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Pertussis Pseudo-outbreak Linked to Specimens Contaminated by *Bordetella pertussis* DNA From Clinic Surfaces

**WHAT’S KNOWN ON THIS SUBJECT:** Pertussis is a poorly controlled vaccine-preventable disease. Verifying outbreaks is challenging owing to nonspecific clinical presentations and imperfect diagnostic tests. Exclusive reliance on highly sensitive polymerase chain reaction has been associated with pseudo-outbreaks.

**WHAT THIS STUDY ADDS:** Contamination of specimens with vaccine derived *Bordetella pertussis* DNA from pediatric clinic surfaces likely resulted in misdiagnoses. Standard practices, liquid transport medium, and lack of polymerase chain reaction cutoffs for discerning weakly positive (contaminant) DNA are contributory, but modifiable factors.

**BACKGROUND AND OBJECTIVES:** We investigated a pertussis outbreak characterized by atypical cases, confirmed by polymerase chain reaction (PCR) alone at a single laboratory, which persisted despite high vaccine coverage and routine control measures. We aimed to determine whether *Bordetella pertussis* was the causative agent and advise on control interventions.

**METHODS:** We conducted case ascertainment, confirmatory testing for pertussis and other pathogens, and an assessment for possible sources of specimen contamination, including a survey of clinic practices, sampling clinics for *Bordetella pertussis* DNA in clinics from vaccine, clinic standard specimen collection practices, use of liquid transport medium, and lack of clinically relevant PCR cutoffs.

**RESULTS:** Between November 28, 2008, and September 4, 2009, 125 cases were reported, of which 92 (74%) were PCR positive. Cases occurring after April 2009 (n = 79; 63%) had fewer classic pertussis symptoms (63% vs 98%; P < .01), smaller amounts of *B pertussis* DNA (mean PCR cycle threshold value: 40.9 vs 33.1; P < .01), and a greater proportion of PCR-positive results (34% vs 6%; P < .01). Cultures and serology for *B pertussis* were negative. Other common respiratory pathogens were detected. We identified factors that likely resulted in specimen contamination at the point of collection: environmentally present *B pertussis* DNA in clinics from vaccine, clinic standard specimen collection practices, use of liquid transport medium, and lack of clinically relevant PCR cutoffs.

**CONCLUSIONS:** A summer pertussis pseudo-outbreak, multifactorial in cause, likely occurred. Recommendations beyond standard practice were made to providers on specimen collection and environmental cleaning, and to laboratories on standardizing PCR protocols and reporting results, to minimize false-positive results from contaminated clinical specimens. *Pediatrics* 2012;129:e424–e430

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**KEY WORDS**
*Bordetella pertussis*, cross contamination, disease outbreak, polymerase chain reaction, false positive result, diagnostic errors

**ABBREVIATIONS**
CDC—Centers for Disease Control and Prevention
cycle threshold—CT
insertion sequence 481—IS481
nasopharyngeal—NP
polymerase chain reaction—PCR
Regan-Lowe—RL

All authors participated in the concept and design, analysis and interpretation of data, drafting or revising of the manuscript, and have read and approved the manuscript. Dr Mandal had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Dr Mandal analyzed the data and drafted the manuscript. Ms Martin supervised the investigation.

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Despite high vaccination coverage, the United States has experienced a resurgence of pertussis, a highly infectious and outbreak-prone respiratory disease caused by the bacterium *Bordetella pertussis*. Clinical diagnosis is difficult, as early signs and symptoms are often nonspecific and laboratory diagnostics are imperfect. Culture is the gold standard, but the speed of polymerase chain reaction (PCR) has superseded culture. PCR methods are not standardized and few laboratories have defined their lower limits of detection, so positive cutoffs and reporting ranges vary. The highly sensitive nature of PCR means that extrinsic contamination of reagents, assays, or specimens with DNA can lead to false-positive results.

