Duration of Shedding and Secondary Household Transmission of Shiga Toxin–Producing *Escherichia coli* O26 During an Outbreak in a Childcare Center, Oregon, October–December 2010

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We assessed shedding duration and secondary household transmission of Shiga toxin 1-positive *Escherichia coli* O26 during a childcare-associated outbreak. No severe illness was noted. Shedding duration was 15–46 days (median, 29). No secondary transmission to household members was identified. Value of isolating asymptomatic infected children with this low-virulence infection remains uncertain.

**Key words.** Child Day Care Centers; *Escherichia coli* O26; Infectious Disease Outbreaks; Shiga Toxin

Non-O157 Shiga toxin-producing *Escherichia coli* (STEC) are an increasingly recognized cause of illness and can cause symptoms comparable to those caused by STEC O157 (O157), including hemolytic uremic syndrome (HUS) [1]. The virulence of non-O157 STEC strains is incompletely understood and varies with serotype and production of virulence factors, such as Shiga toxin types 1 (Stx1) or 2 (Stx2), intimin, and others [2]. STEC O26 (O26) is the most commonly reported non-O157 STEC in the United States [3]. Worldwide, few O26 outbreaks have been reported, and data regarding outbreaks in childcare settings are limited [4–10]. Measures to prevent person-to-person spread of STEC in childcare settings are based on experiences with O157 and include identification and exclusion of infected persons. Most states require exclusion of children with diarrhea from childcare centers until symptoms resolve, and exclusion of STEC-infected children until they have tested negative for STEC on at least 2 consecutive stool samples collected at least 24 hours apart [11]. No consensus exists on whether all non-O157 outbreaks require similar interventions.

In October 2010, 2 cases of Stx1-positive enteric infection in children ages 6 and 18 months were reported to a local health department in Oregon. The children had attended the same childcare center while symptomatic and had been excluded from attending in accordance with Oregon policy. The Oregon State Public Health Laboratory (OSPHL) confirmed by polymerase chain reaction (PCR) that both isolates carried stx1, but not stx2, genes and were serogroup O26, with indistinguishable pulsed-field gel electrophoresis (PFGE) patterns. We investigated to assess the extent of illness, duration of shedding, and secondary transmission to members of households with O26-infected children.

**SUBJECTS AND METHODS**

We defined a case as culture-confirmed O26 infection in a person who worked at or attended the childcare center during October 2010.
The center employed 13 staff, including 1 cook, and had 76 attendees (age range: 6 weeks–12 years) in 6 classrooms segregated by age. Four preschool classrooms were physically separated from 2 school-age classrooms, and preschool attendees had limited contact with school-age attendees. The 2 reported cases were in 2 different preschool classrooms.

Consent was obtained from all parents and staff. A single stool specimen was collected from each staff member and preschool classroom attendee, and from each of their siblings in the 2 other classrooms. Specimens were collected in Cary-Blair® transport media (Remel, Lenexa, Kansas); broth cultures were screened for stx using PCR essentially as previously described [12]; stx-positive broths were cultured; isolates were sero-grouped and PFGE performed using restriction enzymes XbaI and BlnI.

Asymptomatic stx-positive children were cohorted in 1 preschool classroom. Infected staff and parents of infected children were interviewed using a standard questionnaire to collect demographics and symptom information. No environmental sampling or food testing was done.

Duration of O26 shedding was assessed among children by collecting stool specimens weekly—twice weekly when possible—for stx testing using PCR, until 2 consecutive samples were negative. Detection of Shiga toxin genes was used as a proxy for O26 shedding, with no follow-up culture on stx-positive samples. Minimum duration of stx shedding was defined as the interval between first and last stx-positive samples.

Assessment of O26 transmission to household members of infected children was approved by the Institutional Review Boards (IRB) of the Centers for Disease Control and Prevention (CDC) and the Oregon Health Authority. Informed consent was obtained and participants’ demographics and gastrointestinal symptoms experienced since October 1, 2010 were collected using a standard questionnaire. Participants provided a single stool specimen for stx testing at OSPHL using PCR, with isolation and serotyping planned on stx-positive samples.

RESULTS

Staff and attendees reported no gastrointestinal illness during the 2 weeks before onset of the first case. No hygiene violations were found at the center. Persons with diarrhea or vomiting were excluded from the center in accordance with Oregon policy; surfaces were cleaned with bleach solution; hand washing was emphasized and commercial alcohol-based hand sanitizers were distributed in response to the outbreak.

Sixty-one (69%) of 89 persons in the center were screened for stx, including all 13 staff, all 41 attendees in the 4 preschool classrooms, and all 7 siblings of preschool attendees who attended school-age classrooms. Including the 2 initially reported cases, 9 (19%) of 48 children and 1 of 13 staff tested positive for STEC. All isolates were serogroup O26.

