drug Administration's (FDA) Center for Food Safety and Applied Nutrition (CFSAN), in collaboration with the Florida Fish and Wildlife Conservation Commission (FWC), and additional testing by FDA's Northeast Regional Laboratory indicated that pufferfish containing potentially toxic concentrations of saxitoxins (2--53 μg saxitoxin equivalent toxicity/g) are

present in the Indian River in the area of the Titusville Pier. Lower concentrations of saxitoxin have been found in pufferfish from the South Banana River.

All pufferfish related to these cases were caught in the Indian River Lagoon; 11 patients caught pufferfish off the Titusville Pier. The FWC banned retaining pufferfish caught from Volusia, Brevard, Indian River, and St. Lucie counties on April 25; the ban will remain in effect until mid-July. Information about this ban was provided at the Titusville Pier on April 30, 2002.

No filets associated with the Florida illnesses were available for testing to confirm the presence of saxitoxin. However, ongoing statewide sampling of pufferfish in Florida has indicated that pufferfish containing saxitoxins are limited to the Indian and South Banana rivers. Because saxitoxin poisoning is usually associated with mollusks, CFSAN and the Florida Department of Agriculture and Consumer Services sampled approximately 100 hard-shell clams from aquaculture lease sites at five locations along the Indian River Lagoon, including the Titusville area in Florida. Clams from two additional locations were chosen as control samples and were collected from the west coast of Florida in the Gulf of Mexico. All samples tested negative for saxitoxin.

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Editorial Note:

The initial report described 10 illnesses that were associated with pufferfish ingestion in three states (New Jersey, Virginia, and Florida) and were consistent with exposure to saxitoxins (*1*). One case (New Jersey) was associated with commercially purchased

pufferfish from Titusville, and all other cases resulted from recreationally caught pufferfish in the Titusville area. Laboratory analysis by the Canadian Institute for Marine Biosciences of partially eaten pufferfish from the New Jersey incidents confirmed the presence of saxitoxin and two analogs (*2*). These results were confirmed with additional analysis by FDA's Northeast Regional Laboratory (*3*).

Some previous intoxications by pufferfish in Florida were attributed to tetrodotoxin (*4*). Seven cases of pufferfish poisoning were reported in Florida during 1951--1974, including three fatalities (*5, 6*). These case reports were associated with ingestion of locally caught species of pufferfish *Sphoeroides*. A 1963 study of pufferfish from the east coast of Florida (from the Indian and Banana Rivers, including the Titusville area) demonstrated that pufferfish were toxic to mice (*4*). Although the species tested in this study was listed as *Sphoeroides maculatus* (northern pufferfish), there was confusion over the identification of this species with *S. nephelus* (southern pufferfish) (Figure 1). Northern pufferfish extend only as far south as Jacksonville, Florida (*7*), and are not known to exist in the Indian and Banana rivers.

The illnesses described in this report occurred after ingestion of pufferfish but are consistent with the presence of saxitoxin, a paralytic shellfish toxin usually associated with ingestion of filter-feeding shellfish. Concentration of saxitoxin in the pufferfish tested from the Titusville area varies. Saxitoxin has been reported in pufferfish from the Far East (*8*) and the Philippines (*9*). Shellfish containing 2--10 μg saxitoxin/g previously have caused illness (*10*), but saxitoxin has not previously been reported in Florida. The severity of illnesses in persons described in this report varied probably because of the concentration of saxitoxin in a particular pufferfish and/or the amount of pufferfish eaten.

Approximately 100 species of pufferfish are known worldwide, and nine species are present in Florida. Southern pufferfish populations have been increasing in the Northern Indian River during the previous 5 years (FWC, unpublished data, 2002). The southern pufferfish that have been caught recently near Titusville are normally present in this area of Florida, but they have not been implicated previously in fish poisoning events.

Sportfishers in Florida need to be educated that potentially toxic pufferfish might be in the Titusville area. Warnings about the presence of certain species of potentially toxic pufferfish should be posted in commonly fished areas. Because many sportfishers

vacation in Florida and transport fish home to other states, health-care providers should be aware that rapid onset of neurologic symptoms after a meal of pufferfish could be caused by saxitoxin. Ingestion of paralytic shellfish toxins produces neurologic symptoms that are sensory, cerebellar, and motor. The most common symptoms are tingling and burning of the mouth and tongue, numbness, drowsiness, and incoherent speech. These symptoms usually occur 30 minutes to 2 hours after ingestion of the fish, depending on the amount of toxin ingested. In severe cases, ataxia, muscle weakness, respiratory paralysis, and death can occur (*10*). Ill persons should contact their local poison control center and proceed to a hospital ED. Hospital EDs and poison control centers should contact the local health department if persons report neurologic symptoms after eating pufferfish.

Acknowledgments

This report is based on data contributed by R Weisman, Pharm D, JL Schauben, PharmD, V Speranza, PharmD, Florida Poison Information Center; D Johnson, MD, Bur of Environmental Epidemiology, Florida Dept of Health. T Litovitz, MD, American Association of Poison Control Centers, Washington, DC. Office of Regulatory Affairs and Center for Food Safety and Applied Nutrition, Food and Drug Administration.

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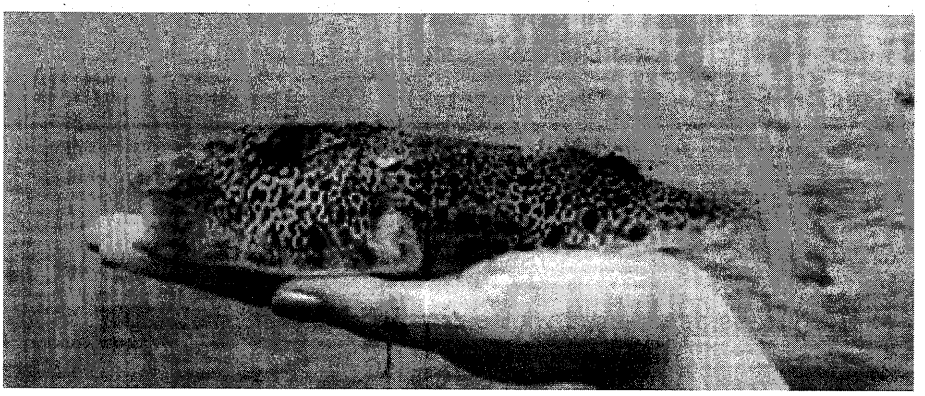
FIGURE 1. A southern pufferfish (*Sphoeroides nephelus*)



Photo/Florida Marine Research Institute, Florida Fish and Wildlife Conservation Commission

MMWR51 (15):321-323

フロリダフグによる神経障害
2002 年

2002 年 1 月より、フロリダ州タイタスビル市近海で捕獲されたフグによる神経症状の発症が全米中毒情報センター協会（the American Association of Poison Control Centers）の有毒物質曝露監視システム（Toxic Exposure Surveillance System : TESS）に 10 例（フロリダ州 5、ニュージャージー州 3、バージニア州 2）報告されている。

フロリダ州：34 歳男性、タイタスビル市近海で捕獲されたフグを食べた後、嘔吐及び下痢を来し、翌日両手のしびれ感及び刺痛を訴えて 1 月 2 日に救急外来（ED）を受診した。フロリダ州タンパ市の中毒情報センターへその旨、報告があった。患者は、輸液静注により入院 2 日後に回復した。

バージニア州：50 歳男性と息子 24 歳。タイタスビル市へ釣りに出かけ、フグを捕獲。調理して食べた後、唇と舌のしびれ感及び刺痛を訴え、バージニア州リッチモンド市の中毒情報センターへ連絡した。在宅で経過を観察し、息子は 3～4 日で症状が消失したが、父親は翌日、顔面、首及び肩へ症状が広がり、回復に 2 週間以上を要した。

ニュージャージー州：65 歳女性、家族がタイテスビル市近海で捕獲したフグを食べ、数分後、唇周辺に刺痛を感じ、2 時間後嘔吐を来した。ニュージャージー州中毒情報教育システム（New Jersey Poison Information and Education System）の指示で ED を受診し、胸部痛、頻拍及び血圧上昇に対し、ニトログリセリンを局所投与された。4 時間から 6 時間後には上行性筋肉麻痺を来し、呼吸機能も低下したため人工呼吸を行った。約 72 時間後には抜管し、退院に至った。

カナダ学術研究会議海洋生物科学研究所（Institute for Marine Biosciences, National Research Council, Canada）は、LC-タンデム質量分析により残っていたフグからテトロドトキシンは検出しなかったが、神経毒素サキシトキシン及びその類似物（N-sulfocarbamoylsaxitoxin 及び decarbamoylsaxitoxin）を検出した。また、分割標本（split specimen）は、ニューヨーク市クイーンズ区にある食品医薬品局地域研究室（regional Food and Drug Administration (FDA) laboratory）に提出された。

Editorial Note《編集注記》

ニュージャージー州保健・高齢者サービス局（Department of Health and Senior Services : NJDHSS）は、ニュージャージー州での2例を記述する報告を4月11日に発行し、4月12日にはタイタスビル市近海で捕れたフグを食べる際には十分注意するよう警告を発した。次いで4月15日に米国食品医薬品局（FDA）がタイタスビル市近海で捕れたフグに対する健康勧告（health advisory）を発令した。フロリダ州保健局（Department of Health）は現在、農業・消費者サービス局（Department of Agriculture and Consumer Services）及び魚類・野生生物保護委員会（Fish and Wildlife Conservation Commission）の協力を得て、各種のフグ及び海洋生物がサキシトキシンをどの程度持っているかについて調査すると共に、ニュージャージー州、フロリダ州、FDA及び米国疾病管理予防センター（CDC）では、この状況の詳細調査を継続している。

April 19, 2002 / 51(15); 321-3

Neurologic Illness Associated with Eating Florida Pufferfish, 2002

Since January 1, 2002, human illness after eating pufferfish caught in waters near Titusville, Florida, has been reported (Figure 1). The illnesses were manifested by neurologic symptoms consistent with exposure to paralytic shellfish toxins. Laboratory analysis in early April confirmed the presence of saxitoxin in uneaten pufferfish. This report presents selected case examples and summarizes all cases reported to the Toxic Exposure Surveillance System of the American Association of Poison Control Centers (TESS).

Case Reports

Florida. On January 2, the poison control center in Tampa, Florida, received a call from an emergency department (ED) physician about a man aged 34 years who had numbness and tingling of his hands. On January 1, he had experienced vomiting and diarrhea after eating approximately eight mouthfuls of pufferfish recreationally caught in waters near Titusville. The man was admitted to the hospital for observation and was administered intravenous fluids. His symptoms gradually resolved, and he was released 2 days after admission.

Virginia. On March 12, a man aged 50 years and his son aged 24 years returned from a fishing trip to Titusville, where they had caught several pufferfish. Approximately 3 hours after they had cooked and eaten the fish, they contacted the Richmond poison control center complaining of numbness and tingling of the lips and tongue. The two men decided to monitor their symptoms at home. The younger man's symptoms were limited to oral numbness and resolved in 3--4 days. The older man's symptoms progressed during the evening to include numbness and tingling in the face, neck, and shoulders; the next day, he still had numbness in his mouth. The symptoms reportedly resolved over 2 weeks.

New Jersey. On March 18, a woman aged 65 years was brought to the hospital ED by her husband. Hours earlier, they had eaten a meal of pufferfish that a family member

had caught in Titusville. Several minutes after eating the fish, both persons experienced tingling around their lips. During the next 2 hours, the woman's symptoms worsened, and she developed vomiting. They contacted the New Jersey Poison Information and Education System and were advised to go to the hospital ED. The woman developed increasing chest pain and had mild tachycardia and blood pressure of 160/70 mmHg; she was treated with topical nitroglycerine. During the next 4--6 hours, she developed an ascending muscular paralysis. A test of her respiratory function indicated carbon dioxide retention and a rapid decrease to <20% of normal vital capacity for a woman her age. She was electively intubated and placed on a ventilator. Over the next day, she regained her reflexes and voluntary movement. She was extubated at approximately 72 hours and discharged.

Laboratory Findings

Uneaten fish samples recovered in New Jersey were submitted for toxin analysis to the Institute for Marine Biosciences, National Research Council, Canada. Liquid chromatographic-tandem mass spectrometric analysis of uneaten fish samples did not detect tetrodotoxin in any of the pufferfish samples (*1*). However, the analysis confirmed that the fish contained the paralytic shellfish toxin, saxitoxin, and two analogs, N-sulfocarbamoylsaxitoxin and decarbamoylsaxitoxin. Liquid chromatography with postcolumn oxidation and fluorescence detection confirmed these analytical results (*2*).

A split specimen also was submitted to the regional Food and Drug Administration (FDA) laboratory in Queens, New York. Presence of a sodium channel-blocking toxin was confirmed by cell bioassay (*3*). These results are consistent with the presence of saxitoxin or tetrodotoxin.

Toxic Exposure Surveillance System

Since January 1, TESS has identified 10 illnesses of presumed pufferfish poisoning (five from Florida, three from New Jersey, and two from Virgnia). All ill persons reported eating pufferfish originating from the Titusville area (Indian and Banana rivers). All reported at least one of the following symptoms: tingling in the mouth and lips or fingertips, numbness, or peripheral neuropathy. All cases eventually resolved. Efforts are ongoing to identify additional cases.

Reported by: *S Marcus, MD, MY Wong, B Ruck, PharmD, New Jersey Poison Information and Education System, Univ of Medicine and Dentistry of New Jersey/New Jersey Medical School, Newark; E Bresnitz, MD, State Epidemiologist, New Jersey Dept of Health and Senior Svcs. R Hammond, PhD, Florida Dept of Health. H Rogers, PhD, M McGeehin, PhD, Div of Environmental Hazards and Health Effects, National Center for Environmental Health, CDC.*

Editorial Note:

Neurologic illnesses associated with eating pufferfish (i.e., blowfish, sea squab, and Fugu [*Terodontidae* and *Diodontidae* families]) are not common in the United States. Pufferfish are harvested recreationally and commercially in the United States and internationally. Fish harvested in the United States are often transported to different states for sale. Several of the approximately 100 species of pufferfish contain neurotoxins (i.e., tetrodotoxin and/or saxitoxin); most pufferfish caught in U.S. waters are not known to be toxic, although there have been reports of toxic pufferfish.

The cases in this report occurred after eating pufferfish but are consistent with paralytic shellfish poisoning (PSP). In the United States, PSP is associated with eating filter-feeding shellfish or mollusks. Approximately 10 outbreak-associated PSP cases are reported to CDC each year. Ingestion of paralytic shellfish toxins produces neurologic symptoms that are sensory, cerebellar, and motor. The most common symptoms are tingling and burning of the mouth and tongue, numbness, drowsiness, and incoherent speech. These symptoms occur 30 minutes to 2 hours after ingestion of the fish, depending on the amount of toxin ingested. In severe cases, ataxia, muscle weakness, respiratory paralysis, and death can occur (*4*).

Saxitoxin and tetrodotoxin together and saxitoxin alone in freshwater pufferfish have been reported in waters near Thailand (*5*) and Bangladesh (*6*). Saxitoxin and its analogs are produced by dinoflagellates of the *Gonyaulacoid* family and by some freshwater cyanobacteria (*7*). Shellfish are contaminated when toxin-producing organisms multiply in the water and form a bloom, and water-siphoning shellfish---principally clams, mussels, and scallops---filter out organisms to feed and absorb any toxins produced. Generally, nonfilter feeders such as fish, lobsters, crabs, and shrimp are considered safe to eat, even if caught in contaminated waters. However, pufferfish eat molluscs and might accumulate or even magnify the toxin (*8*).

Saxitoxin is heat- and acid-stable and does not alter the odor or taste of food. This toxin cannot be destroyed by cooking or freezing. It is rapidly absorbed through the human gastrointestinal tract and excreted in urine. The molecule is complex and contains a guanidinium moiety. This portion of the molecule is believed to block the opening of the voltage-sensitive Na+ channel, preventing the rapid entrance of sodium into the cell at depolarization. The rapid movement of sodium is necessary for propagation of neural impulses and mediation of cellular function. The outcome of blockage at this site is motor paralysis.

Tetrodotoxin is a powerful neurotoxin that has been detected in many pufferfish species; its presence is usually associated with season, geographic location, sex, and organ tissue. Tetrodotoxin might be produced by *Vibrio* species or other bacteria that bioaccumulate in the pufferfish (*9*). Tetrodotoxin has been detected in pufferfish throughout the Pacific Ocean and the Baja California coastal region. This is the first report to CDC of neurotoxic pufferfish in the Atlantic Ocean.

Health-care providers should be aware that rapid onset of neurologic symptoms after a meal of pufferfish could be caused by saxitoxin. Ill persons should be advised to proceed to a hospital ED and contact their local poison control center.

On April 11, the New Jersey Department of Health and Senior Services (NJDHSS) issued a report describing two of the New Jersey cases. On April 12, NJDHSS issued a warning about eating pufferfish originating from the Titusville area. On April 15, FDA also issued a health advisory on pufferfish caught from this area. The Florida Department of Health, in collaboration with the Florida Department of Agriculture and Consumer Services and the Florida Fish and Wildlife Conservation Commission, is assessing the extent of the presence of saxitoxin in pufferfish and other marine species. New Jersey, Florida, FDA, and CDC are continuing to investigate this situation.

Acknowledgments

This report is based on data contributed by G Wolf, J Blumenstock, New Jersey Dept of Health and Senior Svcs. SR Rose, PharmD, Virginia Poison Center. JL Schauben, PharmD, V Speranza, Pharm D, Florida Poison Information Center. T Litovitz, MD, American Association of Poison Control Centers, Washington, DC. Office of Regulatory Affairs and Center for Food Safety and Applied Nutrition, Food and Drug

Administration. M Quilliam, PhD, D Wechsler, Institute for Marine Biosciences, National Research Council, Canada, Halifax.

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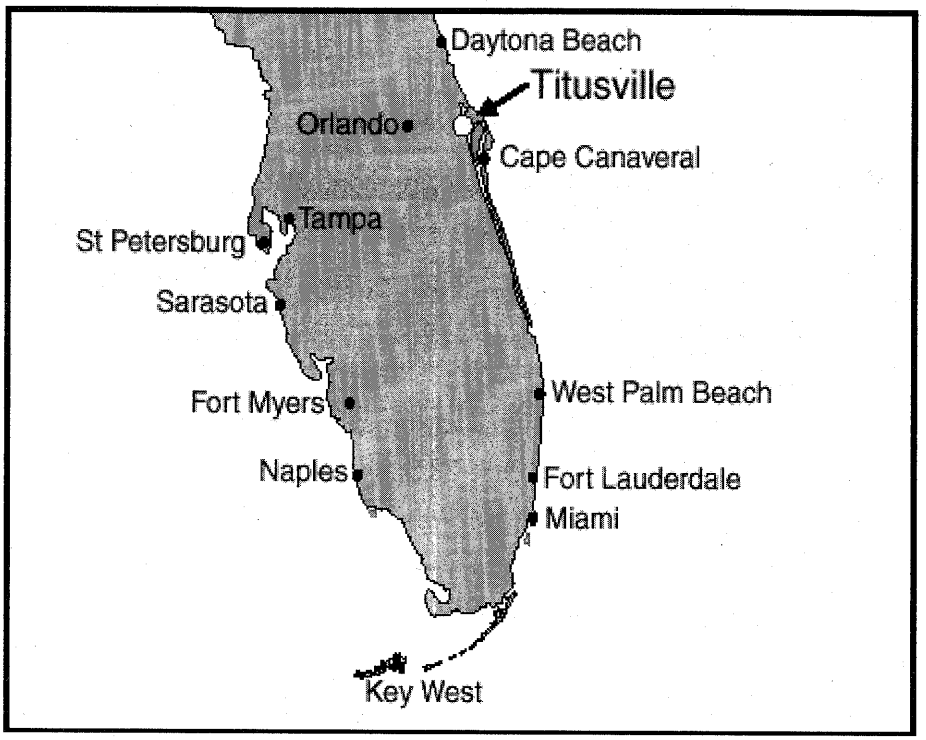
FIGURE 1. Location of Titusville, Florida



MMWR49 (18):398-400, 2000

サバ亜目魚によるヒスタミン中毒
ペンシルベニア州，1998 年

1998 年 12 月、ペンシルベニア州チェスター郡の衛生局（the Chester County Health Department : CCHD）はレストランでスコンブロイドフィッシュ（サバ亜目の魚）により 4 例の中毒が発生した旨についての報告を受けた。本報告では、この事件について実施された CCHD、ペンシルベニア州農業局（PDAg）及び衛生局（PDOH）による調査結果について紹介する。

本事件は、1998 年 12 月 3 日、チェスター郡の某レストランでマグロとほうれん草のサラダを喫食した成人 4 名が喫食約 5 分～2 時間後に発症したもので、症状は口周囲の熱感、顔面紅潮、吐き気、下痢、発汗、頭痛、嘔吐等であった。患者 1 名が地域の救急センターを受診、ディフェンヒドラミン、シメチジン及びエピネフィリンによる治療を受けたが、他の 3 名は医師を受診せず数時間後に回復した。臨床的並びに疫学的所見からスコンブロイドフィッシュ中毒が疑われたため、事件のあったレストランに残されていたマグロについて PDOH で検査を実施した結果、大腸菌群及び大腸菌が分離されると同時に、ELISA（酵素免疫吸着測定）法により 50 ppm を超えるヒスタミン（新鮮な魚に含まれるヒスタミンは通常 10 ppm 未満）が検出された。

一方、CCHD 及び PDAg により、マグロに対する追跡調査がなされた結果、問題となったマグロは 1998 年 11 月の後半にメキシコ湾で商業的に捕獲されたキハダマグロで、捕獲は延縄によるものであった。捕獲当時の海水温度は 25.8 ℃で、マグロは捕獲時に氷詰にし、陸揚げ後、11 月 24 日にトラックで食品加工業者まで輸送されたことが判明した（温度 0～1 ℃）。また、同時に捕獲された 785 ポンドのマグロが同日、ペンシルベニア州の卸売業者に配送された。この卸売業者が荷を受領したのは 11 月 27 日で、マグロの温度は 2 ℃であった。そして、11.1 ポンド大の切り身（肉眼的にみて良好な状態）にして小売業者を経て 11 月 27 日にレストランに納入された。納入されたマグロは、さらに 30 個に分割、冷凍保存され、必要時に解凍して使用された。11 月 28 日～12 月 4 日にかけて客に提供され

たのはこのうちの 17 個で、患者が発生したのは 12 月 3 日に提供されたホウレン草と和えたツナサラダを食べた（報告のあった）4 名のみであった。

CCHD 及び PDAg により実施された卸売～小売段階に至る査察検査では、特に HACCP（危害分析重要管理点方式）上、問題視されるような逸脱は認められなかった。しかし、延縄によるマグロの捕獲に関しては米国食品医薬品局（FDA）が設定した水産物に対する HACCP 規定がないため、おそらく今回の事故は HACCP 規定のない漁獲から陸揚げまでの最初の段階で毒素の蓄積が起きたものと推定された。

Editorial Note《編集注記》

1997 年 12 月 18 日以降、FDA によって全ての魚加工業者はその加工工程に関して危害分析を実施し、特定された各危害を管制するために HACCP を実施するよう要請されている。

May 12, 2000 / 49(18); 398-400

Scombroid Fish Poisoning --- Pennsylvania, 1998

In December 1998, the Chester County Health Department (CCHD) in Pennsylvania received reports of four cases of scombroid fish poisoning among patrons at a local restaurant. This report summarizes the investigation of these cases by CCHD, the Pennsylvania Department of Agriculture (PDAg), and the Pennsylvania Department of Health (PDOH). Findings from this investigation suggest that initial processes that are not regulated by the Food and Drug Administration (FDA) (i.e., from hooking the fish to unloading the fish on the dock) may permit scombrotoxin formation.

On December 3, 1998, four adults became ill after eating tuna-spinach salad at the restaurant. Symptoms of illness included a burning sensation in the mouth, a metallic taste, facial flushing, nausea, diarrhea, sweating, and headache; symptoms occurred approximately 5 minutes to 2 hours after eating the salad. One patient was taken to the local emergency department and treated with diphenhydramine, cimetidine, and epinephrine. The other three patients were not examined by physicians and their symptoms resolved within a few hours. A presumptive diagnosis of scombroid fish poisoning was made based on clinical and epidemiologic features of the illness.

A sample of the remaining fish obtained from the restaurant was sent to PDOH for testing. The fish was positive for coliform and *Escherichia coli*, and tests were positive for histamine levels >50 ppm (fresh fish normally contain histamine levels of <10 ppm [*1*]) using an enzyme-linked immunoabsorbent assay.

CCHD and PDAg conducted a traceback investigation of the source of the tuna. The wholesale-to-retail chain of events involved transporting the fish across national, state, and municipal borders and involved five transporters and four processors. The tuna was from a 40--60 lb yellow-fin tuna caught by a commercial fishing boat in the Gulf of Mexico during late November 1998. The fish was caught using the long-line method, which uses a mainline up to 60 miles long with a series of suspended hook lines. The water temperature where the fish was caught was 78.5 F (25.8 C). The catch of tuna was shipped from the fishing boat in iced vats by truck to a processor

on November 24. The average temperature of the fish was 32 F--33 F (0 C--1 C). Of this catch, 785 lbs of tuna were shipped the same day to the wholesaler in Pennsylvania. The wholesaler received the shipment on November 27, and the average temperature of the fish was recorded as 36 F (2 C). Three of these fish were delivered to the retail supplier; two large fillets, weighing 11.1 lbs each and noted to be in good physical appearance, were delivered to the restaurant on November 27. The fish was divided into 30 portions, kept in the freezer, and removed for thawing as needed for use. During November 28--December 4, 17 portions of the fish were served. The only four persons reporting illness ate the tuna-spinach salad on December 3.

CCHD and PDAg reviewed the records of each distributor involved in the wholesale-to-retail process of the tuna. All of the fish plants involved were inspected regularly by the FDA and/or PDAg and have Hazard Analysis and Critical Control Point (HACCP) procedures. No deviations in HACCP procedures in the wholesale-to-retail distribution of the tuna could be identified. However, the long-line method of fishing is not covered as part of the FDA Seafood HACCP regulations.

Reported by: *JP Maher, MD, JA Worth, J Arvay, K Raum, Chester County Health Dept, West Chester; L Iampetro, Pennsylvania Dept of Health Bur of Laboratories; JR Welte, Food Safety and Laboratory Svcs, Region 7, Pennsylvania Dept of Agriculture. Food and Drug Administration. Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; and an EIS Officer, CDC.*

Editorial Note:

Scombroid fish poisoning has been associated primarily with the consumption of tuna, mahi-mahi, and bluefish. It is caused by histamine and other products produced by certain bacteria on some types of fish; these bacteria grow in warm temperatures and produce the enzyme histidine decarboxylase that converts free histidine in fish flesh to histamine and other products (*2--6*).

National surveillance data on scombroid fish poisoning is based on outbreaks of acute foodborne disease reported by state health departments to CDC (*7, 8*). During 1988--1997, scombroid fish poisoning was reported in 145 outbreaks involving 811 persons from at least 20 states (*7, 8*); however, many cases probably are not reported.

Since December 18, 1997, all processors of fish are required by FDA to conduct a hazard analysis of their operation and to implement a HACCP plan to control each identified hazard (*9*). The HACCP plan must be specific for each location where fish and fish products are processed and for each species processed (*10*). The fish implicated in these scombroid fish poisonings was caught by the long-line method of fishing, which consists of suspending a monofilament line, up to 60 miles long, with up to 3000 baited hooks in the water. The retrieval process may take up to 12--14 hours, and the fish may be retained on the lines up to 20 hours. Although no deviations in HACCP procedures were documented in this outbreak, the time from hooking the fish to unloading the fish on the dock is not covered by HACCP. Conditions permitting histamine production could have occurred while the fish were in warm water suspended on the long line.

Scombrotoxin formation also could have resulted from fish handling practices anywhere along the distribution chain after the fish was caught to serving at the restaurant. The reportedly good color and appearance of the fish at the retailer and the lack of other reported illnesses may indicate that scombrotoxin formation occurred at the restaurant during processing and handling of the fish.

This outbreak suggests interventions that could reduce the risk for scombroid poisoning. First, consideration should be given to limiting the amount of time that fish can remain on the line during the long-line method of fishing. Second, efforts should focus on maintaining adequate refrigeration of fish during distribution and in restaurants to prevent conditions favorable for scombrotoxin production. The key to prevention of scombroid fish poisoning is continuous icing or refrigeration at $\leq$32 F ($\leq$0 C) of all potential scombrotoxin-producing fish from the time they are caught until they are cooked.

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MMWR47(33):692-694,1998

魚のシガテラ中毒
テキサス州，1997 年

1997 年 10 月 21 日、テキサス南東中毒センターは地域の医師からフリーポート港にドッグ入りした貨物船乗組員に発生した集団中毒患者（悪心、嘔吐、下痢、筋肉無力）の治療法について問い合わせを受けた。

調査によると、10 月 12、13 日の両日、ノルウェーの貨物船乗組員に胃腸炎患者が発生、ドッグ入りした後の 10 月 22 日に船員 27 名中 23 名（85%）に聞き取り調査した結果、17 名（74%）が次のような症状を呈していることが判明した。17 名が下痢（100%）、14 名が腹痛（82%）、13 名が悪心（76%）、13 名が嘔吐（76%）。患者は 10 月 12 日午後 7 時頃にカマスを食べ、その 2～16 時間（平均 4.5 時間）後に発症、14 日までに罹患した全乗組員にシガテラ中毒特有の神経症状（筋肉痛・無筋力 15 名、口唇麻痺または掻痒 13 名、手または足の掻痒 11 名、温度感覚異常 11 名、めまい 10 名等）が出現した。10 月 21 日、17 人名全員が医療機関を受診し、症状を減弱するための対処療法を受けたが入院者はいなかった。

テキサス州衛生局（the Texas Department of Health : TDH）により実施された船員の喫食調査の結果、10 月 11 日のバハマの Cay Sal Bank 付近で船員が釣りあげたカマスを喫食した 17 名全員が発症していたのに対し、非喫食者 8 名については全員非発症であったことが判明し、それが原因と推定された。船員は同時にアカフエダイとハタも喫食していたが、これらは疫学的にいずれも発症とは関連性が認められなかった。患者 16 名について実施された糞便の菌培養（サルモネラ、赤痢菌、カンピロバクター、エルシニア、ビブリオ）は全て陰性であったが、原因と推定されたカマスと同じ時に捕獲され、冷凍室に残されていたカマス及びアカハタ 3 点についてハワイ大学で実施された魚毒検査ではいずれもシガトキシン陽性であった。

Editorial Note《編集注記》

米国では、1983～1992 年までの間に計 129 件（患者数 508 名）の魚によるシガテラ中毒が米国疾病管理予防センター（CDC）に報告（主にハワイ、フロリダ）されているが、死亡した事例はまだ報告されていない。

August 28, 1998 / 47(33); 692-694

Ciguatera Fish Poisoning -- Texas, 1997

On October 21, 1997, the Southeast Texas Poison Center was contacted by a local physician requesting information about treatment for crew members of a cargo ship docked in Freeport, Texas, who were ill with nausea, vomiting, diarrhea, and muscle weakness. This report summarizes an investigation of this outbreak by the Texas Department of Health (TDH), which indicated that 17 crew members experienced ciguatera fish poisoning resulting from eating a contaminated barracuda.

On October 12 and 13, gastrointestinal illness developed in crew members aboard a Norwegian cargo ship. After the ship had docked, on October 22 interviews were conducted with 23 (85%) of 27 crew members. A case was defined as ciguatera fish poisoning if there was a combination of gastrointestinal symptoms (i.e., nausea, vomiting, diarrhea, or abdominal cramps) and neurologic symptoms (i.e., muscle pain, weakness, dizziness, numbness or itching of the mouth, hands, or feet) in a crew member after eating fish on October 12. Of the 23 interviewed, 17 (74%) crew members reported the following symptoms: diarrhea (17 {100%}), abdominal cramps (14 {82%}), nausea (13 {76%}), and vomiting (13 {76%}). Symptoms occurred within 2-16 hours (median: 4.5 hours) after eating fish at approximately 7 p.m. on October 12. By October 14, all ill crew members had experienced neurologic symptoms characteristic of ciguatera poisoning: 15 (88%) reported muscle weakness and pain; 13 (76%), numbness or itching of the mouth; 11 (65%), pruritus of the feet and/or hands; 11 (65%), temperature sensation reversal; 10 (59%), dizziness; and eight (47%), aching or loose-feeling teeth.

On October 21, all 17 ill crew members sought medical care at a clinic. None of the crew members were hospitalized; treatment consisted of supportive measures to reduce discomfort from symptoms. All patients were men aged 23-46 years.

Based on food histories from the 23 crew members, TDH suspected consumption of a barracuda caught by crew members while fishing near the Cay Sal Bank of the Bahamas on October 11 as the source of illness. Seventeen crew members ate the barracuda, and all became ill. None of the eight crew members who did not eat

barracuda became ill. Although crew members also ate red snapper and grouper at the same meal, neither of these fish were linked epidemiologically with illness.

Results of cultures of stool samples from 16 crew members were negative for Salmonella, Shigella, Campylobacter, Yersinia, and Vibrio. Three samples of leftover raw barracuda and red snapper that were caught simultaneously with the barracuda that was eaten were recovered from cold storage and then tested for ciguatoxin using an experimental membrane immunobead assay at the Department of Pathology, University of Hawaii. The samples from both fish tested positive for ciguatoxin.

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Editorial Note:

Ciguatera poisoning occurs throughout the Caribbean and tropical Pacific regions, where outbreaks have been reported among both residents and tourists. From 1983 through 1992 in the United States, 129 outbreaks of ciguatera poisoning involving 508 persons were reported to CDC; no ciguatera-related deaths were reported (1,2). Most outbreaks were reported from Hawaii (111) and Florida (10), although outbreaks and sporadic cases in California (two), Vermont (one), New York (one), and Illinois (one) also have been associated with consumption of fish imported from tropical waters (3,4). The outbreak described in this report was recognized in an area not typically associated with ciguatera intoxication and underscores that ciguatera poisoning can occur among travelers returning from areas where ciguatera is endemic or among persons consuming fish imported from those areas.

Ciguatera toxins are produced by dinoflagellates, which herbivorous fish consume. These fish are then eaten by large, predatory reef fish (e.g., barracuda, grouper, and amberjacks), which appear to be unharmed by the toxin; because the toxins are lipid-soluble, they accumulate through the food chain. The toxin may be most concentrated in the head, viscera, and roe. Ciguatoxin-containing fish may be highly localized; islands may have some reefs where the fish are inedible because of the

toxin and other reefs where the fish are unaffected. No deep-sea fish (e.g., tuna, dolphin, or wahoo) have been found to carry ciguatoxin.

As in this outbreak, ciguatera fish poisoning is diagnosed by the characteristic combination of acute gastrointestinal symptoms (developing within 3-6 hours after ingestion of contaminated fish; watery diarrhea, nausea, and abdominal pain occur and typically lasting approximately 12 hours) and neurologic symptoms (circumoral and extremity paresthesia, severe pruritus, and hot-cold temperature reversal) in persons who eat large, predatory reef fish. Neurologic symptoms may be worsened by alcohol consumption, exercise, sexual intercourse, or changes in dietary behavior, such as dieting or high-protein meals (5; R.W. Dickey, Ph.D., Center for Food Safety and Applied Nutrition, Food and Drug Administration, personal communication, 1998). Occasionally, hypotension, respiratory depression, and coma develop in patients. Mean duration of acute illness is typically 8.5 days, although neurologic symptoms may last for months (6). Because there is no approved human assay for ciguatoxin, the diagnosis is based on clinical findings and by the detection of toxin in samples of fish. No known antidote for ciguatoxin poisoning has been proven, and treatment is primarily for relief of symptoms. Intravenous mannitol may be effective early in the course of illness, but the results of a randomized, placebo-controlled trial of mannitol therapy have not been reported (7-9).

Ciguatoxins are odorless, colorless, tasteless, and unaffected by either cooking or freezing; therefore, persons living in or traveling to areas where ciguatera toxin is endemic should follow these general precautions: 1) avoid consuming large, predatory reef fish, especially barracuda; 2) avoid eating the head, viscera, or roe of any reef fish; and 3) avoid eating fish caught at sites with known ciguatera toxins. Persons traveling to areas where ciguatera is endemic should contact local health officials for more specific recommendations pertaining to that area. Fishermen should avoid known ciguatera-contaminated areas, and vendors should not sell fish caught in those areas.

Ill persons with suspected ciguatera poisoning should promptly seek medical care and save any uneaten portions of fish in a freezer. Suspected cases should be reported to state or local public health officials to assist with the investigation and control of a possible outbreak. Additional information is available about ciguatoxin testing of implicated fish from the Gulf Coast Seafood Laboratory of the Food and Drug Administration (FDA) in Dauphin Island, Alabama, telephone (334) 694-4480, or the

University of Hawaii, Honolulu, telephone (808) 956-8682. For general information about seafood safety, call FDA's Seafood Hotline, telephone (800) 332-4010.

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MMWR47 (50):1091-1093, 1998

バッファローフィッシュの喫食により発生したハフ病
米国，1997 年

ハフ病は、ある種の魚の喫食後に起こる原因未解明の横紋筋の変性を伴う症候群で、未確定の魚毒が関与しているとされる。この横紋筋変性は、筋肉細胞成分の血行放出を招く骨格筋の障害によって引き起こされる臨床的な症候群である。米国では、1997 年バッファローフィッシュ（Ictiobus cyprinellus：コイ科の淡水魚で、ミシシッピー川またはその支流の川底に棲息）の喫食により計 6 例のハフ病の患者発生が確認された（カリフォルニア州 4 例、ミズーリ州 2 例）。本報告はこれらの症例を調査した結果のまとめである。

症例 1、2 は、1997 年 3 月 8 日、バッファローフィッシュのフライを喫食したカリフォルニア州ロサンゼルス在住のウクライナ人姉妹（70、73 才）、症例 3 は、3 月 9 日に同じくバッファローフィッシュのフライを喫食したロサンゼルス在住のウクライナ人夫婦（共に 35 才）の夫、症例 4、5 は、6 月 8 日にバッファローフィッシュ及びコイのすり身の入った料理を喫食したミズーリ州セントルイス在住のウクライナ人夫婦（66 才、58 才）、症例 6 は、8 月 8 日にバッファローフィッシュのフライを喫食した米国生まれの 87 才の男性が、それぞれ喫食後 6～21 時間後に筋肉痛・硬直などを伴って発症（死亡者なし）したもので、調査の結果、症例 1～3 及び 6 の患者が喫食した魚は、ルイジアナ州内の河川で 25 名の漁師により捕獲された魚を、ルイジアナにある同一の卸売業者が販売したもの、症例 4、5 の患者が喫食した魚は、ミズーリ州セントルイスの半径 100 マイル以内の河川で捕獲されたものであることが確認された。米国食品医薬品局（FDA）では、現在回収された魚の毒素について確認及び同定作業中であるが、本毒素はその発生状況からみて耐熱性の物質で、特に危険性を増大させる調理法はないと思われる。

December 25, 1998 / 47(50); 1091-3

Haff Disease Associated with Eating Buffalo Fish -- United States, 1997

Haff disease is a syndrome of unexplained rhabdomyolysis following consumption of certain types of fish; it is caused by an unidentified toxin. Rhabdomyolysis is a clinical syndrome caused by injury to skeletal muscle that results in release of muscle cell contents into the circulation (1). In 1997, six cases of Haff disease were identified in the United States (four in California and two in Missouri) among persons who ate buffalo fish (Ictiobus cyprinellus), a bottom-feeding species found mostly in the Mississippi River or its tributaries. This report summarizes the investigation of these cases.

Los Angeles County, California

Patients 1 and 2. On March 8, two Ukrainian sisters (patients 1 and 2), aged 70 and 73 years, respectively, and the husband of patient 2 (aged 75 years) ate fried buffalo fish. Eight hours after the meal, patient 1 experienced neck pain followed by stiffness in her arms. On arrival, emergency medical technicians noted both women were rigid, unable to move, and extremely sensitive even to light touch. On evaluation at a local hospital, the serum creatine kinase (CK) of patients 1 and 2 were 25,000 IU/L and 9454 IU/L, respectively (normal: less than 120 IU/L); the muscle/brain (MB)-fraction at the peak of the CK was 2.7% and 0.5% (normal: less than 5%). Patient 1 was treated with intravenous hydration and bicarbonate. Patient 2, who had a history of angina pectoris, also complained of chest pain. During hospitalization, an angiogram revealed occlusion of a coronary artery requiring dilatation. She was treated with nitrates and coumadin. The man did not become ill. Both sisters recovered. Main sequelae were newly diagnosed hypertension (patient 1) and diminished muscular strength (patient 2).

Patient 3. On March 9, a husband and wife (both aged 33 years) from Ukraine ate fried buffalo fish purchased from the same market where patients 1 and 2 purchased their fish. Eight hours after the meal, the husband experienced left-sided chest pain that radiated to his left arm and increased with deep inspiration. He was admitted to the

same hospital as patients 1 and 2. A comprehensive cardiovascular examination did not reveal abnormalities except an elevated CK (4140 IU/L) with a CK-MB of 1.4% at the peak of the CK. He reported no history of angina pectoris and had not smoked for 2 years. He did not receive any special treatment. Following discharge, the patient has reported occasional chest pain that he had not noticed before this episode. His wife did not become ill.

St. Louis, Missouri

Patients 4 and 5. On June 8, a Ukrainian husband and wife (aged 66 and 58 years, respectively) ate a dish consisting of ground buffalo fish and carp. One hour later, the wife vomited. Six hours after the meal, they developed generalized body aches and muscle stiffness. On evaluation at a local hospital, the CK of patients 4 and 5 exceeded 17,700 IU/L, and the CK-MB were 4.8% and 4.5%, respectively. The husband had severe pain on inspiration, resulting in respiratory insufficiency requiring assisted ventilation. His wife was treated with intravenous fluids and mannitol. Following the acute episode, the husband complained of more frequent headaches, and his wife continued to experience tearing eyes, easy fatigability, and pruritus after eating seafood.

Bakersfield, California

Patient 6. On August 8, an 87-year-old U.S.-born man vomited 30 minutes after eating one third of a fried buffalo fish. Twenty-one hours later, he awoke with extreme stiffness and generalized muscle tenderness. At a local emergency department, his CK was 2226 IU/L with a CK-MB of 2.1%. The patient was treated with intravenous fluids and analgesics. Following this episode, the patient suffered 6 months of muscle weakness, primarily in his legs.

Follow-Up Investigations

The origin of the buffalo fish eaten by patients 1, 2, 3, and 6 was traced to the same wholesaler in Louisiana who receives fish from approximately 25 fishermen who fish rivers in Louisiana. The fish for patients 4 and 5 were caught within a 100-mile radius of St. Louis, Missouri. The Food and Drug Administration is attempting to identify a toxin from recovered fish samples. The case histories suggest that the toxin is heat stable; no particular mode of preparation seems to increase risk for disease.

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Editorial Note:

Editorial Note: During the 1920s, the name "Haff disease" was given to an illness characterized by severe muscle pain and stiffness that affected approximately 1000 persons living along the Koenigsberg Haff, a brackish inlet of the Baltic Sea (1). Subsequent similar outbreaks were identified in Sweden and the former Soviet Union (2–4). Although the etiology was not determined, epidemiologic investigations linked illness to ingestion of fish, especially burbot.

The first reported case of Haff disease in the United States occurred in Texas in 1984 (M. Tormey, Los Angeles Department of Health Services, personal communication, 1997); five additional cases were reported in California during 1984–1986. All U.S. cases have been associated with eating buffalo fish.

Haff disease typically presents as a paroxysm of rhabdomyolysis, with accompanying muscle tenderness, rigidity, and dark brown urine. However, as in patient 3, milder presentations also occur. Although the median incubation period for the patients in this report was 8 hours (range: 6–21 hours), symptoms generally appear approximately 18 hours after eating fish.

Laboratory features of Haff disease include a markedly elevated CK level with an MB fraction of less than 5%. Levels of other muscle enzymes (e.g., lactate dehydrogenase, glutamate oxalate transaminase, and glutamate pyruvate transaminase) also are elevated. Myoglobinuria is often mistaken for gross hematuria (5). Diagnosis is based on a compatible clinical history.

Treatment is supportive and consists of administering large volumes of fluid early in the course of illness to prevent myoglobin toxicity to the renal tubules (5). Possible complications include electrolyte disturbances, renal failure, and disseminated intra-vascular coagulation. Symptoms usually resolve within 2-3 days. Historically, the case-fatality rate is approximately 1% (1).

Clinicians and public health practitioners are encountering an increasing variety of foodborne illnesses, in part because of a diversification of food preparation and eating habits. International travelers, members of ethnic groups with unique cuisines, and consumers of both imported and domestic specialty food items may be at risk for foodborne illnesses that are rare or have not been reported previously in the United States. Clinicians should be aware of food exposures that pose a risk to their patients and routinely obtain food histories, even from those patients whose illness may not appear to be food-related.

Physicians who identify or suspect cases of Haff disease, based on the clinical presentation, laboratory parameters, and food history, should report them to public health authorities for initiation of traceback and recall of implicated food items. State health departments are requested to report to the Foodborne and Diarrheal Diseases Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC, telephone (404) 639-2206.

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MMWR52 (18): 413- 416

汚染された牛挽肉によるニコチン中毒
ミシガン州，2003 年

2002 年 12 月 31 日または 2003 年 1 月 1 日にミシガン州のあるスーパーマーケットで販売された牛挽肉を食べた直後に、口内焼灼感、悪心、嘔吐、めまい等が発現したとの苦情が 4 世帯 18 名から寄せられ、スーパーはその挽肉（約 1,700 ポンド）を回収する旨をミシガン州農務局食物・乳製品部（Department of Agriculture (MDA), Food and Diary Division）及び米国農務省（USDA）に届け出した。

リコール製品は、州外の加工業者から購入（USDA 食品安全検査局（FSIS）検疫済）した挽肉をさらにそのスーパーで挽いたものであった。MDA 食物・乳製品部は、州内の保健に関わる政府諸機関に対し、リコールについての告示を定期的に行った。1 月 3 日に販売期限が 1 月 1 及び 2 日の挽肉を回収し始め、8 日に販売期限が 1 月 3 日の肉までを回収すると消費者に発表した。約 120 名が該当する挽肉を返品し、また本人や家族が発病したとの報告が 36 名からあった。

1 月 10 日、スーパーが依頼した民間の研究所において、挽肉のサンプルから 300 mg/kg のニコチン（致死量 0.5～1.0 mg/kg または 30～60mg）が検出されたとスーパーから MDA 及び USDA に報告があった。検出されたニコチンは高濃度であり、さらに他のチェーン店や加工工場から発病や汚染の報告はないことから、汚染源はスーパーの店内であると考えられたため、ニコチン含有殺虫剤による意図的な汚染の可能性が浮上し、連邦捜査局（FBI）も捜査に乗り出した。

1 月 23 日、地元の保健局は各病院の救診科に警報を発し、スーパー周辺地域住民に対する適切な医療措置の検討を行った。地元保健局はまた、疫学的調査を実施し、中毒例は 12 月 31 日または 1 月 1 日に購入した挽肉を食べた後、2 時間以内に口唇や喉の焼灼感、めまい、悪心、嘔吐、腹痛、下痢、発汗、視力障害、頭痛、しびれ感、疲労感、不安感、不眠、頻呼吸症、呼吸困難、頻拍、頻脈性不整脈等から 1 つ以上の症状を呈した症例と定義した。本人、家族または友人が挽肉を食べて発病した 148 名を対象とした聞き取り調査では、92

名 （平均 31(調査対象 1－76)歳、92 名中 46 名（50%）が女性）がこの定義に合致し、65%の症例がスーパーのある町の住民であった。

2 月 12 日、この店の従業員が 200 ポンドの肉を殺虫剤（ブラックリーフ 40（Black Leaf 40））で汚染した容疑で逮捕、起訴された。

Editorial Note《編集注記》

ブラックリーフ 40 は、40%のニコチンを含有しており、その毒性から 1992 年に米国環境保護庁（EPA）より製品登録を取り消されている。本事例は、政府機関の動きという観点からみれば、地元及び州の公衆衛生局、州農務局、連邦農務省、連邦捜査局という 5 つの政府機関を巻き込んだ大事件となった。

May 9, 2003 / 52(18); 413-416

Nicotine Poisoning After Ingestion of Contaminated Ground Beef --- Michigan, 2003

On January 3, 2003, the Michigan Department of Agriculture's (MDA) Food and Dairy Division and the U.S. Department of Agriculture (USDA) were notified by a supermarket of a planned recall of approximately 1,700 pounds of ground beef because of customer complaints of illness after eating the product. On January 10, the supermarket notified MDA that their laboratory had determined that the contaminant in the ground beef returned by customers with reported illness was nicotine. This report summarizes the investigation of these cases, which identified approximately 100 affected persons, and discusses actions taken to prevent additional illness, including the arrest of a person charged with deliberately poisoning the ground beef at the supermarket.

The recall was prompted by complaints from four families comprising 18 persons who became ill immediately after eating product sold on December 31 or January 1. Reported symptoms included burning of the mouth, nausea, vomiting, and dizziness. One person reported having been seen in the emergency department (ED) and treated for atrial fibrillation. The recalled product had been ground in the store using ground beef purchased from an out-of-state processor inspected by USDA, Food Safety Inspection Service. MDA made routine notifications about the recall to local and state health departments. The product recall was issued on January 3 for beef with a sell-by date of January 1 and January 2, followed by a press release on January 8, which expanded the recall to beef with a sell-by date of January 3. After the initial recall notices, approximately 36 persons reported to the supermarket that they or their families had experienced illness after eating the product, and approximately 120 persons returned recalled product.

Company officials submitted samples of ground beef provided by the ill families to a private laboratory, where product testing for foodborne pathogens was negative. Additional testing for chemical contamination was conducted at a large regional

medical center. On January 10, company officials notified MDA and USDA that nicotine had been presumptively identified in the ground beef samples tested by the second laboratory, which reported an assay result 1 week later of approximately 300 mg/kg nicotine in the submitted samples. The high nicotine concentrations found in the tested meat products prompted concerns of intentional contamination with a pesticide, which sometimes contain nicotine as an additive. USDA and the Federal Bureau of Investigation joined the investigation because interstate commerce could have been involved and intentional contamination was suspected. Because a legal investigation was initiated, federal authorities requested that information be released to the public only as necessary to avoid compromising any future criminal case. On January 17, the supermarket issued another press release and recall notice stating the implicated product contained an unspecified, nonbacterial contaminant that could not be made safe by cooking.

Contamination of the product was believed to have occurred at a single store rather than the meat processing plant. The product was distributed directly from the plant to many other stores, including other stores in the supermarket chain; neither the processing plant nor any other store in the supermarket chain received complaints of illness. No nicotine-containing pesticides were reportedly used or sold in the store where the recalled product was sold.

On January 23, the local health department alerted hospital EDs and selected medical practices serving the area where the store was located. On January 24, after receiving confirmatory test results, the company issued another press release naming nicotine as the contaminant. This announcement was published and broadcast by local media.

The local health department conducted an epidemiologic investigation, including interviews of persons reporting illness, to assess the consistency of the clinical presentation and to establish a case definition. A case was defined as one or more symptoms (i.e., burning sensation to lips, mouth or throat, dizziness, nausea, vomiting, abdominal pain, diarrhea, sweating, blurred vision, headache, body numbness, unusual fatigue or anxiety, insomnia, tachypnea or dyspnea, and tachycardia or tachyarrythmias) in persons who ate ground beef product purchased from the supermarket on either December 31, 2002, or January 1, 2003, with symptom onset occurring within 2 hours of eating the product.

A total of 148 interviews were conducted with persons who reported they had experienced illness after eating the product and of family members and friends who also might have eaten the contaminated meat. Of those interviewed, 92 persons had illness consistent with the case definition. Patients had a median age of 31 years (range: 1--76 years), and 46 (50%) were female; 65% of the patients lived in the town where the implicated store was located. The majority of illness occurred during the time that the contaminated product was sold. Cases were identified as late as 49 days after the last date of potential sale, indicating that some persons froze and then ate the contaminated product after the first recall was issued. Of the 92 patients, four (3%) sought medical treatment, including two who reported to their personal physicians with complaints of vomiting and stomach pains and two who were evaluated in EDs. The two who were treated in the EDs included a man aged 39 years with atrial fibrillation and a woman aged 31 years who had nausea, vomiting, and complaint of rectal bleeding. Information is being collected on an additional 16 persons to assess whether their illnesses are consistent with the case definition, including a pregnant woman aged 24 years who was hospitalized for 1 day with episodic vomiting.

On February 12, a grand jury returned an indictment for arrest of a person accused of poisoning 200 pounds of meat at the supermarket with an insecticide called Black Leaf 40, which has a main ingredient of nicotine. The person was an employee of the supermarket at the time of the contamination.

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Editorial Note:

Deliberate contamination of food during its production and preparation has been reported infrequently (*1, 2*). Unintentional contamination of food by chemicals occurs sporadically, including reports of contamination by pesticides (*3, 4*). Unintentional poisoning by nicotine has been reported, usually among children who eat cigarettes (*5*), in suicide attempts (*6*), or among tobacco workers who experience "green tobacco sickness" (*7*). One homicide by nicotine was reported to have occurred in Eastern Europe (*8*).

Acute nicotine toxicity is associated with overstimulation of nicotinic receptors. Burning in the throat with nausea and vomiting occurs quickly after ingestion. More toxic manifestations include cardiac tachyarrythmias, seizures, and hypertensive crisis. The lethal dose of nicotine in adults is from 0.5 to 1.0 mg/kg of body weight or a total dose of 30--60 mg. Toxic symptoms might been seen at doses as low as 2--5 mg of nicotine; however, persons might have widely different levels of tolerance to the toxic effects of nicotine. Small children might develop symptoms after exposure to as little as 1 mg of nicotine. Nicotine is used in a limited number of pesticides because of its toxic properties; nine nicotine-containing pesticides are registered for use in Michigan, and none of the product labels list nicotine at more than 14%. Black Leaf 40 contained 40% nicotine, and the EPA canceled its product registration in 1992 because of its toxicity.

This investigation involved the private sector (i.e., the food retailer) and five government agencies, including local and state public health departments, the state agriculture department, and two federal agencies. Public health officials undertook an epidemiologic investigation that involved contacting affected persons and providing information to the public and clinicians about the health threat. It also was necessary to conduct a legal investigation in a rapid and relatively closed manner. Frequent contacts among the parties allowed for negotiation and consensus around most issues.

This incident underscores the importance of ensuring the safety and security of food supplies. Vigilance and heightened awareness for human poisonings caused by hazardous levels of chemical in the food supply are essential. Clinicians should immediately report clusters of poisonings to public health officials, especially when presenting symptoms are unusual. Public health response capabilities addressing hazardous chemicals in food and other media need to be strengthened. Multiple agency coordination and cooperation between health, agriculture, and law enforcement officials at the local, state, and federal levels are critical for the detection and response to similar events, whether they are intentional or unintentional (*9*).

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γ－ブチロラクトンの摂取による不慮の事故
ミネソタ州、ニューメキシコ州、テキサス州，1998～1999 年

γ－ブチロラクトン（GBL）は、催眠、成長ホルモン促進、強精、抑鬱、老化防止、運動競技力向上といった目的に市販されている。GBL は、体内に摂取されると、米国食品医薬品局（FDA）から臨床試験目的以外には使用が禁止されているγ－ブチルヒドロキシ酪酸（GHB）に代謝される。これまでに知られている GHB による中毒症状としては、徐脈、低体温、中枢神経系の抑制、運動不良などが代表的なものである。本報告では、1998 年 10～12 月にかけてミネソタ州の 2 か所の救急外来（ED）から報告された「Revivarant」（液量オンス当たり GBLI. 82g 含有と表示）などの製品によって発生した GBL 中毒 7 症例並びにこの他 1998 年 10 月～1999 年 1 月に、ニューメキシコ及びテキサス州の中毒センターに報告された 34 例の中毒症例の概要について紹介した。

FDA は、1999 年 1 月 21 日、GBL 含有製品を製造した会社に対し、製品の回収を求めると同時に、消費者に対してこれらの製品を摂取しないよう新聞等を通じて警告した。

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Adverse Events Associated with Ingestion of Gamma-Butyrolactone Minnesota, New Mexico, and Texas, 1998-1999

Products containing gamma-butyrolactone (GBL) * are marketed for many claimed purposes, including to induce sleep, release growth hormone, enhance sexual activity and athletic performance, relieve depression, and prolong life. GBL is converted by the body into gamma-hydroxybutyrate (GHB), a drug banned outside of clinical trials approved by the Food and Drug Administration (FDA). Recognized manifestations of GHB toxicity include bradycardia, hypothermia, central nervous system depression, and uncontrolled movements (1). This report describes seven cases of GBL toxicity involving the product "Revivarant," which is labeled as containing 1.82 g of GBL per fluid ounce, reported from two hospital emergency departments (EDs) in Minnesota during October-December 1998 and summarizes an additional 34 cases of GBL toxicity reported to poison centers in New Mexico and Texas during October 1998-January 1999.

Minnesota

Patient 1. On November 26, 1998, a 24-year-old man vomited and had seizures shortly after drinking 3-4 oz of Revivarant. His behavior became unusual, and he alternated between extreme agitation and profound calm. Paramedics noted that his skin was warm, flushed, and profusely diaphoretic, and he had bradycardia (pulse as low as 45 beats per minute {bpm}). Systolic blood pressure was 110 mm Hg. Transcutaneous oxygen saturations (SpO2) were 96% on room air, and blood glucose by fingerstick was 90 mg/dL. During transport to an ED, he had periods of combativeness lasting 30 to 60 seconds followed by coma lasting 1-3 minutes. In the ED, he was unconscious with spontaneous eye opening, a positive withdrawal reflex, and no speech (Glasgow Coma Scale of 7); rectal temperature was 94.8 F (34.9 C). A urine toxicology screen and blood ethanol test were negative. He was intubated and admitted to the intensive-care unit (ICU) with a diagnosis of toxic encephalopathy. During the next 7 hours, his heart rate increased from 42 to 116 bpm and he became

more alert. He had no recollection of events except for having ingested Revivarant. He was discharged with normal mental status.

Patient 2. On December 12, 1998, a 46-year-old woman had a seizure and lost consciousness after drinking approximately 2.7 oz of Revivarant in conjunction with ethanol. Paramedics found her unconscious and in severe respiratory depression with a pulse of 54 bpm. Oxygen was administered by mask; she had an SpO2 of 87%. On arrival in the ED, physical examination identified sinus bradycardia (54 bpm); temperature of 96.1 F (35.6 C); and miotic pupils. A serum ethanol level was 0.11%. She was admitted to the ICU, mechanically ventilated through the night, and awoke in improved condition the next morning; she was discharged with no memory of the events.

Patient 3. On November 8, 1998, a 31-year-old man drank approximately 1 oz of Revivarant, four beers, and a large sip of wine. Shortly thereafter, he gradually lost consciousness and subsequently fell. He regained consciousness but had involuntary muscle movements and episodes of confusion. Paramedics noted that he was ambulatory but confused. On physical examination in the ED, he was agitated, anxious, and unable to recall the preceding events. His shoulders twitched, and he had a small abrasion below his left eye. He had a pulse of 64 bpm and hypothermia (oral temperature of 95.2 F {35.1 C}). Breath ethanol level was 0.08%. He denied previous GBL use or illicit drug use. He recovered completely and was discharged.

Patients 4 and 5. On October 31, 1998, a 24-year-old man (patient 4) and a 26-year-old man (patient 5) each drank 10-13 oz of Revivarant while drinking alcohol at a bar. On leaving the bar, witnesses observed them fall and become unresponsive. On arrival at the ED, they alternated between somnolence and confusion. When awake, neither patient could consistently follow commands. Patient 4 had fecal incontinence. Vital signs for both patients were within normal limits. Breath ethanol levels were 0.09% (patient 4) and 0.15% (patient 5). Neither patient had a history of using medications or illicit drugs. After 2 hours of observation, the patients recovered but were unable to recall most of the evening's events.

Patients 6 and 7. On December 12, 1998, a 19-year-old woman (patient 6) and a 22-year-old woman (patient 7) were brought to an ED by friends because of vomiting and decreased levels of consciousness. These symptoms followed ingestion of Revivarant (2 oz by patient 6 and an unknown amount by patient 7). Patient 6 had

drank one beer; patient 7 had had no ethanol. Vital signs were normal except for respiratory depression. On physical examination, patient 6 was lethargic and disoriented. Patient 7 exhibited intermittent periods of extreme agitation, necessitating chemical treatment and physical restraint, punctuated by moments of calm during which her attention focused on minor details. Mental changes for both patients resolved, and they were discharged approximately 4 hours after arrival.

New Mexico

From October 3, 1998, through January 29, 1999, the New Mexico Poison Center identified 14 cases of adverse events resulting in an ED visit among persons who had ingested GBL-containing products. Ten (71%) of the cases were reported in January. Patients' ages ranged from 14 to 36 years; nine were male. Products used included "Firewater" (11 cases), "Blue Nitro Vitality" (two), and "RenewTrient" (one). The approximate amount ingested ranged from 1 to 10 oz (mean: 3 oz). Five (36%) persons also had ingested ethanol and/or other drugs. Most of the patients were discharged from the ED within 13 hours of arrival; three were hospitalized. The most common symptoms and signs were nausea/vomiting (10 [71%]), obtundation (nine [64%]), bradycardia (seven [50%]), prolonged unconsciousness (six [43%]), syncope (six [43%]), seizures (four [29%]), confusion (four [29%]), combativeness (four [29%]), respiratory depression (three [21%]), amnesia (two [14%]), and euphoria (two [14%]). One person had cardiac arrest, one had respiratory arrest, and one had a motor-vehicle crash associated with the effects resulting from use of a GBL-containing product. No deaths were reported.

Texas

From October 2, 1998, through January 24, 1999, Texas poison-control centers identified 20 adverse events resulting in ED visits among persons who had ingested GBL-containing products. Twelve (60%) of the cases were reported in January. Patients' ages ranged from 11 to 41 years; 13 were male. Products known to have been used included "RenewTrient" (six cases), "Revivarant" (four), "Revivarant-G" (two), and "Blue Nitro Vitality" (two). Ten persons also ingested ethanol and/or other drugs. Ten patients were admitted to the hospital from the ED. The most common symptoms and signs were obtundation (13 [65%]), prolonged unconsciousness (nine [45%]), respiratory depression (nine [45%]), anxiety/nervousness (seven [35%]), nausea/vomiting (six [30%]), confusion (six [30%]), tremors/twitching (four [20%]),

tachycardia (three [15%]), and combativeness (three [15%]). One person had respiratory arrest; no deaths were reported.