This article describes the investigation of a respiratory illness outbreak that occurred in rural southwest Colorado from late November 2008 to August 2009. Clinics in a major population center were the main providers of health care for 3 contiguous counties (population 90,000). Elevated rates of disease were restricted to these 3 counties. The outbreak was attributed to *B. pertussis* based on positive PCR results from clinical specimens collected largely from one clinic (Clinic A) and tested at a single laboratory (Laboratory A), a national diagnostic reference laboratory serving many states. The outbreak persisted despite appropriate control measures. Many cases reported prior pertussis vaccination and atypical symptoms. Efforts in July 2009 to confirm the outbreak by additional laboratory testing (PCR and serology) were inconclusive.

**METHODS**

A case-patient was defined as a person who met the Council of State and Territorial Epidemiologists pertussis case definition (pertussis case definition available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5517a3.htm) or was PCR confirmed, was resident in 1 of 3 counties served by health facilities in the outbreak town, and was reported as a pertussis case between November 28, 2008, and September 4, 2009. Cases were ascertained by querying the state's electronic disease-reporting system and by active case-finding in local clinics for new cough illness between August 12, 2009, and September 4, 2009.

We abstracted demographic, clinical, and immunization data from the state's electronic disease-reporting system. We collected supplemental immunization and clinical symptom data through a questionnaire administered in person or by telephone interviews. Immunization status was verified using the state's vaccine registry. A case was considered to be age-appropriately vaccinated if the case had documented receipt of pertussis vaccines as per the recommended schedule.

To investigate whether there was any clustering of cases by clinic, the total number of PCR tests performed and the proportion positive at Laboratory A were obtained for providers who performed most of the specimen collection in the outbreak area.

Confirmatory testing, including other respiratory pathogens, was performed at the Centers for Disease Control and Prevention (CDC). Blood for serologic testing was requested from cases whose cough onset was 2 to 8 weeks before the interview. For cases coughing fewer than 2 weeks at interview, we requested blood, and a nasopharyngeal (NP) specimen for culture and PCR for *Bordetella* species, and PCR for human metapneumovirus, adenovirus, rhinovirus, parainfluenza virus 3, *Mycoplasma pneumoniae*, and *Chlamydia pneumoniae*.

To assess the potential for laboratory contamination of NP specimens, Laboratory A provided any remaining frozen NP swabs in liquid transport medium from cases that had previously tested PCR positive. NP swabs and liquid transport media were distributed by Laboratory A to providers as prepackaged kits. These specimens were retested at CDC for *Bordetella* species and other respiratory pathogens. Uninoculated (sterile) NP swabs obtained from clinics were used as controls. To assess the validity and reliability of PCR at Laboratory A and potential for laboratory contamination, paired (ie, duplicate) testing was implemented from August 28, 2009, to September 4, 2009. NP aspirates from new cases of cough illness were split into 2 aliquots: one for CDC *Bordetella* PCR and culture and the other for Laboratory A *Bordetella* PCR. Specimens were collected, stored, and transported using identical methods. We also reviewed PCR data from Laboratory A, including standard operating procedures, PCR assay protocols, positivity rates over a 2-year period, including the outbreak months, and cycle threshold (Ct) values on outbreak cases. PCR Ct values are a quantification of the amount of DNA present in a specimen: low Ct values indicate large amounts of DNA and vice versa.

NP aspirates were collected using N-Pak kits (M-Pro, Annandale, MN) and dispensed into sterile screw-cap tubes. Sterile rayon-tipped NP swabs (Copan Diagnostics, Murrieta, CA) were dipped into each aspirate and inserted into Regan-Lowe (RL) transport agar tubes (Remel, Lenexa, KS). For aspirate specimens on which paired testing was conducted, a second and third rayon-tipped swab were dipped into the aspirate and inserted into separate liquid universal transport media destined for CDC and Laboratory A. If aspiration collection was unsuccessful, an NP swab was taken and inserted into an RL transport tube.

NP specimens were plated onto RL agar plates with and without cephalexin and incubated for up to 10 days at 37°C. DNA was extracted by using the Roche Compact MagNApure (Roche Applied Science, Indianapolis, IN). Real-time multigene PCR assays were performed on the ABI
Serological testing was performed using an immunoglobulin G anti–pertussis toxin enzyme-linked immunosorbent assay. The diagnostic cutoff for recent pertussis infection was ≥94 IU/mL; values between 49 and 93 IU/mL were considered indeterminate, and values <49 IU/mL were deemed negative.