O26-positive children attended 3 of the 4 preschool classrooms. Median age of infected children was 1 year (range: 6 months–5 years); 45% were female. Four children (40%) reported diarrhea, with onsets during October 13–22. All children recovered within 2 weeks (median: 5.5 days; range: 3–10 days); none was hospitalized or suffered HUS. The staff member was asymptomatic; the cook tested negative.

All isolates tested positive for stx1 only and shared indistinguishable PFGE patterns. Isolates from the initial 2 patients were confirmed O26:H11, stx1 only, intimin-positive at CDC.

Duration of shedding was studied in all 9 O26-infected children. Two tested positive for stx on the initial stool sample only, making estimate of duration of shedding impossible. One had an interval of 12 days between first and last stx-positive stool samples, after which the parents declined to provide additional specimens. Six were followed until 2 consecutive stool samples tested negative. Duration of stx-shedding for these 6 children was 15–46 (median, 29) days (Fig. 1). No intermittent shedding was identified (ie, stx-positive sample after a negative one).

Five of 10 households where O26-infected children lived agreed to stx screening. Fourteen (82%) of 17 household members (age range: 1–41 years, median 21) submitted stool specimens; none tested positive for stx, and none reported gastrointestinal symptoms.

![Figure 1. Minimum duration of Shiga toxin genes shedding among 6 childcare attendees.](https://academic.oup.com/jpids/article-abstract/1/4/329/950964)
Cohort measures were discontinued after 2 weeks because no severe illness was noted, no stx2-positive STEC was identified, and strict compliance with hygiene procedures was observed. No symptoms were reported at the center after cohort measures were discontinued.

**DISCUSSION**

Few O26 outbreaks have been reported in childcare settings [4–10]. This outbreak mirrored those previously reported regarding lack of severe disease [4, 8, 10] and the presence of stx1, but not stx2, in the isolates identified. Findings on shedding duration are also similar to previous reports. In childcare settings in Argentina, O26 was detected in stool specimens 37 days after symptom onset [6, 7]; in Colorado, Brown et al. found O26 14–52 days after symptoms onset [8]. Low rates of secondary transmission to household members have also been suggested. Hiruta et al. and Sonoda et al. reported rates of 0 and 3%, respectively [4, 9]; in Colorado, 49% of case household members reported diarrheal illness, but they were not tested for STEC.

With the exception of O157 infections, STEC disease severity cannot be reliably predicted based on STEC serogroup or Shiga toxin type [13]. Although some evidence suggests that non-O157 Stx1-only strains can cause bloody diarrhea, the frequency of Stx1-only strains causing HUS is very low [14]. Further research is needed to characterize the virulence of Stx1-only, non-O157 STEC, including O26.

The burden of control measures used in childcare center outbreaks must be weighed against the disease-prevention benefit. Aggressive control measures appear warranted for O157 but may not be necessary in certain non-O157 STEC outbreaks. Until additional data regarding the virulence and transmissibility of non-O157 STEC are obtained, public health policies regarding screening, cohort measures, and exclusion in non-O157 childcare-associated outbreaks should remain flexible. We recommend that symptomatic children be excluded until symptoms resolve, and incidence of new infections be monitored. Routine hand washing should be emphasized. In the absence of severe disease or a virulent pathogen, requiring 2 consecutive negative stool samples to lift exclusion might not be justifiable. The value of screening asymptomatic children to segregate those who are infected remains unproven.

Of note, this outbreak was detected through routine screening of stool specimens for Shiga toxin at 2 clinical laboratories and would have been missed had stool testing been limited to culture for O157. However, Shiga toxin screening should not replace culture for O157.

CDC recommends simultaneous culture for O157 and—to detect non-O157 STEC—Shiga toxin assay for all stool specimens submitted to clinical laboratories [15].

Limitations in this investigation included our inability to test school-age attendees; this might have underestimated the extent of O26 infection in the center. Additionally, we assessed O26 shedding through PCR assay for stx genes. However, a pathogen’s viability and infectivity cannot be inferred from a positive PCR. It is also possible, though unlikely, that stx shedding could be from other STEC that were missed in culture. Finally, episodes of household transmission might have been missed: half of the households declined to participate in screening, and assessment of household transmission by testing a single stool specimen might have missed intermittent shedding. Furthermore, despite expedited IRB approval, screening of households did not begin until 24 days after identification of the outbreak. By that time, 4 of 9 children were stx-negative.

**CONCLUSION**

This childcare-associated outbreak of O26 stx1 infection was notable for mild symptoms and prolonged shedding. Low virulence, coupled with prolonged shedding makes exclusion of infected attendees until 2 consecutive stool samples are negative difficult to justify. The value of testing asymptomatic children to guide cohorting is uncertain. Further studies of STEC virulence factors and person-to-person transmission in childcare centers will improve public health decision making. Until then, exclusion of symptomatic attendees and strict attention to hygiene in childcare centers should be emphasized.

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