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Editorial Note:

GBL is metabolized to GHB in the body, but because of better absorption GBL has greater bioavailability than GHB on an equimolar basis (2). Clinical effects of GHB appear to be dose-related and include reports of vomiting, hypotonia, tremors, seizures, aggression, impairment of judgment, coma, respiratory depression, hypothermia, and bradycardia (1). GHB mixed with ethanol acts synergistically to produce central nervous system and respiratory depression (3). Symptoms usually resolve with supportive care within 2-96 hours (4). Death occurring when GHB was the sole intoxicant also has been reported (5). Toxic effects of GBL would be expected to be similar or identical to those of GHB, but previous clinical experience is limited (6, 7). There is no antidote for GHB; treatment consists of supportive therapy until symptoms of toxicity subside. A withdrawal syndrome, which can include insomnia, tremor, and anxiety, has been reported following discontinuance of GHB in chronic, high-dose users (8).

GBL is an industrial and household solvent of acrylate polymers, and unintentional poisonings have been reported (6, 9). It also is marketed as a dietary supplement at health food stores and on the World-Wide Web under several trade names. Although

labeled as dietary supplements, GBL-containing products are illegally marketed, unapproved new drugs that have been involved in at least 55 reports of adverse events, including one death (10). On January 21, 1999, FDA asked manufacturers to recall their GBL-containing products and warned consumers through press releases to avoid taking these products (10). Public education efforts should inform consumers that FDA review procedures for drugs are different than those used for dietary supplements. Consumers should be alert to the potential dangers of these products and understand that terms such as "natural" do not necessarily imply safety. Physicians should counsel patients about these products and be prepared to recognize and treat the toxic reactions that some might produce. Chronic GBL users should be monitored for withdrawal symptoms when discontinuing use of the product. Depending on the severity of the withdrawal symptoms, medical intervention may be required. Physicians are encouraged to report serious adverse events associated with these products to FDA's MedWatch program, telephone (800) 332-1088.

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Also is known as dihydro-2(3H)-furanone; 4-butanolide; 2(3H)-furanone, dihydro; tetrahydro-2-furanone; and butyrolactone gamma.

MMWR52 (26): 613-615

複数の州における非低温殺菌牛乳の摂取によるサルモネラ（ネズミチフス菌：*Salmonella* Typhimurium）感染症の発生 イリノイ州、インディアナ州、オハイオ州、テネシー州，2002～2003 年

2002 年 12 月 10 日、オハイオ州クラーク郡統合保健区（Clark County Combined Health District）と 同州保健局（the Ohio Department of Health：ODH）は、ネズミチフス菌（*Salmonella Enterica* serotype Typhimurium）に感染した小児 2 例の報告を受けた。感染源の調査により、2002 年 11 月 27 日～12 月 13 日に、ある酪農場のレストランで購入した非低温殺菌牛乳との関連が明らかになった。2002 年 11 月 30 日から 2003 年 2 月 18 日の期間に、ODH の研究所は 94 のネズミチフス菌の臨床分離株を受理し、パルス・フィールド・ゲル電気泳動（PFGE）により検査した結果、60 株が区別できないパターンであった。加えて、イリノイ州、インディアナ州、テネシー州からのパターンがオハイオ州でのパターンと一致した。

その後、その酪農場の従業員や客を対象として、ODH とクラーク郡統合保健区によって調査が行われ、4 州（イリノイ、インディアナ、オハイオ、テネシー、いずれも隣接している）の計 62 例（男 28、女 34、年齢中央値 18（1～70）歳）のネズミチフス菌感染症患者を確認した。62 例中 54 例（87.1%）は下痢、痙攣、発熱、悪寒等の症状を有し、発症日は 2002 年 11 月 30 日～2003 年 1 月 14 日であった。感染源確認のため、症例対照研究を行った所、危険因子の単変量解析で生乳の摂取のみが疾患と有意に相関した。また検査した食品 32 サンプル中、スキムミルクやバター等 5 サンプルがネズミチフス菌陽性であった。酪農場の動物や環境サンプルはネズミチフス菌陰性であったが、農場従業員 4 例で無症候性ネズミチフス菌感染が明らかになった。

2002 年 12 月 13 日、クラーク郡統合保健区を含む地元の保健政府機関からの命令により、この酪農場は全生乳製品の販売を中止した。

2003 年 1 月 13 日、オハイオ州農業局（the Ohio Department of Agriculture：ODA）は酪農場に対して全ての生乳製品販売の永久中止と農場従業員の衛生状態や衛生設備等の改善を勧告した。米国では 27 州が未だに生乳の販売を許可している。生乳の危険性について

の消費者教育と現行の政策に対する再検討が必要である。

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Multistate Outbreak of *Salmonella* Serotype Typhimurium Infections Associated with Drinking Unpasteurized Milk --- Illinois, Indiana, Ohio, and Tennessee, 2002--2003

On December 10, 2002, the Clark County Combined Health District and the Ohio Department of Health (ODH) were notified of two hospitalized children infected with *Salmonella Enterica* serotype Typhimurium. Initial investigation implicated consumption of raw, unpasteurized milk purchased at a local combination dairy-restaurant (dairy) during November 27--December 13, 2002, as the cause. This report summarizes the subsequent investigation. Because 27 states still allow the sale of raw milk, and organizations continue their efforts to allow marketing and sale of raw milk to the public directly from the farm (*1, 2*), consumer education about the hazards of raw milk and a careful review of existing policies are needed.

The dairy comprised a working dairy farm, restaurant, snack bar, and petting zoo with goats, cows, calves, lambs, and pigs. At the time of the epidemiologic investigation in December 2002, the workforce comprised 211 workers, including 16 members of the owner family. In 2002, the dairy was the only place in Ohio that sold raw milk in jugs and served raw milk and milk shakes made with raw milk legally to customers. In 2001, approximately 1,350,000 customers visited the dairy.

During November 30, 2002--February 18, 2003, ODH laboratory received 94 *S.* Typhimurium clinical isolates for pulsed-field gel electrophoresis (PFGE) testing. Of these, 60 had an indistinguishable pattern. In addition, patterns from Illinois, Indiana, and Tennessee matched the Ohio pattern.

A case of *S.* Typhimurium was defined as PFGE--matched *S.* Typhimurium isolated during November 30, 2002--February 18, 2003, from clinical samples from a person

with an epidemiologic link to the dairy. Case finding was conducted by reviewing laboratory culture results from hospital, private, and ODH laboratories, comparing PFGE patterns of *S.* Typhimurium isolates with background isolates statewide and nationwide, screening dairy workers, interviewing meal companions, and alerting public health officials of the outbreak nationwide by using CDC's *Epidemic Information Exchange* (*Epi-X*).

A total of 62 persons had illness consistent with the case definition, including 40 customers, six household contacts, and 16 (7.6%) of 211 dairy workers; patients were from four states (Illinois, Indiana, Ohio, and Tennessee); the median age was 18 years (range: 1--70 years), and 34 (54.8%) were females. Of the 62 patients, 54 (87.1%) reported signs and symptoms of illness, including diarrhea (52 [96.3%]), cramps (41 [75.9%]), fever (37 [68.5%]), chills (29 [53.7%]), body aches (29 [53.7%]), bloody diarrhea (27 [50.0%]), nausea (25 [46.3%]), vomiting (24 [44.4%]), and headache (21 [38.9%]). A total of 50 (80.6%) exhibited more than one symptom. Disease onset occurred during November 30, 2002--January 14, 2003 (Figure).

A case-control study was conducted to verify the initial findings implicating raw milk and to identify other potential sources of infection. The 40 case-patients who were dairy customers were included in the study. Controls were a convenience sample of well meal companions of case-patients. Because of numerous potential exposures to *S.* Typhimurium, dairy workers were excluded from the study; secondary infections among friends or households contacts of case-patients also were excluded. Food histories were obtained through telephone interviews by using a standard questionnaire. State and local investigators reviewed milking, bottling, and capping procedures and collected and tested samples from the food, stools of dairy cows, and the environment.

A total of 40 case-patients and 56 controls were eligible for the case-control study. The median age of case-patients was 8 years (range: 1--69 years); 24 (60.0%) were females. The median age of controls was 35 years (range: 1--74 years); 34 (60.7%) were females. In the univariate analysis of potential risk factors, only consumption of raw milk was associated significantly with illness. Among 39 case-patients and 55 controls for whom date of milk purchase was known, 37 (94.9%) and 16 (29.1%), respectively, consumed raw milk (odds ratio [OR] = 45.1; 95% confidence interval [CI] = 8.8--311.9). Consumption of other food items, visiting the petting zoo, and petting animals were not associated with illness.

Of the 32 food samples tested, five were positive for *S.* Typhimurium, including three raw skim milk samples, one sample of butter made from raw milk purchased by a customer, and one sample of cream. Skim milk samples were taken from milk either bought or bottled on November 29. The PFGE pattern for all five food isolates matched the outbreak pattern. The 31 animal stool samples collected from cows providing milk and the 23 environmental samples taken from dairy equipment and storage sites were negative for *S.* Typhimurium.

The review of the dairy operation and results of worker screening tests revealed that four barn workers had asymptomatic *S.* Typhimurium infection. Barn workers milked the cows, bottled the milk, and made ice cream.

On December 13, 2002, following an order from local health authorities, the dairy discontinued the sale of all raw milk products. On January 13, 2003, the Ohio Department of Agriculture (ODA) recommended that the sale of all dairy products made with raw milk, including bottled raw whole milk, skim milk, and cream, be discontinued permanently. Several sanitation improvements, primarily for the barn workers, also were recommended, including more frequent hand washing, replacement of the some of the equipment and utensils (e.g., mixing bowls), and enhanced general cleaning in the entire property.

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Editorial Note:

Each year in the United States, foodborne disease causes an estimated 76 million illnesses. Of these, an estimated 1.4 million are caused by *Salmonella*, resulting in approximately 16,000 hospitalizations and 580 deaths (*3*). Raw bulk tank milk can contain one or more species of pathogenic bacteria, including *Salmonella* spp. (*4, 5*). During 1972--2000, a total of 58 raw milk--associated outbreaks were reported to CDC, of which 17 (29%) were caused by *Salmonella* spp. (*1, 6*).

This report describes a large multistate outbreak of *S.* Typhimurium transmitted through consumption of raw milk and milk products. Although animal and environmental samples were negative for *S.* Typhimurium, four barn workers were infected with *S.* Typhimurium. In addition, all *S.* Typhimurium isolates from clinical specimens and foods had indistinguishable PFGE patterns. The source for contamination was not determined; however, the findings suggest that contamination of milk might have occurred during the milking, bottling, or capping process.

In 2002, intrastate sale of raw milk for human consumption was legal in 28 states, including Ohio (*1*). As of October 1997, Ohio law did not allow the sale of raw milk except for dairies that were engaged continuously in the business of selling or offering for sale raw milk directly to consumers before October 31, 1965 (*7*). The dairy in this outbreak had been in operation since 1958 and was the only place in Ohio selling raw milk legally. After ODA issued its recommendations, the dairy voluntarily relinquished its license for selling raw milk. As a result, no businesses now sell raw milk to the public legally in Ohio.

Molecular subtyping of *S.* Typhimurium isolates had an important role in identifying cases that were part of this outbreak and defining its extent (*8*). Typhimurium is one of the most common *Salmonella* serotypes isolated from persons in Ohio, and without the specificity of PFGE typing, identifying cases that were part of the outbreak would have been difficult.

Despite the known association of raw milk with disease-causing organisms, some consumers believe that raw milk is of better quality than pasteurized milk (*9*). In several states, producers circumvent regulations and provide raw milk to consumers by establishing cow-leasing programs in which farmers keep and milk cows owned by individuals (CDC, unpublished data, 2003). Consumer education about the hazards of raw milk consumption is needed. Retail milk regulations should be reviewed and strengthened, if needed, to minimize exposure of the public to the hazards of raw milk consumption.

Acknowledgments

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Edwards, Clermont County General Health District, Batavia; L Hines, Delaware General Health District, Delaware; M Fornadley, Franklin County Board of Health, Columbus; D Brannen, A Woodard, Greene County Combined Health District, Xenia; S Lester, Hamilton County General Health District, Cincinnati; A McCoppin, Highland County Health Dept, Hillsboro; C Alderman, Madison County Health District, London; J Close, J Reed, Combined General Health District of Montgomery, Dayton; E Meyer, Preble County General Health District, Eaton; R Hart, Sidney-Shelby County Health District, Sidney; V Johnson, Warren County Combined Health District, Lebanon; B Mitchell, L Holbert, Ohio Dept of Health.

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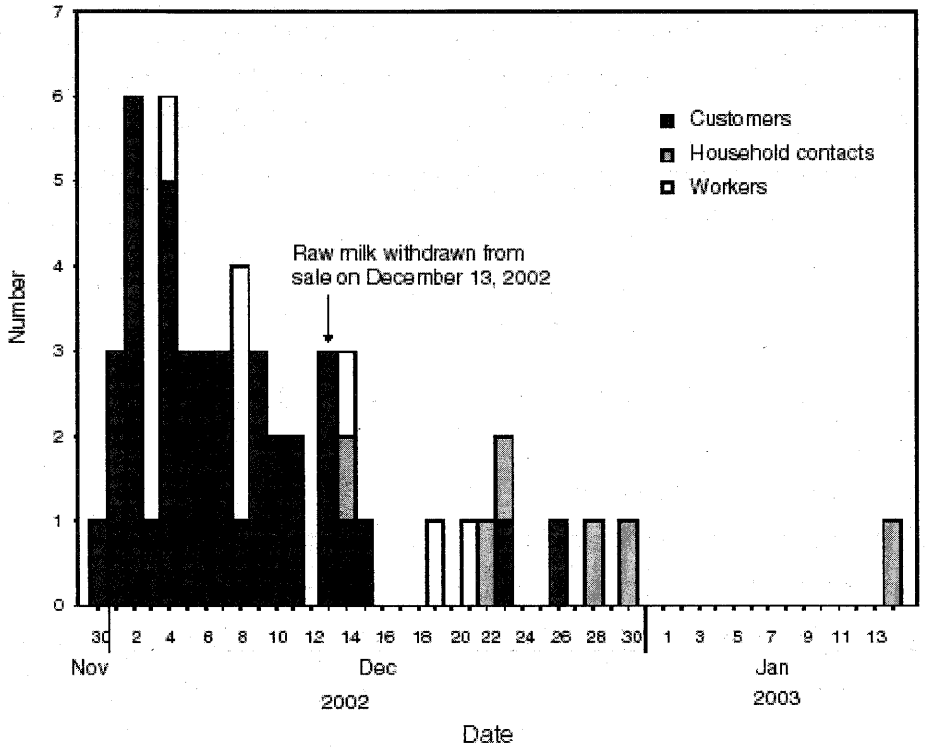
FIGURE. Number* of patients with symptomatic *Salmonella* serotype Typhimurium infections, by date of illness onset — Illinois, Indiana, Ohio, and Tennessee, November 30, 2002–January 14, 2003



* N = 53.

MMWR51 (51):1149- 1152

殻付卵によるサルモネラ（腸炎菌：*S.* Enteritidis）感染症の発生 米国，1999～2001 年

米国でのサルモネラ（腸炎菌、*Salmonella* Serotype Enteritidis：SE）感染症の流行は1980 年代に出現し、北東部及び中部大西洋岸地域にて増加した。1990 年代初期には北東部で減少傾向に転じたが太平洋岸地域に拡大した。米国疾病管理予防センター（CDC）に報告された発症例数は 1995 年にピークに達し（人口 10 万人あたり 3.8 人）、1999 年には 1.9 人に低下したが、その後、横這い状態が続いており、その原因として生あるいは未調理の卵との関連が示唆されている。

2001 年 2 月～3 月、サウスカロライナ州内の刑務所 4 施設で急性胃腸炎が大発生した。2,317 名の収容者の内、688 名が腹痛、下痢、悪心等の消化器症状を発症し、便検体からサルモネラ（*S.* Enteritidis）2、13a 及び 23 型が検出された。サウスカロライナ州保健・自然環境管理局（the South Carolina Department of Health and Environmental Control：SCDC）が行った二つの症例対照研究から 3 月 2 日の昼食に出されたツナサラダとの関連性が示唆された。サラダには固ゆで卵が入っており、4 施設全ての卵の出荷元である農場 A を検査した所、患者と同一の 2 型サルモネラ（*S.* Enteritidis）が検出された。

2001 年 6 月、CDC の統計的アウトブレイク探知アルゴリズム（the Statistical Outbreak Detection Algorithm）は、ノースカロライナ州でサルモネラ（*S.* Enteritidis）による急性胃腸炎の発症例が増加傾向にあることを示唆した。これを受けて、ノースカロライナ州保健・福祉サービス局公衆衛生部（Department of Health and Human Services, Division of Public Health）は州内の発症例の調査を開始し、ノースカロライナ州公衆衛生部公衆衛生研究所（the North Carolina State Laboratory of Public Health）は 2001 年 7 月中に 51 例、8 月中に 31 例を報告した。2000 年における発症例は 7、8 月共にわずか 11 例だけであった。パルス・フィールド・ゲル電気泳動（PFGE）により症例対照研究（発症例 53 例、非発症例 78 例）を実施した所、卵の摂取との関連が示唆された。発症した 53 例中 21 例はサウスカロライナ州での症例と同様に PFGE でパターン A を示し、同様にサルモネラ（*S.* Enteritidis）も 13a 型であった。

サルモネラ（*S.* Enteritidis）感染の予防には、農場に対するサルモネラ（*S.* Enteritidis）抑制プログラムへの参加の奨励、貯蔵時、輸送時の卵の冷蔵保存や、調理者、消費者への教育が重要となると考えられている。

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Outbreaks of *Salmonella* Serotype Enteritidis Infection Associated with Eating Shell Eggs --- United States, 1999--2001

A *Salmonella* serotype Enteritidis (SE) epidemic emerged in the 1980s, when increasing numbers of infections were detected in the Northeastern and Mid-Atlantic regions of the United States (*1*). In the early 1990s, while SE rates in the Northeast began to decline, the SE epidemic expanded to the Pacific region (*2*). Nationwide, the number of SE isolates reported to CDC peaked at 3.8 per 100,000 population in 1995. Although rates of culture-confirmed SE infection reported to CDC declined to 1.9 by 1999 (Figure 1), rates did not decline further through 2001, and outbreaks continue to occur. Investigations of outbreaks and sporadic cases have indicated repeatedly that, when a food vehicle is identified, the most common sources of SE infection are undercooked and raw shell eggs (*3, 4*). This report describes two SE outbreaks associated with eating shell eggs and underscores the need to strengthen SE-control measures.

South Carolina, 2001

During February--March 2001, outbreaks of gastroenteritis occurred among inmates in four prison facilities of the South Carolina Department of Corrections (SCDC). The first outbreak occurred in a men's facility (M1) on February 6. The three other outbreaks, all occurring on March 2, affected a second men's facility (M2) and two women's facilities (F1 and F2). Among 2,317 inmates in the four prisons, 688 reported to prison infirmaries with gastrointestinal symptoms (e.g., abdominal cramps, diarrhea, and nausea). Stool specimens from ill inmates yielded SE phage types 2, 13a, and 23. No illness was reported among SCDC staff members.

The South Carolina Department of Health and Environmental Control conducted two case-control studies in M2 and F1, which shared a common kitchen. A case-patient

was defined as any SCDC inmate who reported to the prison infirmary with acute gastrointestinal symptoms. Case-patients were selected at random from a list of ill inmates. Controls were inmates without illness who were selected at random from an inmate roster provided by the prisons and who were matched by prison facility. A tuna salad served for lunch on March 2 was eaten by 88% of the male case-patients (odds ratio [OR] =7.0; 95% confidence interval [CI] =1.8--30.5) and by 89% of the female case-patients (OR=16.7; 95% CI=4.1--74.7). The tuna salad was prepared with eggs that were reportedly hard-boiled by kitchen staff, who also were inmates. At the time of the outbreaks, all eggs used by the four involved SCDC facilities were supplied from a single vendor. Eggs supplied to M2 and F1 were traced back to the vendor's farm (Farm A). In February 2001, eggs submitted from Farm A to the South Carolina Egg Quality Assurance Program tested positive for SE phage types 2,13a, 22, 23, and 28. Phage type 2 was the predominant SE strain isolated from both ill patients and eggs from Farm A. To protect the inmates, SCDC switched to pasteurized egg products in April 2001.

North Carolina, 2001

In June 2001, the Statistical Outbreak Detection Algorithm at CDC signaled an increase in SE cases reported from North Carolina. The Division of Public Health in North Carolina was alerted and began to review SE cases throughout the state. The North Carolina State Laboratory of Public Health reported 51 cases in July and 31 in August, compared with 11 cases in each of those months during 2000. Cases occurred throughout the state.

A case-control study was performed. A case was defined as culture-confirmed SE in a resident of North Carolina reported during July 1--September 7, 2001. One to two neighbor controls were matched to each case. SE isolates were subtyped by pulsed-field gel electrophoresis (PFGE) and phage typing. Analysis of 53 patients and 78 controls indicated that illness was associated with eating eggs (matched odds ratio [MOR] =2.8; 95% CI=1.1--9.5). Isolates from 21 (40%) of 53 patients had PFGE pattern A. Analysis restricted to patients with pattern A indicated a stronger association with egg consumption (MOR=10.7; 95% CI=1.3--88.1). PFGE pattern A also was identified in isolates from patients in the South Carolina SE outbreak. All isolates from SE patients in both outbreaks that were PFGE pattern A also were phage type 13a. Among 14 random, nonoutbreak phage type 13a SE isolates tested subsequently at CDC, seven distinct PFGE patterns were identified; none was PFGE pattern A. A traceback of

implicated eggs purchased from retail outlets in North Carolina was inconclusive for implicating a farm.

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Editorial Note:

During 1990--2001, state and territorial health departments reported 677 SE outbreaks, which accounted for 23,366 illnesses, 1,988 hospitalizations, and 33 deaths (CDC, unpublished data, 2002). Among the 309 outbreaks reported with a confirmed vehicle of transmission, 241 (78.0%) were associated with shell eggs, accounting for 14,319 illnesses. Of these, 10,406 illnesses occurred during 1990--1995, and 3,913 occurred during 1996--2001. The overall decrease in SE incidence and the decrease in the number of illnesses related to egg-associated SE outbreaks during the last decade might be attributed in part to the implementation of prevention measures, including on-farm control programs, egg refrigeration, and consumer and food worker education. However, reported cases did not decline during 1999--2001, and outbreaks associated with shell eggs continue to occur.

In the South Carolina outbreak, eggs from a farm that tested positive for SE in February 2001 were distributed to the prisons in March, despite the farm's participation in a voluntary, state-sponsored SE-control program. This farm withdrew from the state program in April 2001. Phage type 2 was the most common SE strain isolated in the South Carolina outbreak. This uncommon phage type, which has accounted for 3% of SE outbreaks with reported phage type since 1985, also was found on Farm A. Cases in the outbreak in North Carolina shared the same SE PFGE pattern and phage type (13a) as some of the South Carolina outbreak cases, suggesting a possible link to the same farm.

Eggs that reportedly were hard-boiled and used in a tuna salad were the implicated vehicle in the South Carolina outbreak. A recent study demonstrated that unless SE-containing eggs are exposed to boiling water until the yolk is completely solidified,

SE can survive the cooking process (*5*). Cross contamination of the tuna salad by inmate food handlers also was possible.

To achieve sustained decreases in egg-associated SE illnesses, a concerted prevention effort is needed from farmers to consumers (*6*). A key factor in this effort is the implementation of farm-based measures to reduce SE contamination of eggs during production. The implementation of such control programs in Northeastern states in the early 1990s might have contributed to subsequent decreases in human SE isolation rates in New England and Mid-Atlantic regions (*7*). One important control measure is microbiologic testing of hen houses for the presence of SE. If SE is found on a farm during routine environmental testing, eggs may be diverted to pasteurization. Evidence suggests that proper implementation and oversight of farm-based control programs can result in a reduction of SE infections among flocks in poultry houses (*8*). Farm participation in current SE-control programs is voluntary, and the components of programs vary. Future shell-egg safety measures should include greater participation in farm-based control programs with microbiologic testing.

Both outbreaks described in this report occurred in the Southeastern region of the United States. Compared with declining rates of SE infections in other regions of the United States, the incidence of SE in Southeastern states increased by 50% from 2000 to 2001 (Figure 2). Ongoing surveillance of SE outbreaks will be necessary to detect changes in trends of SE infection in this region. Expansion of SE-prevention measures will be an important part of efforts to prevent SE infections in the Southeast. This includes actively encouraging farms to participate in SE-control programs, promotion of proper refrigeration of eggs during storage and transportation, and education of food handlers and consumers about food preparation (see box). Retail and consumer buyers can specify that suppliers provide only eggs produced from farms managed under an SE-control program that is recognized by a state regulatory agency or a state poultry association.

The outbreak in South Carolina prisons was the largest SE outbreak in 2001. Because persons residing in institutions depend entirely on their institutions for meals, the supply of contaminated foods to these settings can place large populations at risk for developing foodborne diseases. Persons residing in institutions, especially elderly persons in nursing homes or assisted living facilities, are at higher risk for dying from outbreak-associated SE infections (*9*). During 1990--2001, a total of 83 SE outbreaks occurred in institutional settings*, representing 12% of reported SE outbreaks. Of the

33 outbreak-associated deaths, 22 (67%) occurred in institutional facilities, underscoring the importance of using pasteurized egg products or in-shell pasteurized eggs for all recipes requiring pooled, raw, or undercooked shell eggs for institutionalized persons.

Additional information about preventing SE infections associated with eating raw or undercooked shell eggs is available at http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salment_g.htm, http://www.cfsan.fda.gov/~dms/fs-eggs.html, http://www.cfsan.fda.gov/~dms/fs-eggs2.html, and http://www.cfsan.fda.gov/~dms/fs-eggs4.html. Information for retail and food service establishments and institutional facilities about handling and cooking shell eggs is available in the Food Code at http://www.cfsan.fda.gov/~dms/foodcode.html.

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* Institutional settings include nursing homes, independent living facilities, assisted living facilities, childcare settings, campus cafeterias, prisons and correctional facilities, and shelters.

BOX. Recommendations for preventing *Salmonella* serotype Enteritidis (SE) infections associated with eggs

For egg producers:

- Flock-based SE-control programs that include routine microbiologic testing should be adopted and implemented by industry nationwide.

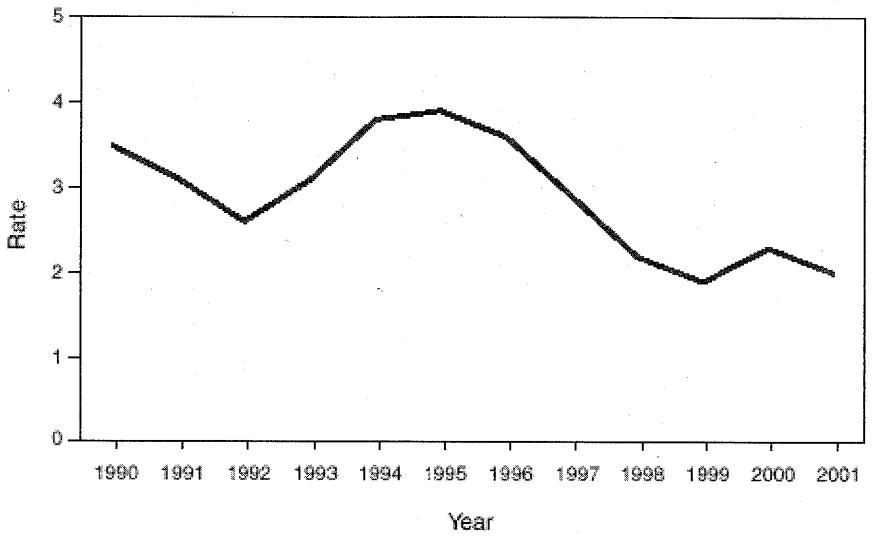
For retail and food service establishments and institutional settings:

- In retail and food service establishments, pasteurized egg products or pasteurized in-shell eggs are recommended in place of pooled eggs or raw or undercooked shell eggs. If used, raw shell eggs should be fully cooked. If shell eggs are served undercooked, a consumer advisory should be posted in accordance with the Food Code.
- In hospitals, nursing homes, adult or childcare facilities, and senior centers, pasteurized egg products or pasteurized in-shell eggs should be used in place of pooled eggs or raw or undercooked eggs.
- Eggs should be purchased or received from a distributor refrigerated and stored refrigerated at ≤45° F (≤7° C) at all times.

For egg consumers:

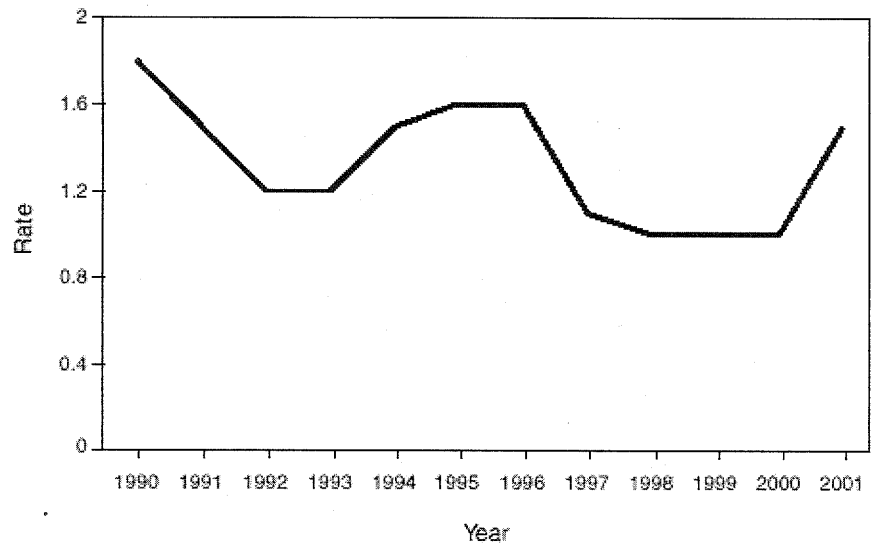
- Consumption of raw or undercooked eggs should be avoided, especially by young children, elderly persons, and persons with weakened immune systems or debilitating illness.
- Eggs should be cooked until both the white and the yolk are firm and eaten promptly after cooking.
- Hands, cooking utensils, and food-preparation surfaces should be washed with soap and water after contact with raw eggs.
- Eggs should be purchased or received from a retail store or distributor refrigerated and stored refrigerated at ≤45° F (≤7° C) at all times.

FIGURE 1. Isolation rate* of *Salmonella* serotype Enteritidis (SE), by year — United States, 1990–2001



* Per 100,000 population.

FIGURE 2. Isolation rate* of *Salmonella* serotype Enteritidis (SE), by year — Southeastern region†, 1990–2001



* Per 100,000 population.
† Alabama, Delaware, Florida, Georgia, Kentucky, Maryland, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, and West Virginia.

MMWR51 (46):1044- 1047

複数の州におけるメキシコ産カンタループメロンの摂取による サルモネラ（*S.* Poona）感染症の発生 米国及びカナダ，2000～2002 年

2000 年から 2002 年の間、米国及びカナダの複数の州でメキシコから輸入されたカンタループメロンの摂取に関連するサルモネラ（*S.* Poona）感染症が大発生した。発生は最初にカリフォルニア州保健局（the California Department of Health Services）（2000 年、2001 年）とワシントン州保健局（the Washington State Department of Health）（2002 年）で確認され、米国内の 12 州及びカナダの住民が影響を受けた。

2000 年 4 月～6 月には 6 州で 47 例（1～95 歳）、2001 年 4 月～5 月には 5 州で 50 例（カリフォルニア州で 28 例中、小児は 19 例（1～5 歳）、成人は 9 例（39～91 歳））、2002 年 3 月～5 月には 14 州で 58 例（4 ヶ月～91 歳）のサルモネラ（*S.* Poona）陽性患者が確認された。各感染症の発生において、分離菌株はパルス・フィールド・ゲル電気泳動（PFGE）パターンにより区別できなかった。

2002 年 10 月 28 日、米国食品医薬品局（FDA）は全米の港に着いた全てのメキシコ産カンタループメロンをそのまま留めておくよう輸入警告を出した。さらに FDA は、今後もメキシコ政府と共に生のカンタループメロンの生産、梱包、船舶輸送に関する食品安全性プログラムに取り組む予定である。

追跡調査及び規制措置

FDA は州及び州の食品規制機関と連携して、3 件の食品事故で患者が購入したカンタループメロンの追跡調査を実施した。各事件に関して、輸送業者及びメキシコの（カンタループメロン生産）農場を突き止めた。2000 年及び 2001 年の食品事故を受けて、FDA はメキシコの農場の現地調査を実施したが、これはカンタループメロンの生育、収穫、梱包、及び冷却処理中の細菌汚染を最小限に抑えるための方法としては不適切であったと結論づけた。2001 年の食品事故に関連して、FDA は 5 月 31 日までは当該製品の輸入を一時停止し、荷主は自主的にメキシコから輸入したカンタループメロンを回収した。荷主及びメキ

シコの関連農場は留置されたままである。2002 年の食品事故に関連し、輸入業者は自主的にメキシコから輸入したカンタループメロンを回収し、FDA は関連農場を留置した。

Editorial Note《編集注記》

1991 年に起きたカンタループメロンによる食品事故を受け、FDA は輸入カンタループメロン及びメロンのサンプルのおよそ 1%から、様々なサルモネラ血清型を分離する微生物調査を実施した。2000 年及び 2001 年の食品事故の後、FDA はメキシコの農場調査を行い、消費者に警告するための広報を発行、当該農場を留置し、輸入カンタループメロンのサンプリング調査を実施した。FDA はメロンの汚染除去に関する資料を小売産業、レストラン、メロンをカットする加工業者に提供している。

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Multistate Outbreaks of *Salmonella* Serotype Poona Infections Associated with Eating Cantaloupe from Mexico --- United States and Canada, 2000--2002

Three multistate outbreaks of *Salmonella* serotype Poona infections associated with eating cantaloupe imported from Mexico occurred in the spring of consecutive years during 2000--2002. In each outbreak, the isolates had indistinguishable pulsed-field gel electrophoresis (PFGE) patterns; the PFGE patterns observed in the 2000 and 2002 outbreaks were indistinguishable, but the pattern from 2001 was unique among them. Outbreaks were identified first by the California Department of Health Services (2000 and 2001) and the Washington State Department of Health (2002) and involved residents of 12 states and Canada. This report describes the investigations, which led ultimately to an import alert on cantaloupes from Mexico. To limit the potential for cantaloupe contamination, the Food and Drug Administration (FDA) continues to work with the Mexican government on a food-safety program for the production, packing, and shipping of fresh cantaloupes.

April--June 2000 Outbreak

A total of 47 confirmed cases of *S.* Poona infections with indistinguishable PFGE patterns were identified from California (26), Washington (10), Nevada (five), New Mexico (three), Oregon (two), and Colorado (one), with illness onset occurring during April 14--June 2. The median age of ill persons was 7 years (range: 1--95 years); 28 (60%) patients were aged <10 years, and nine (19%) were aged >60 years. Twenty-four (51%) patients were male and nine (19%) were hospitalized.

A matched case-control study was conducted; 20 case-patients were matched by age category to 37 community controls. A case was defined as laboratory-confirmed infection with *S.* Poona of the outbreak PFGE pattern in a person with illness onset during April--June. By multivariable modeling, illness was associated only with eating

cantaloupe (matched odds ratio [MOR] =6.7; 95% confidence interval [CI] =1.3--34.0), with 16 (80%) case-patients versus seven (19%) controls reporting eating cantaloupe. Cantaloupe was purchased either pre-cut or whole.

April--May 2001 Outbreak

In April, an initial cluster of *S.* Poona was identified in California. Isolates had a rare biochemical trait, the inability to produce hydrogen sulfide (H_2S), and PFGE patterns that were indistinguishable. A total of 50 cases of H_2S-negative *S.* Poona infections were identified in residents of California (28), Washington (eight), Nevada (seven), Arizona (six), and Oregon (one). Demographic and illness-history data from the 28 California patients indicated that illness onset occurred during April 6--May 28. The age distribution was bimodal; the 19 children had a median age of 3 years (range: 1--5 years) and the nine adults had a median age of 80 years (range: 39--91 years). Fifteen (54%) patients were female. Ten (36%) patients were bacteremic; one infant girl had *S.* Poona isolated from a urine specimen. Nine (33%) patients were hospitalized, and two patients (a man aged 78 years and a woman aged 91 years) died with *Salmonella* septicemia.

A matched case-control study was conducted; 11 case-patients from California (seven), Nevada (two), Arizona (one), and Washington (one) were matched by age category to 19 community controls. Case-patients had laboratory-confirmed infections of the outbreak strain of H_2S-negative *S.* Poona and illness onset during the first 2 weeks of April. Illness was associated only with eating cantaloupe (MOR=7.4; 95% CI=1.0--178.0). Eight (80%) case-patients and six (33%) controls recalled eating cantaloupe. Cantaloupe was purchased either pre-cut or whole.

March--May 2002 Outbreak

A total of 58 cases with *S.* Poona isolates with indistinguishable PFGE patterns were identified in California (21), Washington (nine), Oregon (five), British Columbia (four), Colorado (three), Nevada (three), Manitoba (two), Missouri (two), Ontario (two), Saskatchewan (two), Texas (two), Arkansas (one), Minnesota (one), and Vermont (one). Illness onset occurred during March 30--May 31; the median age of patients was 6 years (range: 4 months--91 years); 32 (55%) were aged <10 years, and 11 (19%) were aged >60 years. A total of 31 (55%) were female. Ten patients were hospitalized.

A matched case–control study was conducted; 27 case–patients were matched by age category to 54 community controls. A case was defined as *S.* Poona infection with the outbreak PFGE pattern in a person aged ≥2 years with illness onset during March 15––May 3. The only exposure significantly associated with illness was eating cantaloupe; 20 (74%) case–patients recalled eating cantaloupe compared with 11 (20%) controls (MOR=15.5; 95% CI=3.3––125.0). Case–patients (50%) were more likely than controls (13%) to eat cantaloupe purchased whole (MOR=5.8; 95% CI=1.6––23.3) or to eat cantaloupe in a fruit salad or as a garnish (28% versus 5%) (MOR=6.5; 95% CI=1.2––63.0). No other factors were significantly associated with illness.

Traceback and Regulatory Action

FDA, in conjunction with state and provincial food regulatory agencies, conducted traceback investigations of cantaloupe purchased by patients in all three outbreaks. In each instance, point–of–sale sources of cantaloupe were traced back to shippers and then to farms in Mexico. In response to the 2000 and 2001 outbreaks, FDA conducted on–farm investigations in Mexico and concluded that measures were not in place to minimize microbial contamination in the growing, harvesting, packaging, and cooling of cantaloupe. Possible sources of contamination include irrigation of fields with water contaminated with sewage, processing (cleaning and cooling) produce with *Salmonella*–contaminated water, poor hygienic practices of workers who harvest and process the cantaloupe, pests in packing facilities, and inadequate cleaning and sanitizing of equipment that comes in contact with cantaloupe. In association with the 2001 outbreak, FDA detained product imported by the shipper on May 31, and the shipper voluntarily recalled its imported Mexican cantaloupe. The shipper and the implicated farm in Mexico remain on detention. In association with the 2002 outbreak, the importer voluntarily recalled the implicated Mexican cantaloupe, and FDA placed the implicated farms on detention. On October 28, 2002, FDA issued an import alert on cantaloupe from Mexico that detains all products offered for entry at all U.S. ports.

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Center for Food Safety and Applied Nutrition and the Office of Regulatory Affairs, Food and Drug Administration. C Braden, MD, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; G Djomand, MD, M Reller, MD, W Chege, MD, EIS officers, CDC.

Editorial Note:

Salmonella infections have been linked to melons at least since 1990 when *Salmonella* serotype Chester traced to cantaloupe caused 245 illnesses in 30 states (*1*). The cantaloupe were imported from either Mexico or Guatemala. In 1991, an outbreak of cantaloupe-associated *S.* Poona infections caused 400 illnesses in 23 states (*2*). Illness was associated with eating pre-cut cantaloupe in fruit salads or from salad bars. Although industry sources identified the lower Rio Grande Valley in Texas as the probable source of the implicated cantaloupe, some might have come from Mexico. In response to this outbreak, FDA conducted a microbiologic survey that isolated a variety of *Salmonella* serotypes from approximately 1% of sampled imported cantaloupe and watermelon (*2*). In 1997, an outbreak of *Salmonella* serotype Saphra infections affected 25 persons in California. Illness was associated with cantaloupe imported from Mexico (*3*). After the 2000 and 2001 *S.* Poona outbreaks, FDA conducted farm investigations in Mexico, issued press releases to warn consumers, placed implicated farms on detention, and conducted sampling surveys of imported cantaloupe. The 1999 and 2000 FDA surveys of imported produce indicated that 5% of cantaloupe sampled (eight of 151) was contaminated with *Salmonella* (*4*). A 2001 survey of imported produce indicates that of 29 cantaloupes from Mexico tested, none yielded *Salmonella*, *Shigella*, or *Escherichia coli* O157:H7 (FDA, unpublished data, 2001). The interpretation of the 2001 survey is limited by of the small sample size.

S. Poona is a relatively rare serotype that is responsible for 1% of human *Salmonella* isolates reported in the United States in 2001; however, of the six cantaloupe-associated *Salmonella* outbreaks, four were attributed to infections with *S.* Poona. Typically, human infection with *S.* Poona is associated with reptile exposure (*5, 6*). The three outbreaks attributed to *S.* Poona-contaminated cantaloupe traced to Mexican farms suggest the possibility of a unique natural reservoir in the Mexican farm environment, possibly from reptiles such as iguanas drawn to feed on melon crops that enter the packing sheds and contaminate the equipment. Subsequently, water used in the washing and cooling process might spread the contamination.

FDA provides information about the decontamination of melons to the retail industry, food-service establishments, and commercial processors of pre-cut melon (*7, 8*). The use of sodium hypochlorite or other permitted antimicrobials in combination with brushing is recommended. The potential for microbial contamination also might be reduced by using only good-quality fruit that is free from open wounds or defects that might allow bacteria to contaminate the interior of the fruit (*9*). Additional research is needed to determine the effectiveness of consumer produce-washing practices. Consumers should be sure that fresh-cut melons are refrigerated or surrounded by ice; leftover cut melons should be discarded if left at room temperature for >2 hours. Additional information for consumers is available at http://www.fda.gov/bbs/topics/answers/2002/ans01167.html.

On October 28, 2002, in response to the three outbreaks during 2000--2002 and analytical results from the sampling of imported Mexican cantaloupe, FDA issued an import alert that detains all cantaloupe from Mexico offered for entry at all U.S. ports. FDA will continue to work with the Mexican government on a food-safety program for the production, packing, and shipping of fresh cantaloupe. The Mexican government is developing a certification program based on sound agricultural and manufacturing practices that would allow FDA to identify farms that have adopted and implemented such a food-safety program.

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MMWR51 (31):683-684

サルモネラ（*S.* Javiana）感染症の発生 フロリダ州オーランド，2002 年 6 月

2002 年 7 月 16 日、フロリダ州オーランドのテーマパーク A で 6 月 25 日～29 日に開催された 2002 年 U.S.Transplant Games の参加者の内、2 人にサルモネラ（*S.* Javiana）感染症が確認された。パルス・フィールド・ゲル電気泳動（PFGE）により検査した結果、どちらも区別できないパターンであった。U.S.Transplant Games は臓器及び組織移植（心、肝、腎、肺、膵、骨髄）レシピエントによる運動競技会であり、米国及び他の 5 ヶ国から移植レシピエント選手 1,500 人を含む約 6,000 人が参加していた。

この報告は、現在進行中のサルモネラ（*S.* Javiana）感染症の発生についての疫学調査及びこれまでの検査結果をまとめたものである。患者の確認と調査のため、7 月 20 日から E メールアドレスが判明している参加者 1,100 人を対象とし、web ベースでの調査を開始した。患者の定義は、オーランドを訪れた者で 6 月 25 日から 7 月 7 日までに発熱又は下痢の症状が現れた場合とした。

8 月 1 日までに 369 人（34%）から回答が得られ、32 州で 141 例の患者が確認された。この 369 世帯から各 1 人を選び、より詳細な情報を収集した。これら 369 人中 82 例（22%）が発病していた。患者の年齢は 4～71 歳（中央値 47 歳）で、48 例（59%）は移植レシピエントであり、43 例（52%）は免疫抑制療法を受けていた。主な症状は下痢（93%）、腹痛（79%）、発熱（51%）であった。これら患者の内 51 例（66%）は、オーランド滞在中テーマパーク A にあるリゾートに宿泊し、75 例（91%）はその中の施設で食事をしていた。

7 月 31 日に行った 2 回目の調査で、患者群では、さいの目切りのローマトマトを含む食事を食べた者が健常者群に比して有意に多いことが明らかになった。

予備的微生物学的試験では、さいの目切りトマトの糞便汚染が示唆されている。感染症を発生させた菌株の PFGE パターンを PulseNet 上に提示した所、さらに 9 州でサルモネラ（*S.* Javiana）感染症 18 例が確認された。このうち聞き取り調査を行った 16 例では、1 例が競技会参加者であり、他の 12 例は 6 月の最後の週にテーマパーク A を訪れていた。

州及び地方の健康局は、感染症発生との疫学的関係を確認するため、さらなる患者につ

いて調査中である。

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Outbreak of *Salmonella* Serotype Javiana Infections --- Orlando, Florida, June 2002

On July 16, 2002, the Minnesota Department of Health identified two cases of *Salmonella* serotype Javiana infections among persons who had attended the 2002 U.S. Transplant Games held at theme park A in Orlando, Florida, during June 25--29. Isolates from both patients were indistinguishable by pulsed field gel electrophoresis (PFGE). The U.S. Transplant Games is a 4-day athletic competition among recipients of solid organ transplants (i.e., heart, liver, kidney, lung, and pancreas) and bone marrow transplants. Approximately 6,000 persons from the United States and five other countries, including 1,500 transplant-recipient athletes, participated in the games. This report summarizes the results of an ongoing epidemiologic and laboratory investigation that has identified 141 ill persons in 32 states who attended the games.

For case ascertainment and investigation purposes, a web-based survey was distributed electronically on July 20 to 1,100 attendees with known e-mail addresses, including athletes, donors, family members, and transplant professionals. Anonymous e-mail addresses for these persons were obtained from the organizers of the games. A case was defined as fever or diarrhea with onset during June 25--July 7 in a person who visited Orlando. A total of 369 (34%) persons responded by August 1; of these, 296 (80%) responded by July 22. Ninety-four (25%) persons reported that at least one household member had an illness that met the case definition, representing 141 ill persons.

For each of the 369 households, detailed information was collected for one person who was selected on the basis of birth date. Among these persons, 82 (22%) reported illness. The median age of ill respondents was 47 years (range: 4--71 years); 48 (59%) were transplant recipients, and 43 (52%) were receiving immunosuppressive therapy. Dates of illness onset ranged from June 26 to July 7. Predominant symptoms included diarrhea (93%), abdominal pain (79%), and fever (51%). Three (4%) respondents were hospitalized.

All survey respondents were asked about places they stayed, events they attended,

and foods they ate while in Orlando. Fifty-one (66%) ill persons stayed at resorts located in theme park A during their time in Orlando, and 75 (91%) reported eating food items at establishments located in theme park A. On July 31, a second web-based survey containing questions about potentially suspect food items available in theme park A was distributed electronically to the 369 persons who responded to the first survey. Ill persons were asked about specific foods eaten during the 3 days before illness onset, and well persons were asked about the middle 3 days of the games (June 26--28). By August 2, a total of 222 (60%) persons had responded to the second survey; 41 had been ill. Univariate analysis demonstrated that ill persons were significantly more likely to report eating foods containing diced Roma tomatoes than were well persons (44% of ill versus 14% of well persons; adjusted odds ratio=4.3; 95% confidence interval=2.1--9.1). Preliminary microbiologic evaluation indicates fecal coliform contamination of the diced tomatoes.

To identify other potential cases of *S.* Javiana, the PFGE pattern for the outbreak strain was posted on PulseNet, the National Molecular Subtyping Network for Foodborne Disease Surveillance. A total of 18 additional infections caused by *S.* Javiana with an indistinguishable PFGE pattern were identified in nine states (Illinois, Massachusetts, Michigan, Minnesota, New Hampshire, North Carolina, Pennsylvania, Tennessee, and Virginia). Of 16 patients who were interviewed, one was a games participant, and 12 others had visited theme park A during the last week of June but did not attend the games. Dates of illness onset ranged from June 24 to July 8. State and local health departments are investigating additional cases to establish epidemiologic links to the outbreak.

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Editorial Note:

Salmonellosis causes an estimated 1.4 million illnesses each year in the United States (*1*). *S.* Javiana is the fifth most common *Salmonella* serotype in the United States and accounted for 3.4% of *Salmonella* isolates reported to CDC during 2001 (CDC, unpublished data, 2002). The majority of persons infected with *Salmonella* have

diarrhea, fever, and abdominal cramps 12--72 hours after exposure. The illness usually lasts 4--7 days, and the majority of persons recover without treatment.

Persons with impaired immune systems are at increased risk for having a more severe illness, atypical symptoms, and complications of infection. Among organ transplant recipients, salmonellosis is associated strongly with antirejection therapy (*2*), and febrile illness with bacteremia is a more common presentation (*3*). Organ transplant patients are at increased risk for focal manifestations of illness including meningitis, urinary tract infections, abscesses of soft tissues, septic arthritis, osteomyelitis, and vascular infections, including infections of vascular grafts (*4--6*). Recurrence of nontyphoidal salmonellosis is common among this population and might occur in up to 35% of renal transplant recipients (*2, 3*).

Physicians caring for recipients of solid organ and bone marrow transplants should be aware of possible exposure to *S.* Javiana at the 2002 U.S. Transplant Games and should consider obtaining cultures (i.e., stool, blood, and urine) from ill patients with this exposure. The optimal therapy for *Salmonella* infection in transplant recipients is not known (*4*). However, because of the increased susceptibility to infection and the potential for complications, physicians might consider empiric antimicrobial therapy in transplant recipients with suspected salmonellosis from whom appropriate cultures have been obtained. The strain of *S.* Javiana responsible for this outbreak is susceptible to several commonly used antimicrobials, including trimethoprim-sulfamethoxazole, ciprofloxacin, and ceftriaxone. Physicians should report culture-confirmed cases of salmonellosis to their local health department.

The use of a web-based survey in this investigation allowed a substantial number of persons who were dispersed geographically to be asked about potential exposures in a relatively short period of time. Twelve culture-confirmed cases of *S.* Javiana among visitors to theme park A who did not attend the games were identified through PulseNet, indicating that the number of ill persons in this outbreak is probably much larger than what has been identified in the surveyed Transplant Games population. The combination of molecular subtyping, web-based technology, and routine public health surveillance facilitated the outbreak investigation.

The findings in this report are subject to at least two limitations. First, a web-based investigation limited responses to only those attendees with known e-mail addresses and Internet access. Second, although responses were received from both well and ill

persons, households with ill persons might have been more likely to respond to a web-based survey. Therefore, it is difficult to calculate an accurate attack rate among attendees of the games.

Preliminary findings of the epidemiologic investigation have implicated fresh, pre-packaged diced Roma tomatoes supplied to theme park A as the probable vehicle for this outbreak. Efforts are under way to identify the source of these tomatoes and possible routes of contamination. Tomatoes are not a commonly recognized vehicle for *Salmonella,* and no evidence exists for widespread contamination of tomatoes available for purchase. However, tomatoes have been implicated in at least one previous outbreak of *S.* Javiana infections (*7*), and cut surfaces of tomatoes and other fresh fruits and vegetables can support the growth of *Salmonella* and other enteric pathogens (*8,9*). Produce is recognized increasingly as a source of *Salmonella* infections in the United States, and consumers should wash tomatoes and other produce items thoroughly before eating. The Food and Drug Administration guidelines for safe produce-handling practices are available at http://www.cfsan.fda.gov/~lrd/tpproduc.html.

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This report is based on data contributed by R Baker, MS, Florida Dept of Health. C Langkop, MSPH, Illinois Dept of Public Health. T LaPorte, MS, Massachusetts Dept of Public Health. S Bidol, MPH, Michigan Dept of Community Health. L Anderson, MD, New Hampshire Dept of Health and Human Svcs. P Jenkins, North Carolina Dept of Health and Human Svcs. J Murphy, DVM, Virginia Dept of Health.

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多剤耐性サルモネラ（*S.* Newport）の発生
米国，2002 年 1 月～4 月

2002 年 1 月～4 月、米国 5 州において 47 例（ニューヨーク州 34 例、ミシガン州 5 例、ペンシルベニア州 4 例、オハイオ州 2 例、コネチカット州 2 例、2～81 歳）よりサルモネラ（*S.* Newport）が分離された。これら 47 例中 46 例における罹患期間は 3～60 日間（中央値 9 日間）、主要症状は下痢（100%）、腹痛（91%）、発熱（78%）、血便（52%）、嘔吐（48%）であった。全体で 33 例（72%）が抗菌薬による治療を受け、17 例（37%）は入院した。分離菌 3 株について米国疾病管理予防センター（CDC）が抗菌感受性試験を行った結果、これらの菌株はアモキシシリン／クラブラン酸、アンピシリン、セフォキシチン、セフチオフル、セファロチン、クロラムフェニコール、ストレプトマイシン、スルファメトキサゾール、テトラサイクリンに耐性を示した。さらに、この内 2 株についてはカナマイシンにも耐性を示し、セフトリアキソンに対しても 1 株が感受性低下、1 株が耐性を示した。

感染症発生はそもそも、2002 年 2 月 11 日にニューヨーク州のある郡保健局から同州保健局（the New York State Department of Health : NYSDOH）に 7 例のサルモネラ（*S.* Newport）感染症が報告されて初めて認知されることとなった。NYSDOH の研究所によるパルス・フィールド・ゲル電気泳動（PFGE）パターンの検査では、6 例が区別できず、1 例がバンドの 1 つに違いがあった。追加事例がパルスネット（PulseNet）を通じてコネチカット州、ミシガン州、オハイオ州、ペンシルベニア州から報告された。感染症発生の原因を検討するため NYSDOH が行った症例対照研究では、感染源として生あるいは加熱調理していない牛挽肉の曝露が関与していることが明らかになった。この調査結果は米国農務省（USDA）食品安全検査局（FSIS）に報告された。FSIS によってニューヨーク州の 12 患者によって摂取された牛挽肉の追跡調査が行われ、その牛挽肉の出所らしき食肉工場を突き止めた。流通記録、製粉記録、購買に関する情報が確かめられたが、怪しい牛挽肉ロットは見つけられず、また感染症発生が起こった期間中に加工されたままの牛挽肉のサンプルも得られなかった。2002 年 4 月 19 日、USDA は公衆衛生警報を発令し、消費者に食品安全ガイドラインの徹底を呼びかけた。FSIS は、現在サルモネラ（*S.* Newport）によ

る食肉感染を引き起こす恐れのある手順について試験中である。

Editorial Note《編集注記》

多剤耐性サルモネラ（*S.* Newport）感染症は、セフトリアキソンによる治療に無効である可能性があるため、医師はこれら菌株の出現について報告を受ける必要がある。また、加熱調理していない牛挽肉を食べるのは控え、生の牛挽肉を取り扱った後には手を洗うべきである。

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Outbreak of Multidrug-Resistant *Salmonella* Newport --- United States, January--April 2002

During January--April 2002, *Salmonella* serotype Newport was isolated from 47 persons in five states: New York (34 cases), Michigan (five), Pennsylvania (four), Ohio (two), and Connecticut (two). Antimicrobial-susceptibility testing of three isolates by CDC revealed resistance to amoxicillin/clavulanate, ampicillin, cefoxitin, ceftiofur, cephalothin, chloramphenicol, streptomycin, sulfamethoxazole, and tetracycline. In addition, two of three isolates were resistant to kanamycin; two had decreased susceptibility or resistance to ceftriaxone. To determine the cause of the outbreak, the New York State Department of Health (NYSDOH) and CDC conducted a case-control study. This report summarizes the results of this investigation, which implicated exposure to raw or undercooked ground beef as the vehicle of transmission. The findings also highlight the emergence of multidrug-resistant *S.* Newport in the United States. These strains exhibit decreased susceptibility or resistance to ceftriaxone, thereby complicating empiric therapy for serious *Salmonella* infections. Clinicians should be informed of the emergence of these *S.* Newport strains, and persons should refrain from eating undercooked ground beef and wash their hands after handling raw ground beef.

The outbreak was identified on February 11, when a county health department notified NYSDOH of seven cases of *S.* Newport infection. Pulsed-field gel electrophoresis (PFGE) testing by the NYSDOH laboratory revealed that six isolates had an indistinguishable pattern, and one isolate had a single band difference. NYSDOH defined a case as isolation of *S.* Newport with a PFGE pattern that was indistinguishable or one band different from the outbreak pattern. Additional cases were reported from Connecticut, Michigan, Ohio, and Pennsylvania through the National Molecular Subtyping Network for Foodborne Disease Surveillance (PulseNet).

A total of 47 cases from the five states was identified. The median age of infected persons was 45 years (range: 2--81 years); 33 (70%) were females. Symptom onsets

occurred during January 1--April 4, with 33 (73%) occurring during February 1--15. Of the 47 patients, 46 were interviewed. The median duration of illness was 9 days (range: 3--60 days). Predominant symptoms included diarrhea (100%), abdominal pain (91%), fever (78%), blood-tinged stools (52%), and vomiting (48%). Six (13%) patients reported other symptomatic household members. A total of 33 (72%) patients received antimicrobial agents, and 17 (37%) were hospitalized. One patient from New York with leukemia developed sepsis and died; *S.* Newport was identified in both blood and stool cultures from this patient. A total of 44 isolates had an indistinguishable PFGE pattern after analysis with two enzymes (*Xba*I and *Avr*II); three isolates differed by one band.

To identify exposures associated with illness, NYSDOH and CDC compared 36 patients (28 from New York, four from Michigan, and four from Pennsylvania) with 85 controls, who were interviewed through random-digit--dialing in case-patients' home area codes and frequency-matched by age group. A multivariate logistic regression analysis indicated that 22 (67%) of 35 case-patients had eaten ground beef during the 3 days before illness onset compared with 31 (53%) of 58 controls (odds ratio [OR] =2.3; 95% confidence interval [CI] =0.9--5.7). Case-patients and controls were asked about eating raw or undercooked ground beef during the 3 days before illness onset. Of the 26 case-patients who answered definitively, 12 (46%) had eaten raw or undercooked ground beef compared with one (1%) of 80 controls (OR=50.9; 95% CI=5.3--489.0). A total of 11 patients recalled the type of ground beef eaten; seven (64%) had eaten lean or extra-lean ground beef. The U.S. Department of Agriculture (USDA) Food Safety and Inspection Service (FSIS) was notified after this investigation implicated ground beef as a potential vehicle for exposure.

One New York patient had a leftover, frozen, uncooked meatloaf prepared with the same package of ground beef that was used to prepare meals eaten during the 3 days before onset of symptoms. A culture of the meatloaf yielded *S.* Newport with a PFGE pattern indistinguishable from the outbreak pattern. Traceback by FSIS of ground beef eaten by 12 New York patients identified a meat packing plant that could have supplied the meat eaten by all those identified in the outbreak. Review of distribution records, grinding logs, and purchasing information did not identify any specific lot of ground beef, and no intact ground beef sample processed by the plant during the outbreak period was available for testing by FSIS. On April 19, USDA issued a Public Health Alert reminding consumers of food safety guidelines. FSIS is examining practices that might contribute to contamination of meat by this pathogen.

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Editorial Note:

An estimated 1.4 million cases of Salmonellosis occur annually in the United States (*1*). *S.* Newport is the third most common *Salmonella* serotype in the United States. During 1997--2001, the number of laboratory-confirmed *S.* Newport infections reported to CDC increased from 1,584 (5%) of 34,608 reported *Salmonella* infections to 3,152 (10%) of 31,607 (CDC, unpublished data, 2002). The increasing number of *S.* Newport infections in the United States appears to be associated with the emergence and rapid dissemination of multidrug-resistant strains of *S.* Newport.

Since 1996, the National Antimicrobial Resistance Monitoring System (NARMS) for Enteric Bacteria has identified an increasing number of *S.* Newport isolates that are resistant to at least nine of 17 antimicrobial agents tested: amoxicillin/clavulanate, ampicillin, cefoxitin, ceftiofur, cephalothin, chloramphenicol, streptomycin, sulfamethoxazole, and tetracycline. In addition, these isolates exhibit decreased susceptibility (minimal inhibitory concentrations [MIC] $\geq$16mg/ml) or resistance (MIC $\geq$64mg/ml) to ceftriaxone, an antimicrobial agent commonly used to treat serious infections in children. Isolates with this resistance pattern have plasmids that carry a bla_{CMY} gene. These genes produce AmpC-type enzymes, which confer resistance to penicillin-inhibitor combinations (e.g., amoxicillin/clavulanate), cephamycins (e.g., cefoxitin), and expanded-spectrum cephalosporins (e.g., ceftiofur and ceftriaxone). To distinguish this type of resistance from other multidrug-resistant strains, these strains are referred to as Newport MDR-AmpC. In 1998, one (1%) of 78 *S.* Newport isolates tested in NARMS was Newport MDR-AmpC compared with 33 (26%) of 128 in 2001. Although the full clinical significance of Newport MDR-AmpC is unknown, treatment of these infections with ceftriaxone might be ineffective. In addition, antimicrobial-resistant *Salmonella* infections have been associated with an increased hospitalization rate, morbidity, and mortality (*2, 3*).

During 2001--2002, several state health departments, including California, Connecticut, and Massachusetts, documented association of exposure to dairy farms, ill cattle, and cheese made from unpasteurized milk with increased human Newport MDR-AmpC infections (*4--6*). In the outbreak described in this report, most patients for whom information is available ate lean or extra-lean ground beef; dairy cattle are an important source of lean or extra-lean ground beef (*7*). These data suggest that cattle, particularly dairy cattle, might be a source for human Newport MDR-AmpC infection.

This report is the first to associate eating of ground beef, specifically raw or undercooked ground beef, with Newport MDR-AmpC infection. Recent U.S. surveys indicate that 11%--28% of persons report eating raw or undercooked ground beef, and approximately one third of persons do not use safe food-handling practices to prevent cross-contamination in the kitchen (*8*).

The USDA Pathogen Reduction/Hazard Analysis and Critical Control Points (PR/HACCP) inspection system in meat and poultry plants has reduced *Salmonella* prevalence in raw ground beef from 7.5% in 1998 to 2.8% in 2001(*9*). The emergence of Newport MDR-AmpC suggests that further measures might be necessary. Potential strategies include 1) evaluating practices on the farm to determine factors that might contribute to multidrug-resistant *S.* Newport and developing interventions to eliminate these factors; 2) implementing the Public Health Action Plan to Combat Antimicrobial Resistance (*10*); 3) encouraging industry to implement processes such as steam pasteurization or irradiation of ground beef; and 4) increasing efforts to educate consumers on the importance of safe handling and cooking practices.

State health departments and veterinarians should investigate clusters of *S.* Newport and perform antimicrobial-susceptibility testing to determine if isolates are Newport MDR-AmpC. Epidemiologic investigations and PFGE comparison of outbreak isolates will help to identify food vehicles associated with Newport MDR-AmpC and to identify control points for reducing these infections. Because treatment with ceftriaxone might be ineffective, clinicians should be informed of the emergence of Newport MDR-AmpC strains. Persons should not eat undercooked ground beef and should wash their hands after handling raw ground beef.