To assess the potential for clinic contamination of specimens, 2 clinics agreed to participate in an environmental assessment, including Clinic A, which had the highest proportion of positive PCR results. Surveys were administered to the lead nurse on NP specimen collection practices; general cleaning; infection-control procedures; room use for examination and vaccination; and storage, administration, and brands of pertussis vaccines. Clinic environmental sampling for B pertussis DNA was performed. Specimens were systematically collected from surfaces in exam rooms and nurses’ stations. Sterile, flexible, flocked NP swabs (Copan) were wetted in a tube containing 500 μL of 10 mM Tris HCl, pH 8.0, sterile buffer and rubbed along surfaces with a twisting motion for 15 seconds before reinsertion into the tube. DNA extraction was by the Roche MagNAPure LC and testing followed CDC’s IS481 real-time PCR assay.

Statistical analysis was performed in SAS 9.2 for Windows (SAS Institute, Inc, Cary, NC). Differences between proportions were tested using Pearson’s $x^2$ tests or Fisher’s exact test, and differences between means were tested using the Student $t$ test. The Wilcoxon test was used for nonparametric data.

RESULTS

Over 9 months, 127 suspect cases were reported through routine surveillance and 26 additional suspect cases were enrolled via 3 provider offices. We completed questionnaires on 83 (65%) suspect cases from routine surveillance and 25 (98%) suspect cases identified through active case finding. In total, 125 cases (115 of 127 and 10 of 26) met the investigation outbreak case definition. The epidemic curve (Fig 1) shows 2 distinct clusters: 1 in winter (November 2008–April 2009; $n = 46$) and 1 in summer (May–August 2009; $n = 79$). PCR-confirmed cases comprised 92 (74%) cases and 64 (70%) of these occurred during the summer.

Compared with the winter cases ($n = 46$), summer cases ($n = 79$) were less likely to report at least 1 classic pertussis symptom (63% vs 98%; $P < .01$) or cough for ≥14 days (70% vs 100%; $P < .01$), but more likely to have visited Clinic A (96% vs 67%; $P < .01$) and to have age-appropriate vaccinations (75% vs 57%; $P = .05$) (Table 1). There was a decline in the monthly proportion of cases reporting paroxysms over the outbreak period (slope = −0.06; $R^2 = 0.74$; data not shown).

We looked at the distribution of PCR-positive results among providers in the outbreak area to see if this suggested a single source. During the outbreak period, 609 PCR tests were submitted to Laboratory A from providers. The proportion of PCR-positive results increased between winter and summer (6% to 34%; $P < .01$). Five providers requested 567 (93%) tests, of which a single provider, Clinic A, ordered the most tests (376/567, 66%), and had the highest overall proportion of positive tests (21%), with a range of 4% to 67% over the outbreak
period. The number of tests ordered by the other 4 providers ranged from 39 to 54, with a positive proportion range of 0% to 12%. Figure 2 charts the monthly number of positive PCR tests at Clinic A versus other providers. Clinic A experienced an increase in the number of positive tests in the summer, contributing 100% of all positive tests from the outbreak area during May and June 2009, against a decline in the number of PCR tests ordered from all clinics and cough illness presentations (data not shown).

Confirmatory testing at CDC was performed on specimens from 5 summer retrospective cases (5 sera) and 26 new cases of cough illness, providing 10 sera and 25 NP specimens (for culture and PCR). In addition, PCR retesting at CDC was performed on 14 Laboratory A–positive specimens from July 2009. At CDC, no specimens were positive for *B pertussis* by culture, PCR, or serology, but other respiratory pathogens were detected (Table 2). The 9 indeterminate PCR results had similar Ct values on both Laboratory A and CDC’s PCR assays, but were reported differently: Laboratory A did not define an indeterminate range for Ct values indicating fewer than 1 bacterium, reporting these as “positive.” These 9 indeterminate specimens comprised 7 retests of Laboratory A–positive specimens and 2 specimens from new patients on whom Clinic A staff used an NP swab and liquid transport medium, rather than an NP aspirate kit and semisolid transport medium.