Acknowledgments

This report is based on data contributed by G Balzano, S Kondracki, Bur of Communicable Disease Control; BJ Anderson, C Hidalgo, G Smith, Emerging Infections Program; R Woron, MPH, B Gerzonic, MBA, Bur of Community Sanitation and Food Protection; T Root, Wadsworth Center, New York State Dept of Health. S Hrabowy, MPH, Michigan Dept of Health. M Deasy, A Weltman, MD, Pennsylvania Dept of Health. S Nowicki, MPH, Ohio Dept of Health. Q Phan, MPH, J Hadler, MD, Connecticut Dept of Public Health. Office of Public Health and Science, Office of Field Operations, Congressional and Public Affairs Office, Food Safety and Inspection Svc, US Dept of Agriculture. S Van Duyne, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; A Shane, MD, EIS Officer, CDC.

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アルファルファ摂取によるサルモネラ（*S.* Kottbus）感染症の発生 アリゾナ、カリフォルニア、コロラド及びニューメキシコ州, 2001年2月～4月

2月1日から5月1日にかけて、カリフォルニア州保健サービス局（the California Department of Health Services : CDHS）によって計23例（カリフォルニア州のいくつかの郡とアリゾナ州）のサルモネラ（*S.* Kottbus）感染症が確認され、2001年3月12日、パルス・フィールド・ゲル電気泳動（PFGE）パターンによりサルモネラ（*S.* Kottbus）が分離された。

症例は年齢（中央値36（範囲9～72））歳、男7名、女16名、であり、21例が急性の下痢、3例が尿路感染症を発症し、内3例が入院した。症例はサルモネラ（*S.* Kottbus）感染症と診断され、23例中15例（63%）が発症1週間前にアルファルファを食べていた。その後も症例数は増え、最終的にカリフォルニア24例、アリゾナ6例、コロラド1例、ニューメキシコ1例の発症を認めた。感染源と考えられるロットの種は、オーストラリアから2000年11月に輸入され、3月29日まで使用した後、4月17日までに自主的に回収された。植物の種は、非処理農業用水、肥料、野生動物の排泄物等による汚染の可能性があり、これまでにも種から発芽した芽野菜摂取によるサルモネラや病原大腸菌O157:H7感染症が報告されている。そのため米国食品医薬品局（FDA）は20,000 ppm次亜塩素酸ナトリウムにて15分の処理を勧告しているが、今回の汚染除去記録では2,000 ppm、15分処理であった。

今回の感染症発生を受けて、米国疾病管理予防センター（CDC）及びFDAは生の芽野菜による感染の危険性を訴え、CDHSと同州教育局（the California Department of Education）は学校給食でのアルファルファ使用停止を提言した。

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Outbreak of *Salmonella* serotype Kottbus Infections Associated with Eating Alfalfa Sprouts --- Arizona, California, Colorado, and New Mexico, February--April 2001

On March 12, 2001, the California Department of Health Services (CDHS) identified a cluster of *Salmonella* Kottbus isolates with indistinguishable pulsed-field gel electrophoresis (PFGE) patterns. During February 1--May 1, CDHS identified 23 patients with *S.* Kottbus infections in several California counties and an additional patient from Arizona. This report summarizes the results of the investigation of this outbreak, which identified cases in four states and implicated alfalfa sprouts produced at a single facility.

The median age of case-patients was 36 years (range: 9--72 years); 16 patients (67%) were female. Twenty-one patients developed an acute diarrheal illness, and three patients had urinary tract infections. Three patients were hospitalized.

Using a standardized questionnaire, a matched case-control study was conducted. A case was defined as culture-confirmed *S.* Kottbus infection with onset after January 2001 in a California resident with an isolate having the outbreak PFGE pattern. The first 10 reported California patients were matched with two controls by age group, sex, and city prefix code. Fifteen (63%) of 23 patients ate alfalfa sprouts during the week before becoming ill. A significant association was found between eating alfalfa sprouts and illness (matched odds ratio: 5.5; 95% confidence interval=1.2--26.1). No other food or restaurant exposure was significantly associated with illness. Following the case-control study, 32 patients infected with the outbreak strain of *S.* Kottbus were identified in California (24), Arizona (six), Colorado (one), and New Mexico (one).

A traceback investigation identified a single sprout producer as the source of the contaminated sprouts. Review of the sprouter's production records indicated that a single seed lot was temporally associated with the dates of illness onset. A culture of

a sample of this seed lot yielded *S.* Kottbus. These seeds were imported from Australia in November 2000, but no further information about the distribution of this seed lot was available. Cultures from two floor drains in the production facility also yielded *S.* Kottbus. Patient, seed, and environmental isolates all had indistinguishable PFGE patterns.

Although the implicated seed lot was last used on March 29, the sprouter issued a voluntary recall of all sprout products on April 17, and ceased all sprout production pending further internal review of their production processes. Review of decontamination and distribution records indicated that at least some seeds underwent heat treatment followed by a 2,000–ppm sodium hypochlorite treatment for 15 minutes. The U.S. Food and Drug Administration (FDA) recommends decontamination of seeds with one or more treatments (e.g., soaking in a 20,000–ppm calcium hypochlorite for 15 minutes) that have been approved for reduction of pathogens in seeds (*1, 2*). The effectiveness of alternative seed decontamination has not been established. The sprout producers subsequently agreed to use only the FDA–recommended 20,000–ppm soak when sprout production resumed.

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Editorial Note:

S. Kottbus is a rarely reported cause of salmonellosis in the United States. During 1968--1998, a median of 42 *S.* Kottbus isolates were reported each year to CDC through the Public Health Laboratory Information System (*3*). This was the second outbreak of *S.* Kottbus since 1985 and the first outbreak associated with sprouts.

Since 1995, 15 outbreaks of *Salmonella* spp. and two outbreaks of *Escherichia coli* O157:H7 infections associated with sprouts have been reported to CDC. Despite public health advisories about the risks for eating raw sprouts, persons at high risk for systemic infection continue to eat sprouts (*4*). Two of the patients in this outbreak

were immunocompromised, and one was a young child. In each case, persons perceived raw sprouts as a "healthy" food item.

Sprouts may be contaminated during seed production, germination, sprout processing, or consumer handling and preparation (*5, 6*). On the farm, sprouts seeds may become contaminated through the use of untreated agricultural water, improperly composted manure as fertilizer, excretion from domestic or wild animals, runoff from domesticated animal production facilities, or improperly cleaned harvesting or processing machines (*5, 6*). The association of specific seed lots with illness suggests that seeds are the most likely source for this and most other sprout-related outbreaks (*4*). Conditions suitable for seed sprouting also are ideal for increasing pathogenic bacterial counts by several logs.

The use of a 20,000-ppm calcium hypochlorite soak before sprouting might reduce the risk for sprout-related illness (*4*). However, use of this high-dose soak is not completely effective, and outbreaks continue to occur (*7*). Cracks and crevasses in the sprout seed may trap pathogenic bacteria, making them inaccessible to lethal concentrations of disinfectants (*5*). Because >20,000-ppm calcium hypochlorite soaks can impair seed germination (*5*), alternative methods are needed to reduce the risk for human disease following sprout consumption. In this outbreak, some of the implicated sprouts were from seeds that had undergone a combination of heat treatment and a 15-minute, low-dose calcium hypochlorite soak (2,000 ppm). The subsequent outbreak suggests that this hybrid technique using a heat treatment combined with a low-dose hypochlorite solution might not reduce adequately pathogenic bacterial colony counts in alfalfa seeds. Reducing pathogenic bacterial counts on seed during production and harvest could improve the effectiveness of postharvest decontamination.

Public education efforts about the risks for eating uncooked sprouts need to be continued, particularly among vulnerable populations (i.e., the elderly, young children, and immunocompromised persons). CDC and FDA recommend that persons at high risk for systemic infections not eat raw sprouts. For persons who continue to eat sprouts, FDA recommends cooking before eating to reduce the risk for illness (*8*).

In response to this outbreak, CDHS and the California Department of Education recommend that schools stop serving uncooked sprouts to young children. Public health officials should promote awareness of the role of raw sprout consumption in

foodborne disease and consider package labeling as a method for improving consumer awareness. In addition, designation of sprout seed production for human consumption at seed planting could further reduce the risk for sprout-associated outbreaks (*5*). If sprout seed producers knew which sprout seed crops were dedicated for human consumption before harvest, producers could focus on reducing potential contamination in the field. Avoiding seed contamination in the field might reduce the risk for consumer exposure to foodborne pathogens.

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摂食方法又は不十分な殻付卵によるサルモネラ（腸炎菌：*S.* Enteritidis）感染症の発生 米国，1996～1998 年

1980～1990 年代にかけて、米国ではサルモネラ（腸炎菌、*Salmonella* Serotype Enteritidis：SE）感染症が人の感染症の重要な原因として浮上していた。米国疾病管理予防センター（CDC）に報告された SE 分離株の検出頻度も、1976 年の 10 万人当たり 0.6 から 1996 年には 3.6 まで増加し、散発及び集団発生における症例対照研究でこの発生増加が生あるいは加熱調理が十分なされていない卵に密接に関連していることが明らかにされた。一方、1996～1998 年に CDC に報告された、培養で確認された SE 患者の罹患率は 2.2（対 10 万人）までに減少したが、SE の集団食中毒は相変わらず発生が続いている。

本報告では、1996～1998 年の間にカリフォルニア州ロサンゼルス郡（1997 年 8 月、患者 13 名、原因食品：チーズケーキ、原因菌：ファージ 4 型 SE）、コロンビア特別区（1997 年 10 月、患者 43 名、原因食品：ラザニア、原因菌：ファージ 8 型 SE）ネバダ州クラーク郡（1997 年 11 月、患者 91 名、原因食品：ブロッコリーのオランデーズソース和え、原因菌：ファージ 13A 型 SE）及びアリゾナ州マリコパ郡（1998 年 7 月、患者 58 名、原因食品：メキシコ料理の chiles rellenos、原因菌：ファージ 6A 型 SE）で発生した 4 件の集団発生例について各州の衛生局が調査した結果を述べると同時に、SE 患者の減少に貢献するであろう制御手法についても考察を加えた。

なお、SE は、米国にて 1980 年代北東部の州で最初に問題視されて以降、全米に拡大し、1985～1998 年の間に計 796 件（患者 28,689 名、入院 2,839 名、死亡者 79 名）の集団発生が報告されている。これらの集団発生のうち原因が特定された事件は 360 件で、そのうち 279 件（82%）は生あるいは加熱調理が十分なされていない卵が関与して発生したものであった。

米国における SE の罹患率及び集団発生をさらに減少させるためには、農場から食卓までの管理を完全とするために複数の対応が求められている。SE 防止問題に向けて、昨年 12 月 10 日、大統領の食品安全諮問委員会は西暦 2005 年までに卵関連の SE による感染症の

半減を目指した卵の安全行動計画を発表した。この行動計画の目的は、消費者のSE含有食品に対する曝露を減少させること、人及び家禽類のSE感染症サーベイランスの質の向上・拡大、SE集団発生の認知及び調査開始を促進するための連邦・州・地域関係部局間のコミュニケーションの改善、調査の推進及び科学的根拠に基づく材料を用いた教育の推進等に焦点を当てたものである。

Editorial Note《編集注記》

卵がSEに関連している場合、州衛生局及び（又は）FDAは発生源の追跡調査を実施し、SEを検出するために鶏舎周辺のサンプリングを行う。

1990年代初頭、米国農務省、卵製品産業、州農業局及び学術界は共同でQAPs計画を実施した。

現在、農務省は卵を45F（7.2℃）以下で貯蔵及び輸送し、消費者は冷蔵保存する旨、表示するよう命じている。2000年までにFDAによって完成する予定の最終案は、小売店での卵の販売は45F（7.2℃）以下の冷蔵保存が行われるよう要請するだろう。

食品小売店、レストラン、療養施設及びデイケア・センターに関するFDAの食品コードは、シーザーサラダ、オランダイズ・ソース、アイスクリーム、熱処理されていない卵滋養強壮飲料等の食品に使われる生の殻付卵の代わりに低温殺菌された卵を使用するよう提唱している。

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Outbreaks of *Salmonella* Serotype Enteritidis Infection Associated with Eating Raw or Undercooked Shell Eggs -- United States, 1996-1998

During the 1980s and 1990s, *Salmonella* serotype Enteritidis (SE) emerged as an important cause of human illness in the United States. The rate of SE isolates reported to CDC increased from 0.6 per 100,000 population in 1976 to 3.6 per 100,000 in 1996 (Figure 1). Case-control studies of sporadic infections and outbreak investigations found that this increase was associated with eating raw or undercooked shell eggs (1). From 1996 to 1998, the rate of culture-confirmed SE cases reported to CDC declined to 2.2 per 100,000; however, outbreaks of illness caused by SE continue to occur. This report describes four SE outbreaks during 1996-1998 associated with eating raw or undercooked shell eggs and discusses measures that may be contributing to the decline in culture-confirmed SE cases.

Los Angeles County, California

In August 1997, the Los Angeles County Department of Health Services (LACDHS) received reports of gastrointestinal illness in members of a Girl Scout troop and some of their parents. The ill persons had eaten food prepared in a private residence by the scouts. Stool cultures taken from 12 ill persons yielded SE; selected isolates tested were phage type 4.

An investigation by LACDHS found that of 17 persons at the dinner, 13 had gastrointestinal illness consistent with salmonellosis. Cheesecake served at the dinner was associated with illness; all 13 ill persons and two well persons ate the cheesecake (attack rate=87%; relative risk [RR] =undefined; p=0.04). The cheesecake contained raw egg whites and egg yolks that were cooked in a double boiler until slightly thickened. California Department of Health Services and Department of Food and Agriculture investigated the farm that supplied the eggs and found SE

contamination. Of 476 environmental cultures taken from manure, feed, and water, 21 (4.4%) yielded SE; all positive cultures were from manure. Nineteen isolates were phage type 4, and two were phage type 7. SE also was isolated from one (0.5%) of 200 pooled egg samples obtained at the farm. On the basis of these findings, the layer flock was depopulated to prevent further SE cases.

District of Columbia

In October 1997, the District of Columbia Bureau of Epidemiology and Disease Control (DCBEDC) received reports of gastroenteritis among 75 attendees at seven events (a workshop dinner, nursing home luncheon, and five meals in private residences) at which lasagna from the same commercial manufacturer was served. Forty-three cases of illness compatible with salmonellosis were identified among attendees. Stool cultures from nine patients yielded *Salmonella* group D or SE; at least one culture-confirmed case was associated with each event. Isolates tested from attendees at five events were phage type 8. Three patients were hospitalized; none died.

DCBEDC interviewed 48 of the 75 attendees. Of the 47 persons who ate lasagna at the events, 39 became ill; the only person who did not eat lasagna did not become ill (attack rate=83%; RR=undefined; p=0.19). Lasagna was not associated statistically with illness but was implicated because it was the only food item common to all events. Cultures of two leftover lasagnas and one lasagna made on the same day but not eaten yielded SE phage type 8. The lasagnas were prepared commercially by a company in Gaithersburg, Maryland, using fully cooked meat or spinach sauce and a mixture of raw shell eggs, ricotta and mozzarella cheeses, and spices. Although the lasagnas were not labeled with a manufacture date, investigators determined that most, if not all, of the lasagnas implicated were made on the same day from a single batch of the egg-cheese mixture. The product was then frozen (except for one event in which the lasagnas were kept refrigerated as a special order) and held without further cooking until purchased. In at least four of six events for which lasagnas were purchased frozen, the lasagna was not thawed before reheating.

A traceback investigation led to two egg processors. Sampling of the farms that supplied eggs to these processors showed that five of 13 poultry houses had environmental samples positive for SE. In compliance with recommendations from DCBEDC, the manufacturer voluntarily switched to using pasteurized eggs in

egg-containing foods.

Clark County, Nevada

In November 1997, 91 persons who ate either of two meals served 2 weeks apart at a hotel restaurant in Las Vegas, Nevada, developed gastroenteritis. Fifteen patients were hospitalized; none died. Stool cultures taken from ill persons yielded SE; selected isolates tested were phage type 13A.

An investigation by the Clark County Health District found 28 culture-confirmed and 63 probable salmonellosis cases. A case was defined as diarrheal illness in a patient who ate at restaurant A on November 13 or November 27. Two separate case-control studies implicated broccoli with hollandaise sauce: one study among persons who ate at the restaurant on November 13 (odds ratio [OR] =25.5; p=0.04) and a second among persons who ate at the restaurant on November 27 (OR=27.8; p=less than 0.001). Broccoli with hollandaise sauce was offered on a special menu that rotated biweekly. The hollandaise sauce was prepared from pooled shell eggs, cooked to a temperature inadequate to kill SE, and kept at room temperature for several hours until served.

Maricopa County, Arizona

In July 1998, 58 persons developed gastroenteritis associated with eating at any of four Mexican restaurants that were part of a local chain. Eleven persons were hospitalized; none died. Stool cultures taken from 22 persons yielded *Salmonella* group D or SE; selected isolates tested were phage type 6A.

An investigation by the Maricopa County Environmental Services Department found that 14 (64%) of the 22 persons with culture-confirmed infections had eaten chiles rellenos, a precooked commercial product. Cultures of chiles rellenos from all four restaurants yielded SE with the same phage type as the patient isolates. The chiles rellenos consisted of raw egg-white batter on roasted green chile peppers stuffed with cheese, and were commercially processed in Mexico where they were cooked, packed, and frozen. Local public health officials observed that the internal temperature of the chiles rellenos was not checked after reheating at the restaurants. Improper foodhandling and cross-contamination were presumed responsible for the other cases among persons who did not consume chiles rellenos. Cultures of chiles rellenos from other lots distributed in the United States also yielded SE. The

distributor of the chiles rellenos voluntarily recalled all products.

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Editorial Note:

SE was recognized as a public health problem in northeastern states during the 1980s and has since spread throughout the United States (1). During 1985–1998, state and territorial health departments reported 796 SE outbreaks that accounted for 28,689 illnesses, 2839 hospitalizations, and 79 deaths (Table 1). Of the 360 SE outbreaks with a confirmed source, 279 (82%) were associated with raw or undercooked shell eggs. This report illustrates that outbreaks can occur because of breakdowns in procedures at multiple steps in egg production from farm to table. SE prevention measures include on-farm control programs, refrigeration, consumer and food worker education about food preparation and consumption, adoption of the Food and Drug Administration (FDA) Food Code (2) in restaurants and institutions, and improved surveillance.

On-farm control measures may include actions resulting from egg traceback investigations and quality assurance programs (QAPs). When eggs are implicated in SE outbreaks, state health departments and/or FDA may conduct tracebacks to identify the source farm(s) and conduct environmental sampling of poultry houses to detect SE. When SE is detected in a house, eggs are voluntarily diverted to pasteurization facilities until egg testing has shown negative results for SE. Tracebacks have been successful in removing potentially contaminated eggs from the market. During the early 1990s, the U.S. Department of Agriculture (USDA), the egg industry, state departments of agriculture, and academia collaborated to design QAPs (3). Elements may include purchasing chicks from SE-monitored breeders, stringent rodent and pest

control, cleaning and disinfecting hen houses, routine environmental cultures with diversion of eggs to pasteurization if SE is found, and proper refrigeration of eggs. Currently, 13 states participate in voluntary QAPs (4). Northeastern states were the first to implement such QAPs. The decrease in SE outbreaks from 61 in 1989 to eight in 1998 (Figure 2), and in sporadic cases in the region may reflect these collaborative preventive efforts.

Ensuring that eggs are sold soon after being produced and that they are kept refrigerated are important steps in reducing egg-associated SE illness. Although required in 17 states, no federal law exists that requires an expiration or "sell-by" date on egg cartons. Currently, USDA requires that eggs be stored and transported at less than or equal to 45 F (less than or equal to 7.2 C) and that consumer containers be labeled to indicate that refrigeration is required (5). A proposed rule scheduled to be finalized in 2000 by FDA also would require that eggs sold at retail stores be refrigerated at less than or equal to 45 F (less than or equal to 7.2 C) (6).

The education of consumers and food service workers to store, handle, and cook eggs appropriately can prevent many SE infections in humans (see box). FDA has a proposed rule that would require safe handling messages on all egg cartons (6). FDA's Food Code for retail food stores, food service establishments, nursing homes, and day care centers recommends that pasteurized eggs be substituted for raw shell eggs in preparing foods such as Caesar salad, hollandaise sauce, ice cream, and egg-fortified beverages that are not cooked (2). The outbreaks described in this report could have been prevented if pasteurized eggs had been used or if the eggs used in the recipes had been cooked fully. FDA's Food Code recommendations are especially important for children, the elderly, immunocompromised persons, and pregnant women who are at increased risk for severe complications from SE infection. The effectiveness of these recommendations and education efforts are demonstrated by the decline in the number of deaths in health-care facilities, particularly nursing homes (Table 1).

Throughout the 1980s, SE phage type 4 emerged as the predominant phage type in Europe, causing a marked increase in human infections. Phage type 4 had not been seen in the United States except among persons who became ill after international travel. In 1993, the first U.S. outbreak of SE phage type 4 infections occurred in Texas (7), and during the next several years, phage type 4 caused human illness in Arizona, California, Hawaii, Nevada, and Utah. Since then, the isolation rate and number of SE outbreaks in the western United States have increased dramatically; most of these

outbreaks have been phage type 4. SE phage type 4 also has been isolated from eggs and the farm environment of laying flocks implicated as sources for human outbreaks in that region (8). CDC monitors the spread of phage type 4 by phage typing isolates from U.S. outbreaks of SE and sporadic cases.

Further reductions in SE incidence and SE-related outbreaks will require multiple interventions along the entire farm-to-table continuum. To address SE prevention issues, on December 10, 1999, the President's Council on Food Safety announced an Egg Safety Action Plan, which calls for a 50% reduction in egg-associated SE illnesses by 2005 (9). The plan's objectives are aimed at reducing consumer exposure to SE-containing foods; expanding and upgrading surveillance systems for human and poultry SE infection; improving communication among federal, state, and local agencies to accelerate SE outbreak detection and initiation of investigations; conducting research; and educating persons using science-based materials.

Additional information about preventing SE infections associated with eating raw or undercooked shell eggs is available on the World-Wide Web at http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salment_g.htm; http://vm.cfsan.fda.gov/~dms/fs-eggs.html; http://vm.cfsan.fda.gov/~dms/fs-eggs2.html; and http://www.foodsafety.gov/~fsg/ceggs.html.

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Box

Recommendations for Preventing *Salmonella* Serotype Enteritidis Infections Associated with Eating Raw or Undercooked Shell Eggs

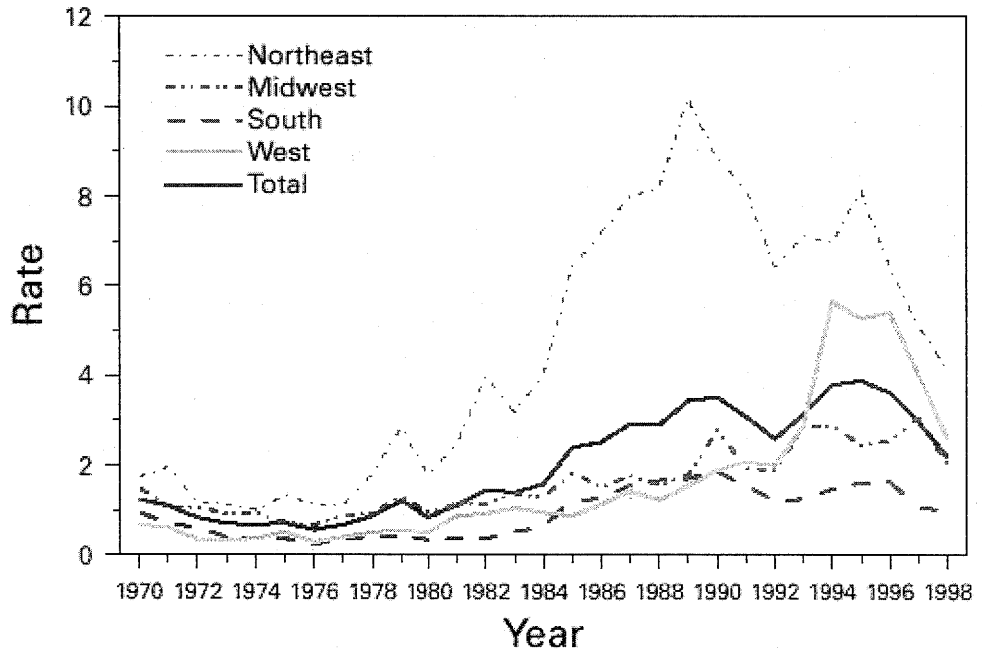
- Eating raw or undercooked eggs should be avoided, especially by young children, the elderly, and immunocompromised persons.
- In hospitals, nursing homes, food service establishments, day care centers, elementary schools, and commercial kitchens, pasteurized egg products should be used in recipes that call for pooled eggs or in which eggs are not thoroughly cooked.
- Eggs should be cooked at greater than or equal to 145 F (greater than or equal to 63 C) for greater than or equal to 15 seconds (until both the yolk and white are firm) and eaten promptly after cooking. Casseroles and other dishes containing eggs should be cooked to 160 F (71 C).
- Hands, cooking utensils, and food–preparation surfaces should be washed with hot water and soap after contact with raw eggs or foods containing raw eggs.
- Eggs should be stored at less than or equal to 45 F (less than or equal to 7.2 C) at all times.

TABLE 1. Characteristics of all outbreaks of *Salmonella* serotype Enteritidis infection and outbreaks in health-care facilities, by year — United States, 1985–1998

	All outbreaks						Outbreaks in health-care facilities*					
			Hospitalizations		Deaths				Hospitalizations		Deaths	
Year	No. outbreaks	No. ill	No.	(%)	No.	(%)	No. outbreaks	No. ill	No.	(%)	No.	(%)
1985	26	1,159	144	(12)	1	(0.09)	3	55	10	(18)	1	(2)
1986	47	1,444	107	(7)	6	(0.42)	6	96	10	(10)	5	(5)
1987	58	2,616	557	(21)	15	(0.57)	8	489	391	(80)	14	(3)
1988	48	1,201	155	(13)	11	(0.92)	8	227	6	(3)	9	(4)
1989	81	2,518	206	(8)	15	(0.60)	19	505	34	(7)	13	(3)
1990	85	2,656	318	(12)	3	(0.11)	12	265	22	(8)	3	(1)
1991	74	2,461	200	(8)	5	(0.20)	8	118	6	(5)	4	(3)
1992	63	2,348	233	(10)	4	(0.17)	2	42	2	(5)	2	(5)
1993	66	2,215	219	(10)	6	(0.27)	6	66	5	(8)	4	(6)
1994	51	5,492	214	(4)	0	—	2	32	6	(19)	0	—
1995	56†	1,312	113	(9)	8	(0.61)	6	147	19	(13)	6	(4)
1996	50	1,460	159	(11)	2	(0.14)	3	64	9	(14)	0	—
1997	44	1,098	124	(11)	0	—	1	13	1	(8)	0	—
1998	47	709	90	(13)	3	(0.42)	3	32	6	(19)	3	(9)
Total	**796**	**28,689**	**2,839**	**(10)**	**79**	**(0.28)**	**87**	**2,151**	**527**	**(25)**	**64**	**(3)**

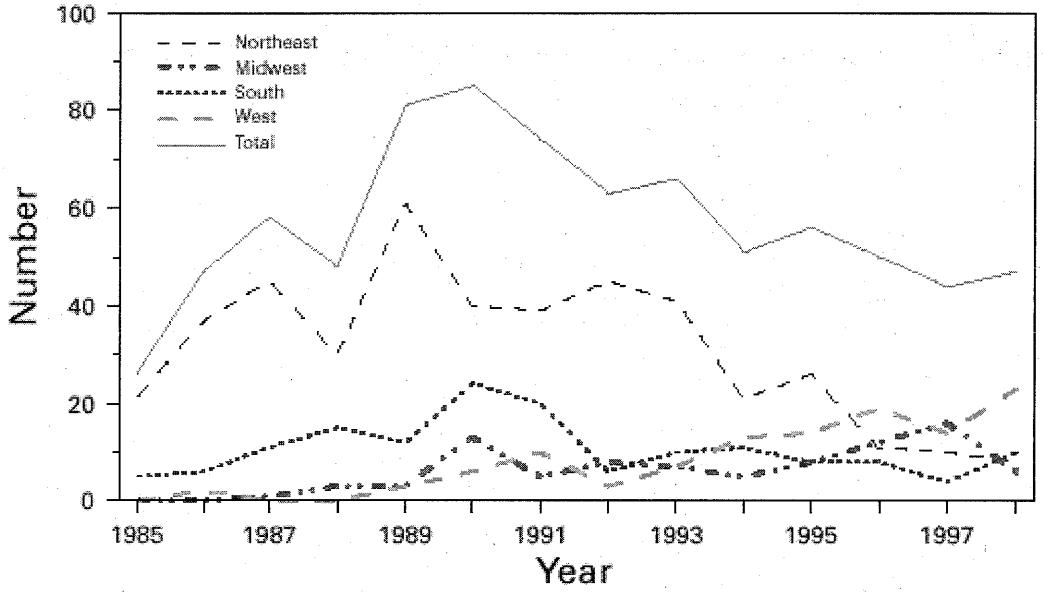
* Includes hospitals and nursing homes.
† Includes one outbreak associated with a Komodo dragon exhibit at a zoologic park.

FIGURE 1. Rate* of isolation of *Salmonella* serotype Enteritidis, by region† and year — United States, 1970–1998



*Per 100,000 population.
†*Northeast*=Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont; *Midwest*=Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin; *South*=Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia; *West*=Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.

FIGURE 2. Number of *Salmonella* serotype Enteritidis outbreaks, by region* and year — United States, 1985–1998†



**Northeast*=Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont; *Midwest*=Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin; *South*=Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia; *West*=Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.*

†n=796.

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未殺菌オレンジジュースによるサルモネラ（*S.* Muenchen）感染症の発生 米国及びカナダ，1999 年 6 月

1999 年 6 月、シアトル及びキング郡の公衆衛生当局（PHSKC）、ワシントン州及びオレゴン州衛生局はそれぞれ独立に両州で発生したサルモネラ（*S.* Muenchen）感染症の発生事件について調査した結果、両感染症発生は全米で市販されている 1 社の未殺菌オレンジジュースに関連していることが判明した。そして、7 月 13 日までに、全米 15 州及びカナダの 2 地域から計 207 名の患者が報告された。この他、7 月 1 日までに報告された 91 名の事例についても調査中である。本報告では、2 州における調査結果並びに他の州、カナダにおける感染症発生の暫定調査結果について紹介する。

ワシントン州：6 月 19 日、3 例のサルモネラ（*S.* Muenchen）感染症患者の発生が州衛生局に報告された。感染症患者に聞き取り調査を実施した所、これらの患者は共通してレストラン A の異なる系列小売店でそれぞれ未殺菌オレンジジュースの入ったフルーツ・スムージーを飲んでいることが判明し、PHSKC 及びワシントン州衛生局により調査が開始された。PHSKC による対照研究の結果、7 月 9 日までに計 85 名の患者発生が確認され、その 67 名は Sun Orchard 製の未殺菌オレンジジュースを飲用しているか、あるいは本ジュースを提供したレストランで食事をしていることが明らかとなった。患者の平均年齢は 27 才（範囲：9 か月～95 才）、51%が男性で、報告された主要症状は下痢（94%）、発熱（75%）、血清下痢（43%）であった。8 名が入院したが死亡者はいなかった。

オレゴン州：6 月 23 日に 1 例のサルモネラ（*S.* Muenchen）感染症患者の発生報告を受けた州衛生局による患者調査で、ポートランドのブランチビュッフェで食事をしたグループ中に患者が 4 名発生していること、また患者発生は Sun Orchard 製の未殺菌オレンジジュースの飲用と密接に関連していることが判明した。その後の調査で、7 月 12 日までに計 57 名の患者が発見された。患者の平均年齢は 36 才（範囲：9 か月～95 才）、54%が男性で、54 名が発症前に未殺菌オレンジジュースを飲用していた。39 名の患者から報告された主要症状は下痢（100%）、発熱（89%）、腹痛（85%）、悪寒（82%）及び血清下痢（59%）で、7 名が入院したが、死亡者はいなかった。

オレンジジュースの回収：Sun Orchard 社は、ワシントン及びオレゴン両州の疫学調査結果並びに米国食品医薬品局（FDA）との協議に基づき、6 月 25 日、自主的に製品の回収を開始した。同社で製造された未殺菌オレンジジュースは、アリゾナなど全米 10 州及びカナダの 2 地域で種々なブランド名で販売され、それ以外の地域でもこれらの製品が 2 次的な流通経路を介して販売されていた。6 月 28 日、FDA 及びワシントン州衛生研究所で実施された未開封製品の検査で、サルモネラ（*S.* Muenchen）が、またレストラン A 小売店のスムージー・ブレンダー及びジュース・ディスペンサーからも同一の菌が検出され、これらのパルス・フィールド・ゲル電気泳動（PFGE）パターンも患者株のそれと一致した。現在、汚染源などにつき調査中である。

他の州及びカナダ：ワシントン州及びオレゴン州以外に、カリフォルニア州（21 名）、ニューメキシコ州（10 名）等、13 州で計 66 名、またカナダのアルバータ及びブリティッシュコロンビアで 12 名の患者が確認された。この他、9 州及びカナダの 2 地域から報告された 78 名についても現在調査中である。

Editorial Note《編集注記》

1998 年に、FDA はジュース製品の安全性向上を目的として、危害分析重要管理点（HACCP）方式及びラベル規定を提案した。提案された HACCP 規定は、微生物病原体の壊滅を確かなものにするために、低温殺菌又は同等の工程を用いて生産するよう命じている。FDA は果物や野菜の非加工ジュースに関連するリスクを消費者に勧告するための警告書を添付するよう、最終的なラベル規定を発行した。

July 16, 1999 / 48(27); 582-585

Outbreak of *Salmonella* Serotype Muenchen Infections Associated with Unpasteurized Orange Juice -- United States and Canada, June 1999

During June 1999, Public Health-Seattle and King County (PHSKC) and the Washington state health department and the Oregon Health Division independently investigated clusters of diarrheal illness attributed to *Salmonella* serotype Muenchen infections in each state. Both clusters were associated with a commercially distributed unpasteurized orange juice traced to a single processor, which distributes widely in the United States. As of July 13, 207 confirmed cases associated with this outbreak have been reported by 15 states and two Canadian provinces; an additional 91 cases of *S.* Muenchen infection reported since June 1 are under investigation. This report summarizes the two state-based investigations and presents preliminary information about the outbreak in the other states and Canada.

Washington

On June 19, state health officials were notified of three cases of *Salmonella* serogroup C2 infection, which were confirmed subsequently as *S.* Muenchen. Interviews of the ill persons revealed one common feature: drinking a fruit smoothie containing unpasteurized orange juice from different outlets of restaurant chain A. PHSKC and the Washington State Department of Health initiated an investigation. A case was defined as illness with onset after June 9, with isolation of *S.* Muenchen from stool or blood or isolation of *Salmonella* serogroup C2 with a pulsed-field gel electrophoresis (PFGE) or restriction fragment length polymorphism pattern that was indistinguishable from the outbreak strain.

In a case-control study by PHSKC of nine ill and 29 well restaurant A patrons, illness was significantly associated with drinking smoothies containing orange juice (100% of cases exposed compared with 14% of controls; odds ratio=undefined, pless than 0.001).

By July 9, 85 persons with onset of illness during June 10–30 were identified in Washington. Sixty-seven patients reported either drinking unpasteurized orange juice produced by Sun Orchard* of Tempe, Arizona or eating at an establishment where the juice was served. Among 79 patients for whom information was available, the median age was 27 years (range: 9 months–95 years), and 51% were male. The predominant symptoms reported were diarrhea (94%), fever (75%), and bloody diarrhea (43%). Eight (10%) patients were hospitalized, and one man had a stroke coincident with his *Salmonella* infection. No patients died.

Oregon

On June 23, the Washington County Department of Health received a report of a case of salmonellosis; the isolate was serotyped subsequently as *S.* Muenchen. An investigation by the Oregon Health Division identified four ill persons among a group of 13 that had eaten a brunch buffet in Portland. A case was defined as diarrhea (three or more loose stools within 24 hours) or vomiting in a person who attended the buffet. Illness was significantly associated with drinking unpasteurized orange juice produced by Sun Orchard (relative risk=undefined; pless than 0.001).

By July 12, 57 persons with *S.* Muenchen infection with onset of illness during June 14–29 were identified in Oregon. The median age was 36 years (range: 9 months–95 years), and 54% were female. Forty-four patients were known to have drank unpasteurized orange juice before illness onset. Among the 39 patients for whom information was available, the predominant symptoms were diarrhea (100%), fever (89%), abdominal cramps (85%), chills (82%), and bloody diarrhea (59%). Seven persons were hospitalized; no patients died.

Recall of Orange Juice

On June 25, on the basis of the epidemiologic information from the investigations in Washington and Oregon and discussions with the Food and Drug Administration (FDA), Sun Orchard voluntarily issued a recall. Unpasteurized orange juice produced by Sun Orchard is distributed to Arizona, California, Colorado, Nevada, New Mexico, Oregon, Texas, Utah, Washington, Wisconsin, and the Canadian provinces of Alberta and British Columbia under the brand names Aloha, Earls and Joeys Tomato's, Markon, Sysco, Trader Joe's, Voila, and Zupan. Other states and provinces received these products through secondary distribution. The juice was distributed to hotels,

restaurants, and supermarkets, and was served in individual glasses as "fresh-squeezed" juice in hotels and restaurants. In addition, a frozen form of the unpasteurized juice was sold under the brand name Vareva for use in restaurants and institutions.

On June 28, samples from a previously unopened container of unpasteurized Sun Orchard orange juice analyzed at an FDA laboratory and the Washington State Public Health Laboratory yielded *S.* Muenchen; samples from the smoothie blender and juice dispenser at an outlet of restaurant A analyzed by the Washington State Public Health Laboratory yielded *Salmonella* serogroup C2. Isolates from both sources had a PFGE pattern that was indistinguishable from strains isolated from patients. Subsequently, orange juice collected from the Sun Orchard factory, cultured in an FDA laboratory and serotyped by the California State Public Health Laboratory, yielded *S.* serotype Javiana, *S.* serotype Gaminara, *S.* serotype Hidalgo, and *S.* serotype Alamo in addition to *S.* Muenchen. Efforts are ongoing to determine the source of all orange juice components, whether they might have been used in other brands, and the source of the *Salmonella* contamination.

Other States and Canada

An outbreak-related case was defined as *S.* Muenchen infection after June 1 in a person who drank unpasteurized orange juice or whose isolate had a PFGE pattern with no more than one band difference from the Washington outbreak strain. In addition to the Washington and Oregon cases, 66 cases were reported in persons in 13 other states: Arizona (four), California (21), Connecticut (one), Florida (one), Illinois (one), Iowa (two), Massachusetts (seven), Michigan (three), Minnesota (six), New Mexico (10), Texas (five), Utah (four), and Wisconsin (one). Cases also were reported from the Canadian provinces of Alberta (four) and British Columbia (eight). Among the 66 patients for whom information was available, the median age was 32 years (range: 6 months-66 years), and 58% were female. Six persons were hospitalized. An additional 78 cases of *S.* Muenchen infection occurring after June 1 reported by nine other states and the two Canadian provinces are under investigation.

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Editorial Note:

S. Muenchen is one of approximately 2400 *Salmonella* serotypes that can cause illness in humans. *Salmonella* infection typically causes gastroenteritis characterized by diarrhea, abdominal cramps, fever, and dehydration. Bacteremia, meningitis, osteomyelitis, and abscesses also can occur. Each year in the United States, 800,000–4 million *Salmonella* infections result in approximately 500 deaths (1). *S.* Muenchen is an infrequently isolated serotype, accounting for approximately 1.6% of human *Salmonella* isolates reported in 1997 to the Public Health Laboratory Information System (2, 3). Oregon typically reports less than 6 isolates per year and Washington less than 10 per year.

Juice has been implicated as the vehicle of transmission in at least 15 outbreaks in the United States in this century involving pathogens, including *Escherichia coli* O157:H7, *Cryptosporidium parvum*, and other *Salmonella* serotypes (e.g., *S.* Typhi and *S.* Hartford) (4). In an outbreak of *E. coli* O157:H7 infections attributed to unpasteurized apple juice, one child died, and 14 children developed hemolytic uremic

syndrome (5). The outbreak described in this report is the second and largest *Salmonella* outbreak associated with unpasteurized orange juice (6). The acidic nature of orange juice (pH of 3.4–4.0) previously was believed to inhibit bacterial growth and protect against foodborne illness; however, recent outbreaks and laboratory investigations have demonstrated otherwise. *Salmonella* serotypes Gaminara, Hartford, Rubislaw, and Typhimurium have survived in orange juice for up to 27 days at pH 3.5 and 60 days at pH 4.1 (7).

In 1998, FDA proposed Hazard Analysis and Critical Control Point (HACCP) and labeling regulations to improve the safety of juice products (8). The proposed HACCP regulation requires juice to be produced using methods such as pasteurization or an equivalent process to ensure that pathogenic microorganisms are destroyed. In the outbreak described in this report, the implicated company had a HACCP plan. Investigations are under way to determine where these control measures failed and how the juice became contaminated. FDA published a final rule for the labeling of fruit and vegetable juices that includes a warning statement to advise consumers of the risks associated with drinking unprocessed juices (9). However, the labeling requirements do not apply to juice or products containing juice that are not packaged (i.e., sold by the glass) in retail establishments, such as the product implicated in this outbreak. In Washington, some consumers were unaware that they were drinking unpasteurized commercial orange juice in their fruit smoothies.

Because the source of contamination of the orange juice is unknown and to facilitate outbreak investigation, local and state health departments are encouraged to investigate all cases of *S.* Muenchen infections occurring since June 1 using a questionnaire from CDC's Foodborne and Diarrheal Diseases Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, telephone (404) 639–2206, and to consider referring isolates for PFGE with the standardized PulseNet *Salmonella* protocol by the Washington State Public Health Laboratory or by another PulseNet laboratory. Health departments also should consider investigating cases of *S.* Alamo, *S.* Gaminara, *S.* Hidalgo, and *S.* Javiana in which illness onset occurred after June 1.

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MMWR47 (22):462-464, 1998

複数の州における焼きオートムギシリアルを原因とするサルモネラ（*S.* Agona）感染症の発生 米国，1998 年 4 月～5 月

1998 年 4 月～5 月、全米の 11 州からサルモネラ（*S.* Agona）感染症が増加していることが報告された。6 月 8 日現在、11 州における報告患者数は 1993 年～1997 年同期の発生件数に比し 8 倍増の 209 名（入院 47 名）を数えた（イリノイ州 49 例、インディアナ州 30 例、オハイオ州 29 例、ニューヨーク州 24 例、ミズリー州 22 例、ペンシルバニア州 20 例、ミシガン州 15 例、アイオワ州 8 例、ウィスコンシン州 6 例、カンザス州 4 例、ウエストバージニア州 2 例)。本報告は、地方・連邦衛生当局によって実施された本集団発生の調査結果をまとめたものであり、Malt-O-Meal 社によって製造された焼きオートムギシリアルがその原因と推定された。

情報の得られた患者 162 名の内、85 名（52%）は女性で、患者の多くは子供又は高齢者（患者の 47%は 10 才以下、21%は 70 才以上)、患者発生は 5 月が主体であった。6 月 8 日現在までに、11 州の衛生当局及び米国疾病管理予防センター（CDC）により実施された 55 家族に対する患者－対照研究（サルモネラ（*S.* Agona）感染症患者と実験対照家族を比較。シリアル喫食と疾病の関連性を条件付き線形ロジスティック回帰により調査した）では、そのうち 46 家族（84%）が Aldi スーパーマーケットでショッピングを、また疾病発症前 3 日間に患者 47 名中 31 名（66%)、対照家族 89 名中 32 名（36%）が Aldi スーパーマーケットで購入した焼きオートムギシリアルを喫食していたことが判明した。

CDC による培養検査で、患者の家に残っていた開封された焼きオートムギシリアルからサルモネラ（*S.* Agona）が検出され、そのパルス・フィールド・ゲル電気泳動（PFGE）パターンは、患者が持つ菌株の主体を占めたパターンと酷似することが確認された（米国食品医薬品局（FDA）でも未開封のサンプル 2 件からサルモネラ（*S.* Agona）を検出)。一方、ミネソタ衛生・農業当局、FDA、CDC による Malt-o-Meal 社の汚染源調査では、同じ生産ラインで時期を異にして種々のブランドの焼きオートムギシリアルを製造していた。同社では、同じ生産ラインで製造された全シリアル製品を自主的に回収し、現在同社の異なる

ブランドのシリアル製品の汚染についても調査中である。なお、これまでにカリフォルニア（11名）、ワシントン（9名）、ニュージャージー（5名）、テネシー（3名）、オクラホマ（3名）、アイダホ（2名）、メリーランド（2名）、ミネソタ（2名）、ネブラスカ（1名）、コネチカット（1名）の各州からもサルモネラ（*S.* Agona）の感染事例が報告され、今回の事件との関連性について調査が進行中である。

Editorial Note《編集注記》

予期していなかったサルモネラ（*S.* Agona）感染症の増加に関して、各州とCDC間の時宜にかなった連携が広域集団感染症の特定を導いた。国立電子研究所（Electric national laboratory）のサルモネラ感染症に関するレポートが、当該事故発生地域の特定を促進した。連邦、州、地方機関の共同調査、CDCとの連携、公衆衛生研究機関の情報システム（PHLIS）を通じた電子レポート、PulseNetを用いた分離株関連の特定は、複数の州間にまたがる食品原因事故の認知及び調査にとっての重要な要素である。

June 12, 1998 / 47(22); 462-4

Multistate Outbreak of *Salmonella* Serotype Agona Infections Linked to Toasted Oats Cereal -- United States, April-May, 1998

During April-May 1998, a total of 11 states reported an increase in cases of *Salmonella* serotype Agona infections; as of June 8, a total of 209 cases have been reported and at least 47 persons have been hospitalized, representing an eightfold increase over the median number of cases reported in those states during 1993-1997. The states reporting increases were Illinois (49 cases), Indiana (30), Ohio (29), New York (24), Missouri (22), Pennsylvania (20), Michigan (15), Iowa (eight), Wisconsin (six), Kansas (four), and West Virginia (two). This report summarizes the outbreak investigation by local, state, and federal public health officials, which implicated Millville brand plain Toasted Oats cereal manufactured by Malt-O-Meal, Inc. as the cause of illness.

Among 162 patients in this outbreak for whom information was available, 85 (52%) were female. Most cases occurred in children and the elderly (47% in persons aged less than 10 years and 21% in persons aged greater than 70 years). Most illnesses began in May.

Officials in the 11 state health departments, in collaboration with CDC, conducted a matched case-control study comparing persons with cases of *S.* Agona infection in April and May with well household members (controls); conditional linear logistic regression was used to examine the relation between consumption of cereal and illness. As of June 8, information from 55 households has been analyzed; 46 (84%) of these 55 households shopped at an Aldi supermarket. During the 3 days before onset of illness, 31 (66%) of 47 patients and 32 (36%) of 89 household controls consumed Millville brand plain Toasted Oats cereal purchased at an Aldi supermarket (matched odds ratio=22; p=0.003). This association remained significant when controlled for age (pless than 0.05). When average daily consumption of Millville brand plain Toasted

Oats cereal purchased from an Aldi supermarket was categorized into three groups (no consumption, less than or equal to 1 cup, and greater than 1 cup), a significant dose response relation was found (p=0.003).

Culture of an open box of Millville brand plain Toasted Oats cereal obtained from the home of a case-patient yielded *Salmonella* Agona at CDC. The pulsed-field gel electrophoresis (PFGE) pattern of this isolate was indistinguishable from the predominant PFGE pattern among outbreak-associated clinical isolates. The Food and Drug Administration (FDA) isolated *Salmonella* Agona from two separate composite samples from unopened boxes. Clinical isolates were susceptible to all antimicrobial agents tested (i.e., ampicillin, trimethoprim-sulfamethoxazole, and ciprofloxacin).

The Minnesota Department of Health, the Minnesota Department of Agriculture, FDA, and CDC are collaborating in the investigation of the Malt-O-Meal, Inc. plant that manufactured the implicated cereal to determine the source of contamination. At this plant on the same production line, multiple brands of plain Toasted Oats are manufactured at different times. Malt-O-Meal has issued a voluntary recall of all plain Toasted Oats cereal produced on the same production line. Investigation is ongoing to determine whether other plain Toasted Oats cereal brands produced by the same company were contaminated. Cases of *Salmonella* Agona infection occurring during the same time have now been reported in California (11), Washington (nine), New Jersey (five), Tennessee (three), Oklahoma (three), Idaho (two), Maryland (two), Minnesota (two), Nebraska (one), and Connecticut (one). These cases are being investigated to determine possible links to this outbreak. CDC recommends that consumers not eat plain Toasted Oats cereal produced by Malt-O-Meal until further investigation has identified the scope, magnitude, and cause of the contamination. Questions about plain Toasted Oats cereals manufactured by Malt-O-Meal should be directed to the company, telephone (800) 590-1810.

Reported by: *State and local health depts. Office of Regulatory Affairs, and Center for Food Safety and Applied Nutrition, Food and Drug Administration. Foodborne and Diarrheal Diseases Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.*

Editorial Note:

Salmonella Agona is one of approximately 2000 *Salmonella* serotypes that can cause illness in humans. An estimated 2–4 million cases of salmonellosis occur in the United States each year, resulting in greater than or equal to 500 deaths (1). Approximately 40,000 of these infections are culture confirmed, serotyped, and reported to CDC by state health departments (1). *Salmonella* infections usually resolve in 5–7 days and do not require antibiotic treatment. Persons with severe diarrhea may require rehydration with intravenous fluids. Antibiotics are required when infection spreads from the intestinal tract. *Salmonella* Agona is an uncommon serotype of *Salmonella*, accounting for approximately 1.5% of human isolates reported to the Public Health Laboratory Information System (PHLIS) (2). Like most other *Salmonella* serotypes, *Salmonella* Agona is found in a variety of animal reservoirs including poultry, cattle, pigs, and animal feed. The first reported U.S. outbreak of *Salmonella* Agona infections was traced to animal feed made with contaminated imported fishmeal in 1972 (3); other outbreaks have been attributed to dried milk (4) and to a commercial peanut-flavored snack (5). This outbreak represents the first time a commercial cereal product has been implicated in a *Salmonella* outbreak, although an infant cereal product was implicated in an outbreak of *Salmonella* senftenberg in the United Kingdom (6). *Salmonella* spp. are relatively resistant to desiccation and can survive for long periods in dry environments such as cereal (7).

Timely communication among the states and CDC about unexplained local increases in *Salmonella* Agona infections, and the relative rarity of this serotype, led to the identification of this multistate outbreak. Electronic national laboratory-based reporting of *Salmonella* infections facilitated prompt recognition of the extent of the outbreak. Cooperative investigations among federal, state, and local agencies, coordination by CDC, electronic reporting through PHLIS, and the rapid identification of related isolates using PulseNet (the national network of public health laboratories that perform DNA "fingerprinting" on foodborne bacteria) are critical components in the recognition and investigation of multistate foodborne outbreaks.

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汚染された上水によるプレシオモナス・シゲロイデスとサルモネラ（*S.* Hartford）感染症 ニューヨーク州リビングストン郡，1996 年

1996 年 6 月 24 日、ニューヨーク州リビングストン郡の保健部門（LCDOH）は 6 月 22 日に私邸において開催されたパーティーの参加者のうち、30 名が集団下痢症を発症したとの報告を受け、病原とされるプレシオモナス・シゲロイデスとサルモネラ（*S.* Hartford）に汚染された上水に関する調査を行った。

このパーティーは 189 人が参加し、地元のコンビニエンスストアーにより料理が供給された。当該コンビニエンスストアーは、6 月 23 日にも 2 件のパーティーに食品ケータリングサービスを行っており、LCDOH はこれら 3 件のパーティー主催者と連絡を取り、他にはどのような疾病も発症していないことを確認した。病原、感染の規模と汚染のメカニズムを確定するため、LCDOH はコホート（集団）調査（コホート群研究法）、環境調査、便の標本（糞便検体）と残りの食物と水のサンプルの生物学的調査（細菌試験）を行った。当日のメニューと参加者リストを入手し、電話で招待客に聞き取り調査し、また料理を提供した店と開催側の雇用者から料理準備に関する情報を得て水源を詳細に調査した。出席者 189 人のうち 98 人が聞き取り調査を受け、56 人に 72 時間以内に下痢の症状が出ていた。便の標本は 14 患者から得られ、9 人からプレシオモナス・シゲロイデス、3 人からサルモネラ（*S.* Hartford）、2 人からその両方が発見された。サルモネラ（*S.* Hartford）の患者 1 人が入院した。料理の中でマカロニサラダ、ポテトサラダ、ベイクドゾティー（baked ziti）の 3 種類が感染に関連していた。マカロニサラダを食べた 56 人の出席者のうち 43 人が、ポテトサラダを食べた 49 人のうち 36 人が、ベイクドゾティーを食べた 46 人のうち 36 人が感染した。6 月 25 日に残された料理が回収され、顕微鏡検査に回された。サルモネラ（*S.* Hartford）はマカロニサラダとベイクドゾティーから分離され、サルモネラ（*S.* Hartford）とプレシオモナス・シゲロイデスはポテトサラダから分離された。ニューヨーク州農業・流通部門（the New York State Department of Agriculture and Markets）は食品提供施設から 9 つの衛生違反（Sanitary Violations）を確認した。また、大腸菌が 6 月 27 日に店の

蛇口の水から分離された。この店は約 10 フィートの深さの井戸が水源であり、周囲の肥料を施された耕地から水が供給されていた。また、家禽農場が約 1600 フィート上流に存在し、その農場の排水は井戸の上方の小川へと注いでいた。27 日に集められた水のサンプルからは塩素の残留物が発見されず、イベント時に塩素消毒がされていなかったことが判明した。19 日と 20 日の雨により病原菌が農場から運ばれ、井戸の水を汚染し、適切に機能していない消毒機により病原菌が調理中の料理に入り込んだと考えられた。感染症発生の後、適切な水の浄化を行うまでこの店は調理を禁止され、店の従業員と一般人はこの水を飲まないように勧告された。

May 22, 1998 / 47(19); 394-396

Plesiomonas shigelloides and *Salmonella* serotype Hartford Infections Associated with a Contaminated Water Supply -- Livingston County, New York, 1996

On June 24, 1996, the Livingston County (New York) Department of Health (LCDOH) was notified of a cluster of diarrheal illness following a party on June 22, at which approximately 30 persons had become ill. This report summarizes the findings of the investigation, which implicated water contaminated with *Plesiomonas shigelloides* and *Salmonella* serotype Hartford as the cause of the outbreak.

The party was held at a private residence on June 22 and was attended by 189 persons. Food was provided by a local convenience store that sells gasoline, packaged goods, sandwiches, and pizza and prepares food for catered events. The convenience store had not catered any parties during the preceding 5 days but catered two parties on June 23. LCDOH contacted the organizers of these events and found no other reports of illness.

To determine the source and extent of the outbreak and mechanism of contamination, LCDOH conducted a cohort study, an environmental investigation, and micro-biologic examinations of stool specimens, leftover food items, and water samples. A menu and guest list were obtained and guests were interviewed by telephone. A probable case was defined as diarrhea (greater than 3 loose stools during a 24-hour period) in a person who attended the party and became ill within 72 hours. Persons with a confirmed case had either *Plesiomonas shigelloides* or *Salmonella* serotype Hartford or both isolated from stool. The caterer and facility employees were interviewed to obtain information on food preparation, and the water source was inspected.

Of the 189 attendees, 98 (52%) were interviewed. Sixty persons reported illness; 56 (57%) of 98 respondents had illnesses meeting the case definition. The mean age for case-patients was 41 years (range: 2-85 years), and 32 (57%) were male. Stool

specimens were obtained from 14 ill attendees: nine yielded only *P. shigelloides*, three only *Salmonella* serotype Hartford, and two had both organisms. One person with culture-confirmed *Salmonella* serotype Hartford was hospitalized. The clinical profiles of the culture-confirmed (n=14) and probable (n=42) cases were similar.

Twenty food and beverage items were served at the party. Three food items were associated with illness: macaroni salad, potato salad, and baked ziti. Of 56 attendees who ate macaroni salad, 43 (77%) became ill, compared with 17 (40%) of 42 who did not eat macaroni salad (relative risk {RR} =2.6; 95% confidence interval {CI} =1.5-4.4). Of 49 guests who ate potato salad, 36 (73%) became ill, compared with 20 (44%) of 45 who did not eat potato salad (RR=2.1; 95% CI=1.2-3.6). Of 46 attendees who ate baked ziti, 36 (78%) became ill, compared with 20 (42%) of 48 that did not eat baked ziti (RR=2.7; 95% CI=1.5-4.9).

Leftover food samples of these three items were collected on June 25 and sent for microbiologic examination. *Salmonella* serotype Hartford was isolated from the macaroni salad and baked ziti. Both *Salmonella* serotype Hartford and *P. shigelloides* were isolated from the potato salad. *Escherichia coli* was isolated from a water sample collected on June 27 from the tap in the store. Water samples collected on July 8 from the well that supplied water to the store contained both *Salmonella* serotype Hartford and *P. shigelloides*.

Preparation of the salads and the baked ziti began on June 21, and prepared food items were stored in a walk-in cooler overnight. On June 22, the ziti was prepared by heating the tomato sauce, pouring it over the meat and pasta, and heating in an oven for 50 minutes at an unknown temperature. The ziti remained in the oven with the heat off until it and the salads were transported to the party.

All foodhandlers denied gastrointestinal illness with onset before June 22. However, three foodhandlers reported illness beginning after June 22; all three reported having eaten foods prepared for the party. *P. shigelloides* was recovered from stool specimens from these three workers only.

The New York State Department of Agriculture and Markets found nine sanitary violations at the caterer's facilities. The water source, an unprotected dug well approximately 10 feet deep, served only the store. The well was fed by shallow ground water and may have received surface runoff from surrounding tilled and manured farm

land and water from adjacent streams. A small poultry farm was located approximately 1600 feet upstream of the well. Farm field drainage systems discharged into the source water stream just above the well. A water sample collected at the store on June 27 showed no chlorine residual, indicating that the pellet chlorinator was off-line at the time of the event. The pellet chamber was empty and the system did not contain any filtration mechanism. Well water used for food preparation (i.e., rinsing pasta used in salads, mixing ingredients, cooking food items, and cleaning equipment) was probably contaminated as a result of rainfall on June 19 and June 20 that transported pathogens from the surrounding farmland. The improperly maintained chlorinator allowed these pathogens to reach the food preparation area. After the outbreak, the store was prohibited from preparing food until an adequate water-treatment system that met drinking water standards could be provided. Store employees and the public were instructed not to drink the water.

Reported by: *R Van Houten, D Farberman, J Norton, J Ellison, Livingston County Dept of Health, Mt. Morris; J Kiehlbauch, PhD, T Morris, MD, P Smith, MD, State Epidemiologist, New York State Dept of Health. Foodborne and Diarrheal Diseases Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.*

Editorial Note

The findings in this report implicated a deficient water supply system as the cause of an outbreak of diarrheal illness caused by *Salmonella* serotype Hartford and *P. shigelloides*. Unfiltered, untreated surface water led to contamination of food during its preparation.

Most infections with *P. shigelloides* have been associated with drinking untreated water, eating uncooked shellfish, or with travel to developing countries (1-3). *P. shigelloides* (previously *Aeromonas shigelloides*) are ubiquitous, facultatively anaerobic, flagellated, gram-negative rods (3). Although they are widespread in the environment, few waterborne or foodborne outbreaks have been reported (4). *P. shigelloides* have been isolated from a variety of sources, including wild and domestic animals (2). Infection is characterized by self-limited diarrhea with blood or mucus, abdominal cramps, and vomiting or fever (5). Symptoms usually occur within 48 hours of exposure. Fecal leukocytes and erythrocytes have been found on stool smears (1); however, the exact mechanism of the diarrhea (secretory versus inflammatory) is unknown.

Salmonella serotype Hartford is a rare serotype that has been isolated from porcine and bovine sources. In May 1995, freshly squeezed, unpasteurized commercial orange juice was implicated as the cause of an outbreak (6). Contamination was thought to have originated from inadequate sanitization of the exterior surfaces of oranges.

In this outbreak, the well water most likely became contaminated with both *P. shigelloides* and *Salmonella* serotype Hartford through runoff from nearby farms. The outbreak could have been prevented if effective public health measures had been in place. Routine testing of well water for total fecal coliform bacteria, turbidity, and chlorine residual may enable early detection of fecal contamination and rapid decontamination. Filtration and chlorination of potable water systems have substantially reduced waterborne outbreaks and subsequent morbidity and mortality. Where possible, water sources subject to contamination from agricultural runoff should not be used for drinking or food preparation. Disinfection and filtration of water from any source can further reduce the risk for waterborne illness.

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MMWR50 (26):560-562, 2001

メキシコ風自家製チーズによるリステリア症の発生 ノースカロライナ州，2000年10月～2001年1月

2000年11月、ノースカロライナ州ウィンストンーセーラムのある病院は、2週間という期間内に3例のリステリア症患者を収容した。患者はいずれも最近移民したばかりのメキシコ人で、同月13日に病院は地元の保健局に報告を行った。ノースカロライナ州保健医療サービス局公衆衛生部門疫学部（the North Carolina General Communicable Disease Control Branch）は、フォーサイス郡保健局、同州農業・消費者サービス局（Department of Agriculture and Consumer Services : NCDA&CS)、同州自然環境・資源局、米国食品医薬品局（FDA)、米国疾病管理予防センター（CDC）の協力を得てリステリア菌（リステリア・モノサイトゲネス）の調査を行った。

2000年10月24日から2001年1月1日に、ウィンストンーセーラムの住民12例でリステリア菌感染を確認した。12例全例がラテンアメリカ系であり、11例（18～38歳）はメキシコ生まれの女性であった。そのうち10例は妊娠中で、リステリア菌感染により5例は死産となり、3例は未熟児を出産し、2例の新生児では感染がみられた。感染源は、地方の酪農場から直接購入した非加工乳による自家製のメキシコ風フレッシュソフトチーズと推定された。

この仮定の下、NCDA&CSは、リステリア菌汚染の原因を探るために、ある加工品質等級済の酪農場（manufacturing grade dairy farm）を調査した。同局は、49頭の乳牛から、そして酪農場の一時的貯蔵タンク（bulk milk storage tank）から牛乳のサンプルを採取した。しかしテスト結果から、乳牛はリステリア菌に感染していないことを明らかになり、環境汚染が原因である可能性を示唆した。

今回の感染症発生の結果、ノースカロライナ州の保健に関する政府諸機関は一般人が酪農場から業者を通さず非加工乳を直接購入することを禁止し、地元の商店のオーナーに未調整乳製品である自家製フレッシュソフトチーズの販売は違法であることを念押しした。ベビー・ラブ・プログラム等既存の教育プログラムを通じて、妊娠中に低温殺菌されていないフレッシュチーズを摂取することがいかに危険であるか指導されている。また、リス

テリア症が報告義務のある疾病のリストに加えられた。現在、全米28州が酪農場から業者を通さず非加工乳を一般人が直接購入することを許可している。全ての州がこの慣行を禁止しない限り、自国での文化様式を大切にする移民者に対する適切な教育に努力が払われてはいるが、メキシコ風フレッシュソフトチーズ及びその他の非低温殺菌乳製品によるリステリア菌の感染症発生は引き続き起こるであろう。

July 06, 2001 / 50(26); 560-2

Outbreak of Listeriosis Associated With Homemade Mexican-Style Cheese --- North Carolina, October 2000--January 2001

On November 13, 2000, health-care providers at a hospital in Winston-Salem, North Carolina, contacted the local health department about three cases of listeriosis within a 2-week period in recent Mexican immigrants. The North Carolina General Communicable Disease Control Branch, in collaboration with the Forsyth County Health Department, the North Carolina Departments of Agriculture and Consumer Services (NCDA&CS) and Environment and Natural Resources, the Food and Drug Administration (FDA), and CDC investigated this outbreak of *Listeria monocytogenes* infections. This report summarizes the results of the investigation, which implicated noncommercial, homemade, Mexican-style fresh soft cheese produced from contaminated raw milk sold by a local dairy farm as the causative agent. Culturally appropriate education efforts are important to reduce the risk for *L. monocytogenes* transmission through Mexican-style fresh soft cheese.

A case was defined as *L. monocytogenes* (isolated from a normally sterile site or with placental tissue staining positive using immunohistochemical techniques) in a mother of a stillborn or premature infant (<37 weeks' gestation), or a mother with a febrile illness, who was a Winston-Salem resident during October 24, 2000--January 1, 2001. Through active case finding, 12 cases were identified. On initial interview, most patients reported eating unlabeled Mexican-style fresh soft cheese bought at local markets or from door-to-door vendors. A case-control study was conducted to determine risk factors for illness; the questionnaire addressed symptoms, diet, and grocery-shopping histories during the month preceding illness. *L. monocytogenes* isolates from patients, raw milk, and cheese were tested using pulsed-field gel electrophoresis (PFGE). Environmental inspections of homes, local markets, and dairy farms were conducted.

All 12 patients were Hispanic; 11 were women with a median age of 21 years (range: 18--38 years), and one was a 70-year-old immunocompromised man. All but one infection were laboratory confirmed. The 11 women did not speak English, were born in Mexico, and had resided in the United States for a median of 2 years (range: 0--5 years). One had traveled outside Forsyth County during the month preceding illness. Ten women were pregnant, and infection with *L. monocytogenes* resulted in five stillbirths, three premature deliveries, and two infected newborns. The 11th woman was 5 months postpartum when she presented to a local hospital with meningitis caused by *L. monocytogenes*. She had no preexisting medical conditions. The male patient, who presented with a brain abscess, was receiving corticosteroid therapy after brain tumor surgery. On hospital admission, the 11 women reported symptoms that included fever (nine), chills (nine), headache (nine), abdominal cramps (five), stiff neck (five), vomiting (three), and photophobia (two).

The male patient was excluded from the case-control study because of difficulty finding suitable controls. In the case-control study, a mother and her fetus or newborn were counted as one case-patient. Controls were identified at a Women, Infants, and Children program office and through the county's record of women enrolled in the state's Baby Love Program, which provides outreach and prenatal-care home visits. A median of four controls (range: three to six controls) per case was selected. Controls were restricted to female Hispanic Winston-Salem residents and matched to patients by age and pregnancy status.

Patients were more likely than controls to have eaten any cheese purchased from door-to-door vendors (matched odds ratio [MOR] =17.5; 95% confidence interval [CI] =2.0--152.5); queso fresco, a Mexican-style fresh soft cheese (MOR=7.3; 95% CI=1.4--37.5); and hotdogs (MOR=4.6; 95% CI=1.1--19.4). Illness was not associated with purchases at specific markets or supermarkets, eating raw fruits or vegetables, deli products, other cheeses (e.g., American, cheddar, mozzarella, and blue/Gorgonzola), or other dairy products.

Various members of the Hispanic immigrant community made the Mexican-style fresh soft cheese from raw milk in their homes. Inspectors found unlabeled homemade cheese in all three of the small local Latino grocery stores they visited in Winston-Salem. In addition, many persons regularly sold the cheese in parking lots and by going door-to-door. Owners of two local dairies reported selling raw milk. Milk samples were obtained from these two Forsyth County dairies and from three dairies

in neighboring counties. *L. monocytogenes* isolates were obtained from nine patients, three cheese samples from two stores, one cheese sample from the home of a patient, and one raw milk sample from a manufacturing grade dairy. All 14 isolates had indistinguishable PFGE patterns, indicating a common link.

NCDA&CS conducted an investigation at a manufacturing grade dairy farm to determine the potential source of *L. monocytogenes* contamination. NCDA&CS collected milk samples from all 49 cows in the herd and samples from the bulk milk storage tanks. Milk from each cow was tested for somatic cell count to identify mastitic cows. Milk from each cow also was tested for presence of *L. monocytogenes*. Repeated testing did not identify any cow with milk confirmed positive for *L. monocytogenes,* suggesting that the cows were not infected and that *L. monocytogenes* may have originated from environmental contamination.

As a result of this outbreak, North Carolina health authorities stopped the sale of raw milk by the dairy farm to noncommercial processors and educated store owners that it is illegal to sell unregulated dairy products. Officials cited the outbreak as sufficient reason to strengthen laws prohibiting the sale of raw milk except to regulated processors. Using already established programs (e.g., Baby Love Program), North Carolina officials recommended reinforcing and expanding the community awareness of the hazards of eating unpasteurized fresh cheese while pregnant. Finally, steps were taken to add listeriosis to the list of reportable diseases in North Carolina.

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Editorial Note:

The investigation of this outbreak implicated Mexican-style fresh soft cheese made from unpasteurized milk and hotdogs, two vehicles commonly identified as causes of *L. monocytogenes* outbreaks. The laboratory investigation resulted in isolation of *L. monocytogenes* from patients, cheese, and raw milk at a dairy farm. Molecular

subtyping identified indistinguishable PFGE patterns, establishing the link between human disease, the cheese, and the source of the raw milk used to make the cheese.

Because of the health risks associated with the consumption of raw milk and raw milk products, FDA requires pasteurization of all dairy products sold across state lines except cheese made from raw milk that has to be aged a minimum of 60 days (*1,2*). Despite North Carolina laws prohibiting the sale and consumption of raw milk and raw milk products, such practices persist in some communities as a result of consumers' taste preferences and for cultural reasons. The popularity of queso fresco, a Mexican-style fresh soft cheese made from unpasteurized milk, has resulted in several outbreaks in Hispanic communities since the 1980s. In 1985, an outbreak of septic abortions attributed to *L. monocytogenes* occurred among Hispanics in Los Angeles and Orange counties, California (*3*). In 1997, three outbreaks of multidrug resistant *Salmonella* serotype Typhimurium DT104 complex strains occurred in Hispanic communities in northern California and Washington (*4, 5*).

Because queso fresco in these communities is produced in private homes, food safety regulations are difficult to enforce. Education of milk and cheese producers and consumers about the increased risk for acquiring infections, particularly *L. monocytogenes*, from consuming unpasteurized milk or fresh soft cheese made from unpasteurized milk, complemented by regulatory action, are the keys to making cheese safe. Successful communication of public health messages to the Hispanic community about the risk for eating Mexican-style fresh soft cheese made from raw milk can be challenging because of language and other social barriers.

The findings in this report are subject to at least four limitations. First, interviewers were not blinded to the status of the persons they were interviewing. Second, efforts were made to select controls from the same population as case-patients; however, controls were selected on the basis of use of public health service programs. Most controls were selected from a county registry for a free prenatal care program that does not require documentation to obtain service. Third, during the study, rumors spread in the community that the suspected vehicle of infection was homemade Mexican-style fresh soft cheese. Finally, patients may have had better recall of potential exposures than controls.

Following a listeriosis outbreak in Yakima County, Washington, an education program to train grandmothers, the primary cheese producers in that community, in the safe

production of soft cheeses was introduced and was well received. A licensing requirement for commercial cheese makers and appropriate regulatory action also may curtail the sale of fresh soft cheese made from unpasteurized milk. Twenty-eight states permit the sale of raw milk directly from farmers to consumers (*6*). Until all states prohibit such sales, outbreaks associated with eating queso fresco and other unpasteurized dairy products may continue despite efforts to educate consumers, especially those who do not speak or read English and whose cultural dietary habits favor such products.

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リステリア症の集団発生
米国，2000 年

2000 年 5 月以降、リステリア菌（リステリア・モノサイトゲネス：LM）により全米 10 州で 29 例の感染を引き起こした（ニューヨーク州 15 例、ジョージア州 3 例、コネチカット州・オハイオ州及びミシガン州各 2 例、カリフォルニア州・ペンシルベニア州・テネシー州・ユタ州及びウィスコンシン州各 1 例）。菌の分離は、5 月 17 日から 11 月 26 日の間で、患者の 9 割は 7 月 15 日以降に感染している。

分離株は、いずれもパルス・フィールド・ゲル電気泳動（PFGE）－パルスネットパターン番号（制限酵素）－GX6A16.0014(Asc1)、GX6A12.0017(Apa1)、及びリボタイピング（DUP－1053）で同じパターンを示した。

報告された患者 29 名中 8 名は周産期患者、残りの 21 名はそれ以外の患者で、後者の年齢中央値は 65 才（範囲 29～92 才）で 13 名（62%）が女性であった。29 名中死亡したのは 4 名、また 3 名に流産あるいは死産がみられた。

5 つの州及び 2 つの地方衛生局と米国疾病管理予防センター（CDC）が症例対照研究を実施した結果、調理済の七面鳥の肉が感染源として浮上した。発症前 30 日間における調理済の七面鳥の肉の喫食は患者群で 17 名中 13 名（76%）であったのに対し、対照群では 24 名中 5 名（21%）と有意差がみられた。患者 11 名が七面鳥の肉を購入した 13 店舗・デリカテッセンに対する州関係当局による調査で、これらの店舗・デリカテッセンでは州当局の検査を受けている、少なくとも 27 施設で製造された七面鳥の肉を販売していたことが判明した。また、七面鳥の肉を製造した 27 施設の内、2 施設は患者 11 名中 10 名に関連しており、この 2 施設の内、1 施設は他の 1 施設の七面鳥の肉も製造していた。12 月 8 日に米国農務省（USDA）では問題の製造所の調査を開始する一方、製造所の 1 つでは 12 月 12 日に調理済即席食品の販売を停止、また 14 日には汚染の恐れのある七面鳥の肉及び鶏肉の自主的回収措置を取った。

December 22, 2000 / 49(50); 1129-1130

Multistate Outbreak of Listeriosis --- United States, 2000

Since May 2000, 29 illnesses caused by a strain of *Listeria monocytogenes* (LM) have been identified in 10 states: New York (15 cases); Georgia (three); Connecticut, Ohio, and Michigan (two each); and California, Pennsylvania, Tennessee, Utah, and Wisconsin (one each). Dates of LM isolation ranged from May 17 through November 26 with 26 (90%) infections occurring since July 15. When subtyped, the LM isolates from these cases were indistinguishable by pulsed-field gel electrophoresis (PulseNet pattern numbers GX6A16.0014 by *Asc*1 and GX6A12.0017 by *Apa*1) and ribotyping (DUP-1053). This report summarizes the investigation, which linked these cases of listeriosis to eating deli turkey meat.

Eight perinatal and 21 nonperinatal cases were reported. Among the 21 nonperinatal case-patients, the median age was 65 years (range: 29--92 years); 13 (62%) were female. The 29 cases have been associated with four deaths and three miscarriages/stillbirths.

A case-control study conducted by five state and two local health departments and CDC implicated eating deli turkey meat as the probable source of infection. Thirteen (76%) of 17 case-patients and five (21%) of 24 controls ate deli turkey meat during the 30 days before illness onset (Mantel-Haenszel weighted odds ratio=8.0; 95% confidence interval=1.2--43.3). State health and agriculture departments investigated 13 stores and delicatessens where 11 patients reported purchasing turkey; these stores and deli-catessens carried turkey meat produced by at least 27 federally inspected establishments. Two establishments were linked to 10 of 11 patients; one of these establishments produced turkey meat for the second establishment.

On December 8, investigators from the Food Safety and Inspection Service, U.S. Department of Agriculture (USDA) began investigating the implicated establishments. On December 12, Cargill Turkey Products, Inc. (Waco, Texas) stopped shipping ready-to-eat foods and, on December 14, voluntarily recalled processed turkey and

chicken deli meat that might have been contaminated.

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Editorial Note:

LM infection causes an estimated 2500 serious illnesses and 500 deaths in the United States each year. Infected pregnant women may experience only a mild, influenzalike illness; however, infections during pregnancy can lead to premature delivery, miscarriage, stillbirth, or serious infection of the newborn. Other persons at increased risk for infection are those aged ≥65 years, persons with cancer, diabetes, kidney disease, acquired immunodeficiency syndrome, or who take immunosuppressive medications. Manifestations of illness include meningitis and sepsis. Healthy persons aged <65 years rarely are affected.

The risk for a person developing *Listeria* infection after eating a contaminated product is very small. Persons who have eaten a recalled product but do not have symptoms do not require tests or treatment even if they are in a highrisk group. However, persons in a high-risk group who have eaten contaminated product and become ill within 2 months with fever or signs of serious illness should consult a physician.

Guidelines for preventing listeriosis are similar to those for preventing other foodborne illnesses. The general recommendations are 1) cook thoroughly raw food from animal sources (e.g., beef, pork, or poultry); 2) wash raw vegetables thoroughly before eating; 3) keep uncooked meats separate from vegetables and from cooked foods and ready-to-eat foods; 4) avoid raw (unpasteurized) milk or foods made from raw milk; and 5) wash hands, knives, and cutting boards after each handling of uncooked foods. Persons at high risk for listeriosis may choose to 1) avoid soft cheeses (i.e., feta, Brie, Camembert, blue-veined, and Mexican-style cheese such as queso fresco). Hard cheeses, processed cheeses, cream cheese, cottage cheese, or yogurt need not be avoided; 2) cook leftover foods or ready-to-eat foods (e.g., hot dogs) until steaming hot; and 3) avoid foods from deli counters (e.g., prepared salads, meats, and cheeses) or thoroughly reheat cold cuts before eating.

Cases of listeriosis with onset since October 1, 2000, should be reported to state and local health departments; information about the recall is available at http://www.fsis.usda.gov/OA/recalls/rec_actv.htm*. Consumers who have recalled meat products, even if they have been stored in freezers, should discard or return them to the point of purchase. High-risk consumers who have processed turkey or chicken deli meat but are uncertain of the brand should call the place of purchase to find out if it might be a recalled product, or discard it. Answers to meat-safety questions are available at the USDA meat and poultry hotline, (800) 535-4555. Listeriosis information is available at http://www.cdc.gov/ncidod/dbmd/diseaseinfo/listeriosis_g.htm.

MMWR47 (51, 52):1117-1118, 1998

複数の州におけるリステリア症の発生
米国，1998 年～1999 年

1998 年 8 月初旬から 1999 年 1 月 6 日にかけて、希少株の 4b 血清型のリステリア菌（リステリア・モノサイトゲネス）による少なくとも 50 件以上の症例が 11 の州から米国疾病管理予防センター（CDC）に報告され、6 人の成人が死亡、2 人の女性が流産した。CDC と州及び地方の保健部門は、ある業者が製造したホットドッグと調理済の食肉が媒体であると同定した。

12 月 22 日、製造元の Bil Mar Foods 社は汚染商品と考えられるホットドッグと調理済の食肉のリコールを行った。CDC はその業者のミシガン州ゼーランドの工場で製造された開封済又は未開封のホットドッグからリステリア菌の発生株を分離し、また同工場で製造された未開封の調理済食肉からも異種株のリステリア菌を分離した。リコール商品の設定番号は、EST P261 又は EST6911 であり、それは全パッケージの端に表示されている。商品名は Ball Park、Bill Mar、Bryan Bunsize、Bryan 3-lb Club Park、Grillmaster、Hygrade、Mr.Turkey、Sara Lee Deli Meat、Sara Lee Home Roast brands であった。協会は他の商品名でリコール商品を受けていた可能性があり、また別の設定番号が付与されている上記の商品名のパッケージと食肉以外の Sara Lee 商品はリコールされていない。

Jan 8, 1999/47 (51&52); 1117-1118

Update: Multistate Outbreak of Listeriosis — United States, 1998-1999

From early August 1998 through January 6, 1999, at least 50 illnesses caused by a rare strain of the bacterium *Listeria* monocytogenes, serotype 4b, have been reported to CDC by 11 states. Six adults have died and two pregnant women have had spontaneous abortions. Reported illness onset dates were during August 2-December 13, 1998. CDC and state and local health departments have identified the vehicle for transmission as hot dogs and possibly deli meats produced under many brand names by one manufacturer. This report updates the investigation of this outbreak (1).

On December 22, the manufacturer, Bil Mar Foods, voluntarily recalled specific production lots of hot dogs and deli meats that might be contaminated. CDC later isolated the outbreak strain of *L. monocytogenes* from an opened and a previously unopened package of hot dogs manufactured at the company's plant in Zeeland, Michigan. In addition, a different strain of *L. monocytogenes* was isolated from unopened packages of deli meats produced at the same plant.

Recalled products bear the establishment numbers EST P261 or EST 6911. The establishment number appears on the outer edge of all packages. The affected products included hot dogs and deli meats with the brand names Ball Park, Bil Mar, Bryan Bunsize, Bryan 3-lb Club Pack, Grillmaster, Hygrade, Mr. Turkey, Sara Lee Deli Meat, and Sara Lee Home Roast brands. Institutions may have received recalled product under other brand names. Packages for the above brand names that carry other establishment numbers are not affected by the recall. Other Sara Lee products that are not meat also are not affected.

Reported by: *Ohio Dept of Health. New York State Dept of Health; Food Safety Laboratory, Cornell Univ, New York City Dept of Health. Tennessee Dept of Health. Massachusetts Dept of Public Health. West Virginia Dept of Health and Human Resources. Michigan Dept of Community Health. Connecticut Dept of Public Health. Health Div, Oregon Dept of Human Resources. Vermont Dept of Health. Div of Public Health, Georgia Dept of Human Resources. Minnesota Dept of Community Health.*

Foodborne and Diarrheal Diseases Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; and EIS officers, CDC.

Editorial Note:

Healthy persons rarely develop severe illness from *Listeria*. The illness primarily occurs in pregnant women, newborns, and persons with impaired immunity caused by serious illness, such as acquired immunodeficiency syndrome or cancer. *Listeria* infections during pregnancy may cause an influenza-like illness with fever and chills, and may lead to loss of the fetus. In other persons, early symptoms can include fever, severe headache, and stiff neck. Illness can begin 2-8 weeks after eating the contaminated food.

Consumers who have the affected product should not eat it, but rather should discard it or return it to the point of purchase. The risk for developing *Listeria* infection after eating a contaminated product is low. Persons who have eaten a contaminated product and do not have any symptoms do not need any special medical evaluation or treatment, even if they are in high-risk groups. However, persons in high-risk groups who have eaten the contaminated product, and within 2 months become ill with fever or influenza-like illness, should inform their physicians about this exposure. Because of this long incubation period, cases may continue to occur and be reported for several weeks after an effective recall.

Consumers who have questions about the recall or the products involved should contact Bil Mar Foods, telephone (800) 247-8339. Persons who have questions about *Listeria* should call their physicians or their local or state health departments or visit CDC's World-Wide Web site, http://www.cdc.gov/ncidod/diseases/foodborn/lister.htm. General questions about meat handling should be directed to the U.S. Department of Agriculture's Meat and Poultry Hotline, telephone (800) 535-4555, Monday through Friday from 10 a.m. to 4 p.m. eastern time.

Reference

1. CDC. Multistate outbreak of listeriosis—United States, 1998. MMWR 1998; 47: 1085-6.

MMWR47 (50):1085-1086, 1998

リステリア症の広域集団発生
米国，1998 年

1998 年 8 月上旬より、リステリア菌（リステリア・モノサイトゲネス：LM）の同一株による感染症が 10 州（オハイオ州 13 例、ニューヨーク州 12 例、テネシー州・マサチューセッツ州・ウエストバージニア州：各 3 例、ミシガン州 2 例、コネチカット州・オレゴン州・バーモント州・ジョージア州：各 1 例）で 40 例確認された。患者の発症あるいは菌の分離日は 8 月 2 日～12 月 2 日で、分離菌の血清型は全て 4b で、パルス・フィールド・ゲル電気泳動のパターン及びリボタイピングを用いたサブタイピングではこれまで米国では人からの検出が稀なタイプに該当することが判明した。患者の情報が得られた 38 名の内、6 名は新生児、32 名は成人（平均 69 才、範囲 18－88 才）で、その 55%は女性であった。新生児 1 名、高齢者 3 名の計 4 名が死亡した。

コネチカット、ニューヨーク、オハイオ及びテネシー州の各衛生当局は、米国疾病管理予防センター（CDC）と共同で、集団感染で確認されたリステリア菌に感染した患者 20 名と異なるリステリア菌に感染した（対照）患者 20 名を対象に、4 週間に渡る喫食調査を主眼とした症例対照研究を実施した。その結果、集団感染患者群（19 名中 16 名：89%）では発症前 1 か月間に対照群（19 名中 6 名：32%）に比し、有意に調理ホットドックを喫食していることが判明し、12 月 19 日には、開封したホットドックから集団感染で確認された菌株と同一の菌株が分離された（このホットドックは集団感染患者の 1 名が発症 4 週間前に喫食）。12 月 22 日、ホットドックの製造会社（Bil Mar Foods）は問題のロットのホットドック及び汚染の可能性のある他肉製品を自主的に回収した。CDC では、現在州及び地方の衛生当局との共同で継続調査を実施中である。

December 25, 1998 / 47(50); 1085-6

Multistate Outbreak of Listeriosis -- United States, 1998

Since early August 1998, 40 illnesses caused by a single strain of *Listeria monocytogenes* (LM) have been identified in 10 states: Ohio (13 cases); New York (12); Tennessee, Massachusetts, and West Virginia (three each); Michigan (two); and Connecticut, Oregon, Vermont, and Georgia (one each). Dates of illness onset or LM isolation ranged from August 2 through December 2. All LM isolates from these cases are serotype 4b and share an unusual pattern when subtyped either by pulsed-field gel electrophoresis or by ribotyping methods. Historically, this pattern is rare among LM isolates from humans.

Among 38 patients for whom demographic data are available, six were newborns and 32 were adults (median age: 69 years; range: 18-88 years); 55% of patients were female. Four deaths occurred, including one fetus and three elderly persons.

In collaboration with CDC, health departments in Connecticut, New York, Ohio, and Tennessee conducted a multistate case-control study comparing 4-week food histories of 20 patients infected with the outbreak strain with those of 20 control patients infected with other LM strains. Sixteen (89%) of 18 cases but only six (32%) of 19 controls consumed cooked hot dogs during the month before illness onset (odds ratio=17.3; 95% confidence interval=2.4-160.0; pless than 0.01). On December 19, the outbreak strain of LM was isolated from an open package of hot dogs. These hot dogs had been eaten by a patient 4 weeks before onset of listeriosis caused by the outbreak strain.

On December 22, the manufacturer, Bil Mar Foods, voluntarily recalled specific production lots of hot dogs and other meat products that might be contaminated. The affected products bear the establishment numbers EST P261 or EST 6911 and include the Ball Park, Bil Mar, Bryan Bunsize and Bryan 3-lb Club Pack, Grillmaster, Hygrade, Mr. Turkey, Sara Lee Deli Meat, and Sara Lee Home Roast brands. The establishment number appears on the outer edge of all packages. Packages for the above brand names that carry any other establishment numbers are not affected by

the recall.

An investigation by CDC is ongoing with local and state health departments. Recent cases of listeriosis should be reported to CDC through state and local health departments. Consumers should return recalled product to the point of purchase.

Reported by: *Local and state health depts. Foodborne and Diarrheal Diseases Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.*

Editorial Note:

The general guidelines for preventing listeriosis are similar to those for preventing other foodborne illnesses, such as salmonellosis. The general recommendations are

- Cook thoroughly raw food from animal sources (e.g., beef, pork, or poultry).
- Wash raw vegetables thoroughly before eating.
- Keep uncooked meats separate from vegetables and from cooked foods and ready-to-eat foods.
- Avoid raw (unpasteurized) milk or foods made from raw milk.
- Wash hands, knives, and cutting boards after each handling of uncooked foods. In addition, persons at high risk for complications from listeriosis (i.e., pregnant women and immunocompromised persons) should
- Avoid soft cheeses (i.e., feta, Brie, Camembert, blue-veined, and Mexican-style cheese). Hard cheeses, processed cheeses, cream cheese, cottage cheese, or yogurt need not be avoided.
- Cook leftover foods or ready-to-eat foods (e.g., hot dogs) until steaming hot.
- Although the risk for listeriosis associated with foods from deli counters is low, pregnant women and immunocompromised persons may choose to avoid these foods or thoroughly reheat cold cuts before eating.

MMWR51 (29):637-639

複数の州における牛挽肉の摂取による病原大腸菌 O157:H7 感染症の発生 米国，2002 年 6 月～7 月

2002 年 7 月、コロラド州健康・環境局（CDPHE）はコロラド州住民における病原大腸菌 O157:H7 感染症の発生を確認した。

この報告は、コロラド州及び他の 6 州で確認された患者 28 例の疫学調査及び検査所見をまとめたものである。コロラド州では、6 月 13 日から 7 月 7 日に発症した 18 例（1～72 歳）より培養法にて病原大腸菌 O157 感染が確認され、分離した菌は 2 酵素の分析によりパルス・フィールド・ゲル電気泳動（PFGE）パターンと一致した。さらに溶血性尿毒症症候群（HUS）と診断された 2 例がこの感染症発生と疫学的に関連していたが、検査にて病原大腸菌 O157 感染は確認されなかった。18 例中 16 例が発症前 7 日間に牛挽肉を食べていることが明らかになり、患者の家から集めた開封済の牛挽肉の培養により病原大腸菌 O157 が検出された。CDPHE の調査により、この牛挽肉は、ConAgra Beef 社により 5 月 31 日に製造されたものを、再度食品チェーン店 A が挽き直した牛挽肉であることが判明した。6 月 30 日、ConAgra Beef 社は 5 月 31 日に製造された牛挽肉製品の全国的な回収を行った。この回収は、米国農務省（USDA）が実施した定期的な細菌検査によって病原大腸菌 O157 が検出されたことに基づいている。CDPHE 及び米国疾病管理予防センター（CDC）が 2 つの制限酵素を用いて実施した PFGE 分析によれば、18 件の食品事故に関連するコロラド州から検出された病原大腸菌 O157 の分離菌株は、感染患者及び ConAgra Beef 社から回収した牛挽肉から分離した大腸菌株と区別できなかった。コロラド州以外での症例を特定するために、食品事故に関連した PFGE パターンを PulsNet に提出した。疫学データと分子サブタイピングに基づき、さらに 6 州（カリフォルニア州、アイオア州、ミシガン州、サウスダコタ州、ワシントン州、ワイオミング州）でコロラド州の患者の集団に関連した病原大腸菌 O157 感染症 8 例が確認された。全体で現在までに 7 例が入院し、5 例は HUS を発症している。その後、回収は 4 月 12 日から 6 月 29 日に製造された生及び凍結牛挽肉と 4 月 12 日から 7 月 11 日に製造された牛肉の付け合わせにまで拡大された。

この病原大腸菌 O157 感染症の発生は、疫学調査における分子サブタイピングを含めた日常的な公衆衛生サーベイランスの重要性を示唆する。

Editorial Note《編集注記》

USDA が実施した細菌検査結果に基づき、複数の州にまたがる感染集団が検出される前に 6 月 30 日の回収が実施された。その後に生じた回収された牛挽肉に関連する疾病の特定は、食肉加工工場の細菌検査の公衆衛生に関する重要性を強化している。

July 26, 2002 / 51(29); 637-639

Multistate Outbreak of *Escherichia coli* O157:H7 Infections Associated with Eating Ground Beef --- United States, June--July 2002

During July 2002, the Colorado Department of Public Health and Environment (CDPHE) identified an outbreak of *Escherichia coli* O157:H7 infections among Colorado residents. This report summarizes the results of an ongoing epidemiologic and laboratory investigation that has linked 28 illnesses in Colorado and six other states to eating contaminated ground beef products recalled by ConAgra Beef Company on June 30, 2002. To date, seven patients have been hospitalized; five developed hemolytic-uremic syndrome (HUS).

For this investigation, a case was defined as culture-confirmed *E. coli* O157 infection in a Colorado resident with symptom onset on or after June 1, and an isolate matching the outbreak pulsed-field gel electrophoresis (PFGE) pattern by two-enzyme analysis. To date, 18 cases have been identified. The median age of patients was 15 years (range: 1--72 years). Dates of symptom onset ranged from June 13 to July 7. Two cases of HUS have been diagnosed among Colorado residents who have epidemiologic links to the outbreak but do not have laboratory-confirmed *E. coli* O157 infection.

Interviews with 16 of 18 patients with confirmed infection revealed that all ate ground beef during the 7 days before illness. All 16 patients ate ground beef that was purchased at grocery chain A during June 10--24. *E. coli* O157 was cultured from an opened package of ground beef collected from a patient's home. A traceback by CDPHE of ground beef collected from a patient's home indicated that it was reground by grocery chain A with meat produced on May 31 by ConAgra Beef Company. On June 30, independent of the outbreak investigation, ConAgra Beef Company issued a nationwide recall of 354,200 lbs of ground beef products produced on May 31. This recall was based on the detection of *E. coli* O157 during routine microbiologic testing conducted by the U.S. Department of Agriculture (USDA).

PFGE analysis conducted by CDPHE and CDC using two restriction enzymes indicated that the 18 outbreak-related human isolates of *E. coli* O157 from Colorado were indistinguishable from isolates of *E. coli* O157 recovered from the opened ground beef package from a patient's home and from the ConAgra Beef Company recalled ground beef product. To identify potential cases outside Colorado, the outbreak-related PFGE patterns were posted on PulseNet, the National Molecular Subtyping Network for Foodborne Disease Surveillance. On the basis of epidemiologic data and molecular subtyping, eight additional *E. coli* O157 cases related to the Colorado cluster have been identified in six states (California, Iowa, Michigan, South Dakota, Washington, and Wyoming). The dates of onset ranged from June 17 to 27. Of the eight patients outside Colorado, six had PFGE patterns that were indistinguishable from the outbreak pattern by two-enzyme analysis, and two were siblings of a PFGE-matched patient. State and local health departments are investigating additional cases to establish epidemiologic and molecular links to the outbreak.

Subsequent to the detection of this multistate outbreak and the initiation of an in-plant inspection of the ConAgra Beef Company by USDA, the nationwide recall of 354,200 lbs of ground beef was expanded to a nationwide recall of 18.6 million lbs of fresh and frozen ground beef and beef trimmings. The expanded recall included fresh and frozen ground beef products produced during April 12--June 29, and beef trimmings produced during April 12--July 11.

Reported by: *P Shillam, MSPH, A Woo-Ming, Colorado Dept of Public Health and Environment. L Mascola, MD, R Bagby, Acute Communicable Disease Control Unit, Los Angeles County Dept of Health Svcs, Los Angeles, California. C Lohff, MD, Iowa Dept of Public Health. S Bidol, MPH, MG Stobierski, DVM, Michigan Dept of Community Health. C Carlson, MS, L Schaefer, L Kightlinger, PhD, South Dakota Dept of Health. S Seys, MPH, Wyoming Dept of Health. K Kubota, MPH, PS Mead, MD, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases; P Kalluri, MD, EIS Officer, CDC.*

Editorial Note:

E. coli O157:H7 was first described as a cause of human illness and associated with undercooked ground beef in 1982 (*1*). Symptoms of *E. coli* O157 infection include bloody and nonbloody diarrhea, vomiting, and abdominal cramps. Illness resolves typically within 7--10 days. A subset of patients, particularly the young and the elderly,

will develop HUS, characterized by microangiopathic hemolytic anemia, thrombocytopenia, and renal failure. Infection with *E. coli* O157 has been associated with exposure to contaminated food and water, person-to-person transmission, and contact with animal reservoirs (*2*). Foods of bovine origin, particularly ground beef, are common causes of sporadic infections and outbreaks of *E. coli* O157 (*2, 3*). Surveys conducted on feed lots demonstrate that cattle can be infected symptomatically with *E. coli* O157, and the prevalence of *E. coli* O157 in feed lots can reach 63%--100%, particularly during the summer, under muddy conditions, or with feeding of barley (*4, 5*).

Although the investigation is ongoing, the findings indicate that this outbreak is associated with the ConAgra Beef Company's recalled ground beef products. Supportive evidence includes 1) reported eating by all Colorado patients of ground beef purchased at grocery chain A; 2) recovery of *E. coli* O157 from leftover meat from a patient's home; 3) traceback of the leftover meat indicating that it was produced at grocery chain A using recalled meat; and 4) PFGE results demonstrating a unique strain of *E. coli* O157 in human isolates, leftover meat, and meat recalled from ConAgra Beef Company.

This outbreak demonstrates the continuing importance of routine public health surveillance combined with molecular subtyping in epidemiologic investigations. The PulseNet database includes molecular fingerprinting patterns of at least 9,800 isolates submitted since 1996. The PFGE pattern of the human and meat isolates in this outbreak was novel in the PulseNet database, facilitating the detection and investigation of seemingly sporadic cases of *E. coli* O157 infection in Colorado and six other states and strengthening the association between the recalled beef products and human illness.

The June 30 recall of meat occurred before detection of the multistate cluster of human infections and was based on results of microbiologic testing conducted by USDA. The subsequent identification of human illness associated with the recalled meat reinforces the importance to public health of microbiologic testing in meat processing plants.

The expanded recall announced on July 19 was one of the largest in U.S. history (*6*). Detailed information on the distribution of recalled meat is not available. The extent to which the recalled meat was repackaged and distributed under other labels is unclear,

potentially making it difficult to identify the affected lots by simple inspection of the package. Grocers and butchers from whom ground beef was purchased might be able to advise concerned customers about the producer and production date of purchased meat. However, consumers should be aware that microbiologic testing in meat processing plants cannot eliminate the risk for contamination of ground beef with *E. coli* O157 and other pathogens. To further reduce the risk for illness, consumers can buy ground beef that is precooked or treated with electron beams. Consumers also can protect themselves by using safe food preparation practices. Frozen ground beef should be thawed in the refrigerator rather than at room temperature. Ground beef should be cooked thoroughly to internal temperatures of at least 160° F (71° C). Using meat thermometers will help ensure that internal temperatures are high enough to kill bacteria. To reduce the risk for cross-contamination, consumers should use soap and hot water to wash hands, utensils, and other surfaces that might have come into contact with raw or undercooked ground beef and other meat products. Additional food safety and product recall information is available from USDA at http://www.usda.gov; telephone 866-849-7438.

Acknowledgments

This report is based on data contributed by L Dippold, REHS, N Haubert, REHS, Tri-county Health Dept, Englewood; K Gieseker, PhD, J Beebe, PhD, S Burnite, Colorado Dept of Public Health and Environment. E Lehnkering, MS, Public Health Laboratory; T Lau, Environmental Health Svcs; R Reporter, MD, Acute Communicable Disease Control Unit, Los Angeles Dept of Health Svcs, Los Angeles; J Farrar, DVM, California Dept of Health Svcs. G Stoltman, PhD, M Boulton, MD, Michigan Dept of Community Health. Spokane Regional Health District, Washington State Dept of Health. A Heryford, MS, W Manley, MS, J Walford, Wyoming Dept of Health. M Lambert-Fair, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.

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MMWR49 (40):911-913, 2000

フレッシュ・チーズカードによる病原大腸菌 0157:H7 感染症の発生 ウィスコンシン州, 1998 年 6 月

1998 年 6 月 15 日、ウィスコンシン州の公衆衛生部門では同州西セントラル地域に居住する住民が 6 月 8 日から 6 月 12 日にかけて病原大腸菌 O157:H7 に感染した旨の報告を受けた（検査室確定患者 8 名及び疑似患者 4 名）。本報告は、本集団発生の調査結果をまとめたものであり、ある酪農プラントで製造されたフレッシュ・チーズカード（curds：凝乳）がその感染源と推定された。

症例対照研究を受けて、ウィスコンシン州の農業局、通商局、消費保護局は、チーズのサンプル、生の食材、梱包された物質を収集し、従業員の食品取り扱い及び衛生管理を調査し、生のミルクからの汚染源を確認するために搾乳工場 A に赴いた。本集団発生では、衛生当局による罹病調査で計 55 名の患者が発見された（家族内の 2 次感染 2 名を含む）。患者は 7 つの郡にまたがって認められ（Chippewa 郡 27 名、Eau Ckaire 郡 16 名）、2 名は他州からこの地域を訪れていた。年齢の中央値は 27 才（範囲 15 ヶ月～90 才）、37 名（67%）が女性で、血便（100%）、腹痛（91%）、疲労感（71%）、悪心（69%）が主要症状であった。罹病期間は 5.1 日（入院）～4.5 日（非入院）の範囲であった。

喫食調査の結果、患者は対照群が 18%であったのに対し、全員がフレッシュ・チーズカードを喫食していること、また患者が喫食したチーズカードはいずれも同じ酪農プラントで製造されたものであることが判明した。喫食したチーズカード（中央値）は 8 個（範囲 1～28 個）で、約 1.6 オンスのチーズを食べたことになる。

一方、チーズ等における環境材料に対する細菌学的検査では、患者 9 名が出席したパーティーで使用されたチーズカードの残品から病原大腸菌 O157:H7 が分離され、パルス・フィールド・ゲル電気泳動（PFGE）による遺伝子パターンは患者から分離された大半の菌株と同一であることが確認された。

Editorial Note《編集注記》

1950 年、FDA はソフトチーズ及びフレッシュチーズの製造業者に対して、低温殺菌乳を

使用すること、又は何年か寝かせるチーズ（Certain aged cheese）にのみ生乳を使用するよう要請した。1986 年、病原大腸菌 O157:H7 感染症は生乳の摂取に関連していた。1987 年、米国食品医薬品局（FDA）は各州間における小売包装の生乳の販売を禁止した。1973 年から 1992 年の間、46 件の生乳関連の食品事故の 40 件（87%）が生乳の販売が許可されている 28 州で発生し、32 件のチーズに関連した食品事故の 11 件は、流通前の汚染に起因するものであった。

October 13, 2000 / 49(40); 911-3

Outbreak of *Escherichia coli* O157:H7 Infection Associated With Eating Fresh Cheese Curds --- Wisconsin, June 1998

On June 15, 1998, the Division of Public Health, Wisconsin Department of Health and Family Services, was notified of eight laboratory-confirmed and four suspected *Escherichia coli* O157:H7 infections among west-central Wisconsin residents who became ill during June 8--12. This report summarizes the outbreak investigation, which implicated fresh (held <60 days) cheese curds from a dairy plant as the source of infection.

A primary case was defined as the first laboratory-confirmed case in a household; a secondary case was one that occurred 3--8 days after a primary case in the same household. A matched case-control study was conducted to assess potential sources of infection. For the purposes of the case-control study, a case was defined as culture-confirmed illness among residents of Chippewa and Eau Claire counties with illness onset during June 7--18. For each case-patient, two community controls matched by sex and age group (range: from <10 years within 2 years to ≥10 years within 5 years) were interviewed by telephone. Case-patients and controls were interviewed about food exposures and potential risk factors for *E. coli* O157:H7 infection within 7 days before onset of illness.

In response to the case-control study, the Wisconsin Department of Agriculture, Trade, and Consumer Protection visited dairy plant A to collect cheese samples, raw ingredients, and packaging materials; to review employee food handling and hygienic practices; and to assess potential sources of contamination from raw milk. Product and environmental samples (e.g., vat surfaces and floor drains) from the dairy plant were screened for phosphatase activity to identify evidence of raw milk.

Fifty-five laboratory-confirmed case-patients were identified, including two from secondary households. Case-patients were from seven Wisconsin counties (27 from Chippewa and 16 from Eau Claire counties); two case-patients were visiting from out

of state. Median age was 27 years (range: 15 months--90 years) and 37 (67%) were female. The most frequently reported symptoms included bloody diarrhea (55 [100%]), cramps (50 [91%]), fatigue (39 [71%]), and nausea (38 [69%]). Mean duration of diarrhea was 5.1 and 4.5 days for 25 hospitalized and 30 nonhospitalized case-patients, respectively.

Eating fresh cheese curds during June 1--17 was reported by all 24 case-patients in Chippewa and Eau Claire counties and eight (18%) of 45 controls (matched odds ratio=undefined; 95% confidence interval=20.6--infinity). Illness was not linked to eating other cheese products (e.g., shredded, sliced, block, or string cheese). Of the 43 laboratory-confirmed case-patients whose cheese curd source could be identified, all had eaten fresh cheese curds produced at dairy plant A; 19 had purchased the curds from an unrefrigerated display at plant A, and 24 had purchased them refrigerated from retail stores that received shipments from plant A. Fifteen (50%) of 30 case-patients who recalled the purchase date had bought the curds on June 5 or 6. The median number of curds eaten was eight (range: one--28), the equivalent of approximately 1.6 oz of cheese.

Thirty-five specimens from plant A that were produced during the outbreak were tested: nine environmental samples, 18 unopened cheese samples, six opened retail packages of curds, and two unopened retail packages of curds. Five of the six opened retail packages of curds and four of the 18 unopened cheese samples were positive for nonbacterial phosphatase (Scharer method). *E. coli* O157:H7 was isolated from an opened package of curds that had been served at a party attended by nine persons with culture-confirmed illness. The contents of this package tested positive for nonbacterial phosphatase. Among 44 *E. coli* O157:H7 case-patient isolates available for pulsed-field gel electrophoresis, 42 were indistinguishable from each other and from the curd isolate.

Dairy plant A had produced four or five vats of pasteurized cheddar and Colby cheese products 5 days a week since 1977. Each vat yielded approximately 1500 pounds of cheese that was pressed into 40-lb blocks, daisies (rounds of cheese), or was packaged as fresh cheese curds. Dairy plant A also produced unpasteurized (raw milk) cheddar cheese daisies every June as part of Dairy Month. Certain raw milk cheese products can be produced and sold legally as long as the cheese is held at $\geq$35 F ($\geq$1.7 C) for at least 60 days before it is sold*. Curds are sold fresh (held <60 days); therefore, curds must be made with pasteurized milk. At least one 1500-lbs vat of raw

milk cheddar cheese was made on May 27 and June 2--5. These vats were used inadvertently to make fresh curds, which were incorrectly labeled "pasteurized" cheddar cheese curds, and distributed and sold in six Wisconsin counties.

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Editorial Note:

Cheese is made in vats by coagulating milk with enzymes and/or acids. After whey is drained, the large cheese clumps are removed and milled into curds, salted, and packaged in small plastic bags for sale. Raw milk consumption has been associated with campylobacteriosis, salmonellosis, *E. coli* O157:H7, yersiniosis, listeriosis, tuberculosis, brucellosis, cryptosporidiosis, and staphylococcal enterotoxin poisoning (*1*). In 1950, the U.S. Food and Drug Administration (FDA) required manufacturers of soft and fresh cheeses to use pasteurized milk and allowed raw milk to be used only for certain aged cheeses (*2*). In 1986, *E. coli* O157:H7 illness was associated with consuming raw milk (*3*). In 1987, FDA banned the interstate sale of raw milk in retail packages. During 1973--1992, 40 (87%) of 46 raw milk-associated outbreaks occurred in the 28 states that permitted the intrastate sale of raw milk (*4*). During the same period, 11 of 32 cheese-associated outbreaks were attributed to contamination before distribution (*5*).

This outbreak investigation illustrates the hazards of using raw milk to produce commercial products that may lead to mislabeling or contaminating pasteurized product by equipment or ingredients. This practice can result in pasteurized products contaminated by equipment or ingredients and in product mislabeling. States that allow the sale of unpasteurized milk or dairy products made from unpasteurized milk should take appropriate steps to reduce the risk for contamination and mislabeling to prevent similar outbreaks.

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MMWR49 (15):321-324, 2000

ティーンエージャーの合宿における病原大腸菌 O111:H8 の発生 テキサス州，1999 年

1999 年 6 月、タラント郡保健部門は、テキサス州保健部門（TDH）に対し、6 月 9 日から 11 日にチアリーダー合宿に参加したティーンエージャーの集団に吐き気、嘔吐、激しい腹部の痙攣、血便を含む下痢、といった症状が生じたことを報告した。2 人が溶血性尿毒性症候群（HUS）のため入院し、他の 2 人が虫垂切除を行った。TDH の研究室と米国疾病管理予防センター（CDC）に送られた便の 2 標本から病原大腸菌 O111:H8 が分離された。本報告ではこの感染症発生の調査について要約する。

他の症例を同定するために、地方の保健部門、病院等にこの感染症発生を報告し、大腸菌による疾患のサーベイランスが行われ、原因を同定するために合宿参加者のコホート調査が行われた。症例は、合宿開始後 14 日間に生じた腹部痙攣を伴う下痢又は出血性の下痢と定義した。参加者は病歴、症状、合宿中の食事や飲料について、調理人と食堂のスタッフはメニュー、食材について聞き取り調査を受けた。コホート調査を行った 650 人のうち、521 人（80%）が聞き取り調査を受け、そのうち 58 人（11%）が症例患者であった。年齢（中間値）は 16 才、95%が女性であった。4 人（7%）が入院し、2 人が HUS を発症、下痢以外の症状として腹部の痙攣、吐き気、頭痛、嘔吐等が挙げられた。合宿初日の夕食と他の飲食物計 21 種が関連しており、19 種が食堂で調理された食材によるものであった。その他には屑入れ容器内の飲料水用の氷と食堂のサラダバーのサラダであった。合宿所の水道からは病原菌は発見されず、製氷機の氷も大腸菌陰性であった。11 人の便標本検査では、2 標本から病原大腸菌 O111:H8 が同定され、酵素免疫測定法（EIA）では 3 つの培養液に志賀毒素が生じた。そのうち 2 つから志賀毒素産生大腸菌のコロニーが生じ、それらは血清型が O111:H8 であった。共にポリメラーゼ連鎖反応（PCR）法により志賀毒素 1 型、2 型の遺伝配列を含んでいることが明らかとなった。病原大腸菌 O157:H7 は同定されなかった。

April 21, 2000 / 49(15); 321-4

Escherichia coli O111:H8 Outbreak Among Teenage Campers --- Texas, 1999

In June 1999, the Tarrant County Health Department reported to the Texas Department of Health (TDH) that a group of teenagers attending a cheerleading camp during June 9--11 became ill with nausea, vomiting, severe abdominal cramps, and diarrhea, some of which was bloody. Two teenagers were hospitalized with hemolytic uremic syndrome (HUS), and two others underwent appendectomies. Routine stool cultures from eight ill persons failed to yield a pathogen. Stools subsequently were sent to laboratories at the Texas Department of Health and CDC, where *Escherichia coli* O111:H8 was isolated from two specimens. This report summarizes the investigation of this outbreak.

To identify additional cases, surveillance for non-O157 Shiga toxin-producing *E. coli* (STEC) illnesses in Texas was enhanced by alerting all local health departments, hospitals, clinical laboratories, and physicians about the outbreak. A cohort study of all campers attending the 3-day camp was conducted to identify the source of the outbreak and to collect data describing the clinical illness. Illness was defined as either diarrhea (three or more loose stools during any 24-hour period) accompanied by abdominal cramps or bloody diarrhea alone, occurring within 14 days after the start of the camp. Campers were interviewed for demographic information, medical histories, and symptoms and about their food and beverage consumption during the camp. Sanitarians inspected the cafeteria where meals were prepared and served to campers and the plumbing system in the dormitory where campers resided. Foodhandlers and other kitchen staff were interviewed about food preparation practices, menus, and the delivery schedules and suppliers for food items served to campers. Foodhandlers submitted stool specimens and rectal swabs for testing. Several food items from the cafeteria were cultured.

Of the 650 campers composing the cohort, 521 (80%) were interviewed. Of these, 58 (11%) had illnesses that met the case definition. The median age of the 58 ill persons was 16 years (range: 12--53 years), and 95% were female. The median length of illness was 5 days; four (7%) persons were hospitalized. Two persons developed HUS. In

addition to diarrhea, reported symptoms included abdominal cramping (100%), nausea (62%), headache (56%), vomiting (38%), bloody diarrhea (37%), and fever with a median temperature of 100 F (38 C) (29%).

Illnesses peaked on the third and final day of camp (Figure 1). Illnesses with bloody diarrhea peaked on the day after the camp ended. No campers reported having a diarrheal illness or contact with a person with diarrhea during the 2 weeks before the start of camp.

One meal (supper on the first day of camp) and 21 other exposures were significantly associated with risk for developing illness. Of these 21 exposures, 19 were specific food items from among 202 foods and beverages served in the cafeteria during the camp and two were more general exposures. Only the two general exposures were significantly and independently associated with illness: consuming any ice from large trash can–style lined barrels that the camp provided in the dormitory lobby for filling water bottles (73% of ill persons versus 43% of nonill persons) (adjusted odds ratio [AOR]=3.4; 95% confidence interval [CI]=1.8--6.3; p=0.0001) and eating any salad from the cafeteria salad bar on at least one occasion (93% of ill persons versus 79% of nonill persons; AOR=3.5; 95% CI=1.4--11.8; p=0.02).

Inspection of the camp's water systems showed no evidence of plumbing cross-connections or failures that might have led to exposures to contaminated water or waste. *Coli*form testing of ice from the ice machines used to fill the barrels was negative. Campers reported dipping their drink containers and arms, hands, and heads into the ice. They also reported observing floating debris in the ice barrels. Inspection of the cafeteria and kitchen indicated that kitchen staff may have improperly followed cooking times and temperatures recommendations when preparing meals.

The laboratory investigation of stools specimens submitted by 11 ill persons yielded *E. coli* O111:H8 from two specimens. Three enrichment broths prepared from these 11 specimens had detectable Shiga toxin when screened with a commercial enzyme immunoassay (EIA). Two of these three EIA–positive stool specimens yielded colonies of Shiga toxin–producing *E. coli*, which were serotyped as *E. coli* O111:H8. Both isolates contained gene sequences for Shiga toxins 1 and 2 by polymerase chain reaction. *E. coli* O157:H7 was not isolated from any camper, foodhandler, or food or water sample. Samples of the implicated ice and salad items served during the camp were not available for testing.

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Editorial Note:

This was the first community outbreak of infections attributable to Shiga toxin-producing *E. coli* O111 reported in the United States. The findings of the investigation suggest a point-source outbreak. Although primary infection from eating a contaminated salad item and then secondary spread through the barrel ice is a plausible hypothesis, the original source of contamination and its means of spread are unknown.

Identification of non-O157 STEC requires techniques not used routinely by clinical laboratories. In this outbreak, a commercially available EIA kit was used to detect and isolate STEC in stool specimens; isolates were then serotyped at CDC.

STEC cause illness in otherwise healthy persons, including severe abdominal cramping (sometimes confused for appendicitis), bloody diarrhea, and HUS. *E. coli* O111 was the second most common non-O157 STEC (after *E. coli* O26) isolated from specimens submitted to CDC for serotyping during 1983--1998 and among isolates from persons with diarrhea collected for an ongoing survey in Minnesota initiated in 1995 (Minnesota Department of Public Health, unpublished data, 2000). STEC cause an estimated 110,000 illnesses each year in the United States, of which $\geq$30% may be attributable to non-O157 serotypes such as O111 (*1*); the burden of disease attributable to non-O157 STEC is unknown.

Most STEC outbreaks in North America have resulted from infection with *E. coli* O157. A household cluster of *E. coli* 0111 infection was reported in 1990 from Ohio (*2*), and outbreaks have occurred in Australia, Europe, and Japan (*3--7*). Despite investigations involving large numbers of persons in well-defined settings, the vehicle of transmission has been epidemiologically implicated and microbiologically confirmed in only one 1995 outbreak in South Australia, which was attributable to mettwurst, a

dried fermented sausage (*3*).

As demonstrated by this outbreak, a commercially available kit could be used to screen stool specimens for Shiga toxin and potential STEC. However, culturing and serotyping the causative organism is critical to identify and better understand these emerging pathogens. To facilitate diagnosis of STEC infections, clinicians should inform health departments about clusters of suspected illnesses that could be attributable to STEC (e.g., bloody diarrhea and HUS). Clinical laboratories should screen stool specimens from persons with either bloody diarrhea or HUS for STEC, routinely or when *E. coli* O157 is not isolated, and attempt to isolate STEC from stools that are positive by the screening test and refer isolates to public health laboratories for serotyping. States should consider adding STEC infections to their notifiable disease lists.

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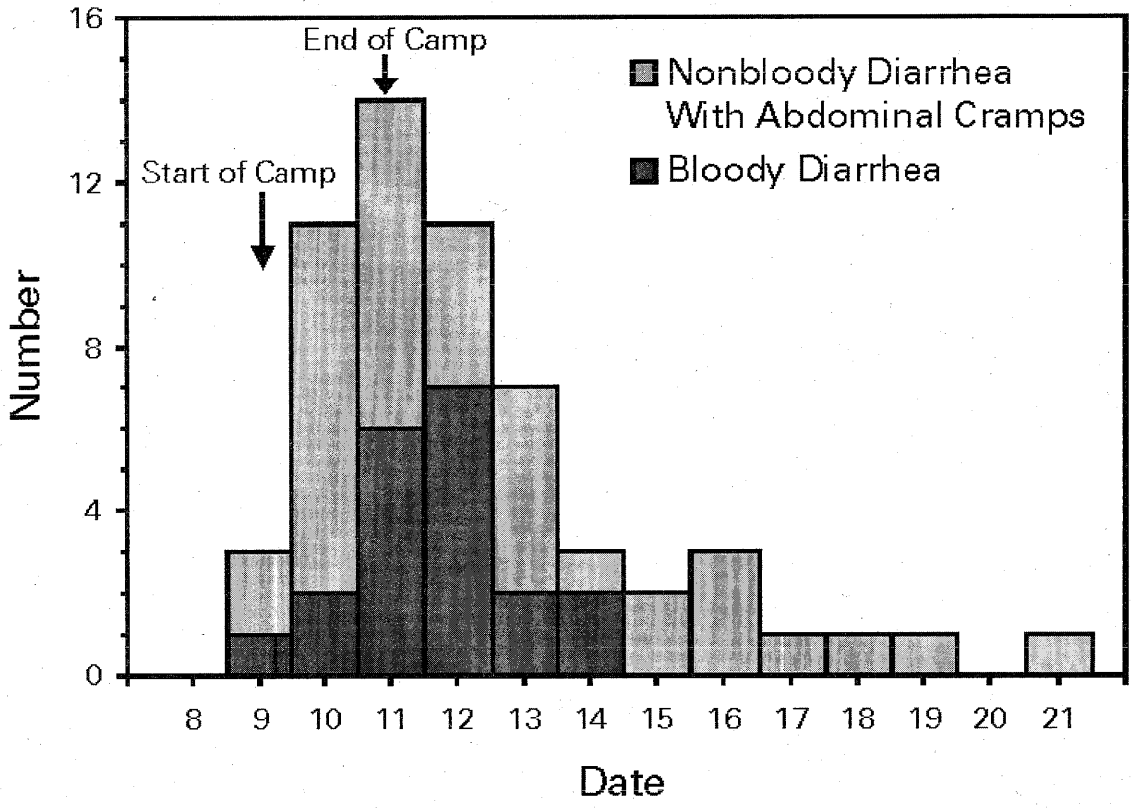
FIGURE 1. Number of *Escherichia coli* O111:H8-associated illnesses at a camp, by date of onset — Texas, June 1999



MMWR52 (2):24- 26

海岸に打ち上げられた鯨の摂食によるE型ボツリヌス中毒の発生 アラスカ州西部，2002年7月

ボツリヌス中毒は、偏性嫌気性菌から産生される毒素による神経麻痺性疾患であり、E型ボツリヌス中毒は魚介類に起因する。2002年7月13～15日、アラスカ州西部のベーリング海に面した漁村において、浜辺に打ち上げられている死後数週間経過した鯨（シロイルカ）が発見された。村人たちは尾の部分を細かく切り、ビニール袋に入れて冷蔵庫で保管し、1日又は2日後に摂食した。

2002年7月17日、アラスカ州西部の診療所よりボツリヌス中毒が疑われる3例が報告され、アラスカ州保健社会サービス局公衆衛生部疫学課（Department of Health and Social Services, Division of Public Health、Section of Epidemiology）による調査が開始された。鯨を食べた14例中8例（57%、男3女5、年齢（中央値）73（範囲13～83）歳）がボツリヌス中毒の診断基準と一致した。いずれも食後36時間以内で発症し、5例が入院、4例に抗毒素が投与され、2例は人工呼吸器を要した。便検体3検体、胃液3検体、血清7検体及び鯨肉7検体が米国疾病管理予防センター（CDC）の国立ボツリヌス中毒監視照会研究所（National Botulism Surveillance and Reference Laboratory）にて検査され、1例の便検体からE型毒素が検出され、鯨肉は全検体でE型ボツリヌス毒素陽性を示した。

1973～1998年、CDCは全部で814のE型ボツリヌス中毒発症例の報告を受け取った（年間24例（14～94例）で、内236例（29%）がアラスカ州にて発生している）。打ち上げられた鯨の死骸の破棄はE型ボツリヌス中毒防止に有効であると示唆する報告があるが、膨大なコストと広大なアラスカ沿岸を逐一監視することは色々な面で割に合わないため、米国魚類・野生生物サービス（U.S. Fish and Wildlife Service）は死骸の定期的撤去を行っていない。打ち上げられた海洋哺乳類の死骸の摂食は避け、生や発酵したアラスカ原住民の料理は、ボツリヌス菌を不活性化するため食事直前に10分以上煮沸するべきである。

January 17, 2003 / 52(02); 24-26

Outbreak of Botulism Type E Associated with Eating a Beached Whale --- Western Alaska, July 2002

Botulism is a neuroparalytic illness caused by toxins produced by the bacterium *Clostridium botulinum*, an obligate anaerobe found commonly in the environment. Intoxication with toxin type E is associated exclusively with eating animal foods of marine (salt or fresh water) origin. Persons who eat raw or fermented marine fish and mammals are at high risk for botulism from type E toxin. On July 17, 2002, the Alaska Division of Public Health investigated a cluster of suspected botulism cases among residents of a fishing village in Alaska. This report summarizes the findings of the outbreak investigation, which linked disease to eating raw muktuk (skin and a pink blubber layer) from a beached whale (Figure). To avoid delays in treatment, health-care providers evaluating patients suspected of having botulism should base treatment decisions on clinical findings. Public health authorities should be notified immediately about any suspected botulism case.

During July 13--15, residents of a western Alaska village on the Bering Sea shore shared a meal consisting of muktuk harvested from a beached adult beluga whale found near their village. The villagers estimated that the whale had been dead for at least several weeks. They cut the whale fluke (tail) into pieces and stored them in zipper-sealed plastic bags in a refrigerator until they were eaten 1 or 2 days later. On July 17, after a physician from western Alaska reported three suspected cases of botulism among patients who had eaten the muktuk, the Alaska Section of Epidemiology began an investigation.

A case of foodborne botulism was defined as illness in a person who had eaten the muktuk and subsequently had symmetric descending flaccid paralysis of motor and autonomic nerves. Persons who ate muktuk were interviewed and examined, and their hospital records were reviewed. Serum, stool, and gastric contents from patients and leftover blubber were tested for botulinum toxin.

Of 14 persons identified who ate the muktuk, eight (57%) had an illness that met the case definition. Five of the eight patients were female; the median age was 73 years (range: 13--83 years). Symptom onset after ingestion of muktuk occurred within 36 hours in all patients (Table). Five patients were hospitalized, four received antitoxin, and two required mechanical ventilation. Three stool, three gastric fluid, and seven serum samples from the eight patients and seven samples of muktuk were tested for botulinum toxin at CDC's National Botulism Surveillance and Reference Laboratory. The diagnostic laboratory received all laboratory specimens on July 26, and results were reported on August 1. Type E toxin was detected in stool from one patient. All seven samples of muktuk were positive for type E botulinum toxin.

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Editorial Note:

This report summarizes a foodborne outbreak of botulism in a western Alaska village that resulted from residents eating muktuk contaminated with type E botulinum toxin. During 1973--1998, a total of 814 cases and an annual median of 24 cases (range: 14--94 cases) of foodborne botulism were reported to CDC (*1*); 236 (29%) of these cases occurred in Alaska (CDC, unpublished data, 2003). Although botulism is a rare disease, its presentation is distinctive (Box. Because of the epidemic potential of foodborne botulism, every case should be reported and investigated immediately.

All patients suspected of having foodborne botulism should be placed in an intensive care setting, monitored regularly for respiratory function deterioration, and provided mechanical ventilation if necessary. Prompt administration of polyvalent equine-source antitoxin can decrease the progression of paralysis and severity of illness but will not reverse existing paralysis. Botulinum antitoxin is available in the United States only through the public health system. Therefore, rapid clinical diagnosis, notification of public health authorities, and timely administration of antitoxin are imperative (*2*). Laboratory confirmation of botulinum intoxication cannot be relied on in making treatment decisions because the standard test, the mouse bioassay, requires approximately 4 days for final results (*2*). In addition, the sensitivity of laboratory testing of clinical samples is low (*3, 4*). In this outbreak, typed toxin was

detected in only 8% of samples from patients who had definitive exposure to contaminated muktuk.

The probable mode of contamination of the whale in this outbreak was either growth and toxin secretion by *C. botulinum* present in the intestinal tract of the whale or traumatic introduction of *C. botulinum* spores into the beached whale tissue from contact with sand, rocks, and driftwood, and subsequent germination and toxin production. *C. botulinum* type E has been found in Alaska coastline soil (*5*), and outbreaks of botulism associated with eating beached marine mammals are documented (Alaska Section of Epidemiology, unpublished data, 2003). A previous report on the accumulation of *C. botulinum* toxins in the North Sea coastal food chain associated with beached whales suggested the disposal of the carcasses as a preventive measure (*6*). However, because of the impracticality of frequent scanning of the vast Alaska shoreline and high costs associated with disposal, the U.S. Fish and Wildlife Service does not remove beached mammal carcasses regularly.

Because of the epidemic potential of foodborne botulism and the status of botulinum toxins as a category A agent of terrorism, health-care providers should be familiar with the presentation of botulism. Treatment is based on clinical diagnosis, and rapid recognition and reporting of cases are the cornerstones of successful public health interventions to prevent additional illnesses. Persons should avoid eating beached marine mammal carcasses and boil raw or fermented Alaska Native dishes ≥10 minutes before eating to inactivate botulinum toxin. Additional information on botulism prevention is available at http://www.phppo.cdc.gov/phtn/botulism/alaska/alaska.asp and http://www.epi.hss.state.ak.us/pubs/botulism/bot_01.htm.