They grouped into a winter cluster with low Ct values (mean 33.1) and a summer cluster with a dramatic shift to high Ct values (mean 40.9); the difference in means was significant at *P* < .01. When CDC Ct cutoffs for determining positivity were applied, most winter cases fell into the positive range with summer cases falling in the indeterminate or negative range, suggestive of low levels of DNA (fewer than 1 bacterium) and possible contamination.

To investigate laboratory contamination, paired testing of NP specimens at CDC and Laboratory A was performed for 9 of the new cough illness cases described previously. Eight of the specimens, collected by using NP aspirate kits, were negative at both laboratories. One specimen was indeterminate at CDC but positive at Laboratory A, although the Ct values, and therefore the amount of DNA detected, were very similar: 39 at CDC and 40 at Laboratory A. This single indeterminate specimen differed from the other 8, having been collected by Clinic A staff using an NP swab and liquid transport medium. Four control liquid transport media tested negative for pertussis by CDC’s PCR. Laboratory A adherence to standard operating procedures likely minimized potential for within-laboratory contamination: culture was performed in a different laboratory from PCR, clean reagents were prepared in areas separate from DNA extraction and amplification, and negative controls were used after every 10 specimens (increased to every 6 specimens beginning July 2009).


### TABLE 1 Characteristics of Winter Versus Summer Cases Meeting the Investigational Outbreak Definition

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Winter</th>
<th>Summer</th>
<th><em>P</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age, y (range)</td>
<td>6 (0–75)</td>
<td>4 (0–35)</td>
<td>.07</td>
</tr>
<tr>
<td>Male gender</td>
<td>18 (45)</td>
<td>35 (50)</td>
<td>.61</td>
</tr>
<tr>
<td>CSTE confirmed case</td>
<td>28 (61)</td>
<td>29 (37)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>CSTE probable case</td>
<td>17 (37)</td>
<td>12 (15)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Cough ≥14 d at time of interview</td>
<td>46 (100)</td>
<td>55 (70)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>≥1 classic pertussis symptom</td>
<td>45 (98)</td>
<td>50 (63)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Paroxysms</td>
<td>44 (96)</td>
<td>49 (62)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Posttussive vomiting</td>
<td>31 (67)</td>
<td>18 (23)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Whoop</td>
<td>16 (35)</td>
<td>8 (10)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Attended Clinic A</td>
<td>24 (57)</td>
<td>48 (75)</td>
<td>.05</td>
</tr>
</tbody>
</table>

**CSTE, Council of State and Territorial Epidemiologists.**

* a Classic pertussis symptoms defined as whoop, paroxysm, or posttussive vomiting reported by patient/parent.

* Denominators may vary because of missing data.
proportion of positive pertussis tests rose sharply. Laboratory A confirmed that this spike in PCR positivity was a result of specimens from the outbreak area alone (personal communication, 2009).

An environmental assessment was conducted at 2 clinics, A and B, to assess the potential for within-clinic specimen contamination. Both clinics used NP swabs and liquid transport media. During fall 2008, Clinic A switched to a diphtheria, tetanus, and acellular pertussis vaccine previously shown to contain *B. pertussis* DNA. Clinicians staff reported not routinely wearing gloves to collect NP specimens or to prepare vaccines, conducting examinations and vaccinations in the same rooms where hand-washing sinks were not always present, although alcohol-based hand sanitizers were, and not performing regular surface bleach cleanings. In Clinic B, in contrast, staff reported glove use for vaccination and specimen collection, sinks were present in all rooms, and periodic bleach cleanings occurred. Additionally, Clinic B used a vaccine in which *B. pertussis* DNA has not been detected.