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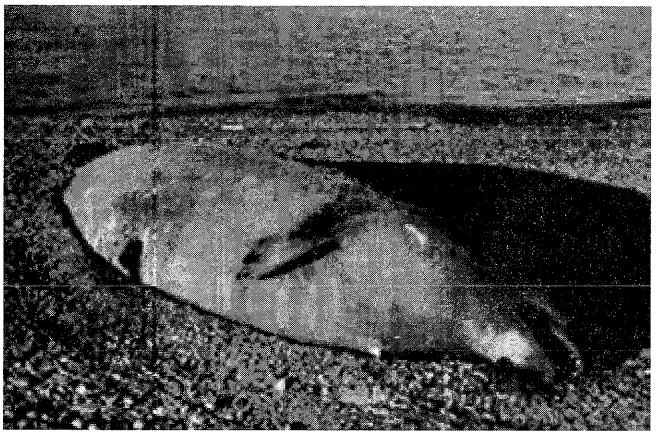
TABLE. Number* and percentage of patients with signs and symptoms of botulism associated with eating a beached whale — Western Alaska, July 2002

Sequelae	No.	(%)
Gastrointestinal symptoms		
Abdominal pain	5	(63)
Constipation	5	(63)
Diarrhea	4	(50)
Nausea or vomiting	7	(88)
Neurologic symptoms		
Blurred vision	5	(63)
Diplopia	1	(13)
Dry mouth	7	(88)
Dysphagia	6	(75)
Dysarthria	4	(50)
Difficulty breathing or shortness of breath	5	(63)
Other symptoms		
Throat pain	3	(38)
Dizziness	6	(75)
Neurologic signs		
Hoarse voice	5	(63)
Ptosis	2	(25)
Pupils fixed and dilated	5	(63)
Urinary retention	1	(13)
Weakness	8	(100)
Other signs		
Bradycardia†	4	(50)
Hypotension§	6	(75)

*N = 8.
†Heart rate <60 beats per minute.
§Systolic blood pressure <100 mmHg.

FIGURE. A juvenile beluga whale beached on the Alaska shoreline



Photo/Natural Resources Canada

BOX. Epidemiology, diagnosis, treatment, and prevention of foodborne botulism

Epidemiology

- Caused by eating foods contaminated with preformed toxins of *Clostridium botulinum*
- Home-canned foods and raw or fermented Alaska Native dishes commonly associated with illness
- During 1973–1998, a total of 814 cases and an annual median of 24 cases (range: 14–94 cases) of foodborne botulism reported in the United States; 236 (29%) in Alaska
- Humans affected by toxin types A, B, E, and rarely F; type E intoxication associated exclusively with eating marine animals
- Classified as a category A terrorism agent

Clinical findings

- Cranial nerve palsies
- Symmetrically descending flaccid voluntary muscle weakness possibly progressing to respiratory compromise
- Normal body temperature
- Normal sensory nerve examination findings
- Intact mental status despite groggy appearance
- Differential diagnosis includes Guillain-Barré syndrome, myasthenia gravis, stroke, drug overdose, and other entities

Laboratory findings

- Normal cerebrospinal fluid values
- Specific electromyography (EMG) findings including
  - normal motor conduction velocities
  - normal sensory nerve amplitudes and latencies
  - decreased evoked muscle action potential
  - facilitation following rapid repetitive nerve stimulation
- Standard mouse bioassay positive for toxin from clinical specimens and/or suspect food; requires up to 4 days for final results

Recommended treatment

- Prompt administration of polyvalent equine-source antitoxin
  - can decrease the progression of paralysis and severity of illness
  - will not reverse existing paralysis
  - available in the United States only through the public health system
- Place suspect cases in an intensive care setting
- Monitor for respiratory function deterioration every 4 hours using forced vital capacity testing
- Provide mechanical ventilation if necessary

Prevention and control

- Boil raw or fermented Alaska Native dishes and home-canned foods ≥10 minutes before eating
- Follow recommended home-canning procedures
- Notify state health department immediately of suspected cases

MMWR50 (32):680-682, 2001

発酵食品の摂食によるボツリヌス中毒の発生 アラスカ州，2001 年

2001 年 1 月 18 日、アラスカ州保健社会サービス局公衆衛生部（Department of Health and Social Service, Public Health Division）はアラスカ南西部の村でボツリヌス中毒の発生が起きた可能性があるとの報告を受けた。

1 月 17 日、アラスカ南西部の村の住民 14 名が発酵したビーバーの尾と足を食べた。その約 20 時間後、14 名中 3 名で口渇、視覚異常、全身の脱力感といったボツリヌス中毒の疑いのある症状が発現した。症状発現から約 6 時間後、この 3 名は A/B 型及び E 型ボツリヌス抗毒素の投与を受け、アンカレッジ市の集中治療室（ICU）に搬送された。2 名は合併症もなく改善したが、他 1 名は 1 ヶ月間、気管開口による管留置と機械換気が必要であった。同じ発酵食品を食べた他の 11 名中 4 名では、口渇や悪心といった軽度のボツリヌス中毒症状が発現した。ICU に入院した 2 名の血清検体と 1 名の便検体より E 型ボツリヌス毒素が検出されたが、他の 11 例では毒素は検出されなかった。E 型毒素は中毒関連食品と思われるビーバーの足からも検出された。

アラスカ州での食事によるボツリヌス中毒には昔から、ビーバーの尾と足をライスペーパーの袋で包んで発酵させ温かな所に保存するという一般的でない発酵法が関連している。1998 年にブリストル湾岸保健公団（the Bristol Bay Area Health Corporation：アラスカ州南西部のアラスカ原住民によって運営されているヘルスケアサービス組織）は、米国疾病管理予防センター（CDC）極寒地調査プログラム（Arctic Investigations Program）の協力を得て、市民参加型のボツリヌス中毒防止計画を作成している。

August 17, 2001 / 50(32); 680-2

Botulism Outbreak Associated With Eating Fermented Food ---Alaska, 2001

On January 18, 2001, the Alaska Division of Public Health was informed by a local physician of a possible botulism outbreak in a southwest Alaska village. This report summarizes the findings of the outbreak investigation, which linked disease to eating fermented food, and describes a new botulism prevention program in Alaska.

A case of foodborne botulism was defined as a clinically compatible illness in a village resident with laboratory confirmation of botulism or a history of eating the same food as a laboratory-confirmed case; 14 persons in the village had eaten fermented beaver tail and paw on January 17. Approximately 20 hours later, three of the 14 had symptoms suggestive of botulism, including dry mouth, blurry vision, and general weakness. Two patients developed respiratory failure and required intubation and mechanical ventilation. One of the two intubated patients suffered cardiac arrest and underwent successful cardiopulmonary resuscitation. Approximately 6 hours after the onset of symptoms, the three patients received types A/B and E botulism antitoxin. They subsequently were evacuated to an intensive care unit (ICU) in Anchorage. Two patients recovered without further complication. The third required tracheostomy tube placement and mechanical ventilation for 1 month; this patient had been hospitalized with botulism in 1997. Of the other 11 exposed persons, four reported minor symptoms compatible with botulism, including dry mouth and nausea, and were admitted to a hospital for overnight observation. One was hospitalized for 10 days with persistent ileus. The remaining seven exposed persons were held for observation for 48 hours.

Clinical specimens from the 14 exposed persons were tested for botulinum toxin at CDC. Type E toxin was detected in serum specimens from two of the ICU patients and in stool from the third. Although they displayed minor symptoms, the other 11 persons had no toxin found in specimens and were not considered laboratory-confirmed cases. Type E toxin also was detected in three beaver paws tested from the implicated meal.

Beaver is hunted in southwest Alaska, and certain parts often are fermented and eaten later. In this outbreak, the tail and paws had been wrapped in a paper rice sack and stored for up to 3 months in the entry of a patient's house. Some of the beaver tail and paw had been added to the sack as recently as 1 week before it was eaten.

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Editorial Note:

This report illustrates how the use of nontraditional fermentation methods is associated with foodborne botulism in Alaska. Botulism results from eating preformed toxin produced by *Clostridium botulinum*. Botulism begins with cranial nerve paralysis, including diplopia, dilated and fixed pupils, dysarthria, dysphagia, and dry throat. Botulism intoxication can result in death, which most often is caused by respiratory failure. The latent period is typically 12--36 hours but can range from 6 hours to 10 days (*1*). *C. botulinum* and closely related organisms produce toxins designated as types A, B, C, D, E, F, and G. Human botulism is most commonly caused by types A, B, and E. Type E is associated with foods of marine or freshwater origin. Alaska's foodborne botulism rates exceed those in any other state and are among the highest in the world (*1*). During 1950--2000, Alaska recorded 226 cases of foodborne botulism from 114 outbreaks. All patients were Alaska Natives, and all cases with known causes were associated with eating fermented foods (*1, 2*). Approximately 27% of U.S. foodborne botulism cases occur in Alaska.

In traditional fermentation, food is kept in a grass-lined hole in the ground or a wooden barrel sunken into the ground or is placed in a shady area above ground for several weeks to months. Since the 1970s, however, plastic or glass containers have been used and fermentation has been done above ground or indoors. The anaerobic condition of sealed containers and warmer temperatures make fermentation more rapid and production of botulism toxin more likely (*3--5*). These nontraditional methods have been associated with increased botulism rates in Alaska during 1970--1989 (Figure 1) (*4, 5*). Although a plastic container was not used in this

outbreak, the beaver tail and paw were fermented in a closed rice sack and stored in a warm area.

Early diagnosis and antitoxin treatment have contributed to the decline of the case-fatality rate from approximately 31% during 1950--1959 to no deaths in Alaska since 1994 (*1*). However, Alaska continues to have high foodborne botulism rates because fermented foods are part of Alaska Native culture. In a 1999 survey, 107 (77%) of 140 Alaska Natives reported having eaten fermented foods at least once in their lifetime (*3*).

In 1998, the Bristol Bay Area Health Corporation, a health-care delivery organization operated by Alaska Natives in southwest Alaska, collaborated with CDC's Arctic Investigations Program to design a community-based botulism prevention strategy, including an education video entitled, "A Helping Hand: Keeping Your Family Safe From Botulism." It features Alaska Native elders and botulism survivors discussing the risks of eating improperly fermented foods and recommends returning to traditional methods. The video also suggests boiling fermented foods for 10 minutes to destroy botulinum toxin. Both an English and an Alaska Native language version of the video were produced and distributed to all village clinics and schools in the Bristol Bay region. Information on botulism prevention also is available at http://www.cdc.gov/phtn/botulism/default/default.htm.

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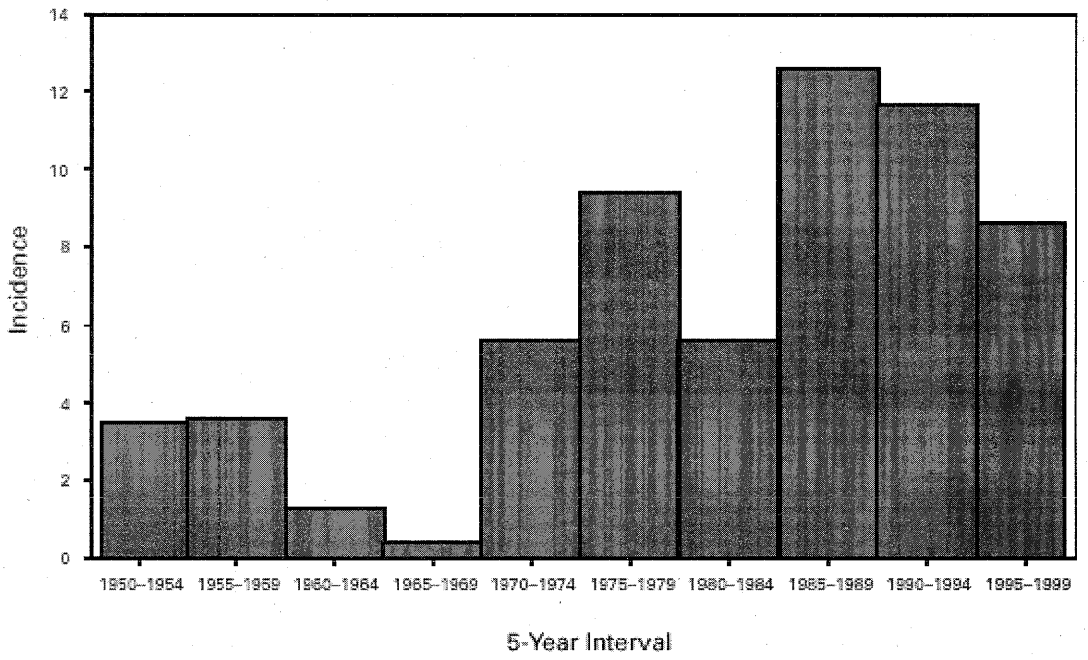
FIGURE 1. Incidence* of foodborne botulism among Alaska Natives, by 5-year intervals — Alaska, 1950–1999



*Per 100,000 population.

MMWR49 (34):778-780, 2000

自家製鶏卵ピクルスによる食餌性ボツリヌス症 イリノイ州，1997 年

1997 年 11 月、地方の臨床医から食餌性ボツリヌス症を疑う 1 症例のあることがイリノイ州の衛生局に報告された。本報告では、この事例の調査結果について紹介する。

本症例は、これまで健康であった 68 歳の男性が 1997 年 11 月 23 日、嘔気及び嘔吐と腹痛を訴えたもので、その後の 2 日間のうちに、復視、発声障害及び呼吸不全を呈し、機械呼吸処置のために入院した。入院時検査で眼球外運動麻痺、広汎性弛緩麻痺などの頭蓋神経異常が確認された。ボツリヌス中毒（推定）と診断され、3 価（A、B 及び E 型）の抗ボツリヌス毒素が投与された。そして、抗毒素投与前に採取された患者血清の検査で B 型毒素が検出された。喫食歴の調査では自家製缶詰類の曝露はなかったが、発病 7 日前に調製した自家製の鶏卵ピクルス（固茹鶏卵、ビート、ホットペパー及び食用酢を密閉容器に入れて室温保存）を摂取し、12 時間後に胃腸炎症状を呈したことが判明した。患者は長期支持療法で回復した。残余の自家製鶏卵ピクルス混合物の検査では、B 型ボツリヌス菌及び B 型毒素がそれぞれ検出された。また、鶏卵中のボツリヌス毒素量はピクルス液の毒素量に比して千倍以上高い結果が示されたが、原材料のビートから毒素は検出されなかった。市販のペッパーの入っていた容器も毒素陰性で、またペッパーから菌も培養されなかった。市販ビートの入っていた容器は入手できなかった。ピクルス液の pH は 3.5 で、ボツリヌス菌の発芽・増殖及び毒素産生を阻止するのに適切な条件であったが、卵黄については不明であった（正常卵では pH6.8）。

September 01, 2000 / 49(34); 778-780

Foodborne Botulism From Eating Home-Pickled Eggs --- Illinois, 1997

During November 1997, the Illinois Department of Public Health was notified by a local physician about a possible case of foodborne botulism. This report summarizes the case investigation, which implicated home-pickled eggs as the cause.

On November 23, 1997, a previously healthy 68-year-old man became nauseated, vomited, and complained of abdominal pain. During the next 2 days, he developed diplopia, dysarthria, and respiratory impairment, necessitating hospitalization and mechanical ventilation. Physical examination confirmed multiple cranial nerve abnormalities, including extraocular motor palsy and diffuse flaccid paralysis. Possible botulism was diagnosed, and a one-vial dose of trivalent (types A, B, and E) antibotulinum toxin was administered. A sample of the patient's serum collected before antitoxin administration demonstrated the presence of type B botulinum toxin. A food history revealed no exposures to home-canned products; however, the patient had eaten pickled eggs that he had prepared 7 days before onset of illness; gastrointestinal symptoms began 12 hours after ingestion. The patient recovered after prolonged supportive care.

The pickled eggs were prepared using a recipe that consisted of hard-boiled eggs, commercially prepared beets and hot peppers, and vinegar. The intact hard-boiled eggs were peeled and punctured with toothpicks then combined with the other ingredients in a glass jar that closed with a metal screw-on lid. The mixture was stored at room temperature and occasionally was exposed to sunlight.

Cultures revealed *Clostridium botulinum* type B, and type B toxin was detected in samples of the pickled egg mixture at CDC's National Botulism Surveillance and Reference Laboratory. *C. botulinum* was cultured from the pickling liquid, beets, and egg yolk. The concentration of preformed type B toxin was 1000 times greater in the egg yolks than in the pickling liquid and was undetected in the beets. Peppers from the original commercial container contained no detectable toxin, and bacterial cultures of the peppers did not yield *C. botulinum*. Beets from the original commercial containers

were not available. The pH of the pickling liquid was 3.5 (i.e., adequate to prevent *C. botulinum* germination and toxin formation. However, the pH of the egg yolk was not determined [normal egg yolk pH: 6.8]).

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Editorial Note:

Botulism is a paralytic illness caused by the neurotoxin produced by the bacterium *C. botulinum*. Paralysis first affects the cranial nerves, then the skeletal muscles; untreated intoxications can lead to dense flaccid paralysis, respiratory failure, and death (*1, 2*).

Although rare and sporadic, foodborne botulism is a persistent cause of morbidity and mortality in the United States. In 1997, an annual survey of state epidemiologists and directors of state public health laboratories identified 24 cases of foodborne botulism with one associated death (CDC, unpublished data, 1998). During 1989--1998, a median of 23 cases (range: 17--42 cases) of foodborne botulism was reported each year with a median of one death (range: 0--2 deaths).

C. botulinum spores are ubiquitous. Safe food preservation methods destroy spores or inhibit their germination and growth. Conditions that promote germination and growth of *C. botulinum* spores include absence of oxygen (anaerobic conditions), low acidity (pH >4.6), temperatures >39 F [4 C]), and high moisture content. Most foodborne botulism cases that occur in the United States are the result of improperly home-canned foods. This is the first reported case of botulism related to eating pickled eggs. The amount of toxin detected in the recovered egg yolk suggested that bacterial growth was concentrated in that portion of the egg. Intact eggs that have been hard-boiled should be free of bacteria or spores. Pricking cooked eggs may introduce *C. botulinum* spores into the yolk. Portions of the yolk that remained anaerobic and inadequately pickled (i.e., not acidified to pH $\leq$4.6) may have allowed *C. botulinum* spores to germinate, grow, and form toxin. Setting the pickling jar in sunlight

provided warmth that facilitated bacterial growth and toxin production.

To reduce the risk for botulism when pickling, food items should be washed and cooked adequately, and utensils, containers, and other surfaces in contact with food, including cutting boards and hands, should be cleaned thoroughly with soap and warm water. Containers (e.g., jars and lids) in which pickling will occur should be sterilized (e.g., placed in boiling water for the prescribed period published in the container instructions) (*3*). Adequate acidification to a pH $\leq$4.6 is essential. Refrigeration at 39 F (4 C) during pickling is advisable, especially in foods that may be acidified inadequately such as whole eggs. Once opened, any canned or pickled food should be refrigerated. Pricking, poking holes, or otherwise handling whole eggs in a manner that might allow spores or bacteria into the yolk should be avoided.

When foodborne botulism is suspected, clinicians and public health investigators should inquire about the preparation and eating of foods preserved by any home method (e.g., canning, pickling, curing, and fermenting). Persons seeking advice on home-food preservation should consult their local county or university cooperative extension service, or contact the U.S. Department of Agriculture Food Safety Hotline, telephone (800) 535-4555. CDC provides epidemiologic consultation and laboratory diagnostic services for suspected botulism cases and authorizes release of botulism antitoxin. Through state health departments, these services are available 24 hours a day from CDC.

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MMWR51 (25):548-549

乳牛借用プログラムを通じて入手した非低温殺菌牛乳の飲用による カンピロバクター（*Campylobacter jejuni*）感染症の発生 ウィスコンシン州，2001 年

2001 年 12 月 7 日、ウィスコンシン州南西部のソーヤー郡保健と福祉サービス局(Sawyer County Department of Health and Human Services）はカンピロバクター（*C. jejuni*）による腸炎 5 例をウィスコンシン州保健家族サービス局公衆衛生部（Department of Health and Family Services, Division of Public Health）に報告した。5 例全例が地元の酪農場で購入した非低温殺菌牛乳を飲んでいた。この報告は、ウィスコンシン州におけるこれら 5 例及び他の症例について、そして州の非低温殺菌牛乳販売禁止規則の抜け穴として利用された乳牛借用プログラム（cow-leasing program）についての調査結果をまとめたものである。

ソーヤー郡とその周辺地域で合計 75 例のカンピロバクター（*C. jejuni*）による腸炎発症例（2～63 歳）が確認された。疾患の徴候及び症状としては、下痢（93%）、腹部痙攣（92%）、発熱（76%）、悪心（40%）、血性下痢（23%）がみられた。70 例（93%）が地元の酪農場で購入した非低温殺菌牛乳を飲んでおり、4 例（5%）は非低温殺菌牛乳を飲んだ患児の母親であった。1 例は小児ケア施設に通う子供で、非低温殺菌牛乳を飲んでおらず、他の患者との接触もなかった。29 例の便検体中 28 検体（97%）でカンピロバクター（*C. jejuni*）の増殖を認めた。患者に牛乳を提供した施設は、36 頭の乳牛を有するグレード A の有機酪農場であった。ウィスコンシン州では非低温殺菌牛乳は法的に販売できないため、その農場は乳牛借用プログラムを通じ、消費者が最初に料金を支払って乳牛の一部を借りるという形式を取ることにより非低温殺菌牛乳を販売していた。12 月 8 日に農場のタンクから採取した牛乳サンプルにてカンピロバクター（*C. jejuni*）の増殖を認め、そのパルス・フィールド・ゲル電気泳動（PFGE）パターンは感染症発生菌株と一致した。

ウィスコンシン州政府は非低温殺菌牛乳を飲まないよう消費者に勧告すると共に、規則を強化し、乳牛借用プログラムを禁じるという対応を見せている。ところで、1995 年時点で全米 28 の州で非低温殺菌牛乳の州内販売が許可されていた。州の規制機関は、非低温殺

菌牛乳の販売を規制する政策見直しの際には、是非とも疾病リスクを考慮に入れるべきである。また非低温殺菌牛乳に関するリスクコミュニケーションも徹底されなければならない。

June 28, 2002 / 51(25); 548-549

Outbreak of *Campylobacter jejuni* Infections Associated with Drinking Unpasteurized Milk Procured through a Cow-Leasing Program --- Wisconsin, 2001

On December 7, 2001, the Sawyer County Department of Health and Human Services in northwestern Wisconsin notified the Wisconsin Division of Public Health about five cases of *Campylobacter jejuni* enteritis. All of the ill persons drank unpasteurized milk obtained at a local dairy farm. This report summarizes the investigation of these and other cases and of a cow-leasing program used to circumvent regulations prohibiting the sale of unpasteurized milk in Wisconsin. The outbreak highlights the hazards of consuming unpasteurized milk and milk products.

A case of *C. jejuni* enteritis was defined as illness in a person from Sawyer County or a surrounding county who had diarrhea or abdominal cramps and fever during November 10--December 18. Case finding was conducted by notifying health-care providers, infection-control practitioners, laboratorians, and the public about the outbreak.

A total of 75 persons had illness that met the case definition (Figure). The patients ranged in age from 2 to 63 years (median: 30 years); 41 (56%) were males. Signs and symptoms of illness included diarrhea (93%), abdominal cramps (92%), fever (76%), nausea (40%), and grossly bloody diarrhea (23%). None of the patients was hospitalized, and none had Guillain-Barre syndrome. A total of 70 (93%) patients reported drinking unpasteurized milk from a local dairy farm. Four (5%) patients did not drink unpasteurized milk but were mothers of ill children who drank unpasteurized milk. One patient was a child who attended a child care facility but did not drink unpasteurized milk or have contact with other patients.

Of the 75 patients, 29 (39%) provided stool specimens; 28 (97%) specimens grew *C. jejuni* (Figure). Of the 28 patients with positive stool specimens, 23 (33%) were patients who drank the unpasteurized milk, four were mothers of patients, and one patient had an unknown mode of infection. Pulsed-field gel electrophoresis (PFGE) was performed on 21 isolates; the patterns were indistinguishable when restricted separately by two enzymes.

The facility that supplied milk to patients was a Grade A organic dairy farm with 36 dairy cows. The farm also had a retail store in which milk and other food products were available. In addition, farm operators provided unpasteurized milk samples at community events and to persons who toured the farm, including children from childcare facilities. Because unpasteurized milk cannot be sold legally to consumers in Wisconsin, the dairy distributed unpasteurized milk through a cow-leasing program. Customers paid an initial fee to lease part of a cow. Farm operators milked the cows and stored the milk from all leased cows together in a bulk tank. Either customers picked up milk at the farm or farm operators had it delivered. On December 8, investigators obtained a milk sample from the farm's bulk milk tank, and cultures of the milk samples grew *C. jejuni* with a PFGE pattern that matched the outbreak strain. Farm operators were ordered to divert all milk to a processor for pasteurization. State inspectors found the farm to meet Grade A standards for a farm shipping milk to a pasteurization plant. Consumers were advised not to drink unpasteurized milk. To ensure that unpasteurized milk will not be distributed to the public in Wisconsin, state officials are enforcing existing regulations and prohibiting cow-leasing programs.

Reported by: *P Harrington, Sawyer County Dept of Health and Human Svcs, Hayward; J Archer, MS, JP Davis, MD, State Epidemiologist, Wisconsin Div of Public Health. DR Croft, MD, JK Varma, MD, EIS officers, CDC.*

Editorial Note:

Unpasteurized milk is an important vehicle for transmission of pathogens including *Campylobacter* spp., *Brucella* spp., Shiga toxin-producing *Escherichia coli* (e.g., *E. coli* O157), *Corynebacterium diphtheriae*, *Salmonella* spp. (including multidrug-resistant strains), *Mycobacterium bovis*, and *Listeria monocytogenes* (*1, 2*). In 1995, intrastate sale of unpasteurized milk was permitted in 28 states (*3*). In California, where the sale of unpasteurized milk is legal, 128 (3%) of 3,999 residents reported drinking unpasteurized milk in 1993 (*4*). Persons who drink unpasteurized milk and milk

products might believe that these products taste better, provide greater nutrition than pasteurized products, and/or decrease the risk for various medical conditions (*4*). However, the benefits of consuming unpasteurized milk and milk products have never been validated scientifically (*5*).

As in this outbreak, in several states milk producers have established cow-leasing programs to circumvent regulations (*6*). Advocates of unpasteurized milk also have published lists of those states that permit the sale of unpasteurized milk for nonhuman consumption. Persons might use such lists to obtain milk covertly in these states.

State regulatory agencies should consider the risk for human illness when reviewing policies regulating the sale of unpasteurized milk. States that permit the sale of unpasteurized milk might consider placing warning labels on such products, as with unpasteurized juice. Because persons might attempt to circumvent existing regulations, further public health research should address how to communicate to consumers the health risks of drinking unpasteurized milk.

Acknowledgments

This report is based on data contributed by E Simak, J Connell, Sawyer County Dept of Health and Human Svcs, Hayward; T Leitzke, M Barnett, B Carroll, G Hewitt, W Resheske, S Steinhoff, C Koschmann, L Kelly, K Manner, Wisconsin Dept of Agriculture, Trade, and Consumer Protection, Madison; T Monson, MS, D Lucas, T Kurzynski, MS, D Hoang-Johnson, L Machmueller, Wisconsin State Laboratory of Hygiene, Madison, Wisconsin. M Beatty, MD, R Tauxe, MD, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.

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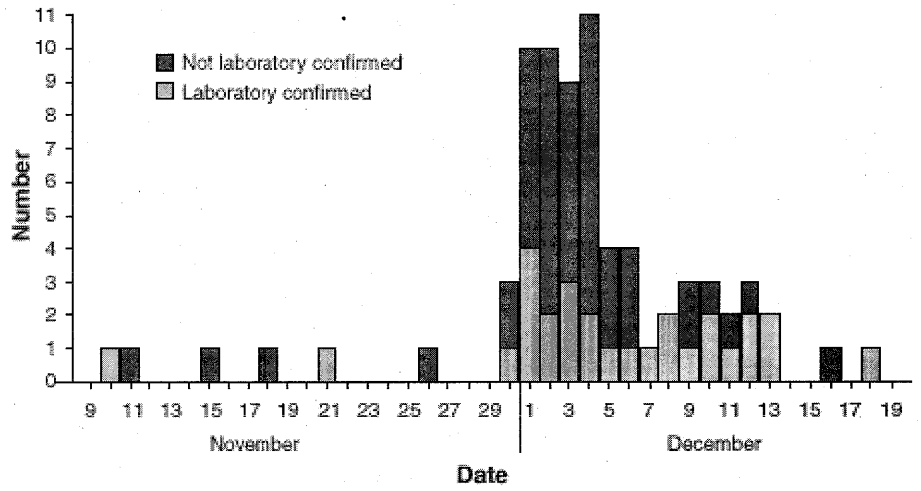
FIGURE. Number of patients with *Campylobacter jejuni* infections, by confirmation status and date of illness onset — Wisconsin, November 10–December 18, 2001*



* n=75.

MMWR47 (7):129-131, 1998

食物の二次汚染によるカンピロバクター腸炎（*C. jejuni*）の発生 オクラホマ州，1996年

1996年8月15日、オクラホマ州ジャクソン郡の某レストランでランチを食べた人々に、カンピロバクター（*C. jejuni*）による腸炎が発生した〔ジャクソン群衛生局（JCHD）はオクラホマ州衛生局（OSDH）に対し、8月16日から20日にかけて発症したカンピロバクター（*C. jejuni*）による腸炎の集団発生について、8月29日に報告した〕。本報告では、調理器具を介して生の鶏肉に付着していたカンピロバクター（*C. jejuni*）がレタスを汚染し、ほとんどそれが原因で感染症が起きたことから、食材により調理器具を使い分ける必要性が強調されている。最初のケースでは、某レストランでランチを食べ、16日に下痢と嘔吐を発症した。聞き取り調査できた25名中14名（56%）に同様の症状が発生していた。平均潜伏期間は3日で内2名は入院した。10名の糞便試料全部からカンピロバクター（*C. jejuni*）が検出された（食品について検査することはできなかった）。OSDHは、14名の症例と11名の非発症例による症例対照研究を実施した。JCHD及びOSDHの職員は、メニューに関する資料の収集、調理実態の観察、調理場の調査を実施するために当該レストランに赴いた。有意に食べられていた食品は、レタス（14名 vs 4名）とラザニア（11名 vs 3名）であった。調理場の検視とコックへの聞き取り調査の結果、生の鶏肉に付着していたカンピロバクター（*C. jejuni*）がレタスやラザニアを汚染したことが推定された。JCHDは当該レストランに対し、調理台を大きくすること、使い捨てタオル容器の設置、異なる食品を調理する時は調理従事者の手洗い及び調理器具の清浄を実施するよう勧告した。

February 27, 1998 / 47(07); 129-131

Outbreak of *Campylobacter* Enteritis Associated with Cross-Contamination of Food -- Oklahoma, 1996

On August 29, 1996, the Jackson County Health Department (JCHD) in southwestern Oklahoma notified the Oklahoma State Department of Health (OSDH) of a cluster of *Campylobacter jejuni* infections that occurred during August 16-20 among persons who had eaten lunch at a local restaurant on August 15. This report summarizes the investigation of these cases and indicates that *C. jejuni* infection was most likely acquired from eating lettuce cross-contaminated with raw chicken. This report also emphasizes the need to keep certain foods and cooking utensils separate during food handling.

A case was defined as illness in a person who had eaten lunch at the restaurant on August 15, 1996, and had onset of diarrhea (i.e., three or more loose stools during a 24-hour period) or vomiting during August 16-20. Of 25 persons available for interview who had eaten lunch at the restaurant on August 15, a total of 14 (56%) had had an illness that met the case definition. The median age of patients was 33 years (range: 5-52 years); 10 (71%) were female. All patients reported diarrhea; 13 (93%), fever; 13 (93%), abdominal cramps; 11 (79%), nausea; five (36%), vomiting; and three (21%), visible blood in their stools. The median incubation period was 3 days (range: 1-5 days). Two (14%) patients were hospitalized. Stool specimens were collected from 10 patients; all yielded *C. jejuni*. No food items were available for testing.

To identify risk factors for illness, OSDH, in collaboration with JCHD, conducted a case-control study of 14 patients and 11 controls (i.e., persons who had eaten lunch with patients at the implicated restaurant on August 15 but did not become ill). Health department staff visited the restaurant to obtain information about menu items, to observe food preparation, and to inspect the kitchen.

All 14 patients and four (36%) controls reported eating lettuce (odds ratio {OR} =48.3; 95% confidence interval {CI} =2.3-infinity; p less than 0.01). Eleven (79%) patients and

three (27%) controls had eaten lasagna (OR=6.7; 95% CI=1.1–42.7; p less than 0.05). Both lettuce and lasagna were statistically associated with illness. Lettuce consumption accounted for all cases, and lasagna consumption accounted for 79% of cases.

Inspection of the restaurant indicated that the countertop surface area was too small to separate raw poultry and other foods adequately during preparation. The cook reported cutting up raw chicken for the dinner meals before preparing salads, lasagna, and sandwiches as luncheon menu items. Lettuce for salads was shredded with a knife, and the cook wore a towel around her waist that she frequently used to dry her hands. Bleach solution at the appropriate temperature (greater than 75 F [greater than 24 C]) and concentration (greater than 50 ppm) was present to sanitize tables surfaces, but it was uncertain whether the cook had cleaned the countertop after cutting up the chicken. The lettuce or lasagna was probably contaminated with *C. jejuni* from raw chicken through unwashed or inadequately washed hands, cooking utensils, or the countertop.

JCHD recommended that the restaurant enlarge its food-preparation table and install a disposable hand towel dispenser and that food handlers wash hands and cooking utensils between use while preparing different foods.

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Editorial Note:

Campylobacter is one of the most common causes of foodborne disease in the United States, causing approximately 2 million cases of gastroenteritis each year (1). Illness associated with *Campylobacter* infection is usually mild, but can be severe and even fatal. Although it did not occur in this outbreak, Guillain-Barre syndrome (GBS), a demyelinating disorder resulting in acute neuromuscular paralysis, is a serious sequela of *Campylobacter* infection (2). Up to 40% of patients with GBS have evidence of *Campylobacter* infection before onset of symptoms (2).

Most illnesses associated with *Campylobacter* infection are sporadic. Common source outbreaks occur, and most have been traced to unpasteurized milk and contaminated drinking water (1). In comparison, most sporadic cases, and those in this outbreak, are associated with improper handling and preparing of poultry (1). *Campylobacter* has been found in up to 88% of broiler chicken carcasses in the United States (1, 3). The infectious dose of *Campylobacter* is low; ingestion of only 500 organisms, easily present in one drop of raw chicken juice, can result in human illness (1). Therefore, contamination of foods by raw chicken is an efficient mechanism for transmission of this organism.

Restaurants provide opportunities for outbreaks of foodborne disease because large quantities of different foods are handled in the same kitchen. Failure to wash hands, utensils, or countertops can lead to contamination of foods that will not be cooked. The food handler involved in this outbreak had not received training in food safety. The Food and Drug Administration has developed guidelines for food handlers to prevent cross-contamination of foods; however, states are not required to adopt these guidelines (4).

Laws mandating certification of food-service employees differ by state. Twelve states have requirements for certification of food-service managers in all jurisdictions, 21 states have requirements in some jurisdictions, and 17 states have no requirements (5). Of 33 states for which information is available, only two have statewide requirements for training of food handlers (5).

States can reduce the risk for foodborne illness in restaurants by ensuring that restaurant employees receive training in food safety. For example, food handlers should be aware that pathogens can be present on raw poultry and meat and that foodborne disease can be prevented by adhering to the following measures: 1) raw poultry and meat should be prepared on a separate countertop or cutting board from other food items; 2) all utensils, cutting boards, and countertops should be cleaned with hot water and soap after preparing raw poultry or meat and before preparing other foods; 3) hands should be washed thoroughly with soap and running water after handling raw poultry or meat; and 4) poultry should be cooked thoroughly to an internal temperature of 180 F (82 C) or until the meat is no longer pink and juices run clear.

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MMWR48 (3):48-51, 1999

ロングアイランド湾で採取された生カキ及び蛤による腸炎ビブリオ感染症の集団発生 コネチカット州、ニュージャージー州、ニューヨーク州，1998 年

1998 年の 7 月～9 月にかけて、ロングアイランド湾で採取された生カキ及び蛤による腸炎ビブリオ感染症がコネチカット、ニュージャージー、ニューヨークの 3 州の住民の間で集団発生した。本事例は、ニューヨーク州の海域で採取された魚介類では最初の集団発生例である。

1998 年 8 月 10 日、海外渡航歴のないニューヨーク市在住の 1 人がコレラに罹患したことがニューヨーク市衛生局（NYCDOH）に報告された。NYCDOH では感染源の調査を開始する一方、8 月 12 日には同報ファックスにより全てのクイーンズ郡の研究所に連絡を取り、8 月 26 日には病院等の関係機関にこの種の腸炎ビブリオ感染症につき注意を呼びかけると共に、確認あるいは疑わしい患者があった場合は NYCDOH まで報告するよう通知した。その結果、新たなコレラ患者の報告はなかったものの、コネチカット、ニュージャージー、ニューヨークの 3 州の住民中に培養検査で確認された計 23 名の腸炎ビブリオ患者がいることが確認された。患者の発症日は 7 月 21 日～9 月 17 日の間であった。

NYCDOH による調査の結果、患者 23 名中 22 名がカキ、蛤あるいは甲殻類を喫食又は取り扱っていたことが判明した（16 名は生のカキあるいは蛤を喫食、2 名は茹でカニ、この他クラブ（カニ）ケーキ、茹でカニとロブスター、ロブスターロールの喫食各 1 名、生カキの取り扱い者 1 名）。喫食から発症までの平均潜伏時間は 19 時間（範囲：12～52 時間）で、患者 23 名中、臨床的情報の得られた 19 名の内 17 名（89%）に胃腸炎、2 名に下肢の浮腫及び水疱を伴う菌血症が認められた。胃腸炎の主要臨床症状は下痢（100%）、腹痛（94%）、悪心（94%）嘔吐（82%）、発熱（47%）、血便（29%）頭痛（24%）、筋肉痛（24%）で、有病期間は平均 5 日であった。

一方、地域及び州衛生当局により実施された感染源調査では、カキあるいは蛤を喫食した患者 16 名中 11 名についてその採取場所が特定された。8 名については、ニューヨーク州ロングアイランド湾沖のオイスターベイで 8 月 4 日～27 日にかけて採取されたもの、また

他の 3 名はロングアイランド以外の場所（内 1 例はワシントン州）で採取されたものを喫食していた。また、オイスターベイ海域の今夏の水温は 1996 年（20.7℃）、1997 年（23.4℃）に比し高温（25.1℃）であったことも判明した。9 月 10 日、ニューヨーク州環境保全局（NYSDEC）は魚介類を採取しないようオイスターベイを閉鎖、また 8 月 10 日以降、同海域で採取された魚介類の回収措置を取った。

オイスターベイで採取された魚介類を喫食した 8 名を含む患者 12 名から分離された腸炎ビブリオはいずれも血清型 O3：K6 に属するもので、ニューヨーク市衛生研究所で 4 つの菌株を対象に実施したパルス・フィールド・ゲル電気泳動（PFGE）ではオイスターベイで採取された魚介類に関連する患者 3 名の菌株は互いに区別できないパターンを示したのに対し、オイスターベイで採取された魚介類と関連のなかった患者の菌株はそれらとは全く別のパターンであった。その後、9 月 11 日～10 月 14 日にかけて 5 回採取されたオイスターベイのカキについて実施した菌の検索では、カキ肉中に≦120 CFU/g の腸炎ビブリオが検出されたが、これらの菌株の PFGE パターンはいずれも集団発生した菌株とは異なるものであった。これらの知見に併せ、海水温も 17.5℃までに低下したことから、NYSDEC は 10 月 22 日、オイスターベイにおける魚介類の商業捕獲を再開した。それ以降、新たな患者の発生報告はなされていない。

Editorial Note《編集注記》

腸炎ビブリオ感染症の発生を監視するために、米国疾病管理予防センター（CDC）は湾岸に生息する腸炎ビブリオの調査システムと FoodNet の連携を図った。

January 29, 1999 /48(03); 48-51

Outbreak of *Vibrio parahaemolyticus* Infection Associated with Eating Raw Oysters and Clams Harvested from Long Island Sound -- Connecticut, New Jersey, and New York, 1998

During July-September 1998, an outbreak of *Vibrio parahaemolyticus* infections associated with consumption of oysters and clams harvested from Long Island Sound occurred among residents of Connecticut, New Jersey, and New York. This is the first reported outbreak of *V. parahaemolyticus* linked to consumption of shellfish harvested from New York waters. This report summarizes the investigation of this outbreak.

On August 10, 1998, a New York City resident with toxigenic *V. cholerae* O1 infection who had not traveled recently was reported to the New York City Department of Health (NYCDOH). NYCDOH initiated an investigation to determine the most likely source of the infection. Using a broadcast facsimile, NYCDOH contacted all Queens County laboratories on August 12 and, on August 26, asked selected infectious diseases physicians and all New York City hospitals and laboratories to consider *V. cholerae* as a potential cause of diarrhea and to report any confirmed or suspected *Vibrio* infections to the NYCDOH. Although no additional *V. cholerae* infections were reported, 23 culture-confirmed cases of *V. parahaemolyticus* were reported among residents of Connecticut, New Jersey, and New York. Dates of illness onset ranged from July 21 through September 17 (Figure_1).

An investigation coordinated by the New York State Department of Health determined that 22 of 23 ill persons had eaten or handled oysters, clams, or crustaceans: 16 ate raw oysters or clams, two ate steamed crabs, one ate crab cakes, one ate boiled crabs and lobsters, one ate lobster roll, and one handled live crabs. The median onset of illness following consumption of shellfish was 19 hours (range: 12-52 hours). Clinical histories were available for 19 of the 23 ill persons; 17 (89%) had gastroenteritis and

two (11%) had bloodstream infections with lower extremity edema and bullae. Among patients with gastroenteritis, reported clinical symptoms included diarrhea (100%), abdominal cramps (94%), nausea (94%), vomiting (82%), fever (47%), bloody stools (29%), headache (24%), and myalgia (24%). Median duration of gastrointestinal illness was 5 days.

Traceback investigations by local and state health departments identified the site of harvest for oysters or clams eaten by 11 of the 16 patients. Oysters or clams eaten by eight patients were harvested from Oyster Bay, off New York's Long Island Sound, during August 4–27. Shellfish tags from oysters and clams eaten by the other three persons indicated harvest areas elsewhere off Long Island or, in one case, Washington state (1) *.

During the outbreak period, mean surface water temperature measurements from 15 Oyster Bay stations was 77.2 F (25.1 C), compared with cooler 1997 and 1996 measurements (74.1 F [23.4 C] and 69.4 F [20.7 C], respectively). On September 10, the New York State Department of Environmental Conservation (NYSDEC) closed Oyster Bay to harvesting of shellfish and recalled shellfish harvested from that area after August 10.

Laboratory testing of 12 *V. parahaemolyticus* clinical isolates, including the eight traced to Oyster Bay, identified O3:K6 serotype. Pulsed-field gel electrophoresis (PFGE) performed on four clinical isolates at the New York City Bureau of Labs indicated that three isolates epidemiologically linked to Oyster Bay had indistinguishable PFGE patterns, and the other isolate not linked to Oyster Bay had a distinctly different pattern. Oysters harvested on five occasions from Oyster Bay during September 11–October 14 contained *V. parahaemolyticus* at less than or equal to 120 colony forming units [cfu] per gram of oyster meat. None of these environmental isolates matched the outbreak strain or other clinical isolates by PFGE. On the basis of these results and a decline in water temperature to 63.5 F (17.5 C), NYSDEC reopened Oyster Bay to commercial shellfish harvesting on October 22. No additional culture-confirmed cases of *V. parahaemolyticus* infection have been reported.

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Editorial Note:

This is the fourth multistate outbreak of *V. parahaemolyticus* infections in the United States since 1997, and the first associated with shellfish harvested from the northeast Atlantic Ocean. Before 1997, foodborne outbreaks caused by *V. parahaemolyticus* had been infrequently reported in the United States (1). During 1997–1998, multistate outbreaks of *V. parahaemolyticus* were associated with consumption of raw or undercooked oysters harvested from the Pacific Northwest and Texas (2; CDC, unpublished data, 1998).

V. parahaemolyticus is a halophilic, gram-negative bacterium that naturally inhabits marine and estuarine waters. *V. parahaemolyticus* infections are usually acquired by persons who eat raw or undercooked shellfish, particularly oysters, or whose skin wounds are exposed to warm seawater. The most common clinical manifestation of infection is self-limited gastroenteritis, but infections may result in septicemia that can be life threatening (3, 4). The concentration of *V. parahaemolyticus* in seawater increases with increasing water temperature and corresponds with a seasonal increase in sporadically occurring cases in warmer months (4). This outbreak and the recent outbreaks of *V. parahaemolyticus* infections in the Pacific Northwest and

Texas occurred during summer months.

To reduce the risk for *V. parahaemolyticus* and other shellfish-associated infections, persons should avoid eating raw or undercooked shellfish, particularly during warmer months. Monitoring of environmental conditions, such as water temperature and salinity, may help determine when shellfish harvesting areas should be closed and re-opened to harvesting.

Guidelines regulating the harvesting of oysters and clams rely on quantitative measurement of *V. parahaemolyticus* levels in oyster or clam meat. However, data from recent outbreaks may require revision of these guidelines. The recommended action level of *V. parahaemolyticus* per gram of oyster meat that must be detected in the absence of human illness before closing oyster beds is greater than 10,000 cfu/g. Oyster samples that were harvested from implicated beds in the Pacific Northwest in 1997 and Oyster Bay in 1998 yielded less than 200 *V. parahaemolyticus* cfu/g of oyster meat, indicating that human illness can occur at levels much lower than the current action level.

Infection with *V. parahaemolyticus* is not a notifiable condition in most states, including New York. This outbreak was detected only coincidentally because of enhanced surveillance during an investigation of a case of *V. cholerae* O1. Health-care providers treating patients with gastroenteritis who have a history of recent ingestion of raw or undercooked shellfish should consider *Vibrio* infection and request a stool culture specifically for *Vibrio*. Clinical laboratories should use thiosulfate-citrate-bile salts-sucrose agar (TCBS), a selective medium for culturing for *Vibrio* spp., when culturing stool specimens for *Vibrio* and should consider using TCBS for routine screening of all stools specimens, at least during summer months.

CDC coordinates a passive Gulf Coast *Vibrio* surveillance system and the Foodborne Diseases Active Surveillance Network (FoodNet) to monitor the incidence of *Vibrio* infections. Because of these multistate outbreaks, all states should consider making infections with *V. parahaemolyticus* and other vibrioses reportable, with referral of clinical isolates to public health laboratories for confirmation and strain subtyping.

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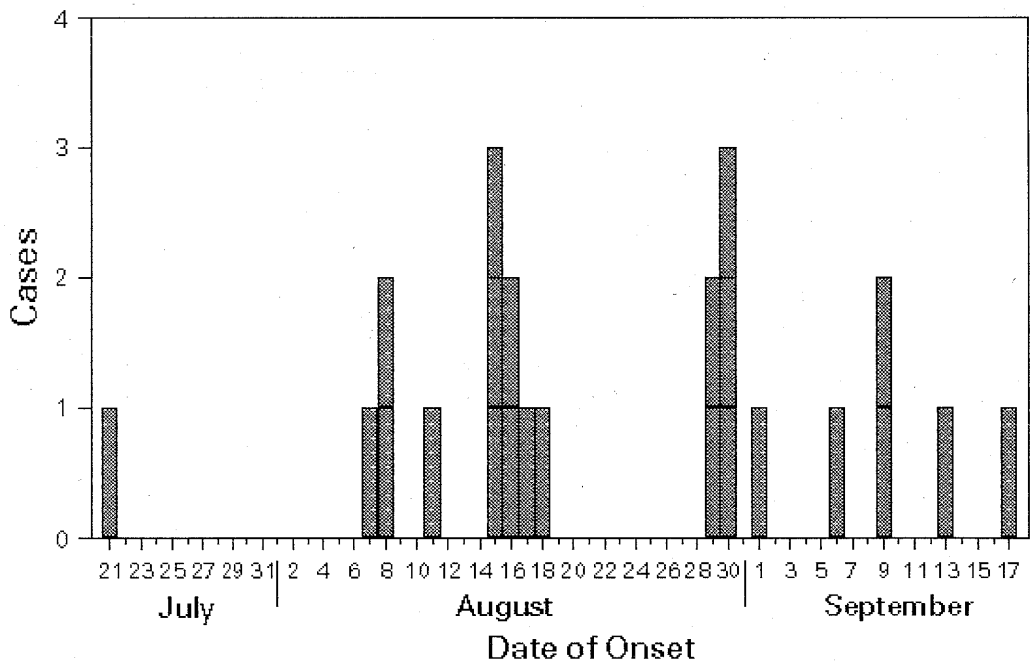
The shipper that provided the oysters harvested elsewhere in Long Island also had received oysters from Oyster Bay at approximately the same time. Although comingling of shellfish is against state regulations, it is known to occur.

FIGURE 1. Cases of *Vibrio parahaemolyticus*, by date of symptom onset — Connecticut, New Jersey, and New York, July-September, 1998



MMWR47 (22):457-462, 1998

生カキの喫食による腸炎ビブリオ感染症の集団発生 太平洋北西部地域，1997 年

1997 年 7～8 月にかけて、北米ではこれまでで最大規模の腸炎ビブリオによる集団感染症が発生した。罹患した患者 209 名はカリフォルニア州、オレゴン州、ワシントン州及びカナダのブリティッシュコロンビア（BC）で採取されたカキの喫食に関連して発生しており、1 名が死亡した。以下は本集団発生について実施された調査結果のまとめである。

ブリティッシュコロンビア： 7 月 1 日～19 日にかけて、BC 地域の研究所は 7 月としては例年の 2 倍にあたる 9 名の腸炎ビブリオ患者の分離株を受け取った。そこで、BC の疾病管理予防センター（BCCDC）ではこれらの患者 9 名中、接触できた 8 名に対して調査を実施した結果、7 名は発症前 24 時間までの間に生カキを、また 1 名はカニを喫食していることが判明し、同月 30 日、BC 衛生局（BCMOH）はカキ、貝類等、軟体動物性の魚介類の生食、不完全調理での喫食を避けるよう警告を出した。同月 31 日、バンクーバー／リッチモンド衛生委員会は、同地域のレストランにおける生食するカキ等の販売禁止措置を取った結果、新たな患者発生は急減した。8 月 19 日、カナダ海洋漁業省（DFO）はカキを採取している BC 沿岸沖を閉鎖した。BCMOH により、その後引き続き実施された調査（BC 居住者への聞き込み）では、7 月 1 日～9 月 26 日までに培養検査で確認された患者 51 名中 42 名の 35 名（83%）が生あるいは半調理のカキを喫食していること、喫食したカキは BC 沿岸で収穫されたもので、それらのサンプルに各種血清型の腸炎ビブリオ汚染（200 個以下/g（CFU））のあることが確認された。なお、8 月 19 日に DFO による BC 沿岸全域でのカキの捕獲禁止措置が取られて以降、新たな集団発生は報告されていない。

ワシントン：7 月 18 日、ワシントン州衛生局（WDOH）は地域衛生局及び患者からの食中毒の報告に基づき、完全に熱処理したカキのみを摂取する旨の勧告を出した。8 月 14 日、新たな食中毒事故が報告されたことを受け、WDOH は商業用のカキ採取者に対して、カキの採取後 4 時間以内に冷蔵処理をするよう通告した。8 月 20 日、一般の人々に対し、完全に熱処理したカキのみを摂取するよう勧告した。8 月 23 日、米国食品医薬品局（FDA）はカキの適切な調理法に関しての陳述書を発行した。WDOH は、5 月 26 日～9 月 9 日までに

培養検査で確認された腸炎ビブリオ感染者 56 名中 54 名について調査を実施した結果、48 名は発症前に軟体動物性魚介類、そのうち 42 名はカキを喫食していること、また 35 名はワシントン州で捕獲された魚介類を喫食していることが判明した。8 月 20 日、太平洋沿岸カキ養殖協会はワシントン州からのカキの出荷を自主的に中止、また WDOH は 8 月 28 日魚介類採取地域にあるカキ養殖場を閉鎖した。同養殖場は 9 月 15 日再開したが、その後の患者発生は報告されていない。

オレゴン：8 月 21 日、オレゴン衛生局（OHD）は地域の衛生局及び微生物研究施設に腸炎ビブリオに関連している疾病又は腸炎ビブリオ分離株を直ちに届け出るよう要請した。その結果、フードネット（Foodborne Disease Active Surveillance Network : FoodNet）により、腸炎ビブリオ患者の報告が増加した。OHD は、7 月 19 日～9 月 27 日の間に発症した腸炎ビブリオ患者 13 名（培養確定済）に対し調査を実施した結果、12 名は軟体動物性魚介類、そのうち 10 名は生カキを喫食していることが判明した。また、喫食したカキの採取地調査では、BC 近海が 4 名、ワシントン州が 4 名、オレゴン州・カリフォルニア州が各 1 名であった。8 月 26 日、関係当局は原因と目されたオレゴン州のカキ養殖場を閉鎖すると同時に、太平洋北西沿岸で採取された魚介類の生での喫食を避けるよう報道機関を通じて要請した。カキ養殖場閉鎖後におけるオレゴン産生カキに関連した患者発生は報告されていない。

カリフォルニア：5～7 月の間に、計 11 名の腸炎ビブリオ確定患者がサンフランシスコ市・郡公衆衛生局からカリフォルニア衛生局（CDHS）に報告された。この事態を受けて、8 月 18 日、サンフランシスコ衛生当局は公衆やレストランに対して、カキ等の魚介類を生のまま摂取しない又は提供しないよう通告した。そして 8 月 19 日、CDHS はワシントン州及び BC 沿岸沖で採取されたカキ等の魚介類の生食を避けるよう警告する等の処置を取った。一方、CDHS では 6 月 9 日～12 月 9 日の間に発症した計 83 名の腸炎ビブリオ確定患者に対し、聞き取り調査を実施した結果、その内 68 名が発症 1 週間までの間にカキを喫食していたことが判明した。これらのうち、59 名はワシントン州及び BC 沿岸沖で採取したカキを喫食していたが、他の 9 名が喫食したものはカリフォルニア州 Tomales 湾（サンフランシスコの北 40 マイルに位置する）で採取されたものであった。

集団発生の要約：ワシントン州及び BC で採取された魚介類の喫食に関連した腸炎ビブリオ感染症は、7 月 20 日～8 月 24 日までの間にユタ（3 名）、アラスカ（1 名）、メリーランド（1 名）、ハワイ（1 名）でも報告されており、この集団発生における北米全体の患者（培

養確認済）は総計 209 名（1 名死亡）にのぼった。患者の発症日は 5 月 26 日～12 月 9 日（中間値 8 月 8 日、図 1）で、患者からは様々な血清型の腸炎ビブリオが検出されたが、その一部はカキから検出された株のそれと合致した。患者の平均年齢は 39 才（範囲 12～85 才、67%）の男性で、主症状は下痢（99%）、腹痛（88%）、嘔気（52%）、嘔吐（39%）、発熱（33%）、血便（12%）であった。

米国海軍によって調べられた 1997 年 5 月 13 日～9 月 9 日までの太平洋沿岸海水表層の平均水温は 54F～66F（12℃～19℃）で、1996 年の同時期に比べて 2F～9F（1℃～5℃）高いものであった。また、原因と目された地域で採取されたカキから腸炎ビブリオが検出されているが、その多くは菌数が 200 個以下/g、最も高いもので 11,000 個/g であった。

Editorial Note《編集注記》

腸炎ビブリオ感染量試験に基づき、米国及びカナダは 1g につき 10,000CFU より少ない腸炎ビブリオ菌を含有するカキの販売を認可しているが、このガイドラインを順守しても今回の事件を防ぐことはできなかった。カナダの衛生局が取った腸炎ビブリオ感染症に関連していると思われるカキ採取海域の閉鎖は有効であり、9 月に BC 海域からカキを採取することを一時停止したことでその後の疾病は急激に減少した一方、米国ではカキに関連した感染症の発生は 12 月まで続いた。

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Outbreak of *Vibrio parahaemolyticus* Infections Associated with Eating Raw Oysters -- Pacific Northwest, 1997

During July-August 1997, the largest reported outbreak in North America of culture-confirmed *Vibrio parahaemolyticus* infections occurred. Illness in 209 persons was associated with eating raw oysters harvested from California, Oregon, and Washington in the United States and from British Columbia (BC) in Canada; one person died. This report summarizes the investigations of the outbreak, which suggest that elevated water temperatures may have contributed to increased cases of illness and highlights the need for enhanced surveillance for human infections.

British Columbia

During July 1-19, the BC Provincial Laboratory received isolates of *V. parahaemolyticus* from nine patients, more than twice the expected number for July. Because of the high number of isolates identified, the BC Center for Disease Control (BCCDC) conducted interviews with the eight patients who could be contacted; seven had eaten raw oysters during the 24 hours before illness onset, and one had eaten crabs. On July 30, the BC Ministry of Health (BCMOH) issued a public health alert advising that molluscan shellfish (e.g., oysters, clams, mussels, and scallops) should not be eaten raw or undercooked. On July 31, the Vancouver/Richmond Health Board banned the sale of raw molluscan shellfish in restaurants in the cities of Vancouver and Richmond, BC. These actions were followed by a rapid decline in the number of new cases. On August 19, the Federal Department of Fisheries and Oceans (DFO) closed all BC coastal waters to the harvesting of oysters.

The BCMOH continued to interview BC residents with culture-confirmed *V. parahaemolyticus* infections; information was obtained from 42 of the 51 persons with illness reported during July 1-September 26. Of the 42, a total of 39 (93%) had eaten molluscan shellfish and 35 (83%) had eaten raw or undercooked oysters during the 4 days before onset of illness; 28 had eaten oysters purchased at restaurants or other

food establishments in BC; and seven had eaten oysters they had harvested. Oysters eaten by ill persons were traced by BCCDC, the Canadian Food Inspection Agency (CFIA), and BCMOH to harvesting areas along the BC coast. Samples of oysters harvested from these areas contained multiple *V. parahaemolyticus* serotypes at less than 200 colony-forming units (CFU) per gram of oyster tissue. No additional outbreak-related illnesses were reported in BC residents after DFO closed the coastal waters to the harvesting of oysters. The closure remained in effect until September 12, after which no additional cases were reported.

Washington

On July 18, on the basis of reports of illness received from local health departments and from ill persons, the Washington Department of Health (WDOH) issued an advisory that persons eat only thoroughly cooked oysters. On August 14, after additional cases had been reported, the WDOH advised commercial harvesters to refrigerate oysters within 4 hours after harvesting, and on August 20, advised the public to thoroughly cook molluscan shellfish from both commercial and noncommercial sources. On August 23, the U.S. Food and Drug Administration (FDA) also issued a statement regarding proper procedures for cooking oysters (1).

WDOH interviewed 54 of the 56 persons who had culture-confirmed *V. parahaemolyticus* during May 26-September 9. Of the 54, a total of 48 (89%) had eaten molluscan shellfish before becoming ill; 42 (88%) reported eating oysters. Product traceback by the WDOH's Shellfish Program determined that 35 case-patients had eaten molluscan shellfish harvested in Washington. On August 20, members of the Pacific Coast Oyster Growers Association voluntarily halted shipments of shell oysters from Washington, and on August 28, WDOH closed oyster beds in major shellfish harvesting areas. The oyster beds were reopened on September 15, and no additional illnesses were reported.

Oregon

On August 21, the Oregon Health Division (OHD) requested that local county health departments and microbiology laboratories provide immediate notification of illnesses associated with or isolations of *V. parahaemolyticus*. The request was prompted by an increased number of *V. parahaemolyticus* cases detected by the Foodborne Disease Active Surveillance Network (FoodNet) (a collaboration between CDC, the U.S.

Department of Agriculture, FDA, and seven states for surveillance of foodborne diseases and related epidemiologic studies) and simultaneous reports from BC and Washington of a *V. parahaemolyticus* outbreak associated with eating raw or undercooked shellfish.

OHD interviewed the 13 persons reported with culture-confirmed *V. parahaemolyticus* infections with onsets during July 19-September 27. Twelve had eaten molluscan shellfish; 10 (77%) had eaten raw oysters. Traceback of the oysters that had been eaten indicated they had been harvested in waters near BC (four cases), Washington (four), Oregon (one), and California (one). On August 26, the implicated oyster harvest bed in Oregon was closed by the Oregon Department of Agriculture; only oysters to be cooked could be harvested. On August 28, OHD, in conjunction with the Food Safety Division of the Oregon Department of Agriculture, issued a press release warning persons not to eat raw molluscan shellfish harvested along the Pacific Northwest coast.

After closure of the implicated oyster harvest bed in Oregon, no additional cases associated with eating raw oysters harvested from Oregon waters were reported. The sale of oysters to be eaten raw was reestablished on September 30.

California

During May-July, the City and County of San Francisco Department of Public Health reported 11 culture-confirmed *V. parahaemolyticus* infections to the California Department of Health Services (CDHS). On the basis of these cases, on August 18, San Francisco health officials issued a health advisory recommending that persons not eat raw shellfish and advising restaurants not to serve raw oysters, clams, or mussels. On August 19, CDHS issued a warning about eating raw oysters, clams, and mussels harvested off the coasts of BC and Washington. CDHS interviewed each of the 83 persons reported with culture-confirmed *V. parahaemolyticus* infections with onset during June 9-December 9. Of the 83, a total of 68 (82%) reported eating oysters during the week before onset of illness. Although 59 persons ate oysters identified through traceback as having been harvested off the coast of Washington and BC, nine persons with culture-confirmed illness ate oysters harvested from Tomales Bay, California (40 miles north of San Francisco).

Summary Findings

During July 20–August 24, culture-confirmed cases of *V. parahaemolyticus* infections associated with eating shellfish harvested from Washington or BC also were reported to the state health departments of Utah (three), Alaska (one), Maryland (one), and Hawaii (one). A total of 209 culture-confirmed *V. parahaemolyticus* infections were reported throughout North America during this outbreak. Dates of illness onset ranged from May 26 through December 9 (median: August 8) (Figure 1). *V. parahaemolyticus* isolates from ill persons included many different serotypes, some of which matched serotypes found in oysters. The median age of patients was 39 years (range: 12–85 years); 141 (67%) were male. Clinical histories were available for 196 persons with culture-confirmed infection: 194 (99%) reported diarrhea; 172 (88%), abdominal cramps; 101 (52%), nausea; 77 (39%), vomiting; 64 (33%), fever; and 24 (12%), bloody diarrhea. Of 137 persons providing information on underlying illnesses, 17 (12%) reported an underlying illness. Two patients were hospitalized; one with *V. parahaemolyticus* isolated from her bloodstream died.

Mean Pacific coastal sea surface temperatures recorded by the U.S. Navy ranged from 54 F–66 F (12 C–19 C) during May 13–September 9, 1997 (B. McKenzie, U.S. Navy, personal communication, 1998). These temperatures were 2 F–9 F (1 C–5 C) above temperatures from the same period in 1996.

Oysters from implicated harvest sites contained *V. parahaemolyticus*, but the number of organisms per gram was often less than 200 CFU. The highest levels were greater than 11,000 CFU in samples tested by CFIA.

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Editorial Note:

The last large outbreak of *V. parahaemolyticus* infections reported in North America occurred in 1982 and resulted in 10 culture-confirmed cases. Although *V. parahaemolyticus* outbreaks are rare, sporadic cases are not infrequent. Most infections are associated with ingestion of raw or undercooked shellfish harvested from both the Gulf of Mexico and the Pacific Ocean.

V. parahaemolyticus is a gram-negative bacterium that naturally inhabits U.S. and Canadian coastal waters and is found in higher concentrations during the summer (2, 3). The outbreak described in this report may have been associated with elevated water temperatures. Because *V. parahaemolyticus* concentrations in oysters and shellfish increase with warmer temperatures, enhanced surveillance at the beginning of summer may lead to earlier recognition and appropriate public health action. Water temperature monitoring may help determine when oyster beds should be closed to harvesting to prevent further outbreaks (4).

Epidemiologic and microbiologic studies conducted during this outbreak primarily implicated eating raw oysters. On the basis of studies suggesting that the infectious dose of *V. parahaemolyticus* might be greater than or equal to 100,000 CFU (5), the United States and Canada allow the sale of oysters if there are less than 10,000 CFU of *V. parahaemolyticus* per gram of oyster. However, adherence to these guidelines did not prevent this outbreak. Closure of implicated shellfish beds by health officials was useful; in Canada, additional human illness rapidly declined following a federally mandated suspension of harvesting of shellfish from BC waters in September. In the

United States, shellfish-associated infections continued to occur into December.

The mean incubation period for *V. parahaemolyticus* is 15 hours (range: 4-96 hours). In immunocompetent persons, *V. parahaemolyticus* causes a mild to moderate gastroenteritis with a mean duration of illness of 3 days. Infection can cause serious illness in persons with underlying disease (e.g., persons who use alcohol excessively or have diabetes, pre-existing liver disease, iron overload states, compromised immune systems, or gastrointestinal problems) (2,6). During this outbreak, most ill persons had no underlying illness. To reduce the risk for *V. parahaemolyticus* and other shellfish-associated infections, persons should avoid eating raw or undercooked shellfish. If persons who eat raw or undercooked shellfish develop gastroenteritis within 4 days of ingestion, they should consult a health-care provider and request a stool culture. Only three states (California, Florida, and Louisiana) require visible posting of alerts regarding the risks associated with eating raw oysters at point of retail sale (2, 7, 8). Although assessment of these regulatory educational strategies have indicated compliance is variable (7), other states might consider posting such alerts.

V. parahaemolyticus is not a reportable disease in all states. During this outbreak, public health officials in Washington and California and in BC promptly became aware of the outbreak through routine reporting; in Oregon, although *V. parahaemolyticus* is not reportable, the outbreak was detected through an active surveillance program. All states should consider making *V. parahaemolyticus* and other vibrioses reportable; standard forms are available from CDC's Foodborne and Diarrheal Diseases Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, telephone (404) 639-2206; fax (404) 639-2205.

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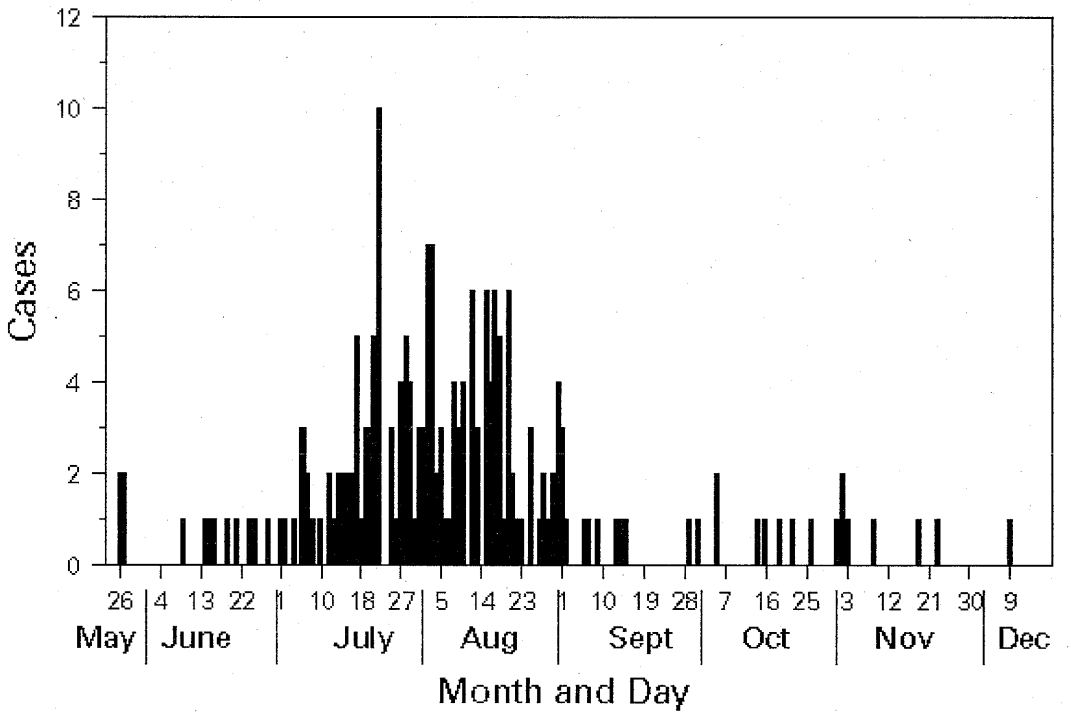
FIGURE 1. Culture-confirmed *Vibrio parahaemolyticus* cases associated with oysters harvested in the Pacific Northwest, by date of illness onset — North America, 1997*



*N=209.

MMWR47 (27):565-567, 1998

食物によるクリプトスポリジウム症の発生 ワシントン州スポーケン，1997 年

1997 年 12 月 29 日、スポーケンの地域保健区（Spokane Regional Health District）は 12 月 18 日スポーケンのレストランのディナーに出席した人々の急性胃腸炎の報告を受けた。症状の特徴は 3～9 日の潜伏期間と下痢であり、寄生虫による疾患の疑いが持たれた。出席者の便の 10 標本の内 8 つがクリプトスポリジウム陽性であった。疫学的調査から、汚染した食材を通じて食物媒体の伝染が生じたことが判明した。遡及的コホート（集団）調査では、62 人の出席者のうち 54 人が発症し、潜伏期の中央値は 6 日であった。症状は下痢、発熱、悪寒、頭痛、身体の痛み、腹部の痙攣等であり、数人が 1 ヶ月又はそれ以上続き、2 人が入院、6 人がヘルスケアを求めた。ビュッフェには 18 種の料理と飲料があり、7 種の料理は火を加えられていなかった。51 人の患者が火を通していないグリーンオニオンを含んだ料理（グラタンサラダ等）を食べていた。15 人の調理人が食材を準備し、給仕した。3～4 週間以内に 14 人の調理人から便の標本を入手し、2 人がクリプトスポリジウム陽性であった。そのうち 1 人は出席者と同様の症状が出て、1 人は無症状であった。他の陰性結果の 1 人を含めて、この 3 人は発症に関与した食材を口にしていた。グリーンオニオンは配送前と利用前共にしっかり洗われていなかった。発症の規模を把握するために保健担当者はスポーケンの医師たちにクリプトスポリジウムの症状を持つ患者の報告を FAX で求めたが、他の症例は報告されなかった。

July 17, 1998 / 47(27); 565-567

Foodborne Outbreak of Cryptosporidiosis -- Spokane, Washington, 1997

On December 29, 1997, the Spokane Regional Health District received reports of acute gastroenteritis among members of a group attending a dinner banquet catered by a Spokane restaurant on December 18. The illness was characterized by a prolonged (3-9 days) incubation period and diarrhea, which led public health officials to suspect a parasitic cause of the illness. Eight of 10 stool specimens obtained from ill banquet attendees were positive for *Cryptosporidium* using both modified acid-fast and auramine-rhodamine staining of concentrated specimens. This report summarizes the epidemiologic investigation of the outbreak, which suggests that foodborne transmission occurred through a contaminated ingredient in multiple menu items.

In a retrospective cohort study, a case was defined as diarrhea or abdominal cramping in a banquet attendee with onset within 10 days after the banquet. Of the 62 attendees, 54 (87%) had illnesses meeting the case definition; they became ill a median of 6 days (range: 3-9 days) after the banquet. Symptoms included diarrhea (98%), fever/chills (61%), headache (59%), body ache (54%), abdominal cramps (50%), nausea (28%), and vomiting (11%). Based on information from initial interviews, the median length of illness was 5 days (range: 1-13 days), but subsequently several persons reported that they had symptoms intermittently for a month or longer. Two persons were hospitalized, and six others sought health care for their illness.

The banquet buffet included 18 separate food and beverage items; seven items contained uncooked produce. No single food was significantly associated with illness. When menu items that contained green onions were combined, foods containing uncooked green onions (au gratin potatoes, romaine salad, and pasta salad) were reportedly eaten by all 51 case-patients who could recall and by three of four persons who were not ill and could recall (undefined relative risk, p=0.07).

The banquet food items were prepared or served by 15 food workers. Stool specimens were available from 14 food workers within 3-4 weeks of the banquet; specimens from two tested positive for *Cryptosporidium*. One of the two food workers was

symptomatic at the same time as banquet attendees; the other was asymptomatic. A stool specimen from another food worker was not available for testing until 5 weeks after the outbreak and was negative; he reported that he worked for 2 days in December while experiencing diarrhea but he could not remember the dates of his illness. All three of these food workers reportedly ate food items served at the banquet associated with the outbreak.

The green onions were not washed before delivery at the restaurant. Food workers at the restaurant reported they did not consistently wash green onions before using them to prepare food or serving them to patrons.

To determine the extent of the outbreak, the health district requested by fax that Spokane area physicians report any patients with symptoms typical of cryptosporidiosis. No other cryptosporidiosis-like illnesses were identified at the time of the outbreak. Two other banquets catered by the restaurant on December 18 and 19 had menus similar to the banquet where the outbreak occurred; no illness was reported in either of these groups.

Reported by: *K Quinn, MPA, G Baldwin, P Stepak, MD, K Thorburn, MD, Spokane Regional Health District; C Bartleson, MPH, M Goldoft, MD, J Kobayashi, MD, P Stehr-Green, DrPH, State Epidemiologist, Washington Dept of Health. Div of Parasitic Diseases, National Center for Infectious Diseases, CDC.*

Editorial Note:

Since 1993, three foodborne outbreaks of cryptosporidiosis have been reported in the United States. In 1993, an outbreak was associated with drinking unpasteurized, fresh-pressed apple cider (1); the apples used for the cider probably were contaminated when they fell to the ground in a cow pasture. In 1995, an outbreak was associated with eating chicken salad that may have been contaminated by a food worker who operated a day care facility in her home (2). In 1996, an outbreak was associated with drinking commercially produced, unpasteurized apple cider (3); the apples used for the cider may have become contaminated when they were washed with well water that had fecal contamination.

The outbreak described in this report had characteristics similar to others in the United States caused by enteric coccidian parasites (*Cryptosporidium parvum* and

Cyclospora cayetanensis) in that case-patients had prolonged diarrhea; the incubation period averaged 6 days; and the attack rates were high (4,5). Physicians and public health officials should have a high index of suspicion for infection with coccidian parasites in patients with severe or prolonged watery diarrhea. Because most laboratories do not routinely test stool for either *Cryptosporidium* or *Cyclospora* (6), specific testing for these organisms generally must be ordered by a physician.

The high attack rate among banquet attendees made finding a statistically significant association with a particular menu item difficult. The strongest association between illness and eating a menu item was observed for food items containing uncooked green onions. This suggests that the onions were a possible source, but the data are inadequate to conclusively implicate them as the vehicle of infection. Available data do not exclude the possibility that multiple menu items may have been contaminated before arriving at the restaurant, contaminated by a food worker, or by cross-contamination during preparation.

This outbreak highlights several key issues for food workers. Uncooked produce should be throughly washed before being placed on kitchen work surfaces to prevent contamination of these surfaces. The FDA Food Code prohibits further bare-handed contact with fruits and vegetables after washing when they are intended for use in "ready-to-eat" foods except where approved by the regulating authority (7). Food preparation surfaces should be washed between preparation of different produce to prevent cross-contamination. Food workers should not work when experiencing a gastrointestinal illness. Persons infected with *Cryptosporidium* may intermittently shed oocysts in stool and remain infectious for up to 60 days after diarrhea has resolved; however, most persons will cease shedding within 2 weeks after resolution of their diarrhea (8). Therefore, food workers should be particularly meticulous about handwashing. Asymptomatic shedding probably occurs in persons exposed to the parasite who have developed some immunity, but the frequency of asymptomatic shedding is unknown.

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MMWR52(40)：956－958

Chitterling（食用のブタ小腸）に曝露した乳児における腸炎エルシニア胃腸炎 イリノイ州シカゴ市，2002年

2002年12月～2003年1月、シカゴ保健局（Department of Public Health：CDPH）は、シカゴの1歳以下の乳児9例において10週間に渡り報告された腸炎エルシニア感染症について調査した。CDPHは、患者を収容した各病院とその医療関係者に警戒を発令し、研究室で確認された全ケースに関する報告を要求した。それに加えて、感染したchitterlingsの小売店からサンプルを採取した。

9例（年齢（中央値）8ヶ月（範囲：生後7週間～13ヶ月））は、2002年11月～12月に発症した。9例中8例は黒人で、chitterlingsを調理した保護者との接触歴があり、内1例はそれを食べた。残る1例は1歳のラテンアメリカ系乳児で、chitterlingsとの関連はみられなかった。曝露時間について情報が得られた7例は発症前の2週間以内（中央値4日間（範囲：1～12日間））にchitterlingsとの直接あるいは間接的接触があった。6例の保護者は、場所は異なるが同じチェーンの食料雑貨店3軒でchitterlingsを購入しており、2例は同じブランド品により曝露した。調理法は保護者により様々で、調理時間は2～12時間であった。発症に関連したchitterlings又はそのロットナンバーは入手できなかった。乳児2例から分離された腸炎エルシニアの血清型はO3であった。乳児1例の便培養結果からはchitterlings関連の腸炎エルシニアと共にネズミチフス菌も分離された。全例共に回復し、症状は胃腸炎に限られていた。米国農務省（USDA）食品安全検査局（FSIS）は、連邦機関による検査を受けた作業場におけるchitterlings検査を規制している。Chitterlingsに曝露した乳児における腸感染症のリスクは持続しており、医療機関は、特に冬季休暇中の黒人家庭の食卓において胃腸炎の原因である腸炎エルシニアに注意する必要がある。

October 10, 2003 / 52(40); 956-958

Yersinia enterocolitica Gastroenteritis Among Infants Exposed to Chitterlings --- Chicago, Illinois, 2002

During December 2002--January 2003, the Chicago Department of Public Health (CDPH) investigated a cluster of *Yersinia enterocolitica* infections reported during a 10-week period among nine Chicago infants aged ≤1 year. This report summarizes the investigation of these cases and underscores the continuing risks for enteric infection among infants exposed to chitterlings (i.e., pork intestines), and the need for health-care providers to be aware of *Y. enterocolitica* as a cause of gastroenteritis, particularly in black children during traditional winter holiday celebrations.

CDPH defined a case of *Y. enterocolitica* gastroenteritis as diarrhea in an infant, with accompanying isolation of *Y. enterocolitica* from stool culture. CDPH alerted hospitals and health-care providers of the cases and requested reports of all laboratory-confirmed cases. Caretakers of the affected infants were interviewed by using a standard case investigation form. Questions were added to determine the source of the chitterlings, brand name, and preparation techniques. CDPH acquired chitterlings from several identified retail outlets for microbiologic testing.

During November--December 2002, nine infants had illness onset; the median age of the infants was 8 months (range: 7 weeks--13 months). Of the nine infants, eight were black and had either eaten chitterlings or spent time in a household in which the dish had been prepared. The one case not associated with chitterlings occurred in a Hispanic infant aged 1 year. All eight infants who were exposed to chitterlings had a history of contact with caretakers who prepared chitterlings, and one had a history of eating chitterlings. For seven infants for whom information about time of exposure to chitterlings was available, all had direct or indirect contact with chitterlings within 2 weeks of illness onset (median: 4 days; range: 1--12 days).

Caretakers of six infants purchased chitterlings from the same grocery store chain but from three separate locations. Two infants were exposed to the same brand of

chitterlings. Caretakers reported different preparation techniques, and preparation times ranged from 2 to 12 hours. No chitterlings or lot numbers associated with the cases were available.

Y. enterocolitica isolates from two infants were serotyped; both were serotype O: 3. Samples of chitterlings obtained for testing yielded *Y. enterocolitica* serotype O: 3 and *Salmonella* serotype Derby. One infant with chitterlings–associated *Y. enterocolitica* also had *S.* Typhimurium isolated from stool culture. All nine infants recovered, and clinical illness was limited to gastroenteritis. Six infants were hospitalized; median duration of hospitalization was 5 days (range: 3–-6 days). The infant who also had coinfection with *S.* Typhimurium required 6 days of hospitalization because of possible intussusception.

Reported by: *RC Jones, MPH, JR Fernandez, SI Gerber, MD, W Paul, MD, Chicago Dept of Public Health; L Williams, Environmental Svcs, Illinois Dept of Public Health. R Turner, DVM, Food Safety and Inspection Svc, US Dept of Agriculture. JT Watson, MD, EIS Officer, CDC.*

Editorial Note:

This report describes nine cases of *Y. enterocolitica* gastroenteritis among Chicago infants, eight of whom were exposed to chitterlings prepared in their homes during the winter holidays. Chitterlings are a known cause of *Y. enterocolitica* gastroenteritis, particularly among black children (*1, 2*). *Y. enterocolitica,* a gram–negative enteric organism commonly present in swine, can cause illness characterized by fever, occasional bloody diarrhea, and abdominal pain. Bacteremia also can occur, especially in infants aged <3 months (*3*). Chitterlings are traditional winter holiday food in certain black families and are readily available in the United States. *Y. enterocolitica* is transferred from raw chitterlings to infants, particularly to bottle–fed infants, through contact with the hands of food preparers (*1, 2*). In Fulton County, Georgia, nearly half of all child caretakers enrolled in an epidemiologic investigation to determine risk factors for *Y. enterocolitica* infection reported household preparation of chitterlings for holiday meals (*1*). In 2002, *Y. enterocolitica* gastroenteritis was reported by active surveillance in FoodNet sites at an incidence of 0.44 per 100,000 population. Incidence has been decreasing for years for undetermined reasons (*4*).

Prevention of yersiniosis should focus primarily on increased consumer awareness of the inherent bacterial contamination of chitterlings as a food product and the risks associated with their preparation and consumption. The Food Safety and Inspection Service of the U.S. Department of Agriculture regulates inspection of chitterlings produced in federally inspected establishments. Preparation of chitterlings requires thorough cleaning before cooking, an extensive process usually performed at home. Special care should be taken when handling raw chitterlings, including careful hand washing by persons cleaning chitterlings before touching children or anything used by children (*5*). Public health officials and clinicians should be alert to the possibility of *Y. enterocolitica* as a cause of gastroenteritis, particularly in black communities during the winter holiday season. Information regarding safe preparation of chitterlings is available at http://www.ph.dhr.state.ga.us/epi/news/oct02/103102.shtml.

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MMWR51 (14):297-300

粉末状乳児用人工乳の使用によるエンテロバクター（*Enterobacter sakazakii*）感染症 テネシー州，2001 年

2001 年 4 月、妊娠 33.5 週で帝王切開にて分娩された男児（1,270g）が、低出生時体重、未熟、呼吸困難のため新生児集中治療室（NICU）に入院した。この乳児は、11 日目に発熱、頻脈、血液循環低下、神経学的異常を呈した。脳脊髄液（CSF）の分析では白血球及び赤血球数の増加と蛋白質濃度上昇、グルコース濃度低下がみられ、CSF 培養にてエンテロバクター（*E. sakazakii*）増殖を認めた。髄膜炎治療のため抗細菌薬の静注を行ったが、神経学的障害は進行し、それから 9 日後に死亡した。

エンテロバクター（*E. sakazakii*）による新生児髄膜炎は珍しいケースであるため、2001 年 4 月 10 日から 20 日に、NICU に入院中の乳児 49 例を対象とし、エンテロバクター（*E. sakazakii*）感染あるいはコロニー形成の有無について調査を行った。テネシー州保健局（Department of Health）と米国疾病管理予防センター（CDC）が協力した。49 例中 10 例でエンテロバクター（*E. sakazakii*）感染あるいはコロナイゼーションが確認された。危険因子の分析では、特殊な粉末状乳児用人工乳（Portagen）の使用だけがエンテロバクター（*E. sakazakii*）感染あるいはコロナイゼーションと有意に関連することが明らかになった。また、感染源特定のための細菌学的検討では、ある同一バッチの Portagen の開封缶及び未開封缶より採取したサンプルにてエンテロバクター（*E. sakazakii*）増殖を認めた。さらなる感染を防ぐため、病院は NICU 患者に原則的に与える人工乳を粉末状のものからすぐに授乳できる液状の無菌人工乳に変更する等、いくつかの変更を行った。

2002 年 4 月 10 日現在、感染や定着のさらなる症状の発見はない。乳児用ミルクの調製はラベル上の指示より詳しい取り扱い説明書に従うべきであり、米国栄養士会（ADA）は適切な乳児用ミルク使用のガイドラインを発行した。加えて、米国食品医薬品局（FDA）は医療担当者に対し、非殺菌の経腸栄養使用による感染リスクの可能性に関する勧告について公式文書を通して発表した。

April 12, 2002 / 51(14); 298-300

Enterobacter sakazakii Infections Associated with the Use of Powdered Infant Formula --- Tennessee, 2001

Enterobacter sakazakii, a gram-negative, rod-shaped bacterium, is a rare cause of invasive infection with high death rates in neonates (*1, 2*). This report summarizes the investigation of a fatal infection associated with *E. sakazakii* in a hospitalized neonate, which indicated that the infection was associated with the presence of the organism in commercial powdered formula fed to the infant. The implicated batch of formula has been recalled by the manufacturer. Clinicians should be aware of the potential risk for infection from use of nonsterile enteral formula in the neonatal health-care setting.

In April 2001, a male infant (2 lbs, 13 oz [1,270 grams]) was delivered by cesarean section at 33.5 weeks' gestation and was hospitalized in a neonatal intensive care unit (NICU) because of low birthweight, prematurity, and respiratory distress. The infant had fever, tachycardia, decreased vascular perfusion, and neurologic abnormalities (e.g., suspected seizure activity) at 11 days. Cerebrospinal fluid (CSF) obtained by lumbar puncture was analyzed and revealed a white blood cell count of 32/mm^3 [normal=0--0.5/mm^3], red blood cell count of 27/mm^3 [normal=0], protein of 292 mg/dL [normal=15--45 mg/dL], and glucose of 1 mg/dL [normal= 40--70 mg/dL]. Culture of CSF grew *E. sakazakii*. The infant was treated with intravenous antimicrobials for meningitis; however, neurologic damage was progressive, and the infant died 9 days later. Because the organism was a rare cause of neonatal meningitis, hospital personnel, in collaboration with the Tennessee Department of Health and CDC, investigated the source of infection.

During April 10--20, 2001 (i.e., the study period), enhanced case surveillance was performed to determine if other infants in the NICU were either infected or colonized with *E. sakazakii*. Patients were assessed for colonization by stool culture; microbiology laboratory records also were reviewed for reports of *E. sakazakii* growth from clinical specimens during the study period. Confirmed infection was defined as any *E. sakazakii*-positive culture from a normally sterile site. Suspected infection was

defined as an *E. sakazakii*-positive culture from a nonsterile site with documented deterioration in clinical status (e.g., increased respiratory rate without other evident cause) in the 24 hours before collection of the specimen for culture. Colonization was defined as an *E. sakazakii*-positive culture from a nonsterile site without documented deterioration in clinical status in the 24 hours before collection of the specimen for culture. A total of 49 infants were screened. Ten *E. sakazakii* infection or colonization events were identified: one confirmed infection in the index patient (culture-positive from CSF), two suspected infections (both culture-positive from tracheal aspirate), and seven colonizations (six culture-positive from stool, one from urine). One patient was colonized at two sites (urine and stool).

A cohort study was performed on the 49 patients who were screened to determine possible risk factors for acquisition of *E. sakazakii* infection or colonization. A case-patient was defined as any NICU patient with *E. sakazakii* infection (confirmed or suspected) or colonization during the study period. Medical records were reviewed to assess possible risk factors during the study period, including gestational age, birthweight, mechanical ventilator use, humidified incubator use, oral medications, and feeding type (total parenteral nutrition, formula [e.g., powdered or liquid], or breast milk) or method (i.e., continuous or intermittent administration). Of the 49 patients identified in the cohort, nine were case-patients and 40 were noncase-patients. Analysis of risk factors identified only use of a specific powdered infant formula product (Portagen [Mead Johnson Nutritionals, Evansville, Indiana]) to be significantly associated with *E. sakazakii* infection or colonization; all case-patients received Portagen compared with 21 of 40 noncase-patients ($p<0.01$).

To determine the source of infection, microbiologic studies were performed on samples of commercially sterile water used for formula preparation and from samples of formula taken from opened cans of Portagen from the same two batches used in the NICU during the study period. Environmental swab cultures were taken from surfaces on which the product had been prepared. Cultures also were performed on unopened containers of Portagen supplied by the manufacturer with batch codes matching those of opened cans. The water was cultured using membrane filtration. The powdered infant formula was cultured using a modification of a previously described enrichment method (*3*). Specifically, for each culture of formula, 100 grams of Portagen were inoculated in phosphate-buffered peptone water, incubated overnight, subcultured, reincubated, and picked and streaked. Colonies that demonstrated a yellow pigment characteristic of *E. sakazakii* were then picked for

identification. Cultures of formula taken from both opened and unopened cans of Portagen from a single batch grew *E. sakazakii*. Water and all environmental cultures were negative. Pulsed-field gel electrophoresis revealed that isolates of *E. sakazakii* from the CSF culture of the neonate with meningitis and from the culture of formula from both opened and unopened containers were indistinguishable.

Hospital personnel reviewed NICU infection-control practices, policies, and procedures for preparation, storage, and administration of powdered infant formula. No breaches in infection control were detected. The product was prepared in the NICU according to manufacturer's instructions. Powdered formula was mixed with sterile water and was immediately refrigerated and used within 24 hours of preparation. The infant with *E. sakazakii* meningitis was given formula by continuous administration; administration or "hang" time (i.e., the amount of time the contents of a formula bag are fed to a patient) did not exceed 8 hours.

To prevent additional infections, the hospital made several policy changes. Principal formula type for NICU patients was changed from powdered formula to a commercially sterile, ready-to-feed liquid formula. Portagen is no longer used; other powdered formula products are reserved for specific needs and, when necessary, are prepared in a designated formula preparation room in the pharmacy. The amount of allowable administration or "hang" time has been reduced from 8 hours to 4 hours. As of April 10, 2002, no additional episodes of infection or colonization have been detected at the reporting hospital.

Reported by: *I Himelright, E Harris, V Lorch, M Anderson, Univ of Tennessee Medical Center at Knoxville; T Jones, A Craig, Tennessee Dept of Health. M Kuehnert, T Forster, M Arduino, B Jensen, D Jernigan, Div of Healthcare Quality Promotion, National Center for Infectious Diseases, CDC.*

Editorial Note:

This report describes an association between fatal infection attributed to *E. sakazakii* and use of a commercial powdered infant formula in a NICU. *E. sakazakii* is a rare cause of invasive disease in neonates; however, when meningitis occurs, severe neurologic complications, including cerebral abscess formation, are common, and death occurs in 33%--80% of cases (*1, 2*). *E. sakazakii* infection, including sepsis, meningitis, or necrotizing enterocolitis, has been associated with use of powdered

infant formula (*4*--*7*). In previous studies and in this report, the organism was detected in either prepared formula, the environment in which it was prepared, or unopened products. This is the first report of *E. sakazakii* infection associated with infant formula prompting recall of a commercial product in the United States. Portagen is a type of formula recommended by the manufacturer for infants with nutritional malabsorption problems and is to be used under the supervision of a health-care provider. The batch of Portagen implicated in this investigation (coded BMC17) was recalled voluntarily by Mead Johnson Nutritionals on March 29, 2002 (*8*). The manufacturer has disseminated a letter to health-care providers about the risk of powdered infant formulas.

Proper handling and use of infant formula products in the health-care setting is an important patient safety issue. Clinicians should be aware that powdered formulas are not sterile products and might contain opportunistic bacterial pathogens such as those in the family *Enterobacteriacae*, including *E. sakazakii* (*3*). These products commonly are used at many hospitals. A recent survey indicated that of 16 responding facilities, nine used powdered formulas in the NICU setting; four (25%) reported powdered formula as a principal source of patient feeding, and five (31%) reported use of powdered formula along with other formula types for principal feeding (National Association of Children's Hospitals and Related Institutions, unpublished data, 2001).

Risk for infection might depend on several factors, including the number of bacteria present in the product, handling after preparation, and underlying patient characteristics (e.g., immunosuppression, prematurity, or low birthweight). Because powdered formula is not sterile and can provide a good medium for growth, prolonged periods of storage or administration at room temperature might amplify the amount of bacteria already present. Health-care providers might be able to reduce risks for hospitalized neonates by choosing alternatives to powdered forms when possible. Preparation of formula should follow manufacturer's instructions, which might require steps beyond those described on the product label. The American Dietetic Association (ADA) has published guidelines for appropriate formula use, including details concerning proper preparation, storage, and administration (*9*). On the basis of these guidelines and input from ADA and the Food and Drug Administration (FDA), interim recommendations have been proposed concerning preparation of powdered infant formula in the NICU setting [see box]. In addition, FDA has disseminated a letter to health-care providers with further recommendations (*10*).

Health-care providers should report invasive disease attributed to *E. sakazakii* in infants aged <12 months, particularly bloodstream infection or meningitis with onset in the health-care setting, to state health departments and CDC (800-893-0485); adverse events associated with infant formula should be reported to FDA's MedWatch program (800-332-1088 or at http://www.fda.gov/medwatch).

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Office of Field Programs, Office of Scientific Analysis and Support, Office of Field Products, Office of Nutritional Products, Labeling and Dietary Supplements, Center for Food Safety and Applied Nutrition, Food and Drug Administration. S Robbins, American Dietetic Association. D Ben-Avram, American Society for Parenteral and Enteral Nutrition. C Braden, R Tauxe, Div Bacterial and Mycotic Diseases, National Center for Infectious Diseases; A Shane, EIS Officer, CDC.

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Box

Summary Interim Recommendations for Preparation of Powdered Infant Formula in the Neonatal Intensive Care Unit Setting

1. Formula products should be selected based on nutritional needs; alternatives to powdered forms should be chosen when possible.
2. Trained personnel should prepare powdered formula under aseptic technique in a designated preparation room.
3. Manufacturer' s instructions should be followed; product should be refrigerated immediately and discarded if not used within 24 hours after preparation.
4. The administration or "hang" time for continuous enteral feeding should not exceed 4 hours.
5. Written hospital guidelines should be available in the event of a manufacturer product recall, including notification of health-care providers, a system for reporting and follow-up of specific formula products used, and retention of recall records.

MMWR49 (36):813-816, 2000

炭疽菌により汚染された食肉の摂食
ミネソタ州，2000年8月

2000年8月25日、ミネソタ州衛生局（MDH）はミネソタ州動物衛生局（MBAH）から同州ロゾー郡のある牧場の去勢牛から炭疽菌（*Bacillus anthracis*）が分離された旨の報告を受けた。この感染牛は、8月20日に牧場で死亡が確認された牛5頭の内の1頭で、組織及び血液培養で分離された菌に対するファージ検査によって炭疽菌と確認されたものである。

7月24日、感染牛を所有している牧場主は起きあがることのできない牛1頭を屠殺、内臓除去及び皮剥処理をした。そして、同地の獣医が家族の食用としてこの牛の屠殺処理を認めたことから、屠体は直ちに注文システムで稼働する食肉加工工場に運ばれ、7月31日及び8月1日に加工処理された。家族2名が、この屠体から加工されたハンバーガーを8月15日、またステーキを8月19日にそれぞれ喫食、他の家族3名も8月20日にハンバーガーを喫食、また食肉を調理したもう1名の家族もこの汚染肉を喫食したと推測された。これらの食肉はよく調理の上、喫食したとされる。汚染肉の喫食の可能性を調査するため、8月25日に実施されたMDHによる家族への聞き取り調査では、2名が胃腸炎症状のあったことを報告（1名は摂取約48時間後に下痢が1日間、他の1名は摂取24から36時間後に腹痛、下痢及び39.1Cの発熱が3日間）したが、両者共、治療を受けることなく回復した。家族は、これ以上食肉を摂取しないこと、医師を受診すること、そしてシプロフロキサシン予防（500mg、1日2回経口投与）を始めるよう勧告された。8月29日、問題の食肉に炭疽菌の汚染があることが確認され、化学的予防の継続を勧告されると共にワクチンの投与がなされた。

一方、ミネソタ州農業局（MDA）は、8月28日に食肉加工業者と接触、問題の食肉処理後の加工肉を凍結処分した。加工工場では、問題の屠体処理後の7頭について同様に加工処理がなされており、これら7頭の食肉加工依頼主はそれら食肉を喫食しないよう勧告されると同時に、焼却処分するため食肉の回収要請を受けた。MDAが8月29日に実施した同加工工場の衛生管理に問題はなく、クリーニングが実施された後に採取された食品加工

工場の加工食肉や環境ふき取りのサンプル検査では、いずれも炭疽菌は陰性であった。

September 15, 2000 / 49(36); 813-6

Human Ingestion of *Bacillus Anthracis*–Contaminated Meat --- Minnesota, August 2000

On August 25, 2000, the Minnesota Department of Health (MDH) was notified by the Minnesota Board of Animal Health (MBAH) of *Bacillus anthracis* isolated from a steer on a farm in Roseau County, Minnesota. The infected steer was one of five dead cattle found in a pasture on August 20. On the basis of phage typing of isolates cultured from tissues and blood samples by the North Dakota State University Veterinary Diagnostic Laboratory, *B. anthracis* was confirmed. This report describes the management of and public health response to human exposure to meat contaminated with anthrax.

On July 24, the farmer who owned the infected steer also had killed, gutted, and skinned a cow that was unable to rise. A local veterinarian approved the slaughter of the cow for consumption by the farmer's family. Immediately after slaughter, the farmer took the carcass (carcass X) to a custom meat-processing plant; on July 31 and August 1, carcass X was processed. Two family members ate hamburgers made from carcass X on August 15 and steaks on August 19; three other family members ate hamburgers on August 20. A sixth member prepared the meals and also may have eaten contaminated meat. All meat was reported to have been well cooked. To investigate the possibility that they had eaten contaminated meat, the family members were interviewed by MDH on August 25. Two reported gastrointestinal illness; one reported 1 day of diarrhea approximately 48 hours after eating meat from carcass X, and the second reported 3 days of abdominal pain, diarrhea, and a temperature of 102.3 F (39.1 C) beginning 24--36 hours after consumption. Both recovered without treatment. The family was advised by MDH not to eat any more of the meat, to contact a physician, and to begin antibiotic prophylaxis with ciprofloxacin (500 mg, orally, twice daily).

On August 29, samples of carcass X tested by the MDH Public Health Laboratory (MDH PHL) were found to contain gram-positive bacilli on microscopic examination. *B.*

anthracis contamination was confirmed at MDH PHL and the U.S. Army Medical Research Institute for Infectious Diseases through culture on blood agar, presence of a capsule, lack of motility, gamma-phage test, and fluorescent antibody to cell wall polysaccharide and capsular antigens. On the basis of this exposure to meat highly contaminated with *B. anthracis*, the family was advised to continue chemoprophylaxis, and vaccination with anthrax vaccine was initiated (Anthrax Vaccine Adsorbed*, Bioport Corporation, Lansing, Michigan).

The Minnesota Department of Agriculture (MDA) contacted the custom meat processing plant on August 28 and placed a hold on all meat processed after carcass X. On August 29, MDA inspected the plant; sanitation practices were satisfactory. Seven carcasses had been processed after carcass X. Owners of meat from the carcasses were advised not to eat any of the meat and were asked to return meat to a central location for incineration; all the meat products were accounted for and none had left Minnesota. Samples from the other carcasses and environmental swabs collected after plant cleaning tested negative for *B. anthracis*.

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Editorial Note:

Anthrax is a zoonotic disease caused by the spore-forming bacterium *B. anthracis*. Human disease usually occurs through cutaneous exposure to infected animal tissue or products. Rarely, inhalation or ingestion of *B. anthracis* spores also leads to anthrax. In the United States during the early part of the 20th century, approximately 130 human cases occurred annually (*1*); two cutaneous infections have been reported since 1992.

Before this exposure, no animal anthrax cases had been reported in northern Minnesota since recordkeeping began in 1909. However, in adjacent areas of North Dakota during 2000, 120--150 cattle have died of anthrax (L. Schuler, North Dakota state veterinarian, personal communication, 2000), and 11 farms have reported anthrax-related cattle deaths in nearby Manitoba, Canada (Figure 1) (J.G. Spearman, Manitoba Department of Agriculture, personal communication, 2000).

Gastrointestinal anthrax in humans occurs 1--7 days after eating raw or undercooked meat from infected animals (*2*), and two forms of gastrointestinal disease have been reported (*3*). Disease affecting the distal gastrointestinal tract results in nausea, anorexia, and fever followed by abdominal pain and bloody stool. The case fatality rate among reported cases ranges from 25%--60% (*2*). Gastrointestinal anthrax never has been documented in the United States because livestock are vaccinated for anthrax in areas where the disease is endemic; animals routinely are inspected by federal and state meat inspectors before, during, and after slaughter; and raw meat is eaten infrequently. Anthrax has not been documented among the persons exposed to *B. anthracis*-contaminated meat described in this report; however, a serologic test to determine presence of infection is pending.

Limited experience with gastrointestinal anthrax complicates recommendations for use of postexposure prophylaxis. An extended duration of therapy is recommended for inhalational exposure because of the persistence of spores resistant to the action of antimicrobial agents (*4, 5*). Upon cessation of chemoprophylaxis, such spores can cause disease several weeks after exposure. No evidence supports the existence of persistent spores associated with gastrointestinal forms of the disease; however, the meat consumed by the family in this report was highly contaminated with *B. anthracis*. Although possible interventions range from close observation to antibiotics alone to antibiotics with vaccination, because the family was at high risk for anthrax infection, management consisted of an extended course of ciprofloxacin combined with administration of anthrax vaccine.

Federal-inspected and state-inspected animal processing facilities are required to perform intensive cleaning after contact with an anthrax-infected carcasses[†] ; veterinary inspection is not provided at custom meat processors. Slaughter house workers who may be exposed to an anthrax-contaminated carcass should receive medical evaluation for symptoms and for possible treatment. Management of anthrax in livestock should include 1) quarantine of the herd; 2) removal of the herd from the

contaminated pasture, if possible; 3) vaccination of healthy livestock; 4) treatment of symptomatic livestock; and 5) disposal of infected carcasses, preferably by burning. Bedding and other material found around the carcass (e.g., soil) should be incinerated with the carcass and buried (*6*).

Veterinarians notified of sudden death in an animal or of an animal unable to rise should consider anthrax as a diagnosis, especially in areas where anthrax is endemic (*6*). However the potential risk for animal anthrax exists in all areas of the United States. Vaccination of livestock in areas where anthrax is endemic is the most effective method of prevention in animals and humans. Cases of anthrax in animals and cases of suspected human exposure should be reported immediately to the state health department, federal animal heath officials, and to CDC's National Center for Infectious Diseases, Meningitis and Special Pathogens Branch, telephone (404) 639-3158.

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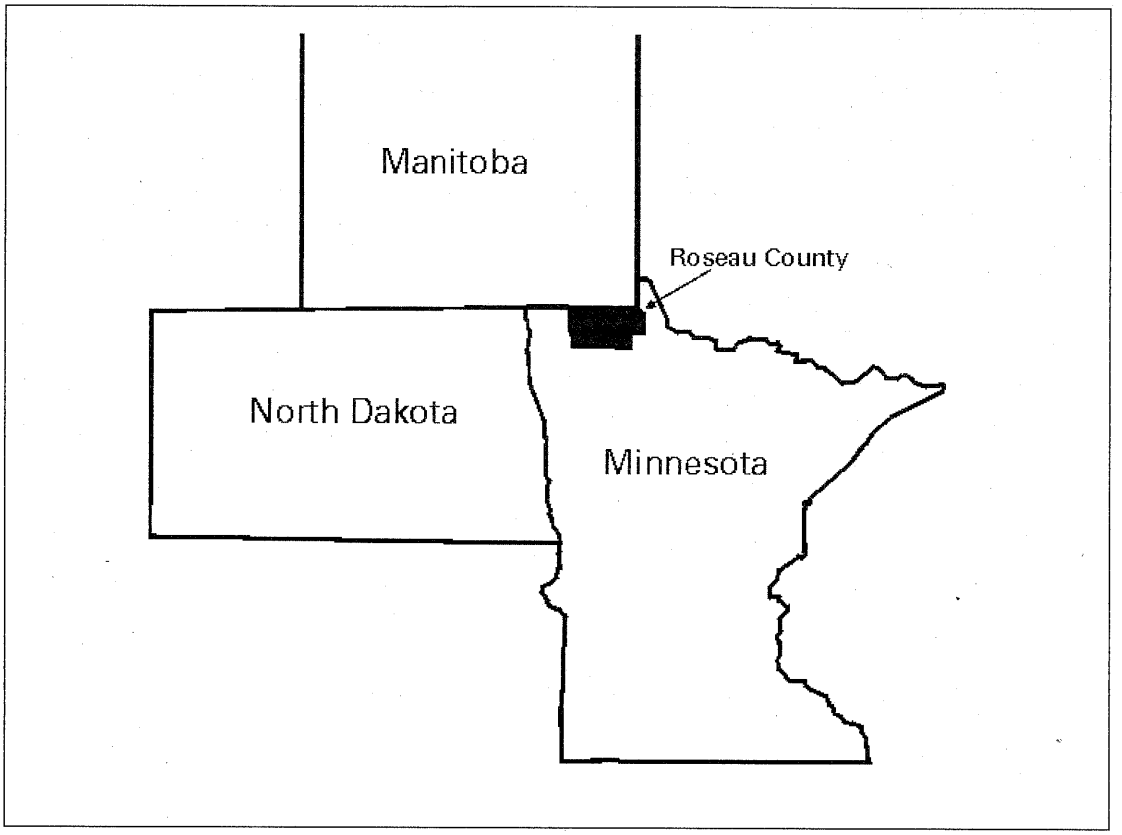
* Use of trade names and commercial sources is for identification only and does not constitute endorsement by CDC or the U.S. Department of Health and Human Services.

† 9 CFR Part 310.9 (2000).

FIGURE 1. Location where *Bacillus anthracis* has been isolated from steer carcasses—Roseau County, Minnesota, North Dakota, and Manitoba, Canada, 2000



MMWR48 (14):285-289, 1999

生パセリの摂食による赤痢菌感染症の発生 米国、カナダ，1998年7月～1998年8月

1998年8月、ミネソタ州保健部門はレストランで発生した2件の赤痢菌感染を米国疾病管理予防センター（CDC）に報告した。疫学的調査により、生の刻みパセリが原因と判明した。同州保健部門と公衆衛生研究所の調査で、7月から8月にかけて6件の同様な発生が確認され、そのうち5件の赤痢菌はミネソタ州の感染症発生と同一のパルス・フィールド・ゲル電気泳動（PFGE）パターンを示した。

米国

ミネソタ州：8月17日、ミネソタ州保健部門は7月24日～8月17日に同じレストランで食事をした2人が細菌性赤痢に感染したとの報告を受けた。このレストランの常連客43人の便標本から赤痢菌が同定され、167人が赤痢症状を示した。レストランの従業員8人に同様な症状が生じ、5人の赤痢菌感染が確定した。感染しなかった常連客との対照調査では、5つの食材（水、氷、ポテト、未調理パセリ、生トマト）が感染に関係しており、多変量解析によりパセリと氷が感染に関係していた。

カリフォルニア州：8月5日、ロサンゼルス郡保健部門は、7月31日に同じレストランで食事をした2人が赤痢菌に感染したと報告を受けた。常連客6人の便標本から、赤痢菌が同定され、3人には赤痢の可能性が示唆された。非感染者と比較すると、感染者は生の刻みパセリのかかった料理を食べていたと考えられる。

マサチューセッツ州：8月11日、マサチューセッツ州保健部門は、7月30日にレストランの昼食パーティー後に6人が発症したと報告を受けた。3人の便標本からは赤痢菌が同定され、3人には赤痢の可能性が示唆された。昼食を取った23人のコホート（集団）調査では、生の刻みパセリのサンドウィッチ等が感染に関係していた。

カナダ

8月10日、オンタリオの厚生大臣は、7月31日～8月3日の料理フェアに参加した3人、1家族が赤痢菌に感染したと報告を受けた。調査により、フェアの売店の料理を食べた他の32人も赤痢菌に感染していることが判明した。20人に食事の経歴を聞いた所、全員が生の刻みパセリの入ったスモークサーモンやパスタを食べていた。

他の調査

上記の発生の他に4件のレストランで赤痢が発生し、218人が赤痢菌に感染していた。聞き取り調査の結果、106人が生の刻みパセリを食べたことが判明した。3件の発生（ミネソタ州、カリフォルニア州、カナダのアルバータ州）で分離された菌株のPFGEパターンが一致した。

追跡、環境調査

関連性の高い7件の事故発生についてパセリの出荷元を確定するため、ミネソタ州と郡の保健部門、米国疾病管理予防センター（CDC）、米国食品医薬品局（FDA）、カナダ食品検査機関（Canadian Food Inspection Agency）は追跡調査を行った結果、メキシコのバハ・カリフォルニアのA農場及びカリフォルニア州の4農場がパセリの出荷元である可能性があった。

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Outbreaks of *Shigella sonnei* Infection Associated with Eating Fresh Parsley -- United States and Canada, July-August 1998

In August 1998, the Minnesota Department of Health reported to CDC two restaurant-associated outbreaks of *Shigella sonnei* infections. Isolates from both outbreaks had two closely related pulsed-field gel electrophoresis (PFGE) patterns that differed only by a single band. Epidemiologic investigations implicated chopped, uncooked, curly parsley as the common vehicle for these outbreaks. Through inquiries to health departments and public health laboratories, six similar outbreaks were identified during July-August (in California {two}, Massachusetts, and Florida in the United States and in Ontario and Alberta in Canada). Isolates from five of these outbreaks had the same PFGE pattern identified in the two outbreaks in Minnesota. This report describes the epidemiologic, traceback, environmental, and laboratory investigations, which implicated parsley imported from a farm in Mexico as the source of these outbreaks.

United States

Minnesota. On August 17, the Minnesota Department of Health received reports of shigellosis in two persons who ate at the same restaurant during July 24-August 17 (Figure_1). *S. sonnei* subsequently was isolated from stool samples of 43 ill restaurant patrons; an additional 167 persons had probable shigellosis (diarrhea {three or more loose stools during a 24-hour period} lasting greater than or equal to 3 days or accompanied by fever). Eight (18%) of 44 restaurant employees had a similar illness; five had laboratory-confirmed *S. sonnei* infection. In a case-control study of 172 ill and 95 well restaurant patrons, five items were associated with illness: water (odds ratio {OR}=1.9; 95% confidence interval {CI}=1.0-3.8), ice (OR=3.7; 95% CI=1.6-8.6), potatoes (OR=2.6; 95% CI=1.5-4.6), uncooked parsley (OR=4.3; 95% CI=2.4-8.0), and raw tomato (OR=1.9; 95% CI=1.0-3.9). In a multivariate analysis, only uncooked parsley

(OR=4.3; pless than 0.01) and ice (OR=6.9; pless than 0.01) remained significantly associated with illness.

California. On August 5, the Los Angeles County Department of Health Services was notified of two persons with shigellosis who ate at the same restaurant on July 31. Stool samples from six ill restaurant patrons yielded *S. sonnei*; an additional three had probable shigellosis (diarrhea {three or more loose stools during a 24-hour period}, or any loose stools accompanied by fever). All 27 foodhandlers denied illness and had stool samples that were negative for *S. sonnei*. In an unmatched comparison with 10 well dining companions, ill patrons were significantly more likely to have eaten foods sprinkled with chopped, uncooked parsley (OR=32.0; 95% CI=1.8-1381.4).

Massachusetts. On August 11, the Massachusetts Department of Health was notified of six persons who reported illness after eating at a restaurant lunch party on July 30. Stool samples from three persons yielded *S. sonnei*; an additional three had probable shigellosis (diarrhea within 4 days of the July 30 meal). Chopped, uncooked parsley was served on chicken sandwiches and in cole slaw served at the lunch. In a cohort study of 23 lunch attendees, illness was significantly associated with eating chicken sandwiches (relative risk {RR} =10.0; 95% CI=2.7-37.2) or eating uncooked parsley with any item (RR=10.0; 95% CI=1.4-70.2). All restaurant employees except one submitted a stool sample for culture; all were negative for *S. sonnei*.

Canada

On August 10, the Ontario Ministry of Health was notified of a family of three persons with *S. sonnei* infection who attended a food fair during July 31-August 3. Laboratory-based surveillance identified 32 additional persons with *S. sonnei* infection who had eaten at a specific kiosk at the fair or at the restaurant that had supplied the kiosk. Of the 35 persons, 20 were questioned about food history; all reported eating a smoked salmon and pasta dish made with fresh chopped parsley. Stool samples from six (38%) of 16 foodhandlers, including the four who handled the parsley, were negative for *S. sonnei*. One child who had eaten at the kiosk was the index patient at a day care center, from which five secondary cases of shigellosis were reported.

Other Investigations

In addition to these four outbreaks, four additional restaurant-associated outbreaks of *S. sonnei* were identified, involving an additional 218 persons with culture-confirmed or probable shigellosis. Of the 111 persons interviewed, 106 (96%) reported eating chopped, uncooked, curly parsley. Isolates from three of these outbreaks (in Minnesota and California in the United States and in Alberta in Canada) matched the outbreak PFGE pattern. In the fourth outbreak (in Florida), one culture-confirmed case was identified; the isolate was not available for PFGE testing.

Traceback and Environmental Investigations

To determine the source(s) of parsley for the seven outbreaks linked by PFGE, state and provincial health departments, CDC, the Food and Drug Administration (FDA), and the Canadian Food Inspection Agency conducted traceback investigations. Farm A in Baja California, Mexico, was a possible source of parsley served in six of the seven outbreaks; four farms in California were possible sources of parsley in two to four of the seven outbreaks.

Field investigations of farm A by FDA and CDC found that the municipal water that supplied the packing shed was unchlorinated and vulnerable to contamination. This water was used for chilling the parsley in a hydrocooler immediately after harvest and for making ice with which the parsley was packaged for transport. Because the water in the hydrocooler was recirculated, bacterial contaminants in the water supply or on the parsley could have survived in the absence of chlorine and contaminated many boxes of parsley. Farm workers and village residents served by this water system reported drinking bottled water or water from other sources. Workers had limited hygiene education and limited sanitary facilities available on the farm at the time of the outbreak.

Foodhandlers at six (75%) of the eight implicated restaurants reported washing parsley before chopping it. Usually parsley was chopped in the morning and left at room temperature, sometimes until the end of the day, before it was served to customers.

Laboratory Investigations

The Minnesota Department of Health laboratory, which has tested isolates of *S. sonnei* by PFGE routinely since 1995, identified a previously unrecognized PFGE pattern of *S. sonnei* and a closely related pattern that differed by a single band

associated with the two outbreaks in Minnesota. The pattern was distributed to other laboratories through PulseNet, the national molecular subtyping network for foodborne disease. In Minnesota and at CDC, strains from all seven outbreaks for which isolates were available for PFGE testing had the outbreak PFGE pattern. Isolates from the seven outbreaks were resistant to ampicillin, trimethoprim–sulfamethoxazole, tetracycline, sulfisoxazole, and streptomycin.

Investigators at the University of Georgia Center for Food Safety and Quality Enhancement conducted studies to determine the effects of temperature and handling on the growth and survival of *S. sonnei* on parsley. Colony–forming units of *S. sonnei* per gram (cfu/g) decreased by approximately 1 log per week on parsley, whether chopped or whole, under refrigeration (39 F [4 C]). In contrast, *S. sonnei* counts increased on parsley kept at room temperature (70 F [21 C]). On whole parsley, the increase was limited to 1 log cfu/g during the first 1–2 days, but on chopped parsley a 3 log cfu/g increase was observed within 24 hours.

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Editorial Note:

S. sonnei is a common cause of gastroenteritis, accounting for 10,262 (73%) of the 14,071 laboratory-confirmed *Shigella* infections reported to CDC in 1996 (1). Humans and other primates are the only reservoirs for *S. sonnei,* and transmission occurs through the fecal-oral route. As few as 10-100 organisms can cause infection, enabling person-to-person transmission where hygienic conditions are compromised. In the United States, *S. sonnei* primarily infects young children and is a common cause of diarrheal outbreaks in child care centers (2). Although reported infrequently, foodborne outbreaks of shigellosis have been associated with raw produce, including green onions (3), iceberg lettuce (4-7), and uncooked baby maize (8).

Before the outbreak described in this report, PFGE was not used routinely by most state public health laboratories to subtype isolates of *S. sonnei,* making it difficult to detect clusters or outbreaks. This investigation demonstrated how the routine use of PFGE and PulseNet can link clusters of *S. sonnei* infections in widely dispersed geographic areas. This same technology is now used widely for comparing isolates of *Escherichia coli* O157:H7. CDC, in consultation with the Minnesota Department of Health, is developing a standard protocol for PFGE subtyping of *S. sonnei* isolates by PulseNet laboratories.

In the outbreak described in this report, isolates were resistant to many antimicrobial agents, including ampicillin and trimethoprim-sulfamethoxazole, which are commonly used to treat shigellosis. This highly resistant pattern is seen more frequently in countries other than the United States. During 1985-1995, antimicrobial resistance among Shigella increased substantially in the United States (9): resistance to ampicillin increased from 32% to 67%, resistance to trimethoprim-sulfamethoxazole increased from 7% to 35%, and resistance to both agents increased from 6% to 19%. A history of international travel was the strongest risk factor for *Shigella* infection resistant to trimethoprim-sulfamethoxazole (9).

The findings in this report indicate that several changes in food storage and food preparation procedures are needed. In restaurants, foodhandling practices such as pooling large batches of parsley for chopping and holding chopped parsley at room temperature increase the risk that sporadic low-level bacterial contamination will lead to outbreaks of gastrointestinal illness. When fresh produce is chopped, the release of nutrients may provide a favorable medium for bacterial growth. The risk for outbreaks can be reduced by storing chopped parsley for shorter times, keeping it refrigerated, and chopping smaller batches (10). Changes in parsley production on the farm (e.g.,

the use of adequately chlorinated water for chilling and icing parsley, education of farm workers on proper hygiene, and possibly the use of post-harvest control measures such as irradiation) may be necessary to ensure that produce is not contaminated with pathogens.

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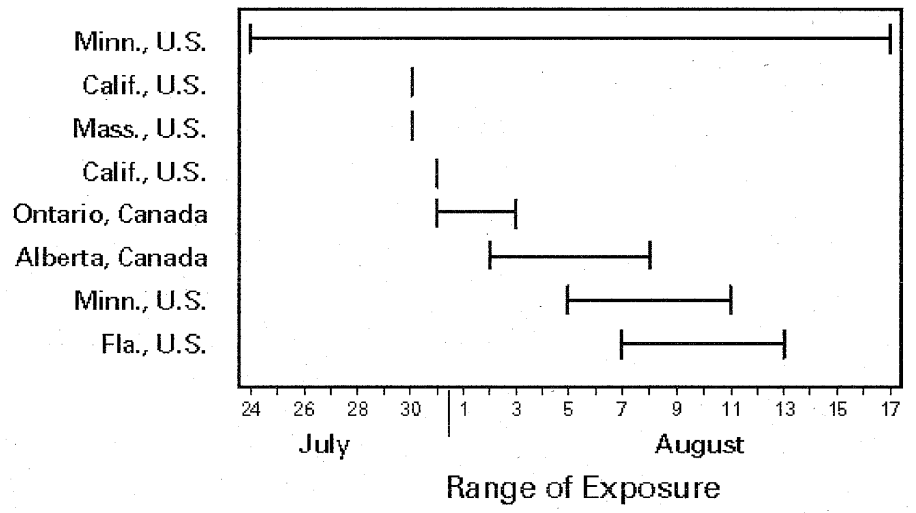
FIGURE 1. Range of dates of exposure for persons infected with *Shigella sonnei* in outbreaks associated with eating fresh parsley — United States* and Canada, July–August, 1998



*Minnesota and California each reported two outbreaks.

MMWR46 (50):1189-1191, 1997

加熱処理済みのハムによるブドウ球菌食中毒の集団発生 フロリダ州，1997 年

1997 年 9 月 27 日、フロリダ州北東部にある地域病院から同州セントジョーンズ郡衛生局に集団食中毒の疑いのある複数の患者がいることが報告された。本事件は、その後のフロリダ州衛生局により実施された調査結果から、9 月 26 日に開かれた退職パーティー参加者に発生したブドウ球菌食中毒と判明した。フロリダ州衛生局では、事件認知後直ちに調査を実施した。以下、調査内容を記載する。

パーティー参加者 125 名に食事履歴、食中毒の発症及び兆候について記載するよう自己記入質問書（self-administered questionnaires）を配布した。パーティーで提供された食事の残り物を回収し、実験分析に回した。また、調理従事者にはパーティーで提供した食事の調理や食材の購入について聞き込みを行った。その結果、パーティー出席者（約 125 名）の中に計 18 名の患者（17 名はパーティー参加者、1 名はパーティーから持ち帰った食品を喫食）のいることが判明した。その臨床症状は、嘔気（94%）、嘔吐（89%）、下痢（72%）などで、潜伏期間は平均 3.4 時間（1～7 時間）、有病期間は平均 24 時間（2～72 時間）、2 名が入院(1 晩)した。出席者に対する喫食調査の結果、発病と密接な関連性が認められたのはパーティーで提供されたハムのみで（発病者 18 名全てが喫食）、他に関連性のある食品は見出せなかった。細菌検査では、残されていた調理ハム及び米飯のピラフについてのブドウ球菌エンテロトキシン検査（逆相受身ラテックス凝集反応）で A 型エンテロトキシンが検出されたが、患者あるいは調理従事者の検査はできなかった。一方、原因と目されたハムの調査では、予め調理されていた 16 ポンドのものを 25 日に 204℃で 1.5 時間加熱、熱いうちにスライサーでカット後冷蔵保存し、26 日のパーティー当日、冷たいまま提供されていたが、ハムのカットに用いたスライサーは日常消毒がなされており、またハムの調理に携わった従事者も特に化膿性疾患などはなく、その汚染ルートは特定できなかった。なお、米飯のピラフはハム調理従事者とは別の人により当日調理されたものであった。

December 19, 1997 / 46(50); 1189-1191

Outbreak of Staphylococcal Food Poisoning Associated with Precooked Ham -- Florida, 1997

On September 27, 1997, a community hospital in northeastern Florida notified the St. Johns County Health Department about several persons who were treated in the emergency department because of gastrointestinal illnesses suspected of being associated with a common meal. This report summarizes the investigation of the outbreak by the Florida Department of Health; the findings implicated staphylococcal intoxication as the cause of illness among some persons who attended a retirement party on September 26, 1997.

Self-administered questionnaires were distributed to the 125 attendees to document food histories, illnesses, and symptoms. A case was defined as nausea and/or vomiting in a person who attended the party or consumed food served at the party and who became ill within 8 hours after eating. Leftover food was collected and submitted for laboratory analysis. Food preparers were interviewed about the purchase and preparation of food served at the party.

Of the approximately 125 persons who attended the party, 98 completed and returned questionnaires. Of these, 31 persons attended the event but ate nothing, and none of them became ill; they were excluded from further analysis. A total of 18 (19%) persons had illnesses meeting the case definition, including 17 party attendees and one person who ate food brought home from the party. Ill persons reported nausea (94%), vomiting (89%), diarrhea (72%), weakness (67%), sweating (61%), chills (44%), fatigue (39%), myalgia (28%), headache (11%), and fever (11%). Onset of illness occurred at a mean of 3.4 hours after eating (range: 1-7 hours); symptoms lasted a median of 24 hours (range: 2-72 hours). Seven persons sought medical treatment, and two of those were hospitalized overnight. Illness was strongly associated with eating ham (risk ratio=26.8 {95% confidence interval=3.8-189.6}). Of the 18 ill persons, 17 (94%) had eaten ham. The ill person who had not attended the party had eaten only leftover ham. None of the other foods served at the party were significantly associated with illness (Table_1).

One sample of leftover cooked ham and one sample of leftover rice pilaf were analyzed by reversed passive latex agglutination to identify staphylococcal enterotoxin and were positive for staphylococcal enterotoxin type A. Samples of stool or vomitus were not obtained from any ill persons, and cultures from nares or skin were not obtained from the food preparers.

On September 25, a food preparer had purchased a 16-pound precooked packaged ham, baked it at home at 400 F (204 C) for 1.5 hours, and transported it to her workplace, a large institutional kitchen, where she sliced the ham while it was hot on a commercial slicer. The food preparer reported having no cuts, sores, or infected wounds on her hands. She reported that she routinely cleaned the slicer in place rather than dismantling it and cleaning it according to recommended procedures and that she did not use an approved sanitizer. All 16 pounds of sliced ham had been placed in a 14-inch by 12-inch by 3-inch plastic container that was covered with foil and stored in a walk-in cooler for 6 hours, then transported back to the preparer's home and refrigerated overnight. The ham was served cold at the party the next day. The rice pilaf was prepared the day of the party by a different person.

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Editorial Note:

Staphylococcal food poisoning, caused by enterotoxin-producing strains of *Staphylococcus aureus*, is one of the most common foodborne illnesses (1). Sudden onset of nausea, vomiting, and diarrhea usually occurs 30 minutes to 8 hours after eating contaminated food; the incubation period may vary in relation to individual susceptibility, amount of toxin in the food, and amount of food ingested. Although the duration of illness is short and almost always self-limited, some deaths have been reported (2).

Although staphylococci are commonly found on environmental surfaces and in a wide variety of mammals and birds, humans are thought to be the primary source of organisms associated with staphylococcal food contamination. Organisms may be present in the nasal passages, throat, hair, and skin of healthy persons, and are abundant in cuts, pustules, and abscesses (2, 3). Staphylococci grow in the

temperature range of 45 F and 118 F (7 C and 48 C); rapid growth and enterotoxin production occurs between 68 F and 99 F (20 C and 37 C). Although growth usually is constrained by the presence of competing organisms, staphylococci thrive in high concentrations of salt and sugar that other organisms cannot tolerate. Staphylococcal enterotoxins are highly resistant to heat. Measures to prevent the growth of *S. aureus* are critical because normal temperatures used in cooking will not destroy the toxins, and foods containing staphylococcal enterotoxin usually look and taste normal (2, 3).

Ham is the most commonly reported vehicle of transmission in staphylococcal food poisoning (1, 4). The salt content of precooked, packaged hams is high, often as high as 3.5%, which provides an ideal growth medium for *Staphylococcus* (2). Although the exact source of contamination for the ham in this outbreak is unknown, the ham could have been contaminated by the food preparer's hands, even though she had no signs of staphylococcal infection. Only one third of food handlers from whom staphylococci are isolated have symptoms consistent with an active staphylococcal infection (4). The ham also could have been contaminated by contact with the slicer because the slicer had not been cleaned adequately. Slicing the ham when the ham was warm increased the surface area and provided a favorable temperature for replication of toxin-producing organisms. In addition, placement of a large quantity of warm, salty ham in a small, tightly closed container prevented rapid cooling and extended the time during which growth and toxin production occurred.

To reduce the incidence of staphylococcal gastroenteritis, potentially hazardous foods such as baked ham must be prepared and served appropriately. The amount of manual handling should be minimal, and food preparers should wash their hands thoroughly before handling food. Food contact surfaces and equipment such as slicers should be cleaned and sanitized. Ham should be sliced cold or, if served warm, immediately before serving to decrease the opportunity for replication of organisms introduced during slicing. Food should be eaten promptly after cooking or refrigerated immediately at a temperature less than or equal to 41 F (less than or equal to 5 C). To permit rapid cooling, food should be stored in small portions in containers that are shallow and loosely covered; this method facilitates adequate air flow and rapid transfer of heat from the food to the container (5).

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TABLE 1. Attack rates and risk ratios associated with buffet foods, by food type — Florida, September 26, 1997

Food	Attack rate (%)		Risk ratio	(95% CI*)
	Ate	Did not eat		
Ham	65.4	2.4	26.8	(3.8–189.6)
Chicken	30.0	25.5	1.2	(0.5– 2.7)
Turkey	38.9	22.4	1.7	(0.8– 3.8)
Rice pilaf	15.4	29.6	0.5	(0.1– 2.0)
Rolls	47.1	20.0	1.4†	(0.8– 2.3)
Eggs	34.8	22.7	1.5	(0.7– 3.3)
Salad platter	31.3	25.5	1.2	(0.5– 2.9)
Nuts	25.0	27.1	0.9	(0.3– 3.3)
Cake	23.5	28.0	0.8	(0.3– 2.2)
Cookies	11.8	32.0	0.4	(0.1– 1.4)
Punch	18.4	37.9	0.5	(0.2– 1.1)

*Confidence interval.
†Summary risk ratio after stratifying on ham consumption.

MMWR50 (23):489-491, 2001

（志賀毒素産生）病原大腸菌 0157:H7 起因と誤診されたカリシウイルス感染症の発生 バージニア州，2000 年

2000 年 2 月 21 日～22 日、バージニア州保健局（Virginia Department of Health : VDH）は、ある大学の保健センターから 2 名の大学生が病原大腸菌 O157:H7 に感染した疑いがあるとの報告を受けた。酵素免疫測定法（EIA）キットにより糞便検体から志賀毒素産生病原大腸菌（STEC）が検出され、その後、カリシウイルスファミリーに属するノーウォーク様ウイルス（流行性胃腸炎ウイルス：NLV）に起因する消化器系疾患を発症した。その前週に消化器症状を訴えた 12 名の学生を調査したところ、いずれも STEC よりウイルスに起因すると思われる症状を呈していた。

疫学・環境調査のサポートのため、バージニア州保健局公衆衛生部門疫学部（Office of Public Health, Office of Epidemiology）から 3 人の職員が地元の保健局に派遣された。職員は 2000 年 2 月 21～22 日の前週に大学保健センターで胃腸症状（gastrointestinal symptom）の診察を受けた 12 人の学生を聞き取り調査した。学生達は 2 月 18 日、レストランで食事した後、72 時間以内に嘔吐と下痢を発症し、潜伏時間は 2.5～49.0 時間（中央値 31.3 時間）、症状は吐き気（97%）、嘔吐（97%）、激しい下腹痛（86%）、寒気（78%）、筋肉痛（67%）、発熱（64%）、頭痛（61%）、下痢（58%）であった。症状の持続時間は 6～120 時間（中央値 26.5 時間）、1 例が入院し、10 例が医療処置を受けた。サンドイッチとサブ（サブマリン・サンドイッチの略称）が症状発生と高い相関性を示した（相対危険度 14.5、95%CI : 2.1～98.1）。

バージニア州総務局統合衛生試験所（Department of General Service, Division of Consolidated Laboratory Service : DCLS）で 2 月 29 日に検査された時点で、2 糞便検体が志賀毒素陽性であったが、病原大腸菌 O157 及び他の STEC は検出されず、その後、逆転写酵素－ポリメラーゼ連鎖反応による検査において 8 検体中 4 検体が NLV 陽性を示した。NLV 感染症の臨床症状は非特異的であり、他の食中毒症状と類似することからその診断基準が現在整備されつつある（症状持続は 12～60 時間、潜伏時間 12～36 時間、症状と特徴

として急激な吐き気、嘔吐、下痢、激しい下腹部及び発熱と倦怠感)。今回のように、非培養式の迅速アッセイにより病原大腸菌 O157 及び志賀毒素産生株を検出し得るが、偽陽性の場合も多く、確定には培養式アッセイが必要と考えられる。

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University Outbreak of Calicivirus Infection Mistakenly Attributed to Shiga Toxin-Producing *Escherichia coli* O157:H7 --- Virginia, 2000

On February 21--22, 2000, the Virginia Department of Health (VDH) was notified by a university student health center of two suspected cases of *Escherichia coli* O157:H7. At a local hospital laboratory, stool specimens from the two ill students tested positive for Shiga toxinproducing *E. coli* (STEC) using a commercially available enzyme immunoassay (EIA) kit. Further investigation revealed that the outbreak of gastrointestinal illness was caused by a Norwalk-like virus (NLV), a member of the calicivirus family. This report summarizes the outbreak investigation and laboratory findings used to identify the causative agent, and highlights the need for follow-up cultures on all specimens testing positive for STEC by EIA and for submission of isolates to state laboratories so that public health agencies can respond appropriately in identifying common source outbreaks.

Three staff members from Virginia's epidemiology office were sent to assist the local health department with the epidemiologic and environmental investigations. VDH staff interviewed 12 students who had sought care for gastrointestinal symptoms at the student health center during the previous week. Most students reported illnesses that appeared more likely to be caused by a virus than by STEC (i.e., vomiting and/or diarrhea lasting 1--2 days that occurred approximately 24--48 hours after eating at an area restaurant [restaurant A]). Other restaurant patrons were located by questioning ill students about persons they knew or recognized at restaurant A on February 18. A case of illness was defined as vomiting or diarrhea occurring within 72 hours of eating at restaurant A. A survey was conducted of 36 ill and 32 well restaurant A patrons. The median incubation period was 31.3 hours (range: 2.5--49.0 hours). Symptoms included nausea (97%), vomiting (97%), abdominal cramps (86%), chills (78%), muscle aches (67%), fever (64%), headache (61%), and diarrhea (58%). The median illness duration was 26.5 hours (range: 6--120 hours). One ill person was

hospitalized and 10 others sought medical care. Eating a sandwich or "sub" (76%) was associated highly with illness (relative risk=14.5; 95% confidence interval=2.1--98.1). No other food item was associated with illness.

The two stool specimens that had tested positive for Shiga toxin at the local hospital laboratory did not yield *E. coli* O157:H7 or other STEC when tested on February 29 at the Virginia Division of Consolidated Laboratory Services (DCLS) using standard biochemical and EIA analysis. Additional stool specimens obtained from ill persons and submitted to DCLS also did not yield Shiga toxin-producing organisms. On subsequent testing by reverse transcriptase-polymerase chain reaction, four of eight specimens were positive for NLV. These results were consistent with the patients' clinical presentation.

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Editorial Note:

In 1995, rapid assays for Shiga toxin first became commercially available. These nonculture assays can detect *E. coli* O157:H7 and other Shiga toxinproducing strains in stool specimens and culture broth (*1*). However, as the findings in this report illustrate, these nonculture rapid assays are subject to false positives, which can result in unnecessary public concern and expenditure of public health resources. Follow-up cultures are needed to confirm the presence of STEC and to obtain isolates for subtyping by pulsed-field gel electrophoresis at state public health laboratories.

Although subtyping is of limited value to the individual patient, it is a useful tool for identifying and responding to common source outbreaks caused by *E. coli* O157:H7 (*2*). Several states require clinical laboratories to submit *E. coli* O157:H7 isolates for this

purpose. Routine submission of all STEC to state public health laboratories also allows enhanced surveillance for illness caused by non-O157 STEC. In 2000, the Council of State and Territorial Epidemiologists adopted a position supporting culture confirmation of positive results from rapid assay tests for pathogens of public health importance (*3*).

Because the clinical signs and symptoms of NLV infection are nonspecific and overlap with other causes of foodborne disease, criteria were developed to aid health-care providers in identifying NLV-associated infection (*4, 5*). These criteria include 1) an illness of 12--60 hours duration, 2) an incubation period of 12--36 hours, and 3) an illness characterized by acute onset of nausea, vomiting, diarrhea, abdominal cramping, and, in some cases, fever and malaise (*4, 6*). Diarrhea is usually more common among adults and vomiting is usually more common among children (*4*). Additional information on NLV is available from CDC's National Center for Infectious Diseases, Division of Viral and Rickettsial Diseases, Respiratory and Enteric Viruses Branch, Viral Gastroenteritis Section at http://www.cdc.gov/od/oc/media/fact/norwalkv.htm.

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MMWR49 (10):207-211, 2000

ノーウォーク様ウイルス胃腸炎の集団発生 アラスカ州及びウィスコンシン州，1999 年

米国では、ノーウォーク様ウイルス（流行性胃腸炎ウイルス：NLVs）は胃腸炎の最も一般的な原因で、毎年約 2,300 万人が罹患している。本症状の患者は、感染してもその免疫が一過性のため、全年齢層に及んでいる。また、集団発生例の多くは食品媒介あるいは人から人への接触感染によるものである。本報告では、アラスカ州における食品媒介による NLVs 集団発生、及びウィスコンシン州における人から人への接触感染による NLVs 集団発生について紹介する。

アラスカ：1999 年 11 月 10 日、アンカレッジにある 1 つの会社から従業員 500 名中、約 20%に急性の胃腸炎を発生しているという届出がアラスカの衛生局にあった。患者は 11 月 8 日、某レストランから仕出しされた会社の昼食を喫食後、悪心、下痢、嘔吐等の胃腸炎症状を呈した。11 月 11 日から当局により 500 名の会社従業員に対する E-mail でのアンケート調査が開始された結果、問題の昼食を喫食した従業員でアンケートに解答した 343 名中 191 名（56%）が規定された症状に合致することが確認された。これら患者の臨床症状は悪心（87%）、下痢（80%）、腹痛（75%）、悪寒（73%）、嘔吐（70%）及び頭痛（65%）で、発病までの潜伏時間（中央値）は 33 時間（範囲 6～96 時間）、また発病期間（中央値）は 24 時間（範囲 5～120 時間）であった。昼食で提供されたポテトサラダを喫食した 236 名中 183 名（78%）が発症し、レストランの客でポテトサラダを喫食した 9 名中 8 名（89%）も発症する等、ポテトサラダの喫食と発症には密接な相関が認められた。問題のサラダは昼食に提供された 2 日前に 2 名の従業員（内 1 名は有病者）によって調製されたもので、発病していた従業員は素手で 12 ガロンの容器中でサラダの材料を混ぜていた。

会社の昼食で発病した 11 名、食品従業員 3 名及びレストランの発病客 2 名の糞便検索を実施した結果、病原細菌は陰性であったが、米国疾病管理予防センター（CDC）で実施された 13 名の RT-PCR（逆転写酵素－ポリメラーゼ連鎖反応）法による遺伝子学的検査ではいずれも NLV 陽性であった（発病会社員 10 名、食品従業員 2 名及びレストラン客 1 名）。また、発病した会社員、レストラン客の発病者各 1 名と問題視された従業員の RT-PCR 結

果によるヌクレオチド配列は完全に一致することも確認された。

ウィスコンシン：1999 年 11 月 30 日～12 月 1 日の間に、大学の寄宿舎の同じフロアで浴場施設を共用して生活している学生 7 名が急性胃腸炎症状を呈した。12 月 1 日からマディソン及びウィスコンシンの衛生当局による調査が開始された結果、寄宿舎に居住する 36 名の学生中、規定された症状に合致した者が 19 名（53%）発見された。観察された臨床症状は、倦怠感（100%）、嘔吐（95%）、悪寒（95%）、体躯痛（84%）、下痢（74%）、腹痛（63%）、筋肉痛（58%）、頭痛（58%）、発熱（44%）で、患者はその発症日から 4 つのグループに分けられた。初発と推定された患者の発病は 11 月 28 日で、同日午後 7 時から 29 日午前 6 時半までの間に頻回の下痢及び嘔吐を呈した。12 名の患者の発病は、11 月 30 日正午～12 月 1 日（2 次感染）、5 名の発病は 12 月 1 日正午～2 日正午（3 次感染）、また 1 名は 12 月 3 日の早朝の発病で、これら患者の平均発病期間は約 24 時間（範囲：3.5～33 時間）であった。学生はいずれも 11 月 27 日～29 日に感謝祭休暇から寄宿舎に帰っているが、疾病と直接関連する特別なイベント、食品や飲物は認められなかった。

患者 8 名から採取された糞便材料 8 件中 7 件について実施された細菌学的検査は、全て陰性であったが 7 件中 5 件の糞便（初発患者、2 次感染者 3 名、3 次感染者 1 名）は RT-PCR 法で NLV 陽性であった。また、CDC で実施された 4 名（初発患者、2 次感染者 2 名、3 次感染者 1 名）の RT-PCR 結果によるヌクレオチド配列も互いに完全に一致していることが確認された。

March 17, 2000 / 49(10); 207-211

Outbreaks of Norwalk-like Viral Gastroenteritis --- Alaska and Wisconsin, 1999

Norwalk-like viruses (NLVs) are the most common cause of epidemic gastroenteritis in the United States, resulting in illness in approximately 23 million persons each year (*1, 2*). Persons of all ages are affected because previous infection confers only short-term immunity (*3*). Most NLV gastroenteritis outbreaks involve foodborne or person-to-person transmission. This report presents investigations of a foodborne NLV outbreak in Alaska and person-to-person transmission in Wisconsin.

Alaska

On November 10, 1999, a company in Anchorage notified the Alaska Division of Public Health that an estimated 20% of its 500 employees had called in sick. Ill workers reported signs and symptoms of acute gastroenteritis (AGE) ---primarily nausea, diarrhea, and vomiting---following a restaurant-catered company luncheon on November 8.

On November 11, questionnaires were e-mailed to 500 company employees; 456 (91%) were returned. A case was defined as nausea, vomiting, or diarrhea occurring in a company employee 1--96 hours after eating the restaurant food. Because the same batches of food served at the luncheon were available at the restaurant during the 4 days following the luncheon, 56 restaurant patrons were identified from credit card receipts to determine whether any were ill. Sanitarians investigated the restaurant facilities and interviewed foodhandlers. Stool samples were collected from ill luncheon attendees, foodhandlers, and ill restaurant patrons.

Of 343 company employees who completed a questionnaire and ate luncheon food, 191 (56%) had an illness meeting the case definition. Among ill luncheon attendees, symptoms included nausea (87%), diarrhea (80%), abdominal pain (75%), chills (73%), vomiting (70%), and headache (65%). Illness onsets occurred a median of 33 hours after

eating the lunch (range: 6--96 hours) (Figure 1), and the median duration of illness was 24 hours (range: 5--120 hours). Of 236 luncheon attendees who ate potato salad, 183 (78%) became ill. In addition, eight (89%) of nine restaurant patrons who responded ate potato salad and became ill. Among luncheon attendees, eating potato salad was associated strongly with illness (odds ratio=42.7; 95% confidence interval=18.6--101.7). The potato salad was prepared 2 days before the luncheon by two foodhandlers, one of whom was ill. The ill foodhandler used bare hands to mix the ingredients in a 12-gallon plastic container.

Stool samples from 11 ill luncheon attendees, three foodhandlers, and two ill restaurant patrons were tested. No bacterial pathogens were isolated. Thirteen specimens tested at CDC by reverse transcriptase-polymerase chain reaction (RT-PCR) were positive for NLV: 10 from ill luncheon attendees, one from the ill foodhandler, and two from restaurant patrons. RT-PCR products from one luncheon attendee, one restaurant patron, and the implicated foodhandler had identical nucleotide sequences.

Wisconsin

During November 30--December 1, 1999, seven students living on the same floor of a university residence hall with shared bathroom facilities developed signs and symptoms of AGE, including nausea, vomiting, and diarrhea. They were taken to local emergency departments. In conjunction with the Madison Department of Public Health and the Wisconsin Division of Public Health, the University Health Service initiated an investigation on December 1.

All residents of the involved floor were administered a symptom, event, and food history questionnaire. A case of AGE was defined as vomiting or three or more loose stools within a 24-hour period in a hall resident during November 27--December 3. Specimen kits were distributed to all hall residents for stool sample collection.

Of the 36 hall residents, all returned from Thanksgiving break during November 27--29. Nineteen (53%) of the residents had illnesses meeting the case definition. Signs and symptoms of illness among the case patients included fatigue (100%), vomiting (95%), chills (95%), body aches (84%), diarrhea (74%), abdominal cramps (63%), muscle aches (58%), headache (58%), and subjective fever (44%). The cases clustered in time into four groups (Figure 2). The probable index case-patient had multiple

episodes of diarrhea and vomiting between 7 p.m. on November 28 and 6:30 a.m. on November 29. Twelve case-patients had illness onsets between noon on November 30 and noon on December 1 (secondary), five had illness onsets between noon on December 1 and noon December 2 (tertiary), and one had illness onset early in the morning of December 3. The mean illness duration was approximately 24 hours (range: 3.5--33 hours). None of the seven patients treated in emergency departments was hospitalized. No particular event, meal, food, or beverage was associated with illness. Only one person with a secondary case reported exposure to someone who was vomiting before illness onset. Three of the five persons with tertiary cases reported exposure to someone who was vomiting during the previous 36 hours.

Stool specimens were obtained from eight case-patients. Seven were tested for bacterial pathogens at the Wisconsin State Laboratory of Hygiene (WSLH), and all were negative. Five of seven stool specimens tested at WSLH were positive by RT-PCR for NLV. The five positive specimens were obtained from the index case-patient, three from secondary case-patients, and one from a tertiary case-patient (Figure 2). The nucleotide sequences of RT-PCR products from four case-patients (the index case-patient and two secondary and the tertiary case-patients) were determined at CDC and were identical.

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Editorial Note:

These two outbreaks illustrate two principal modes of epidemic NLV transmission: consuming contaminated food and person-to-person transmission. In a study of 51 NLV outbreaks, modes of transmission included foodborne (excluding oysters) (37%), person-to-person (20%), consuming contaminated oysters (10%), waterborne (6%), and indeterminate (27%) (*1*). Person-to-person transmission, including direct contact, aerosol, and fomite exposure, generally has been reported in outbreaks involving elder-care settings, hospitals, or cruise ships (*1, 4*).

The low infectious dose of NLVs permits efficient transmission. The Alaska outbreak was associated with eating contaminated potato salad. In this outbreak, an ill foodhandler contaminated enough potato salad to cause illness in at least 200 persons. Evidence from the Wisconsin outbreak, particularly the grouping of cases, suggests that NLV was transmitted person-to-person. Because of the close living quarters and shared bathroom facilities, direct and fomite transmission most likely occurred. Less likely is that secondary case illnesses resulted from aerosol transmission of NLV; only one ill person reported exposure to someone who was vomiting. However, aerosol transmission may have contributed to subsequent NLV spread because three persons with tertiary cases reported exposure to secondary case-patients who were vomiting.

Basic sanitary measures, such as diligent handwashing, can prevent foodborne transmission. In addition, ill workers should be excluded from food handling, and food preparers should minimize direct contact with ready-to-eat foods. Because improper food handling can result in large numbers of persons becoming ill, maintaining food-safety programs is an essential function of public health departments. Constant education of food handling staff and on-site inspections of food preparation facilities, including observation of food handling practices, are basic activities of effective food-safety programs.

Populations in shared living arrangements, such as residence halls, are at greater risk for viral gastroenteritis acquired by person-to-person transmission. Residents in such facilities should use good hygienic practices, especially handwashing. Bathrooms should be kept visibly clean (*5*), and potentially contaminated areas should be cleaned as soon as possible after someone has been ill.

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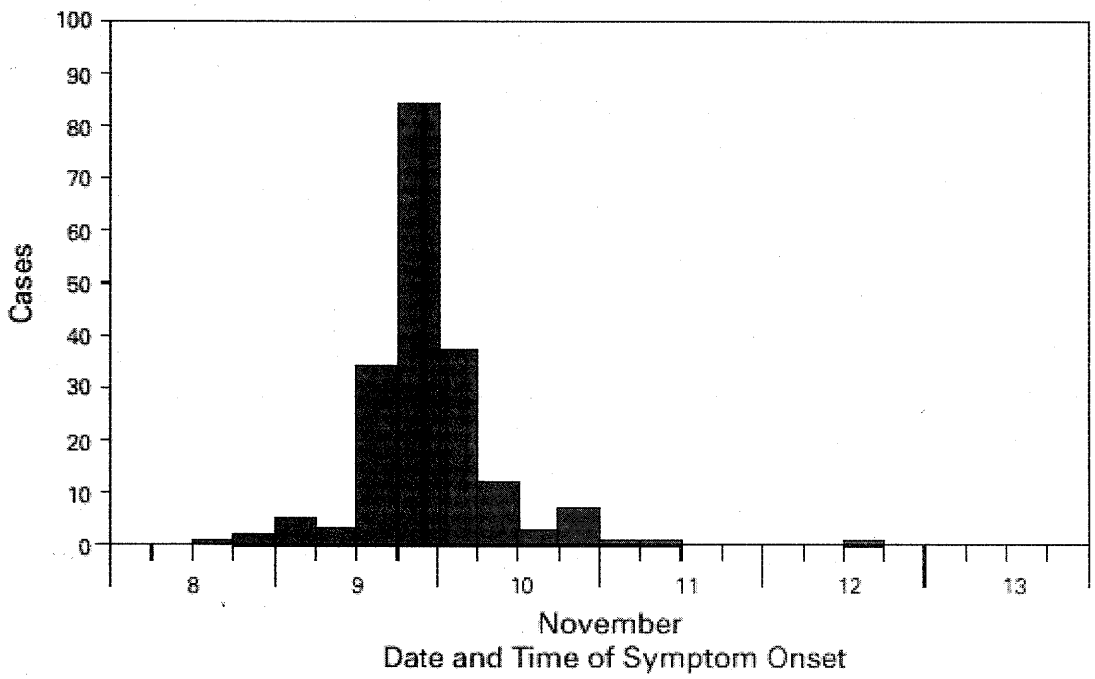
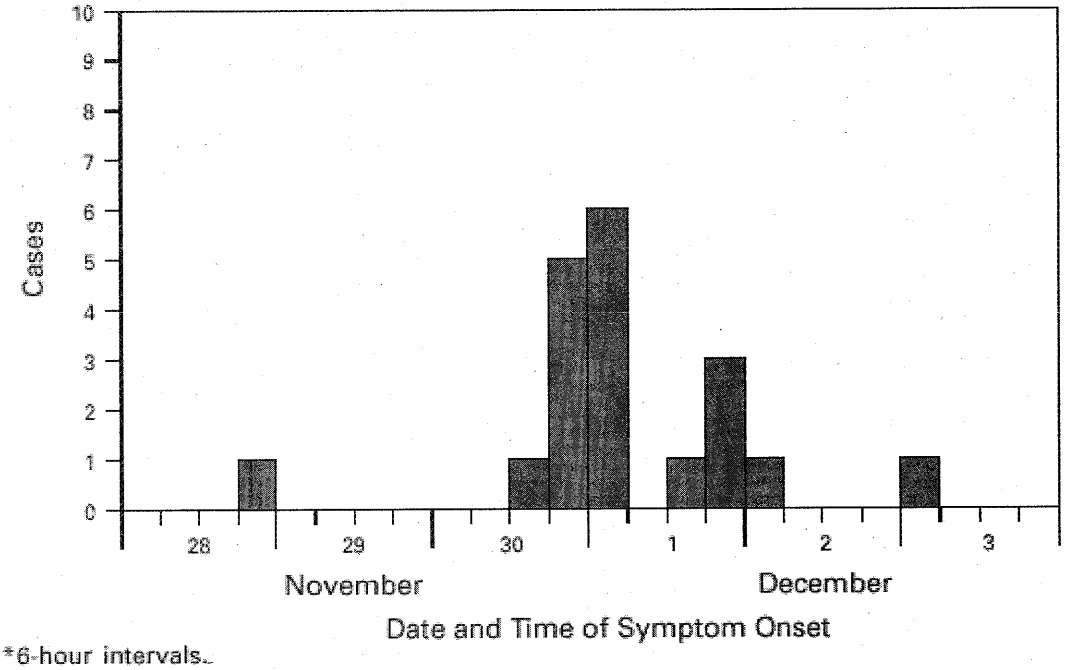
FIGURE 1. Number of cases of Norwalk-like viral gastroenteritis among company employees, by date and time* of symptom onset — Anchorage, Alaska, November 8–13, 1999



*6-hour intervals.

FIGURE 2. Number of cases of Norwalk-like viral gastroenteritis among hall residents, by date and time* of symptom onset — Madison, Wisconsin, November 28–December 3, 1999



*6-hour intervals.

MMWR48 (11):225-227, 1999

米陸軍訓練生におけるノーウォーク様ウイルス胃腸炎の集団発生 テキサス州，1998年

1998年8月27日～9月1日までの間に、テキサス州エルパソにある米陸軍訓練センターの1部隊に所属する835名中99名が胃腸炎（突然の嘔吐、腹痛、下痢及び発熱）のため入院した（未入院者を含めると計126名）。本報告では、8月30日から開始された米陸軍の疫学調査（EPICON）チームがノーウォーク様ウイルス（NLV）によって引き起こされたと指摘した本調査の結果に基づき紹介する。

疾病は、胃腸炎のほか軽度の白血球増多、血小板減少、発熱を伴い、入院期間（中央値）は24時間（範囲12～72時間）であった。入院時採取した糞便の細菌学的、寄生虫学的検査は陰性であったが、米国疾病管理予防センター（CDC）に依頼した24件のRT-PCR（逆転写酵素－ポリメラーゼ連鎖反応）法によるウイルス検査ではその17件から遺伝子タイプ（Genogroup）2に該当するノーウォーク様ウイルス（NVLs）が検出された。

一方、訓練センターにある2か所の食堂の調理従業員に対する聞き取り調査では、食堂1で8月26日にケーキ、パイ及びロールパンを焼いていた従業員1名、また調理には関係していない別の従業員1名が8月27日～29日にそれぞれ発症していることが判明したが、食堂2の従事者で発症を報告したものはいなかった。環境調査では、食堂1のアイスクリーム・ディスペンサーから非病原性の大腸菌群（*Citrobacter diversus* 及び *Enterobacter liquefaciens*）が検出されたが、この試料は室温に放置されていたものであった。また、食堂2のソーダ水容器からも大腸菌群（*Enterobacter cloacae*）が分離されたが、施設内各所から採取された水試料の大腸菌群は全て陰性であった。前週のメニューに基づき、食品嗜好についてのアンケートを入院患者（84名がアンケート実施前10日間に食堂1で喫食）及び無作為に選出された陸軍兵士237名に対して実施された。他方、汚染源を確定するために、8月27～28日に発症した98名を対象に暴露調査を実施した結果、本集団発生は単一暴露型で、その発生に食堂1とソーダ水が密接に関連していることが示された。

Mar 26, 1999/48(11); 225-227

Norwalk-Like Viral Gastroenteritis in U.S. Army Trainees — Texas, 1998

During August 27-September 1, 1998, 99 (12%) of 835 soldiers in one unit at a U.S. Army training center in El Paso, Texas, were hospitalized for acute gastroenteritis (AGE). Their symptoms included acute onset of vomiting, abdominal pain, diarrhea, and fever. Review of medical center admission records for AGE during the previous year indicated that fewer than five cases occurred each month. This report describes the outbreak investigation initiated on August 30 by a U.S. Army Epidemiologic Consultation Service (EPICON) team; the findings indicated the outbreak was caused by a Norwalk-like virus (NLV).

The EPICON team reviewed data from the inpatient records of 90 ill soldiers. AGE was defined as three or more loose stools and/or vomiting within a 24-hour period in a soldier or employee at the training center during August 26-September 1. Illness was accompanied by a minimally elevated leukocyte count, mild thrombocytopenia, and low-grade fever. The median duration of hospitalization was 24 hours (range: 12-72 hours). Stool samples collected from persons with AGE on hospital admission were negative for bacterial and parasitic pathogens. Of 24 stool specimens sent to CDC for viral agent identification, 17 were positive by reverse transcriptase polymerase chain reaction assays for NLVs (genogroup 2).

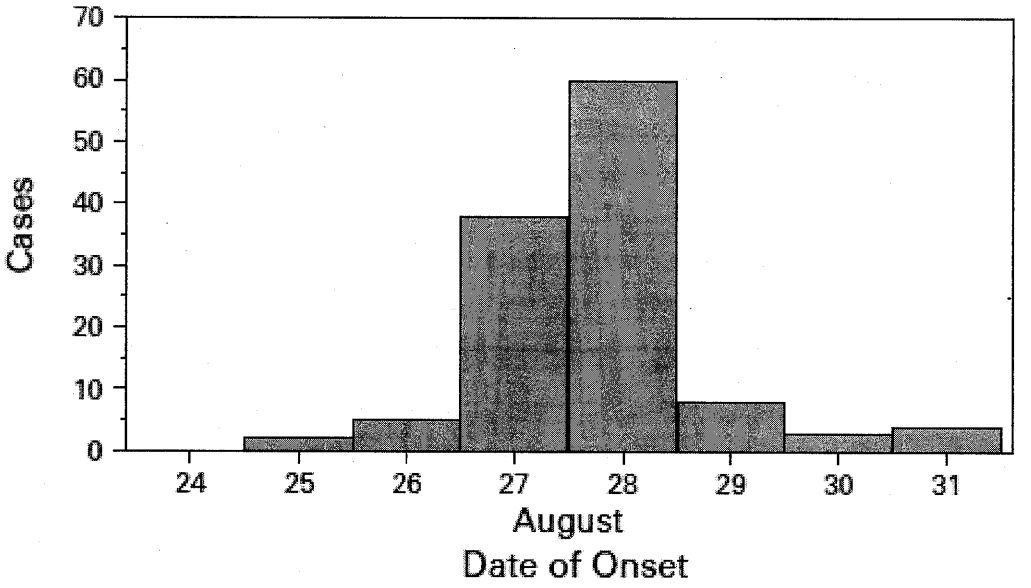
Interviews with foodhandlers in the base's two dining facilities (DF1 and DF2) revealed illness in a confection baker, who had become ill in DF1 while baking crumb cake, pie, and rolls on August 26. One other DF1 employee who was not a foodhandler also reported self-limited gastrointestinal illness during August 27-29. No worker in DF2 reported illness.

Cultures of food specimens from the ice cream dispenser in DF1 grew nonpathogenic coliform bacteria (Citrobacter diversus and Serratia liquefaciens); however, the sample was at room temperature before culture. Enterobacter cloacae coliform bacteria were cultured from the soda fountain in DF2. Water samples taken from multiple sites in the training compound and from elsewhere on post were all negative

for coliform contamination.

A questionnaire about food preferences, based on the previous week's menu, was administered to 86 hospitalized soldiers (84 of whom had eaten in DF1 during the 10 days before answering the questionnaire) and to 237 randomly selected soldiers from the training unit. Of the 237 nonhospitalized soldiers, 41 (17%) did not eat at DF1 during the 10 days before answering the questionnaire; 40 (17%) had illnesses that met the case definition. Thus, cases of AGE were characterized in 126 soldiers (Figure 1).

FIGURE 1. Number of cases of Norwalk-like gastroenteritis in U.S. Army trainees, by date of onset — Texas, August 1998



To determine the point source of the outbreak, cases with onset during August 27–28 (n=98) were analyzed separately for odds ratios (ORs) of selected exposures (Table 1). The univariate OR for illness associated with dining at DF1 during the week before the outbreak was 9.8 (95% confidence interval=2.8–40.2). Two soldiers who ate exclusively at DF2 became ill, and one ill soldier reported not eating at either facility. Food items (crumb cake, pie, cinnamon rolls, and ice cream) and soda fountain dispensers were associated with illness by univariate analysis. Using multivariate analysis, only DF1 and the carbonated beverage dispensers remained strongly associated with illness (Table 1).

TABLE 1. Odds ratios for selected exposures in an outbreak of Norwalk-like viral gastroenteritis in U.S. Army trainees — Texas, August 1998

Exposure	Univariate analysis		Multivariate analysis	
	Odds ratio	(95% CI*)	Odds ratio	(95% CI)
Ever ate at dining facility 1 (DF1) during the week before illness	9.8	(2.8–40.2)	7.3	(2.0–26.4)
Ate preferentially at DF1	3.7	(2.0– 6.9)	2.4	(1.3– 4.5)
Ate at dining facility 2 during the week before illness	1.1	(0.5– 2.3)	0.6	(0.2– 1.4)
Drank carbonated beverages	3.8	(2.0– 7.2)	2.6	(1.3– 5.0)
Ate crumb cake	2.4	(1.2– 4.8)	1.8	(0.8– 3.8)
Ate ice cream	1.7	(1.1– 3.0)	1.1	(0.6– 2.0)
Ate cinnamon roll	1.7	(0.8– 3.7)	1.3	(0.6– 3.0)
Ate pie	1.5	(0.9– 2.7)	1.1	(0.6– 2.0)
Used ice	1.5	(0.8– 2.9)	1.1	(0.6– 2.0)

*Confidence interval.

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Editorial Note:

NLVs, previously known as small round-structured viruses, are the most common cause of nonbacterial gastroenteritis outbreaks in adults (1, 2). Classified in the family Caliciviridae (1, 2), NLVs are transmitted by the fecal-oral route and have been implicated in 42%–71% of viral outbreaks associated with contaminated water and food since the Norwalk virus was identified (1, 3, 4). NLV outbreaks have been caused by eating contaminated raw shellfish and by unsanitary food preparation practices by foodhandlers (1, 3–6). NLVs are hardy, ubiquitous, and extremely persistent in the environment, resisting disinfection and chlorination, and have caused serial gastroenteritis outbreaks (1, 3, 4).

The epidemiologic evidence described in this report indicates that the outbreak was a point-source, propagated, foodborne viral illness. Although cases occurred before the onset of acute illness in the confection baker, he could have been the point source because he probably shed virus before the onset of clinical symptoms. The strong association with drinking carbonated beverages is not easily explained and may

represent increased thirst among ill persons. The use of the Army hospital as a quarantine bay probably decreased secondary propagation of the illness.

Prevention of future outbreaks of NLVs in U.S. military dining facilities or any food service establishment depends on vigilance and rigorous enforcement of simple measures to prevent food contamination. These measures include handwashing, exclusion of ill foodhandlers from the workplace, and basic hygiene and sanitation measures.

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MMWR46 (47):1109-1112, 1997

食用カキによるウイルス性胃腸炎
ルイジアナ州，1996 年 12 月～1997 年 1 月

1996 年 12 月 30 日、ルイジアナ州衛生局(the Louisiana Office of Public Health:LOPH)は、12 月 25 日に生カキを食べた後、胃腸炎を発症した 6 名について報告を受けた。

1996 年 12 月 30 日から 1997 年 1 月 3 日にかけて、さらに 3 例の集団発症が確認された。この 4 例の集団発症において、患者はルイジアナ州で養殖されたカキを食べていた。LOPH は、全ての州の疫学者に対して、食用カキと胃腸炎の明らかな関係について告知を行い、疑わしい症例についての報告を求めた。

胃腸炎の診断基準は、12 月 15 日から 1 月 9 日の間に発症した 24 時間以内に 3 回以上の下痢もしくは嘔吐があった症例とした。アラバマ州、フロリダ州、ジョージア州、ルイジアナ州及びミシシッピー州から、60 の集団発症（493 例）が報告された。60 の集団発症のうち、患者の面接調査が可能であった 34 の集団（290 例）について記述疫学的調査を行った。この 290 例のうち、271 例でカキを食べていた。聞き取り調査を行った 290 例の内、179 例（62%）が、診断基準を満たした。主な症状は、下痢（83%）、激しい腹痛（78%）等であった。潜伏期間（中央値）は 38 時間（範囲：8 時間～90 時間）であった。生カキを食べた 201 例のうち 153 例（76%）が胃腸炎を発症したのに対して、生カキを食べなかった 70 例の内、胃腸炎を発症したのは 13 例（19%）であった（リスク比＝4.0)。

LOPH は、胃腸炎を発症した患者によって食べられたカキの出荷元に関する追跡調査を行った。LOPH はレストラン及び魚介類市場に対して、貝類の取扱い及び貯蔵に関する立入り調査を実施した。漁獲日及び場所を特定するタグや、この生カキの食中毒に間違いなく関連しているサック（カキを入れる袋）の購入者及び小売業者がわかる捕獲者の ID ナンバーを調べた。60 の集団発症に関係したカキについて、26 の小売店、11 の卸売業者、20 の養殖業者が特定された。集団発症の告知後、6 週間目にあたる 2 月 15 日現在、LOPH は 20 の養殖業者の内、3 つの養殖業者に対して聞き取り調査を行った。さらに、他の部署の協力を得て、12 の養殖業者の聞き取り調査を行った結果、8 つのカキ養殖ボートの内、7 つが十分な汚水処理システムを持っていなかった。(LOPH の軟体動物性貝類プログラム

（Molluscan Shellfish Program）が実施した検査によって、1996年11月13日、有毒な水の華（toxic algal bloom）がルイジアナの北東水路で検出された。その結果、LOPHはこの水路を閉鎖し、漁獲者に南東の漁獲場に移るように要請した。さらに11月15日、塩分濃度を下げて北東に存在する水の華を除去するために、淡水迂回路を開門した。それは南東の塩分濃度も下げることになった)。1997年1月3日、LOPHはミシシッピー川の南西に位置する汚染の可能性がある8つの水路の緊急閉鎖を命じ、さらに、LOPHは1月6日、12月22日以降この地域から出荷されたカキの回収を行った。1月23日、養殖業者は営業の再開を許可され、その後カキに関連した胃腸炎は報告されていない。カリシウイルスは、嘔吐あるいは下痢を伴う胃腸炎を引き起こす小さな単鎖RNAウイルスであり、発見が非常に難しい。本報告で得られた知見によって、現行の汚水処理システムとカキの荷札システムの不備が示された。カキに関連した胃腸炎の大量発症を防ぐために、いくつかの適切な対策を講じる必要がある。

November 28, 1997 / 46(47); 1109-1112

Viral Gastroenteritis Associated with Eating Oysters -- Louisiana, December 1996-January 1997

Viral gastroenteritis outbreaks caused by caliciviruses (i.e., Norwalk-like viruses or small round-structured viruses) have been associated with eating contaminated shellfish, particularly oysters (Crassostrea virginica) (1-3). This report describes the findings of the investigation of an outbreak of oyster-associated viral gastroenteritis in Louisiana during the 1996-97 winter season and implicates sewage from oyster harvesting vessels as the probable cause of contaminated oysters.

On December 30, 1996, the Louisiana Office of Public Health (LOPH) was notified about a cluster of six persons who had onset of gastroenteritis after eating raw oysters on December 25. During December 30, 1996-January 3, 1997, three additional clusters were identified. In all four clusters, ill persons had eaten oysters harvested from Louisiana waterways. LOPH notified all state epidemiologists in the United States about the apparent association of gastroenteritis with eating oysters and requested reports of suspected cases.

A case of gastroenteritis was defined as three or more watery stools or vomiting within a 24-hour period, with onset during December 15-January 9. A cluster of oyster-related cases was defined as a group of three or more persons who had shared a common meal, at least one of whom had eaten oysters and at least one of whom developed gastroenteritis. Sixty clusters comprising 493 persons were reported from Alabama, Florida, Georgia, Louisiana, and Mississippi, and all were included in the subsequent traceback investigation. Of the 60 clusters, data were included in the descriptive analysis of the illness only for those 34 clusters for whom all persons in a cluster could be interviewed. The 34 clusters comprised 290 persons who completed interviews and were included in the descriptive analysis; 271 of 290 persons supplied information on oyster consumption.

Onsets of illness occurred during December 21–January 7 (Figure_1). Of the 290 persons interviewed, 179 (62%) had symptoms that met the case definition. The most common symptoms were diarrhea (83%), abdominal cramps (78%), vomiting (58%), headache (50%), and fever (50%). The median incubation period was 38 hours (range: 8–90 hours), and the median duration of illness was 2 days (range: 1–14 days). The median age of case–patients was 42 years (range: 14–83 years), and 111 (62%) were male. The number of reported cases peaked during December 31–January 5 (Figure_1); the harvest dates of subsequently implicated oysters ranged from December 15 to January 1. Of 201 persons who ate raw oysters, 153 (76%) became ill, compared with 13 (19%) of 70 persons who did not eat raw oysters (risk ratio=4.0). Small round–structured viruses were found by direct electron microscopy in fecal specimens from eight of 11 ill persons. Sequence analysis of nucleic acid from eight specimens representing six clusters demonstrated three unique genetic sequences that corresponded with oysters harvested from three separate harvest sites. Small round–structured viruses were detected in oysters, but genetic sequencing could not be conducted.

The LOPH traced oysters eaten by ill persons to retailers, wholesalers, and harvesters. Restaurants and seafood markets were inspected to observe handling and storage of shellfish, and tags that identified the date and site of harvesting and the harvester's identification number were obtained from purchasers and retailers of sacks that were definitely or possibly implicated. Retailer records were cross–referenced with records from wholesalers and harvesters to establish the accuracy of information about harvester and site of harvest. Oysters associated with the 60 clusters were traced to 26 retailers, 11 wholesalers, and 20 harvesters. Records from several wholesalers did not agree with the information on the oyster sack tag.

As of February 15 (6 weeks after notification of the outbreak), LOPH, despite repeated attempts, had been successful in completing interviews with only three of 20 harvesters about the date and specific location of harvesting of potentially contaminated oysters. However, with the assistance of Louisiana Department of Wildlife and Fisheries, 12 additional harvesters were interviewed. Of eight oyster harvesting boats inspected, seven had inadequate sewage collection and disposal systems.

Testing by the LOPH Molluscan Shellfish Program determined that a toxic algal bloom, which causes paralytic shellfish poisoning, was present in Louisiana's northeastern

waterways beginning November 13, 1996; these findings prompted LOPH to close these waterways that day and required harvesters to move to southeastern harvest sites. In addition, on November 15, a freshwater diversion was opened to decrease the salinity and eliminate the algal bloom in the northeastern waters; the diversion also decreased the salinity in the southeastern waters.

On January 3, 1997, LOPH mandated an emergency closure of eight waterways with suspected contamination southeast of the Mississippi River, and on January 6, LOPH recalled oysters harvested from these sites after December 22, 1996. On January 23, 1997, harvesting was permitted to resume, and no additional cases of oyster-associated gastroenteritis were reported.

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Editorial Note:

Caliciviruses are small single-stranded RNA viruses that cause acute gastroenteritis characterized by vomiting and/or diarrhea (4). The viruses are difficult to detect, requiring relatively sophisticated molecular methods to identify the virus in fecal specimens and in oysters. There is no reliable marker for indicating presence of the virus in oyster harvesting waters.

This report represents the third oyster-related gastroenteritis outbreak attributed to calicivirus in Louisiana since 1993. An outbreak in 1993 accounted for cases of illness in 73 persons in Louisiana and approximately 130 persons in other states (5) who had consumed oysters from Louisiana. In that outbreak, a harvester with a high level of immunoglobulin A to Norwalk virus reported having been ill before the outbreak and admitted to dumping sewage directly into harvest waters. The findings of the investigation of that outbreak suggested that one ill harvester could contaminate large quantities of oysters in a relatively large oyster bed (6). An oyster-associated outbreak in 1996 was attributed to a malfunctioning sewage disposal system on an oil rig on which some workers had been ill with Norwalk-like gastroenteritis (LOPH, unpublished data, 1997). However, harvesters dumping feces overboard could not be

excluded as an additional source of oyster contamination. In both outbreaks, recommendations focused on proper sewage disposal and its regulation.

In this outbreak, the link to the large number of wholesalers and retailers suggests that the oyster contamination preceded distribution and probably occurred in the oyster beds. In addition, harvest sites were 12–15 miles from the nearest community sewage outlet, recreational boating was infrequent in December, commercial boating traffic was infrequent because of the shallow depth of the water, and all oil rigs were considered to have had adequate sewage facilities. The only known source of caliciviruses, such as that implicated in this outbreak, is feces from ill persons. Therefore, based on these considerations, the probable source of human sewage found in the implicated waterways was oyster harvesters, who admitted to routinely discharging their sewage overboard, despite recent recommendations in Louisiana for proper sewage collection and disposal (6; LOPH, unpublished data, 1997).

In previous outbreak investigations, molecular tracebacks generally identified a single strain from a single source. A distinguishing feature of this outbreak was its protracted duration and involvement of three geographically separate harvest sites, each associated with a unique strain of calicivirus. These characteristics suggest a contributory role for different oyster harvesters who were concurrently infected with genetically distinct strains of calicivirus, and each of whom dumped their sewage in different waterways, possibly when environmental conditions (e.g., low water temperatures and decreased salinity) facilitated contamination of oysters with calici-viruses.

Findings in this investigation underscore some of the inadequacies in both the current sewage-disposal practices of oyster harvesting vessels and the oyster tagging system designed to reduce the risk for and magnitude of oyster-associated gastroenteritis outbreaks. Oyster-related outbreaks of viral gastroenteritis probably will continue unless seafood regulators and the oyster industry develop, adopt, and enforce standards for the proper disposal of human sewage from oyster harvesting vessels. Traceback investigations of oysters in outbreaks such as this are difficult because of the prevalence of mislabeling in wholesalers' records and on oyster tags and because harvest identification numbers cannot be consistently traced to harvesters. In this investigation, the inability to accurately trace many of the contaminated oysters hampered efforts to contain the outbreak and prevent recurrences and caused a recall of more products than may have been necessary.

Prevention of oyster-related outbreaks of gastroenteritis requires intensified efforts to 1) develop and enforce laws for appropriate sewage containers on oyster harvesting boats with dump-pumpout stations at docks, 2) educate workers in the oyster industry about the consequences of improper sewage disposal, 3) improve record- keeping by oyster harvesters, wholesalers, and retailers to enhance the reliability of traceback investigations, and 4) further assess the relation between environmental conditions and contamination of oysters.

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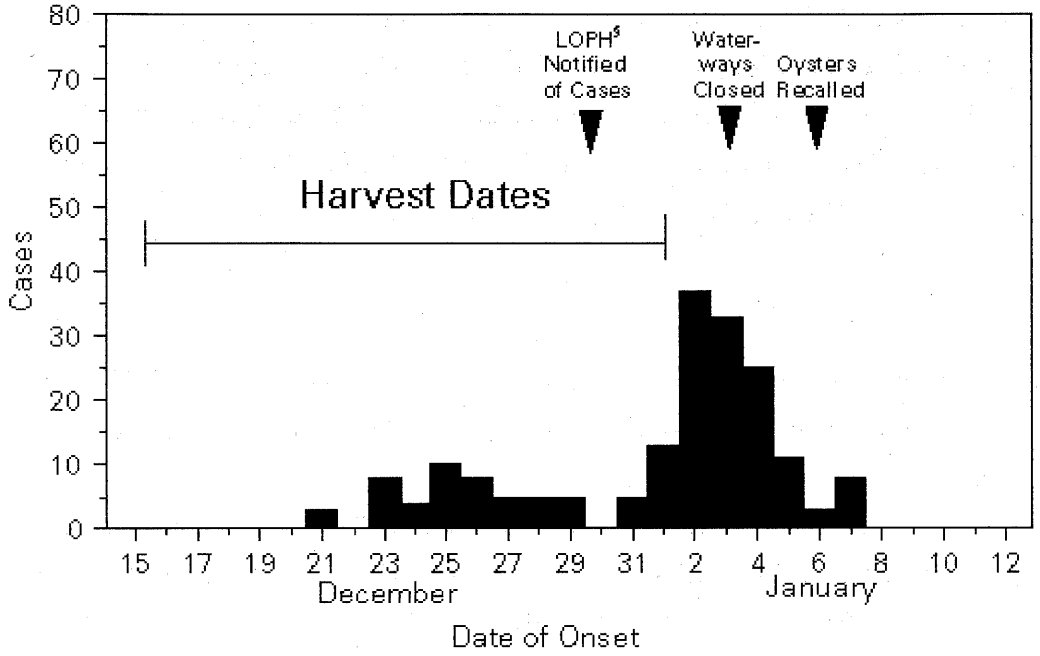
FIGURE 1. Number of cases of gastroenteritis* associated with eating oysters harvested from Louisiana waterways, December 1996–January 1997†



*Defined as three or more watery stools or vomiting within a 24 hour period, with onset during December 15–January 9.
†n=179.
§Louisiana Office of Public Health.

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レストランでのグリーンオニオン(ネギ)によるA型肝炎の集団発生 ペンシルベニア州モナカ市，2003年

ペンシルベニア州保健局と米国疾病管理予防センター（CDC）により、ペンシルベニア州モナカ市のあるレストラン（レストランA）の常連客の間に広がったA型肝炎発生の調査が行われている。11月20日現在、555例に感染が確認され、3例が死亡した。10月14日から11月12日に発症し、発症前2～6週間（9月14日から10月17日、A型肝炎の潜伏期間）にレストランAで食事をしていた207例に対する聞き取り調査では、181例(87%)が10月3日から6日に食事をしていた。レストラン従業員の感染例は、全例共、10月26日以降に発症しているため従業員が感染源とは考えられなかったが、感染した従業員が感染後の10月下旬から11月初旬に勤務していた。そのため、この時期に食事をした客及び感染した従業員と接触のあった約9,000名に対して免疫グロブリンが投与され、レストランAは閉鎖された。感染源特定のため、10月3日から6日に食事をし、10月14日から11月12日に発症したIgM anti-HAV陽性を示す確定診断例181例（4～73歳、中央値34歳）及び同時期に食事をした非発症例83例（2～81歳、中央値28歳）を対象に症例対照研究が実施された。その結果、発症例の98%がグリーンオニオンの入った料理を食べており（非発症例69%）、原因食材としてグリーンオニオンの可能性が示唆された。

現在、米国食品医薬品局（FDA）、CDC、そしてペンシルベニア州内の保健に関する政府諸機関によって、グリーンオニオンのHAV汚染経路の追跡調査が進められているが、現時点ではメキシコ国内の単一、もしくはそれ以上の農場で栽培されたものが原因であることが判明している。FDAは、他にも汚染物を栽培している農場がないか引き続き調査しており、メキシコ政府も追跡調査に協力している。また同局は、11月15日に警報を発令し、消費者に対して昨今のグリーンオニオンによるA型肝炎発生の注意を促した。グリーンオニオンによるA型肝炎感染に特に懸念を抱く消費者に対して、同局は摂取前の念入りな調理をアドバイスし、どのような料理に使おうとしているのかを尋ねることにしている。CDCは現在、ペンシルベニア州内の保健に関する政府諸機関と協力して、グリーンオニオンによるその他のA型肝炎発生例を調査している。11月21日時点で、他の発生例は見つかっ

ていない。

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Hepatitis A Outbreak Associated with Green Onions at a Restaurant --- Monaca, Pennsylvania, 2003

On November 21, 2003, this report was posted on the MMWR *website (http://www.cdc.gov/mmwr). However, two errors* were found. The text of the report printed here has been corrected.*

The Pennsylvania Department of Health and CDC are investigating an outbreak of hepatitis A outbreak among patrons of a restaurant (Restaurant A) in Monaca, Pennsylvania. As of November 20, approximately 555 persons with hepatitis A have been identified, including at least 13 Restaurant A food service workers and 75 residents of six other states who dined at Restaurant A. Three persons have died. Preliminary sequence analysis of a 340 nucleotide region of viral RNA obtained from three patrons who had hepatitis A indicated that all three virus sequences were identical. Preliminary analysis of a case-control study implicated green onions as the source of the outbreak.

Among 207 persons with hepatitis A who were interviewed and who ate at Restaurant A only once during the 2--6 weeks (i.e., the typical incubation period for hepatitis A) before illness, dates of illness onset were between October 14 and November 12. These 207 patrons reported eating food prepared in Restaurant A during September 14--October 17; a total of 181 (87%) persons reported eating at Restaurant A during October 3--6 (Figure). All infected Restaurant A food service workers became ill after October 26, suggesting that a food service worker could not have been the source of the outbreak. However, during late October--early November, these ill food service workers were working in Restaurant A when they could have been infectious. For this reason, immune globulin has been provided to approximately 9,000 persons who ate food from Restaurant A during this time or had exposures to ill persons involved in the outbreak. The restaurant has been closed.

A case-control study was conducted to identify menu item(s) or ingredient(s) associated with illness. A case-patient was defined as a person who had illness onset during October 14--November 12, had laboratory confirmation of acute hepatitis A virus (HAV) infection (i.e., positive IgM anti-HAV), reported eating food prepared at Restaurant A during October 3--6, and had eaten only once at Restaurant A during the 2--6 weeks before illness onset. Controls included persons without hepatitis A who either had dined with case-patients at Restaurant A or were identified through credit card receipts as having dined at Restaurant A during October 3--6. Controls with a previous history of hepatitis A, hepatitis A vaccination, or receipt of immune globulin within 2 weeks after eating Restaurant A food were excluded. Enrolled case-patients and controls were asked about Restaurant A food that they had eaten.

The median age of the 181 case-patients in the study was 34 years (range: 4--73 years), and that of the 83 controls was 28 years (range: 2--81, $p>0.05$). Of 133 menu items, only chili con queso and mild salsa were associated significantly with illness. Mild salsa was eaten by 94% of case-patients, compared with 39% of controls (odds ratio [OR] = 24.2; 95% confidence interval [CI] = 11.4--51.4). Chili con queso was eaten by 15% of case-patients, compared with 3% of controls (OR = 5.2, 95% CI = 1.5--17.8). Both menu items associated with illness contained uncooked or minimally heated fresh green onions. Among 11 case-patients who reported not eating mild salsa, seven ate at least one of the other 52 menu items that contained green onions. Of 103 ingredients used at the restaurant, 12 were associated with illness in a univariate analysis. Of these, 10 had been consumed by <50% of case-patients. Eating a menu item containing green onions was reported by 98% of case-patients, compared with 69% of controls (OR = 20.2, 95%CI = 6.8--59.9). Eating a menu item containing white onions also was associated with illness. However, among the 176 case-patients who reported eating white onions, 174 (99%) also ate green onions. Among the four case-patients and 28 controls who reported not eating green onions, white onions were not associated with illness (OR = 2.5, 95% 0.3--20.9).

During interviews conducted at Restaurant A, food service workers described green onion storage, washing, and preparation practices. Green onions were shipped in 8.5-lb. boxes containing multiple small bundles (6--8 green onions per bundle). Each box was unpacked, and bundles were stored upright (root side down) and refrigerated in a bucket with ice included in the shipment. Green onions were stored $\leq$5 days before processing, which consisted of rinsing intact onion bundles, cutting the roots off, and removing the rubber bands. Green onions from each box were chopped by

machine to yield approximately 8 qts. Chopped green onions were refrigerated for approximately 2 days.

Periodically (i.e., every 1--3 days), salsas were prepared in batches of 40--80 qts. Mild salsa included chopped fresh green onions; hot salsa did not. Salsas were refrigerated in 8-quart containers with a shelf life of 3 days. Mild and hot salsa were ladled into bowls and provided free with tortilla chips upon seating at Restaurant A.

The Food and Drug Administration (FDA), CDC, and the state health departments are investigating the source of the green onions associated with this outbreak and how they became contaminated with HAV. Preliminary traceback information indicates that green onions supplied to Restaurant A were grown in Mexico.

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Editorial Note:

This report describes a large hepatitis A outbreak associated with eating a food item containing green onions at a single restaurant. The majority of ill patrons interviewed as of November 21 were exposed during a 3-day period in early October. No ill food service worker identified could have been the source of the outbreak. The green onions likely were contaminated with HAV in the distribution system or during growing, harvest, packing, or cooling. Traceback investigations completed to date have determined that the green onion source is one or more farms in Mexico.

Both green onions and white onions were associated with illness in the univariate analysis. However, white onions were not associated with illness among those who did not eat green onions. This association with white onions observed in the univariate analysis might not remain when multivariate modeling is completed. Restaurant A purchases previously chopped white onions and adds them to several menu items, including hot and mild salsa. Mild salsa, which contains both green onions and white onions, was associated with illness; however, hot salsa, which contains only white onions, was not associated with illness.

The genetic sequence of the outbreak strain is very similar to viral sequences obtained from persons involved in hepatitis A outbreaks in Tennessee, Georgia, and North Carolina during September 2003 that were linked epidemiologically to green onions. These sequences also were identical or very similar to sequences observed among persons with hepatitis A living along the United States–Mexico border and travelers returning from Mexico, consistent with a source in Mexico (CDC, unpublished data, 2003). Raw green onions from three firms in Mexico have been implicated in the Tennessee and Georgia outbreaks. FDA is still reviewing records to determine if additional firms are involved. The Mexican government is assisting with the traceback investigation in Mexico and the investigation to determine the source of the contamination.

Previous hepatitis A outbreaks linked to green onions have been reported and have involved patrons of a single restaurant (*1*). However, the outbreak at Restaurant A was unusually large. Several characteristics of the way food was prepared and served in Restaurant A could have contributed to the outbreak's size, including 1) multiple opportunities for intermingling of uncontaminated and contaminated green onions in a common bucket for 5 days with the ice in which they were shipped and 2) serving contaminated items with a relatively long shelf life (e.g., mild salsa) to a large proportion of patrons over several days.

HAV is transmitted by the fecal–oral route. Green onions require extensive handling during harvesting and preparation for packing. Contamination of green onions could occur 1) by contact with HAV–infected workers, especially children, working in the field during harvesting and preparation and 2) by contact with HAV–contaminated water during irrigation, rinsing, processing, cooling, and icing of the product. Green onions and other selected produce items (e.g., strawberries [*2*]) might be more vulnerable to contamination because plant surfaces are particularly complex or adherent to viral or fecal particles. Outbreaks of other enteric pathogens linked to green onions have been reported (*3*).

On November 15, FDA issued an alert to consumers about the recent hepatitis A outbreaks associated with green onions (available at http://www.fda.gov/bbs/topics/ANSWERS/2003/ANS01262.html). FDA advised consumers concerned about the possibility of getting hepatitis A from green onions to cook green onions thoroughly before eating and to ask about use of green onions in prepared foods. Unless directed otherwise by public health officials, persons who have

recently eaten green onions do not need postexposure prophylaxis (i.e., immune globulin).

CDC is working with state health departments to identify other hepatitis A outbreaks associated with green onions. As of November 21, no other hepatitis A outbreaks have been identified. To identify other cases related to these outbreaks, state and local health officials should interview persons with hepatitis A with onset after October 1. Persons without typical risk factors for hepatitis A (*4*) should be asked about food and restaurant exposures during their incubation period. Because molecular epidemiologic techniques have been useful for identifying related cases of foodborne hepatitis A in previous outbreaks (*2*), health departments might consider obtaining serum specimens for cases of interest.

An increasing proportion of reported foodborne outbreaks have been linked to fresh produce (*3*). This increase might be attributed to increased consumption of fresh produce or better surveillance techniques. HAV contamination of fresh produce can be reduced by using approaches such as the application of Good Agricultural Practices/Good Manufacturing Practices recommended by FDA (*5*) Recommended control measures include providing sanitary facilities for field workers, ensuring appropriate water quality, use of properly treated manure or biosolids, and ensuring worker health. Reducing HAV transmission in areas where produce is grown and discouraging the presence of children in areas where food is harvested also will reduce opportunities for HAV contamination. Further investigation of this and other hepatitis A outbreaks linked to green onions, including observation of cultivation and harvesting practices, can guide additional specific critical control measures.

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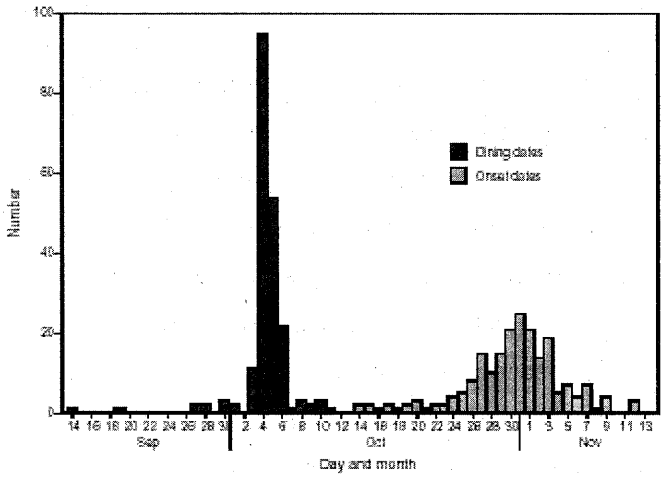
* In the fourth sentence of the fifth paragraph, green onions were stated to have been stored for ≥5 days before processing rather than ≤5 days. In the third sentence of the fifth paragraph of the Editorial Note, the word "of" appeared before "plant surfaces are particularly complex or adherent to viral or fecal particles."

FIGURE. Number of hepatitis A cases*, by date of eating at Restaurant A and illness onset — Monaca, Pennsylvania, 2003



*N = 206. Excludes one case-patient whose illness onset date was not available. Dining dates for three persons who ate at Restaurant A on October 15 (n = one) and October 17 (n = two) are not shown.

MMWR52 (24): 565-567

食品媒介性A型肝炎
マサチューセッツ州，2001年

米国では、1992～2001年に約230,000例のA型肝炎（HAV）症例が報告された。食品取扱い者では年間約8%（数千名）がHAVに感染しており、食品媒介性HAV伝播の可能性は排除できない。

2001年10月26日、マサチューセッツ州公衆衛生局（Department of Public Health：MDPH）に、州内X郡のレストランAの従業員が同月17日よりHAV症状を呈しているとの報告があった。MDPHによる聞き取り調査の結果、この従業員は同月3日から24日に感染したと推定され、その間に調理を手伝っていたことが判明した。同月26日、店主は自主的に休業して清掃を行い、MDPHによって衛生規則違反がないことが確認された。他の従業員にHAV症状はなく（血清学的証拠はないが）、曝露後予防（PEP）目的に免疫グロブリン（IG）を投与後、営業が再開された。

その後、2001年12月3日までに、計46例のHAV感染がX郡内で起こり、MDPHに報告された（発症時期10月29日から11月26日、男31女15、年齢38（範囲5～76）歳）。潜伏期間中に35例（76%）がレストランA、15例（35%）がレストランB、16例（35%）がレストランC、9例（20%）がレストランDで食事をしていた。症例対照研究によりHAVの発症とレストランAでの食事に高い相関性が示唆されたが、レストランAで提供されたどの食品が疾病と関連していたのか疫学的には調査されなかった。

さらに、同じマサチューセッツ州に所在するレストランZの従業員2名がHAVを発症した。2001年11月27日、地元の保健機関はレストランZの従業員に聞き取り調査をし、レストランの検査を行った後、同年11月14日から23日の間にレストランZで非調理もしくは冷製の料理を飲食した者に対して免疫グロブリン接種を勧める公示を発表した。約1,600名が公示に応じ、地元の病院で接種を受けた。

June 20, 2003 / 52(24); 565-567

Foodborne Transmission of Hepatitis A --- Massachusetts, 2001

Hepatitis A virus (HAV) is transmitted typically from person to person by the fecal-oral route. Foodborne transmission occurs when an HAV-infected food handler contaminates food during preparation (*1--3*) or when food is contaminated during harvesting or processing before reaching the food service establishment or home (*4, 5*). Postexposure prophylaxis (PEP) with immune globulin (IG) can prevent hepatitis A among exposed persons if administered within 14 days of exposure. However, the decision about whether to implement PEP for persons who eat food prepared by an infected food handler depends on an assessment of the duties performed by the food handler and personal hygiene while potentially infectious, which are often difficult to determine. This report summarizes the investigation of an outbreak of foodborne hepatitis A in Massachusetts in which a food handler with hepatitis A, who was considered unlikely to transmit HAV, was implicated as the source. The findings underscore challenges faced by local and state health departments when determining whether PEP is appropriate.

On October 26, 2001, the Massachusetts Department of Public Health (MDPH) was notified that a worker at restaurant A in county X had hepatitis A with symptom onset on October 17. On the basis of the date of symptom onset, the worker was considered to have been potentially infectious during October 3--24. The worker's primary responsibility was managerial, but the worker also prepared menu items (primarily sandwiches that were not cooked after preparation) as needed and had worked most recently on October 18. During an interview, the worker reported frequent hand washing and diligent glove use while handling food; supervisors validated the worker's hygiene practices. On the basis of the worker's reported hygiene practices, work duties, and lack of gastrointestinal symptoms, health officials considered HAV contamination of food prepared by this food handler unlikely and did not issue a public notification or recommend PEP for restaurant patrons. The worker denied any change in bowel habits; however, assessment was difficult because the worker had a colostomy and normally produced unformed stool that collected in an ostomy

appliance. The worker reported that the appliance was secured under several layers of clothing and was never changed at work.

On October 26, the restaurant's owners closed and cleaned the restaurant voluntarily. On October 27, an inspection by MDPH found no sanitary code violations. None of the 20 food handlers at the restaurant had symptoms of hepatitis A, although none was tested serologically for evidence of recent HAV infection. The restaurant reopened after 19 food handlers received IG and one was excluded from work.

On November 20, MDPH was notified of six cases of hepatitis A among residents of county X, all with illness onsets during November 8--15. By December 3, a total of 46 persons had been reported in county X, with illness onsets during October 29--November 26 (Figure), compared with no cases during the same period in 2000. The median age of patients was 38 years (range: 5--76 years); 31 (67%) were males. Of the patients who could recall where they had eaten during their hepatitis A incubation period (2--6 weeks before illness onset), 35 (76%) of 46 reported eating at restaurant A, 15 (35%) of 43 at restaurant B, 16 (35%) of 46 at restaurant C, and nine (20%) of 45 at restaurant D. Eating at other restaurants was reported less frequently.

A matched case-control study was conducted to determine whether persons with hepatitis A were more likely than neighborhood controls to have eaten at one of the four restaurants. A case-patient was defined as a resident of county X who had illness onset during October 18--November 29 and had laboratory confirmation of HAV infection (positive IgM anti-HAV). Potential controls were identified by using a web-based neighbor search, matched by age group (2--13 years, 14--22 years, 23--40 years, 41--54 years, and $\geq$55 years) and interviewed by telephone. Potential controls who reported previous hepatitis A vaccination, possible hepatitis A illness during October 18--November 29, or a history of physician-diagnosed hepatitis A were excluded from participation. One neighborhood control was recruited for each of 43 (93%) case-patients; no neighborhood control was found for the remaining three case-patients. Controls were asked about eating food from restaurants from October 1 (4 weeks before the earliest illness onset of any case-patient) to November 12 (2 weeks before the latest illness onset of any case-patient). An exact conditional logistic regression model was used to determine the relation between restaurant patronage and illness; illness was associated with eating food from restaurant A (odds ratio = 29.4; 95% confidence interval = 5.1--infinity) but not food from restaurants B, C, or D. A total of 32 (74%) of the 43 case-patients and seven (16%) of neighborhood

controls reported having eaten food from restaurant A. An epidemiologic study to determine whether any specific foods served at restaurant A were associated with illness was not performed.

Sequence analysis of a segment of HAV RNA isolated from 28 case-patients was performed by using a reverse transcriptase-polymerase chain reaction method (*6*). A total of 25 sequences were identical, including all 21 from case-patients who reported eating food prepared at restaurant A. The remaining four patients reported not eating food from restaurant A during their incubation period. Three additional persons who did not eat at restaurant A had nonidentical viral RNA sequences.

Two case-patients were food handlers at restaurant Z, also in Massachusetts. Each had worked at restaurant Z when they were potentially infectious and prepared foods that were not cooked after handling. On November 27, after interviewing food handlers and inspecting restaurant Z, local health officials issued a public notice offering IG to customers who ate uncooked or cold food prepared at restaurant Z during November 14--23. Approximately 1,600 persons responded to the public notice and were administered IG at a clinic held at a local hospital.

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Editorial Note:

The probable source of the hepatitis A outbreak described in this report was a food handler in restaurant A who worked while infectious and contaminated food that was not cooked subsequently. Although the food handler with hepatitis A was the probable source, transmission from another food handler in restaurant A with unidentified or unreported HAV infection cannot be excluded. This outbreak investigation highlights difficulties faced by public health officials when making hepatitis A PEP decisions. In this investigation, determining the risk for transmission to patrons from the implicated food handler, who handled uncooked foods while potentially infectious, was based on an assessment of self-reported activities such as gastrointestinal symptoms, personal

hygiene, and glove use. The factors that led to transmission despite reportedly good hygiene cannot be determined.

During 1992--2001, approximately 230,000 cases of hepatitis A were reported in the United States (*7*). Although food handlers are not at higher risk for HAV infection because of their occupation, approximately 8% of adults reported with hepatitis A are identified annually as food handlers (CDC, unpublished data, 2003), indicating that thousands of food handlers have hepatitis A each year. Unlike the majority of persons with hepatitis A who transmit HAV only to close contacts, an HAV-infected food handler potentially can transmit HAV to many others and cause a substantial economic burden to public health. The estimated societal cost of a single foodborne outbreak of hepatitis A involving 43 cases was approximately $800,000; >90% of these costs were incurred by the public health department (*8*). Considerable effort is involved in determining the risk for transmission from an HAV-infected food handler to customers.

An interview that includes detailed questions about job duties, work dates, clinical symptoms, and hygiene is the basis for determining the need for PEP. CDC guidelines recommend that PEP can be considered if 1) during the time when the food handler was probably infectious, the food handler both directly handled uncooked foods or foods after cooking and had diarrhea or poor hygiene practices; and 2) patrons can be identified and treated within 2 weeks after the exposure (*9*). However, because good personal hygiene is subjective and difficult to corroborate or might not prevent disease transmission completely, a food handler's report of good hygiene should not be the only criterion for determining whether patron notification and PEP are needed. Other factors that might affect personal hygiene and the potential for HAV transmission should be examined, including the presence of underlying medical conditions. For the outbreak described in this report, the worker's ostomy might have compromised hygiene. HAV transmission from a food handler with a colostomy has been identified previously (D. Perrotta, Ph.D., Texas Department of Health, personal communication, 2003).

A better understanding is needed regarding hygiene practices, clinical symptoms, and viral characteristics that contribute to HAV transmission by contaminated food. However, prevention measures that can reduce the risk for transmission of HAV and other enteric pathogens also should be emphasized, including regular and thorough hand washing, reducing bare-hand contact with foods that are not cooked

subsequently, restricting ill food handlers from working directly with food or food equipment, and providing a sick leave policy so workers can discontinue working while ill (*10*). Hepatitis A vaccination should be encouraged for persons who are both recommended for routine vaccination (i.e., men who have sex with men, illicit-drug users, and persons who plan travel to countries in which hepatitis A is endemic) and are employed as food handlers.

The factors that led to HAV transmission in this outbreak cannot be determined. Until the determinants of HAV transmission through contaminated food are understood better, decisions about providing PEP to customers of food service establishments will continue to be based on data obtained during case interviews and on the judgment and experience of public health officials. Food handlers acquire HAV infection from others within their communities, and reducing food handler transmission of HAV will be achieved ultimately through routine vaccination of persons at risk for HAV infection within these communities.

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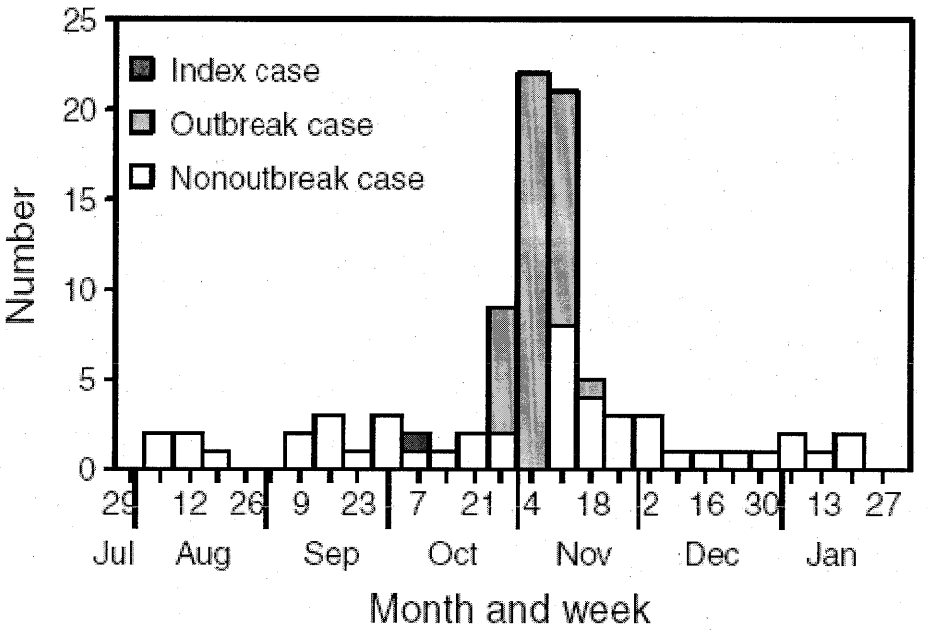
FIGURE. Number of hepatitis A cases, by week of illness onset — County X, Massachusetts, July 29, 2001–January 27, 2002



MMWR49 (50):1131-1133, 2000

食品媒介によるA群ロタウイルス胃腸炎の集団発生 米国コロンビア特別区，2000年3月～4月

2000年3月31日、コロンビア特別区（DC）のある大学内で、3月29日から急性胃腸炎に罹患した学生の増加が学生ヘルスサービス部門からDCの衛生局に届けられた。

発症した一部の学生は、キャンパス内食堂Aでツナあるいはチキンサラダのサンドイッチを食べていることを報告し、3月31日にDC衛生局はこの集団発生に対する調査を開始した。本報告はこの調査結果を要約したものであり、本集団発生は食品によって媒介されたA群ロタウイルスが原因であることが示された。

罹患を報告した学生、聞き込み調査中に罹患が同定された学生及び無作為に選出した学生（健常対照群）に対して電話による聞き込み調査を実施した。

この集団発生は3月27日に始まり、そのピークは3月31日であった。計108名の学生（内55名は電話調査、53名は自己申告）が3月26日から4月11日までの間に胃腸炎症状を呈し、そのうち85名（79%）は規定された症状に合致した。キャンパス内に居住している学生の発症率は5%（77名/1,641名）、キャンパス以外の学生の発症率は0.02%であった。

患者83名には、下痢77名（93%）、腹痛及び不快感75名（90%）、食欲不振69名（83%）、嘔気67名（81%）、倦怠感64名（77%）、嘔吐56名（67%）、頭痛49名（59%）、悪感48名（58%）、微熱48名（58%）、筋肉痛42名（51%）等の症状が現れ、4月2日以降に発症した患者6名には、咽頭炎、咳、喉の鬱血等の症状がみられた。有病期間（中央値）は4日間（範囲1～8日間）で、患者9名（11%）が脱水症状のため静脈補液を受けた。

症例対照研究では、電話調査が完了した患者44名中40名（91%）及び対照群40名中27名（68%）がキャンパスの食堂Aで3月27日から30日の間に何らかのサンドイッチを喫食している結果であった。一方、同じキャンパスの食堂Bでは、3月27日から30日に対照群の55名中18名（33%）が喫食していたのに対し、患者群では49名中4名（8%）が喫食しているのみであった。食堂従業員の喫食歴は記録されていなかったが、6名が疾病を発症していたことを報告した。

学生及び食堂従業員の糞便検査では、細菌、原虫、ノーウォーク様ウイルスは陰性であったが、A群ロタウイルスが27件中9件（33%）から検出された。また、患者4名及び従業員3名由来のロタウイルスは、RT-PCR法によって同じ抗原型（P[4]、G2）と同定された。ロタウイルス（P[4]、G2）陽性従業員3名中2名は、3月27日及び4月2日にそれぞれ発症したことを報告した調理人であったのに対し、他の従業員 1 名は未発症の給仕人であった。

December 22, 2000 / 49(50); 1131-3

Foodborne Outbreak of Group A Rotavirus Gastroenteritis Among College Students --- District of Columbia, March--April 2000

On March 31, student health services at a university in the District of Columbia (DC) notified the DC health department that an increased number of students had become ill with acute gastroenteritis beginning March 29. Some ill students reported eating tuna or chicken salad sandwiches from dining hall A on campus. On March 31, the DC health department initiated an outbreak investigation. This report summarizes results of the investigation, which indicated that group A rotavirus transmitted by food was the cause of the outbreak.

Telephone interviews were conducted with students who reported illness to student health services, with additional ill students who were identified during interviews, and with healthy controls selected randomly from the university registry of students residing on campus. A case of gastroenteritis was defined as three or more episodes of diarrhea and/or two or more episodes of vomiting within a 24-hour period in a student with onset on or after March 20. Controls and case-patients whose illness onset occurred during March 27--31 were questioned about food history, residence and dining hall, source of water, use of a public access computer or sports equipment at the university gym, and attendance at social or athletic events. Electronic records of student meal attendance were available for 49 case-patients with illness onset during March 27--31 and for 55 control subjects.

Twenty-three (79%) of 29 employees of dining hall A were interviewed to identify their work duties and determine whether they were ill. Stool specimens were collected during March 29--April 10 from six ill students and 21 dining hall A employees. Samples were screened for bacterial and parasitic pathogens at a commercial laboratory and for viral pathogens at CDC.

The outbreak among students began March 27 and peaked at 19 cases on March 31 (Figure 1). A total of 108 students (55 were identified by telephone interviews and 53 were self-reported) had gastrointestinal symptoms during March 26--April 11; 85 (79%) had illness that met the case definition. The attack rate among students residing on campus was 5% (77 of 1641), with no significant differences in attack rates by sex, occupancy of residence hall, or grade level. Eight case-patients resided off campus (attack rate: 0.02%). Among the 83 case-patients for whom a complete list of symptoms was reported, 77 (93%) had diarrhea, 75 (90%) abdominal pain or discomfort, 69 (83%) loss of appetite, 67 (81%) nausea, 64 (77%) fatigue, 56 (67%) vomiting, 49 (59%) headache, 48 (58%) chills, 48 (58%) subjective or low-grade fever, and 42 (51%) myalgia. Sore throat, cough, and/or congestion were reported by six case-patients with onsets on or after April 2. The median duration of illness was 4 days (range: 1--8 days). Nine (11%) case-patients received intravenous fluids to treat dehydration.

Of those who completed the telephone interview, 40 (91%) of 44 case-patients and 27 (68%) of 40 controls ate at least one deli sandwich from campus dining hall A during March 27--30 (p=0.017; odds ratio [OR] =4.8; 95% confidence interval [CI] =1.3--22.1). During March 27--30, four (8%) of 49 case-patients ate four or more meals at dining hall B compared with 18 (33%) of 55 controls (p=0.005; OR=0.2; 95% CI=0.04--0.6). Food histories of employees were not recorded; however, six employees reported illness.

Stool specimens of students and employees were negative for bacterial and parasitic pathogens and for Norwalk-like viruses. Using electron microscopy, enzyme immunoassay, and reverse transcriptase-polymerase chain reaction (RT-PCR), nine (33%) of 27 specimens were positive for group A rotavirus. Rotavirus positive stool specimens from four students and three employees were identified as genotype combination P [4], G2 by RT-PCR. Two of the three P [4], G2-positive employees were line cooks who reported having symptoms of gastroenteritis on March 27 and April 2, respectively, while the third positive employee, a deli server, reported no illness.

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Editorial Note:

Group A rotavirus is the most common cause of childhood diarrhea worldwide, infecting >90% of children by age 3 years (*1*). Because rotavirus immunity develops early in life, disease among older children and adults is uncommon (*1*). Although the role of rotavirus in diarrhea outbreaks in adults has not been well studied, it has been documented as the cause of adult diarrheal outbreaks in hospitals (*2*), nursing homes (*3*), isolated communities (*4*), and in travelers (*5*). Also, parents of children infected with rotavirus have been reported to experience acute gastroenteritis (*6*). However, the rotavirus G and P protein–type combinations, the proteins that elicit an immune response in humans, were not characterized in most of these reports.

The rapid increase and gradual decline of the campus outbreak suggest that the infection was foodborne during the first week and was spread person–to–person during the following week. During the first week, illness was associated with eating sandwiches at dining hall A and was associated inversely with eating frequently at dining hall B. The employee who prepared sandwich fillings did not report illness and tested negative for rotavirus. None of the three deli servers who assembled and served sandwiches reported illness; however, one was rotavirus P [4], G2 positive. It is unknown whether the deli server who tested positive was infected before the outbreak among students.

This rotavirus serotype G2 outbreak was unusual for two reasons; food was implicated as the source of infection and the adults affected should have been immune. During April 2000, a gastroenteritis outbreak among adults in Japan also was caused by foodborne transmission of group A rotavirus serotype G2 (*7*). These adults should not have been susceptible to severe rotavirus illness. G2 strains often are found combined with serotype P [4]1B (*8*). The G and P neutralization antigens of serotype G2 strains may allow G2 strains to escape immunity induced by the more common G1, G3, and G4 strains. In addition, G2 has been associated with more severe dehydration during diarrheal episodes in children than other common strains (*9*). These outbreaks of rotavirus gastroenteritis in adults in the United States and Japan raise questions about the persistence of immunity to rotavirus and the virulence of G2 strains. Investigators and clinicians should consider rotavirus as a possible cause of acute gastroenteritis in adults.

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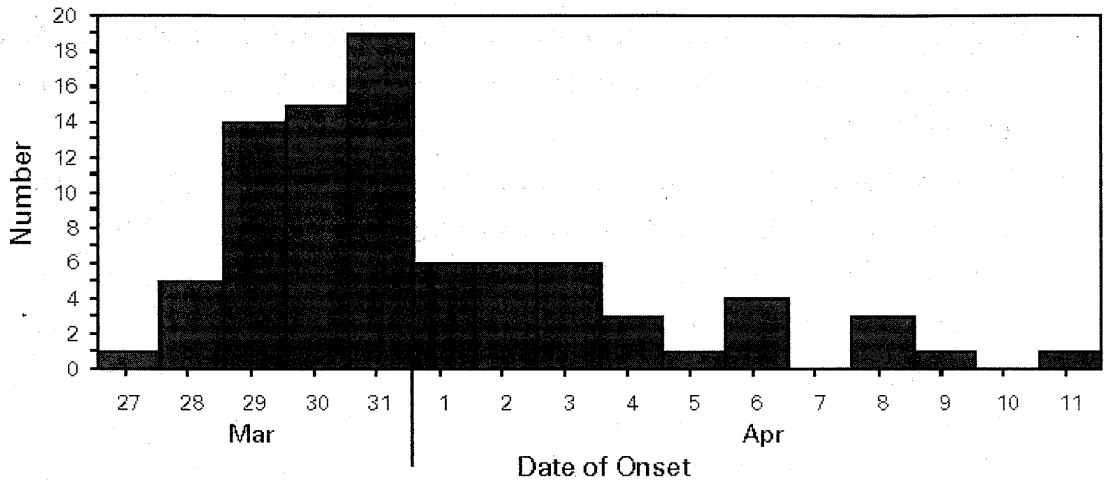
FIGURE 1. Number* of gastroenteritis† cases among college students, by date of illness onset — District of Columbia, March 27–April 11, 2000



* n=85.

† A case of gastroenteritis was defined having three or more episodes of diarrhea and/or two or more episodes of vomiting within a 24-hour period in a student with onset on or after March 20.

MMWR48 (11):228-229, 1999

狂犬病罹患牛に由来する未殺菌乳摂取者に対する予防措置 マサチューセッツ州，1996～1998 年

狂犬病は、罹患動物の咬傷によって媒介されるウイルス性の人畜共通感染症である。マサチューセッツ州では、1996 年以降未殺菌牛乳の摂取による狂犬病曝露の可能性のある事件が 2 件報告された。本報告では両事件の調査結果につき紹介する。

事例 1：1998 年 11 月 12 日、マサチューセッツ州衛生局のウイルス研究所によりウースター郡の 1 牧場で飼育されていた 6 才のホルスタイン種乳牛が狂犬病と診断された。この罹患牛の脳組織について実施されたモノクローナル抗体による診断で、原因ウイルスは米国東部のアライグマ関連ウイルスの変種であることが確認された。この牛は 11 月初めから食欲不振が、11 月 6 日から唾液の亢進が認められるようになり、当初腸閉塞が疑われた。その後、運動失調、興奮、攻撃性がみられ、11 月 8 日に死亡した。この牛は死亡するまで週 12 回搾乳され、他の牛乳と混合された未殺菌乳の一部が消費されていた。衛生当局の調査では、10 月 23 日から 11 月 8 日の間に 66 名が本未殺菌乳を摂取したことが確認され、狂犬病の予防措置を受けた。また、罹患牛の唾液と接触のあった 5 名も同様の予防措置を受けた。ウイルス検出のための罹患牛の乳並びに乳房組織は得られなかった。

事例 2：1996 年 11 月 12 日、マサチューセッツ州衛生局のウイルス研究所によりウースター郡で起きた事例 1 とは異なる牧場で飼育されていた 14 才のジャージー種の乳牛が狂犬病と診断された。この罹患牛もモノクローナル抗体による解析により、米国東部のアライグマ関連ウイルスの変種に感染していることが確認された。本牛は、11 月 6 日にテネスムス（しぶり）、抑鬱状態となり、11 月 10 日に安楽死処置がとられた。本牛は 10 月 26～11 月 2 日まで搾乳され、その間の未殺菌乳摂取者は計 14 名であった。これらの 14 名、また罹患牛の唾液に接触のあった 4 名が狂犬病の予防措置を受けた。

狂犬病ウイルスが未殺菌乳を介して伝播することは理論的にはあり得るが、そのリスクを明確にするには罹患動物から乳並びに乳房組織を採取し、狂犬病ウイルスの存在、生存性、感染性を調べる必要がある。仮に牛乳中に生きたウイルスがその多寡にかかわりなく流入していても、本経路を介した伝播リスクは乳製品の殺菌処理により回避できる。

Editorial Note《編集注記》

国立公衆衛生獣医協会（The National Association of State Public Health Veterinarians）は、凶暴な動物の細胞組織や乳の摂取に反対している。

March 26, 1999 / 48(11); 228-229

Mass Treatment of Humans Who Drank Unpasteurized Milk from Rabid Cows -- Massachusetts, 1996-1998

Rabies is a viral zoonosis that is usually transmitted by the bite of an infected mammal. However, in Massachusetts, two incidents have been reported since 1996 of potential mass exposures to rabies through drinking unpasteurized milk. This report presents the investigations of these two incidents.

Incident 1

On November 12, 1998, the Virology Laboratory of the Massachusetts Department of Public Health (VLMDPH) diagnosed rabies in a 6-year-old Holstein dairy cow from a farm in Worcester County. Further analysis of the cow's brain tissue with monoclonal antibodies revealed the cow was infected with a variant of the rabies virus associated with raccoons in the eastern United States.

The cow had loss of appetite beginning November 4 and hypersalivation beginning November 6. An intestinal obstruction was suspected initially as the cause of illness. However, the cow became ataxic and aggressive and died on November 8.

The cow had been milked 12 times during the week before death. Milk from the cow had been pooled with milk collected from other cows, and an unpasteurized portion was distributed for human consumption. Public health investigations identified 66 persons who drank unpasteurized milk collected from this dairy during October 23-November 8. All 66 received rabies postexposure prophylaxis (PEP). In addition, five persons received PEP because of exposure to the cow's saliva during the 15 days preceding her death. Neither milk nor mammary tissue from the rabid cow was available for examination for the presence of rabies virus.

Incident 2

On November 12, 1996, the VLMDPH diagnosed rabies in a 14-year-old Jersey dairy

cow from a different farm in Worcester County. Analysis with monoclonal antibodies revealed the cow was infected with a variant of the rabies virus associated with raccoons in the eastern United States.

The cow developed tenesmus and depression on November 6 and was euthanized on November 10. The cow had been milked during October 26–November 2. An investigation identified 14 persons who drank unpasteurized milk collected from this cow during this period. All 14 persons received rabies PEP. In addition, four persons received PEP because of exposure to the rabid cow's saliva during the 15 days preceding her death.

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Editorial Note

Management of mass human exposures to rabid animals requires public health officials to balance knowledge of rabies epidemiology, risk for transmission, and pathogenesis with the perceived risk for death among exposed persons. Because of the nearly 100% case–fatality ratio of human rabies and the virtually complete effectiveness of PEP, many mass exposure incidents prompt administration of rabies immune globulin and vaccine, even if the circumstances do not meet the criteria for exposure (1–3).

During 1990–1996, CDC received reports of 22 incidents of mass human exposures to rabid or presumed–rabid animals in the United States, resulting in 1908 persons receiving PEP (median: 33 persons per incident) (4). In Massachusetts during 1991–1995, the median cost for PEP was $2376 per person, including physician and facility charges (5). Prolific administration of PEP in response to these incidents strains the availability of rabies biologics, especially human rabies immune globulin, which has a short shelf–life and tightly controlled distribution by the manufacturers.

An average of 150 rabid cattle have been reported to CDC in the United States each year since 1990 (6). In addition to concerns about rabies transmission from animals to humans through bites, rabid livestock raise the potential for foodborne transmission. The National Association of State Public Health Veterinarians recommends against

consuming tissues and milk from rabid animals (2). However, because rabies virus is inactivated by temperatures below those used for cooking and pasteurization, eating cooked meat or drinking pasteurized milk from a rabid animal is not an indication for PEP.

Rabies virus can be transmitted by direct contact with infected material, such as saliva from an animal infected with rabies, and mucous membranes, including the oral and gastric mucosae (7). In addition to saliva and neural tissue, rabies virus also has been detected in the kidney, prostate, pancreas, and other tissues and body fluids (8). However, saliva and neural tissue are the primary proven vehicles for rabies virus in naturally occurring cases. Anecdotal reports exist of rabies transmission by ingestion of milk from rabid animals (e.g., from a rabid sheep to a nursing lamb) (7). In these reports, the more conventional routes (e.g., bite or mucous membrane exposure) could not be completely excluded.

Transmission of rabies virus in unpasteurized milk is theoretically possible. The risk could be defined better if samples of milk and mammary tissue were collected from rabid livestock and assayed for the presence, viability, and infectivity of rabies virus. Regardless of the amount of viable rabies virus that may be shed in cows' milk, the theoretical risk for transmission of rabies from this route can be eliminated if all dairy products are pasteurized before consumption.

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MMWR52 (7):125- 127

ワイルドゲーム（狩猟）フェスタ参加者における致死的退行性神経疾患 ウィスコンシン州，2002 年

1993～1999 年、ウィスコンシン州北部において、男性 3 名が狩猟で捕獲した動物の肉を食べた結果、退行性神経疾患により死亡した。

症例 1：1992 年 12 月、66 歳の男性がてんかん、健忘症、手の振戦などの症状を呈し、翌年 2 月、錯乱、運動失調、動作時振戦により入院した。MRI（磁気共鳴映像法）では下位傍矢状後頭葉病変、EEG（脳波検査）では進行性脳症を認め、クロイツフェルトーヤコブ病（CJD）と診断され、数ヶ月後に死亡した。神経病理学的所見により CJD に一致する亜急性海綿状脳症を認めた。男性は 1976 年からウィスコンシン州北部やモンタナ州で狩猟し、所有する山小屋で頻繁に獲物のシカ肉を食べていた。国立プリオン病原体監視センター（National Prion Disease Pathology Surveillance Center : NPDPSC）による検査では、プリオン病を示唆する所見は得られなかった。

症例 2：1999 年 5 月、ミネソタ州在住の 55 歳の男性が筆記障害、歩行不安定を呈し、6 月には痴呆、言語障害、ミオクローヌスが認められたため入院した。EEG 所見から CJD と診断され、7 月に症状が悪化、右上肢ジストニアを来し死亡、神経病理学的所見も CJD と一致した。男性は、狩猟はしなかったが頻繁にシカ肉を食べていた。ワイルドゲームフェスタへも 12 回参加したが、1980 年代以降は 1 回のみ参加であった。国立プリオン病原体監視センターによる免疫組織染色及びウェスタンブロット法によりプリオン病が認められた。

症例 3：1992 年 6 月、ウィスコンシン州在住の 65 歳の男性が言語障害、記憶障害、人格障害を呈し、1993 年 1 月にピック病と診断された。5 月には日常生活が不能となり 1993 年 8 月に死亡した。神経病理学的所見では、対側性前頭葉大脳皮質萎縮及び側頭葉軽度萎縮を認めた。国立プリオン病原体監視センターによる検査では CFD を呈する病変は認められなかった。この男性は、症例 1 によるワイルドゲームフェスタでシカ肉を食べ、ウィスコンシン州の他にワイオミング州、ブリティッシュコロンビア州（カナダ）でも狩猟していた。ワイルドゲームフェスタに参加した可能性のある本人及び家族からの聞き取り調査

では、情報が得られた 45 例中 34 例がワイルドゲームフェスタに参加、うち 7 例が死亡（上記 3 例を含む）した。慢性消耗性疾患（CWD）である感染性海綿状脳症（TSE）に感染した野生のシカ肉が感染源と考えられる。なお、症例 2 は因果関係が明らかでなかった。

狩猟者と野生の鹿肉を食べる消費者は、ウィスコンシン州農業、貿易、消費者保護局（Department of Agriculture, Trade, and Consumer Protection）が作成した CWD 曝露予防に関する諸ガイドラインに従うべきである。

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Fatal Degenerative Neurologic Illnesses in Men Who Participated in Wild Game Feasts --- Wisconsin, 2002

Creutzfeldt-Jakob disease (CJD) is a fatal neurologic disorder in humans. CJD is one of a group of conditions known as transmissible spongiform encephalopathies (TSEs), or prion diseases, that are believed to be caused by abnormally configured, host-encoded prion proteins that accumulate in the central nervous tissue (*1*). CJD has an annual incidence of approximately 1 case per million population in the United States (*1*) and occurs in three forms: sporadic, genetically determined, and acquired by infection. In the latter form, the incubation period is measured typically in years. Recent evidence that prion infection can cross the species barrier between humans and cattle has raised increasing public health concerns about the possible transmission to humans of a TSE among deer and elk known as chronic wasting disease (CWD) (*2*). During 1993--1999, three men who participated in wild game feasts in northern Wisconsin died of degenerative neurologic illnesses. This report documents the investigation of these deaths, which was initiated in August 2002 and which confirmed the death of only one person from CJD. Although no association between CWD and CJD was found, continued surveillance of both diseases remains important to assess the possible risk for CWD transmission to humans.

Case Reports

Case 1. In December 1992, a Wisconsin man aged 66 years with a history of seizures since 1969 sought treatment for recurring seizures, increasing forgetfulness, and worsening hand tremors. Electroencephalographic (EEG) examination demonstrated focal epileptiform activity and nonspecific diffuse abnormalities, but no specific diagnosis was made. In February 1993, he was hospitalized for increasing confusion, ataxia, and movement tremors of his extremities. A magnetic resonance image (MRI) demonstrated mild, nonspecific enhancement along the inferior parasagittal occipital lobe. A repeat EEG showed bifrontal intermittent, short-interval, periodic sharp waves, suggesting a progressive encephalopathy; a diagnosis of CJD was suspected. The man

died later that month; neuropathologic examination of brain tissue during autopsy indicated subacute spongiform encephalopathy, compatible with CJD.

The man was a lifelong hunter who ate venison frequently. He hunted primarily in northern Wisconsin but also at least once in Montana. He hosted wild game feasts at his cabin in northern Wisconsin from 1976 until shortly before his death. Fixed brain tissue obtained during the autopsy was sent for analysis to the National Prion Disease Pathology Surveillance Center (NPDPSC) and reexamined at the institution where the autopsy was conducted. Histopathologic examination did not substantiate the diagnosis of prion disease. In addition, 27 brain tissue sections were negative for prions by immunostaining despite positive antibody reactions against other proteins (controls), which indicated that other epitopes in the tissue samples were preserved.

Case 2. In May 1999, a Minnesota man aged 55 years with no previous history of a neurologic disease sought evaluation and treatment following a 3-month history of progressive difficulty in writing and unsteadiness of gait. A computerized tomography (CT) scan and MRI examination of his head did not indicate any abnormality. In June 1999, he was hospitalized following onset of dementia, speech abnormalities, and myoclonic jerking. An EEG indicated left-hemispheric periodic sharp waves and moderate generalized background slowing; CJD was diagnosed clinically. In July 1999, following worsening symptoms and development of right upper extremity dystonia, the patient died. Neuropathologic evaluation of brain tissue during autopsy demonstrated widespread subcortical spongiform lesions, consistent with CJD.

The man was not a hunter but had a history of eating venison. He made an estimated 12 visits to the cabin where the wild game feasts were held, but he participated in only one feast during the mid-1980s. Sections of fixed and frozen brain tissue obtained during autopsy were analyzed at NPDPSC, and prion disease was confirmed by immunohistochemical and Western blot testing. The Western blot characteristics and prion disease phenotype in this patient were consistent with the most common form of sporadic CJD, classified as M/M (M/V) 1 (*3*). Subsequent genetic typing confirmed the presence of methionine homozygosity (M/M) at codon 129 of the patient's prion protein gene.

Case 3. In June 1992, a Wisconsin man aged 65 years sought treatment for progressive slowing of speech, worsening memory, and personality changes. By January 1993, his speech was reduced to one-word utterances. Neurologic

examination showed a flat affect, decreased reflexes, and apraxia. A CT head scan showed mild atrophy, and an EEG was normal. Pick's disease was diagnosed. By May, he was unable to perform any daily living activities; he died in August 1993. Neuropathologic evaluation of brain tissue during autopsy showed symmetrical frontal lobe cerebral cortical atrophy and mild temporal lobe atrophy. No Pick's bodies or spongiform lesions were observed.

The man had a history of eating venison and participated regularly in wild game feasts held at the cabin owned by patient 1. He was a lifelong hunter and hunted mostly in Wisconsin but also in Wyoming and British Columbia. No game was brought to the wild game feasts from his hunting trips outside of Wisconsin. Examination of fixed brain tissue sent to NPDPSC demonstrated no lesions indicative of CJD, and immunohistochemical testing with antibody to the prion protein did not demonstrate the granular deposits seen in prion diseases.

Epidemiologic Investigation

Wild game feasts consisting of elk, deer, antelope, and other game that occurred at a cabin in northern Wisconsin owned by patient 1 began in 1976 and continued through 2002. These feasts typically involved 10--15 participants and usually occurred on weekends before or during hunting seasons in the fall and occasionally in the spring. Wild game brought to these feasts usually were harvested in Wisconsin, but three men who attended these feasts reported hunting in the western United States and bringing game back to Wisconsin. These activities took place in Colorado (near the towns of Cortez, Trinidad, Collbran, Durango, and Meeker), Wyoming (near the towns of Gilette and Cody), and Montana (near the town of Malta). CWD was not known to be endemic in these areas at the time that these hunting activities took place.

Information was obtained for 45 (85%) of 53 persons who were identified as possibly participating in the wild game feasts; all were male. Information was obtained by direct interview or from family members of decedents. Of the 45 persons, for whom information was obtained, 34 were reported to have attended wild game feasts. Seven of the 34 feast attendees were deceased, including the three patients. None of the four other decedents had a cause of death attributed to or associated with a degenerative neurologic disorder. None of the living participants had any signs or symptoms consistent with a degenerative neurologic disorder.

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Editorial Note:

CWD was first described in the United States in the 1960s and classified as a TSE in 1978. Previously localized to a contiguous endemic area in northeastern Colorado and southeast Wyoming, since 2000, CWD has been found in free-ranging deer or elk in Illinois, Nebraska, New Mexico, South Dakota, Wisconsin, and outside the previously known endemic areas of Colorado and Wyoming. CWD has been identified also in captive deer or elk in Colorado, Kansas, Minnesota, Montana, Nebraska, Oklahoma, South Dakota, and Wisconsin (*4*). Because a variant form of CJD, with specific neuropathologic and molecular characteristics that distinguish it from sporadic CJD, has been associated with eating cattle products infected with a prion that causes bovine spongiform encephalopathy (*5*), concern has been raised about the possibility that the prion associated with CWD might be transmitted to humans in a similar way.

In this investigation, because only one of the three cases in Wisconsin had neuropathologic confirmation of a prion disease, no association could be made between case participation in the wild game feasts and the development of CJD. Although patient 2 had confirmed CJD, he was unlikely to have eaten CWD-infected venison at these feasts because venison and other game from outside Wisconsin that was served at these feasts did not originate from known CWD-endemic areas, and the man participated in the feasts only once. In addition, the prion disease in this case was consistent with the most common form of sporadic CJD, without apparent unusual neuropathologic or molecular characteristics that might occur if the prion related to CWD had been responsible for the disease.

The findings in this report are subject to at least two limitations. First, not all members participating in wild game feasts could be identified, and not all persons listed as participating could be contacted for interviews. Second, interviews that were conducted required recall of events that occurred up to 25 years ago, limiting the

detail or accuracy of events. However, the similar responses obtained from different sources support the accuracy of the investigation findings.

A previous investigation of unusually young CJD patients in whom the transmission of CWD was suspected also did not provide convincing evidence for a causal relationship between CWD and CJD (*2*). However, limited epidemiologic investigations cannot rule out the possibility that CWD might play a role in causing human illness. Ongoing surveillance of CJD, particularly in states with CWD, is important to assess the risk, if any, for CWD transmission to humans. Because the confirmation of CJD and the detection of a new prion disease require neuropathologic study of brain tissue, physicians are encouraged to contact NPDPSC (http://www.cjdsurveillance.com/; telephone, 216-368-0587) to confirm diagnoses of CJD and to distinguish its various subtypes. Because of the known severity of TSEs in humans and the possibility that the CWD prion can affect humans, animals with evidence of CWD should be excluded from the human food or animal feed chains. Hunters and wild venison consumers should follow precautionary guidelines available from the Wisconsin Department of Agriculture, Trade, and Consumer Protection (http://datcp.state.wi.us/core/consumerinfo) to prevent potential exposures to the CWD agent.

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ブリート（Burritos）摂食による病因不明の胃腸疾患の発生 米国，1997年10月～1998年10月

1997年10月～1998年10月にかけて、フロリダ州、イリノイ州、インディアナ州、カンザス州、ノースダコタ州、ペンシルヴェニア州において、ブリート摂食による16件の胃腸疾患が流行した。15件が学校で生じており、被害を受けた約1,700人の多くは子供であった。本報告では、米国疾病管理予防センター（CDC）、米国農務省（USDA）、米国食品医薬品局（FDA）が病因を同定するために行った2件の調査を示す。

ジョージア州：1998年3月23日、Hall郡の保健部は、小学生が昼食後に具合が悪くなったという報告を受け、584人中452人の生徒から食事の状況を聴取し、学校での昼食後24時間以内に生じた吐気、腹痛、嘔吐、下痢を症例とした。155人（34%）が症例に適合し、症状は吐気（89%）、頭痛（65%）、腹痛（53%）、嘔吐（29%）、下痢（17%）であった。潜伏期間の中央値は約15分（範囲5～25分）、継続時間の中央値は4.5時間（範囲10分～8時間）であった。ブリートを食べた304人のうち145人（48%）に、食べなかった148人のうち10人（7%）に症状が出た（相対危険度 7.1、95%信頼限界 3.8 - 13.0）。ブリートはA社で製造され、主な成分は牛肉、鶏肉、ぶちインゲン豆、調味料、植物蛋白、トーチラス（メキシコのパンの一種）であった。

フロリダ州：1998年10月8日、Hillsborough郡の保健部は12の小学校で昼食後に生じた疾患の報告を受け、2つの小学校を調査し、10月8日の学校での昼食後、発生した吐気、腹痛、嘔吐を症例とした。27症例が同定され、一方の小学校の14人の症状は、腹痛（88%）、嘔吐（62%）、頭痛（62%）、下痢（39%）であり、もう一つの小学校の13人の症状は、腹痛（82%）、嘔吐（55%）、頭痛（27%）、下痢（18%）、めまい（18%）であった。症例対照研究では、前者の小学校では14人の症例患者中8人、他方では13人中11人がブリートを食べた。ブリートの材料となるトーチラスはB社から供給され、中身の牛肉とぶちインゲン豆は学校の調理室で調理された。

1997年10月～1998年10月の3件の胃腸疾患流行に関係するブリートはA社から供給され、1998年5月～10月の13件の流行に関係するブリートはB社から供給されていた。

主な症状は、吐気、頭痛、腹部の痙攣、嘔吐で、ブリート摂食後 60 分以内に発症し、24 時間未満まで症状が続いたが入院患者は出なかった。 USDA は A 社、B 社にブリートの回収を通告し、約 200 万ポンドが回収された。

Editorial Note《編集注記》

食品事故の調査と追跡調査活動の連携を強化するため、地方衛生局は食品事故と判定されたものについて、直ちに州衛生局を通じて CDC に報告することを推奨されている。CDC は、嘔吐物、血清、糞便及び尿の検体を少なくとも 10 患者から入手し、可能であれば各食品事故の原因である食事の残り物のサンプル、出荷用容器を確保しておくよう提唱している。

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Outbreaks of Gastrointestinal Illness of Unknown Etiology Associated with Eating Burritos -- United States, October 1997-October 1998

From October 1997 through October 1998, 16 outbreaks of gastrointestinal illness associated with eating burritos occurred in Florida, Georgia, Illinois, Indiana, Kansas, North Dakota, and Pennsylvania. All but one outbreak occurred in schools, and most of the approximately 1700 persons affected were children. This report summarizes investigations of two of these outbreaks and describes the collaborative efforts of CDC, the U.S. Department of Agriculture (USDA), and the Food and Drug Administration (FDA) to identify the etiologic agent(s); these outbreaks may have been caused by an undetected toxin or a new agent not previously associated with illness.

Georgia

On March 23, 1998, the Hall County Health Department received a report that students in an elementary school became ill after eating lunch. Health officials obtained food and illness histories from 452 (77%) of the 584 students. A case was defined as nausea, abdominal cramps, vomiting, or diarrhea within 24 hours in a person after eating the school lunch on March 23. Of the 452 students, 155 (34%) had illnesses meeting the case definition. Symptoms most commonly reported were nausea (89%), headache (65%), abdominal cramps (53%), vomiting (29%), and diarrhea (17%). The median incubation period was approximately 15 minutes (range: 5-25 minutes), and median duration of illness was 4.5 hours (range: 10 minutes-8 hours).

The children had access to nine foods during lunch. One hundred forty-five (48%) of 304 who ate burritos, and 10 (7%) of 148 who did not eat burritos became ill (relative risk {RR} =7.1; 95% confidence interval {CI} =3.8-13.0). The burritos were produced by

company A; the main ingredients were beef, chicken, pinto beans, seasoning, textured vegetable protein, and tortillas.

Florida

On October 8, 1998, the Hillsborough County Health Department was notified that students at 12 elementary schools became ill after eating lunch. Health officials conducted investigations at two schools. A case was defined as nausea, abdominal cramps, or vomiting in a person after eating the school lunch on October 8. In both schools, students who initially reported illness and classmates in the three classes with the highest number of cases were interviewed. Twenty-seven cases were identified. The predominant symptoms of the 14 ill children identified in one school were abdominal cramps (88%), vomiting (62%), headache (62%), and nausea (39%). In the other school, symptoms among the 13 identified ill children were abdominal cramps (82%), vomiting (55%), headache (27%), nausea (18%), and dizziness (18%).

In a case-control study at one school, eight (57%) of 14 case-patients and five (13%) of 38 well children ate burritos (odds ratio {OR} =8.8; 95% CI=1.8-47.6). In the other school, 11 (85%) of 13 case-patients and 11 (33%) of 33 well children ate burritos (OR=11.0; 95% CI=1.8-87.6). The tortillas used to make the burritos were supplied by company B; the fillings, beef at one school and beef and pinto beans at the other, were made in the two school kitchens.

Summary Findings

During October 1997-March 1998, burritos from three outbreaks of gastrointestinal illness were traced to company A, and during May-October 1998, burritos from another 13 outbreaks were traced to company B. Three outbreaks were linked to chicken and bean burritos, pork-sausage and egg burritos, and beef burritos; the other 13 were linked to beef and pinto bean burritos. All burritos used tortillas made with wheat flour. The burritos were distributed frozen and prepackaged except in Florida, where the filling was prepared locally.

The major symptoms were nausea, headache, abdominal cramps, and vomiting, typically beginning within 60 minutes after eating a burrito and lasting less than 24 hours. No one was hospitalized.

USDA requested that both companies A and B initiate timely national recalls, and approximately 2 million lbs of burritos were recalled or withheld from distribution. Company A and its tortilla supplier were unrelated to company B and its supplier.

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Editorial Note:

Data from the two outbreaks described in this report and the other 14 outbreaks indicate that the symptoms, incubation period, and duration of illness were similar. The variations in symptoms in the outbreaks in Florida and Georgia could be associated with differences in case finding methods. Epidemiologic investigations in several of the other outbreaks also have implicated burritos, which consisted of meat or vegetable filling wrapped in a tortilla. Data from the Florida outbreak suggest that the etiologic agent was in the tortillas because the filling was made locally. Outbreaks associated with products made by two unrelated companies that used different tortilla suppliers suggest that the agent was an ingredient common to the products made by both companies. No common first-line suppliers were identified; however, whether the source of any ingredients was shared has not been determined.

The short incubation periods suggest that a preformed toxin or other short-acting agent was the cause of illness. Possible agents include bacterial toxins (e.g., *Staphylococcus aureus* enterotoxin and *Bacillus cereus* emetic toxin); mycotoxins

(e.g., deoxynivalenol [DON], acetyl-deoxynivalenol, and other tricothecenes), trace metals, nonmetal ions (e.g., fluorine, bromine, and iodine), plant toxins (e.g., alkaloids such as solanines, opiates, ipecac, and ergot; lectins such as phytohemagglutinin; and glycosides), pesticides (e.g., pyrethrins, organophosphates, and chlorinated hydrocarbons), food additives (e.g., bromate, glutamate, nitrite, salicylate, sorbate, and sulfite), detergents (e.g., anionic detergents and quaternary amines), fat-soluble vitamins, spoilage factors (e.g., biogenic amines, putrefaction, and free fatty acids), or an unknown toxin. Mass sociogenic illness is an unlikely explanation based on the number of different sites where outbreaks have been reported over a short interval and the link to only two companies.

B. cereus emetic toxin and *S. aureus* enterotoxin are common causes of food poisoning, but headache is not usually a prominent feature, and most outbreaks traced to these toxins have incubation periods of 2–4 hours, which is longer than observed in these outbreaks (1,2). Food samples from five outbreaks were negative for *B. cereus* and *S. aureus* by culture and toxin analysis; testing from these same outbreaks for alkaloids, biogenic amines, and pesticides also did not identify the causative agent.

Some metals, such as cadmium, copper, tin, and zinc, can irritate mucosal membranes and cause gastrointestinal illness after short incubation periods; however, only elemental aluminum was mildly elevated in the burrito samples, and there is no evidence that it causes these symptoms (3, 4). Several plant toxins, such as phytohemagglutinin, may survive cooking and cause gastrointestinal symptoms; however, previous outbreaks associated with phytohemagglutinin have been linked to red kidney beans and not pinto beans (5).

Outbreaks with symptoms and incubation periods similar to those described in this report have occurred in China and India, where illness has been linked to consumption of products made with grains contaminated with fungi. These fungi produce heat-stable tricothecene mycotoxins called vomitoxin (6). In China, 35 outbreaks affecting 7818 persons during 1961–1985 were attributed to consumption of foods made with moldy grain (7). Corn and wheat samples collected during two outbreaks had higher levels of DON than those collected at other times. In India in 1987, 97 persons consumed wheat products following heavy rains (8). DON and other tricothecene mycotoxins were detected in the implicated wheat products, and extracted toxins caused vomiting in laboratory tests on puppies (8). High doses of DON are known to cause vomiting in pigs (9). Laboratory testing from burrito samples

from some of the U.S. outbreaks in this report detected DON within the acceptable FDA advisory level of 1 ppm for finished wheat products (10). However, the possibility remains that a mycotoxin is the cause.

To facilitate coordination of outbreak investigation and traceback activities, local health departments are encouraged to report immediately any outbreaks characterized by an incubation period of less than 1 hour, duration of less than 1 day, and symptoms including nausea, headache, abdominal cramps, and vomiting regardless of the suspected vehicle through state health departments to CDC. CDC recommends that vomitus, serum, stool, and urine specimens be obtained from at least 10 ill persons, if possible, in each outbreak and that any leftover food samples and shipping containers be saved.

In addition to testing food specimens for specific toxins and agents, laboratories at USDA, FDA, and CDC are examining these specimens by cell culture assays, biologic toxicity assays, and chemical analyses for toxins. The interagency investigating team seeks to collaborate with groups capable of analyzing suspect burritos and tortillas to identify the etiologic agent. Additional information is available from CDC's Foodborne and Diarrheal Diseases Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, telephone (404) 639-2206.

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米国食品事故対処事例一覧表

危害要因	病 因	発行日	MMWR		標題	病因詳細	発生場所	発生年月	ページ数(ページ番号)			
			Vol	No					邦文	英文	総計	該当番号
毒性化学物質	化学物質	2002.8.9	51	31	タマリンドキャンディと民間療法薬による小児鉛中毒	鉛	カリフォルニア州	1999～2000	2	4	6	1-6
		1998.12.11	47	48	輸入キャンディ及び食品着色料に関連して発生した鉛中毒事例	鉛	カリフォルニア州、ミシガン州	1993, 1997	2	4	6	7-12
	自然毒	2003.8.22	52	33	ヨルガオ中毒の疑い	Datura inoxia(チョウセンアサガオ)中毒	オハイオ州	2002	2	4	6	13-18
		2002.11.29	51	47	カワカワ含有製品による肝毒性の可能性	カワカワ(kava kavaまたはPiper methysticum)	米国、ドイツ、スイス	1999～2002	2	5	7	19-25
		2002.5.17	51	19	最新情報:フロリダフグによる神経疾患	サキシトキシン	フロリダ州	2002	2	5	7	26-32
		2002.4.19	51	15	フロリダフグによる神経障害	サキシトキシン及びその類似物質(N-sulfocarbamoylsaxitoxin、decarbamoylsaxitoxin)	フロリダ州、バージニア州、ニュージャージー州	2002	2	6	8	33-40
		2000.5.12	49	18	サバ亜目魚によるヒスタミン中毒	ヒスタミン(スコンブロイドフィッシュ中毒)	ペンシルベニア州	1998	2	4	6	41-46
		1998.8.28	47	33	魚のシガテラ中毒	シガトキシン(シガテラ中毒)	テキサス州	1997	2	4	6	47-52
		1998.12.25	47	50	バッファローフィッシュの喫食により発生したハフ病	ハフ病(未確定の魚毒)	米国(カリフォルニア州、ミズーリ州)	1997	1	5	6	53-58
	その他の毒性化学物質	2003.5.9	52	18	汚染された牛挽肉によるニコチン中毒	ニコチン(殺虫剤)	ミシガン州	2003	2	5	7	59-65
		1999.2.26	48	7	γ-ブチロラクトンの摂取による不慮の事故	γ-ブチロラクトン	ミネソタ州、ニューメキシコ州、テキサス州	1998～1999	1	6	7	66-72
病原性微生物・寄生虫	サルモネラ	2003.7.4	52	26	複数の州における非低温殺菌乳の摂取によるサルモネラ(ネズミチフス菌:*Salmonella* Typhimurium)感染症の発生	サルモネラ(ネズミチフス菌:*Salmonella* Typhimurium)	イリノイ州、インディアナ州、オハイオ州、テネシー州	2002～2003	2	6	8	73-80
		2003.1.3	51	51	殻付卵によるサルモネラ(腸炎菌:*S.* Enteritidis)感染症の発生	サルモネラ(腸炎菌:*Salmonella* Enteritidis)	米国	1999～2001	2	8	10	81-90
		2002.11.22	51	46	複数の州におけるメキシコ産カンタループメロンの摂取によるサルモネラ(*S.* Poona)感染症の発生	サルモネラ(*Salmonella* Poona)	米国(カリフォルニア州、ワシントン州他、全12州)、カナダ	2000～2002	2	6	8	91-98
		2002.8.9	51	31	サルモネラ(*S.* Javiana)感染症の発生	サルモネラ(*Salmonella* Javiana)	フロリダ州オーランド	2002.6	2	5	7	99-105
		2002.6.28	51	25	多剤耐性サルモネラ(*S.* Newport)の発生	サルモネラ(*Salmonella* Newport)	米国(ニューヨーク州、ミシガン州、ペンシルベニア州、オハイオ州、コネチカット州)	2002.1～4	2	6	8	106-113
		2002.1.11	51	1	アルファルファ摂取によるサルモネラ(*S.* Kottbus)感染症の発生	サルモネラ(*Salmonella* Kottbus)	アリゾナ州、カリフォルニア州、コロラド州、ニューメキシコ州	2001.2～4	1	4	5	114-118
		2000.2.4	49	4	摂食方法又は加熱不十分な殻付卵によるサルモネラ(腸炎菌:*S.* Enteritidis)感染症の発生	サルモネラ(腸炎菌:*Salmonella* Enteritidis)	米国	1996～1998	2	9	11	119-129
		1999.7.16	48	27	未殺菌オレンジジュースによるサルモネラ(*S.* Muenchen)感染症の発生	サルモネラ(*Salmonella* Muenchen)	米国(ワシントン州、オレゴン州他、全13州)、カナダ	1999.6	2	6	8	130-137
		1998.6.12	47	22	複数の州における焼きオートムギシリアルを原因とするサルモネラ(*S.* Agona)感染症の発生	サルモネラ(*Salmonella* Agona)	米国(全11州)	1998.4～5	2	4	6	138-143
		1998.5.22	47	19	汚染した上水によるプレシオモナス・シゲロイデスとサルモネラ(*S.*Hartford)感染症	プレシオモナス・シゲロイデス(*Plesiomonas shigelloides*)、サルモネラ(*Salmonella* Hartford)	ニューヨーク州リビングストン郡	1996	2	4	6	144-149
	リステリア菌	2001.7.6	50	26	メキシコ風自家製チーズによるリステリア症の発生	リステリア菌(*Listeria monocytogenes*)	ノースカロライナ州	2000.10～2001.1	2	5	7	150-156
		2000.12.22	49	50	リステリア症の集団発生	リステリア菌(*Listeria monocytogenes*)	米国(全10州)	2000	1	3	4	157-160
		1999.1.8	47	51,52	複数の州におけるリステリア症の発生	リステリア菌(*Listeria monocytogenes*)	米国(全11州)	1998～1999	1	2	3	161-163
		1998.12.25	47	50	リステリア症の広域集団発生	リステリア菌(*Listeria monocytogenes*)	米国(全10州)	1998	1	2	3	164-166

危害要因	病 因	発行日	MMWR		標題	病因詳細	発生場所	発生年月	ページ数(ページ番号)			
			Vol	No					邦文	英文	総計	該当番号
病原性微生物・寄生虫	病原大腸菌	2002.7.26	51	29	複数の州における牛挽肉の摂取による病原大腸菌O157:H7感染症の発生	病原大腸菌(*Escherichia coli*)O157:H7	米国(コロラド州他、全7州)	2002.6～7	2	5	7	167-173
		2000.10.13	49	40	フレッシュ・チーズカードによる病原大腸菌O157:H7感染症の発生	病原大腸菌(*Escherichia coli*)O157:H7	ウィスコンシン州	1998.6	2	4	6	174-179
		2000.4.21	49	15	ティーンエージャーの合宿における病原大腸菌O111:H8の発生	病原大腸菌(*Escherichia coli*)O111:H8	テキサス州	1999	1	5	6	180-185
	ボツリヌス菌	2003.1.17	52	2	海岸に打ち上げられた鯨の摂食によるE型ボツリヌス中毒の発生	E型ボツリヌス菌(*Clostridium botulinum*)	アラスカ州西部	2002.7	1	6	7	186-192
		2001.8.17	50	32	発酵食品の摂食によるボツリヌス中毒の発生	E型ボツリヌス菌(*Clostridium botulinum*)	アラスカ州	2001	1	4	5	193-197
		2000.9.1	49	34	自家製鶏卵ピクルスによる食餌性ボツリヌス症	B型ボツリヌス菌(*Clostridium botulinum*)	イリノイ州	1997	1	3	4	198-201
	カンピロバクター	2002.6.28	51	25	乳牛信用プログラムを通して入手した非低温殺菌牛乳の飲用によるカンピロバクター(*Campylobacter jejuni*)感染症の発生	カンピロバクター・ジェジュニ(*Campylobacter jejuni*)	ウィスコンシン州	2001	2	4	6	202-207
		1998.2.27	47	7	食物の二次汚染によるカンピロバクター腸炎(*C. jejuni*)の発生	カンピロバクター・ジェジュニ(*Campylobacter jejuni*)	オクラホマ州	1996	1	4	5	208-212
	腸炎ビブリオ	1999.1.29	48	3	ロングアイランド湾で採取された生カキ及び蛤による腸炎ビブリオ感染症の集団発生	腸炎ビブリオ(*Vibrio parahaemolyticus*)(血清型O3:K6)	コネチカット州、ニュージャージー州、ニューヨーク州	1998	2	5	7	213-219
		1998.6.12	47	22	生カキの喫食による腸炎ビブリオ感染症の集団発生	腸炎ビブリオ(*Vibrio parahaemolyticus*)	太平洋北西部地域	1997	3	7	10	220-229
	クリプトスポリジウム	1998.7.17	47	27	食物によるクリプトスポリジウム症の発生	クリプトスポリジウム(*Cryptosporidium*)	ワシントン州スポーケン	1997	1	4	5	230-234
	その他の微生物等	2003.10.10	52	40	Chittering(食用のブタ小腸)に曝露した乳児における腸炎エルシニア胃腸炎	腸炎エルシニア(*Yersinia enterocolitica*)	イリノイ州シカゴ市	2002	1	3	4	235-238
		2002.4.12	51	14	粉末状乳児用人工乳の使用によるエンテロバクター(*Enterobacter sakazaki*)感染症	エンテロバクター(*Enterobacter sakazakii*)	テネシー州	2001	1	6	7	239-245
		2000.9.15	49	36	炭疽菌により汚染された食肉の摂取	炭疽菌(*Bacillus anthracis*)	ミネソタ州	2000.8	2	5	7	246-252
		1999.4.16	48	14	生パセリの摂食による赤痢菌感染症の発生	赤痢菌(*Shigella sonnei*)	米国(ミネソタ州、カリフォルニア州、マサチューセッツ州)、カナダ	1998.7～8	2	7	9	253-261
		1997.12.19	46	50	加熱調理済みのハムによるブドウ球菌食中毒の集団発生	ブドウ球菌(*Staphylococcus aureu*)、A型エンテロトキシン	フロリダ州	1997	1	4	5	262-266
ウイルス	ノロウイルス(ノーウォーク様ウイルス)	2001.6.15	50	23	(志賀毒素産生)病原大腸菌O157:H7起因と誤診されたカリシウイルス感染症の発生	ノロウイルス(ノーウォーク様ウイルス(流行性胃腸炎ウイルス:NLV))	バージニア州	2000	2	3	5	267-271
		2000.3.17	49	10	ノーウォーク様ウイルス胃腸炎の集団発生	ノロウイルス(ノーウォーク様ウイルス(NLVs))	アラスカ州、ウィスコンシン州	1999	2	6	8	272-279
		1999.3.26	48	11	米陸軍訓練生におけるノーウォーク様ウイルス胃腸炎の集団発生	ノロウイルス(ノーウォーク様ウイルス(NLVs))	テキサス州	1998	1	4	5	280-284
		1997.11.28	46	47	食用カキによるウイルス性胃腸炎	カリシウイルス	ルイジアナ州	1996.12～1997.1	2	6	8	285-292
	A型肝炎ウイルス	2003.11.28	52	47	レストランでのグリーンオニオン(ネギ)によるA型肝炎の集団発生	A型肝炎ウイルス(HAV)	ペンシルベニア州モナカ市	2003	2	6	8	293-300
		2003.6.20	52	24	食品媒介性A型肝炎	A型肝炎ウイルス(HAV)	マサチューセッツ州	2001	1	6	7	301-307
	その他のウイルス	2000.12.22	49	50	食品媒介によるA群ロタウイルス胃腸炎の集団発生	A群ロタウイルス	コロンビア特別区	2000.3～4	2	5	7	308-314
		1999.3.26	48	11	狂犬病罹患牛に由来する未殺菌乳摂取者に対する予防措置	狂犬病ウイルス	マサチューセッツ州	1996～1998	2	4	6	315-320
プリオン	プリオン	2003.2.21	52	7	ワイルドゲーム(狩猟)フェスタ参加者における致死的退行性神経疾患	慢性消耗性疾患(CWD)である感染性海綿状脳症(TSE)	ウィスコンシン州	2002	2	5	7	321-327
その他	病因不明	1999.3.19	48	10	ブリート(Burritos)摂食による病因不明の胃腸疾患の発生	病因不明(ブリート摂食)	米国(全6州)	1997.10～1998.10	2	6	8	328-335