*B. pertussis* DNA was widely detected on surfaces in Clinic A (11/18, 61% of sites swabbed) and its satellite clinic, A1 (3/9, 33% of sites swabbed), compared with fewer areas at Clinic B (2/20, 10% of sites swabbed). Large amounts of DNA (Ct value 33.2) were found on nurses’ laptops in Clinic A and to a lesser degree (Ct value range 35.7–41.0) on vaccine refrigerator surfaces and examination room provider areas (worktops, sink areas, glove containers, biohazard bin, stool), patient areas (couch, toys, chairs), and doorknobs, with higher densities in an examination room without a sink. At the smaller satellite, Clinic A1, DNA was detected at the nurses’ station, vaccine refrigerator, and doorknobs (Ct values 39.6–39.9). By comparison, a negligible amount of DNA (Ct value 41.0) was detected on 2 surfaces (door knob and vaccine preparation area) in Clinic B.

**DISCUSSION**

Our findings suggest that although there may have been a true pertussis outbreak during winter 2008–2009, a pseudo-outbreak of pertussis occurred in summer 2009, which was likely attributable to false-positive PCR results, as evidenced by inconsistent clinical symptoms, negative confirmatory testing, detection of other pathogens, and high Ct values suggestive of contamination. Review of Laboratory A PCR data and quality indicators excluded laboratory contamination. The pattern of PCR positivity centering on Clinic A and the environmental findings supported *B. pertussis* DNA contamination of diagnostic specimens in Clinic A.

The environmental assessment indicated that spillage from preparation of diphtheria, tetanus, and acellular pertussis vaccines was the most likely source of high levels of DNA present on surfaces in Clinic A. Children sick with pertussis might have been an additional source, explaining the low levels detected at Clinic B where vaccine containing *B. pertussis* DNA was not reportedly used. We postulate that handling NP swabs without prior hand hygiene or putting on new gloves may have contributed to the transfer of environmental DNA to the swab handle. Following specimen collection, the inoculated swab and handle are inserted into a tube containing liquid transport media, and because this media freely circulates in the

### TABLE 2 Results of CDC Testing for *Bordetella pertussis* and Other Pathogens

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Bordetella pertussis</th>
<th>PCR Positive or Indeterminate for Other Pathogens (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sera (n = 15)</td>
<td>0</td>
<td>15*</td>
</tr>
<tr>
<td>NP culture (n = 25)</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>NP PCR (n = 39)</td>
<td>0</td>
<td>30*</td>
</tr>
</tbody>
</table>

*Bordetella parapertussis* (1)  
Parainfluenza virus type 3 (2)  
Adenovirus (1)  
Rhinovirus (4)  
Chlamydia pneumoniae (3)  
Mycoplasma pneumoniae (6)

* Denotes results including tests done at CDC in July 2009 as part of early efforts to confirm the outbreak; serum: 2 negative; NP PCR: 1 indeterminate and 4 negative. n/a, not applicable.

### FIGURE 3

Cycle threshold (Ct) values of Laboratory A positive PCR tests, December 2008 to July 2009.
tube, any contaminant DNA present on the handle may be washed off into the liquid and extracted for PCR testing. Amplification of low levels of environmental DNA result in high Ct values, which were reported as positive by Laboratory A.

Pseudo-outbreaks and overestimation in true outbreaks associated with contamination in laboratories have previously been described. The role of environmentally present DNA in clinics is less well documented. Some acellular pertussis vaccines contain PCR-detectable B. pertussis DNA and both whole-cell and acellular vaccines have been implicated as a source of DNA on clinic surfaces. Although molecular typing has shown that this DNA can be linked to pertussis vaccines, insufficient DNA in our environmental specimens prevented us from demonstrating this. Segregating examination and vaccination rooms has been suggested as a means of reducing opportunities for contamination of specimens with environmental DNA. Clinic A reported initially using separate, designated rooms, which might explain why false-positive results did not present earlier; following the switch in vaccine brand.

PCR protocols should include clinically relevant cutoff values, and for highly sensitive targets, such as IS481, an indeterminate result category for Ct values consistent with detection of fewer than 1 bacterium. Using other targets with IS481 can improve specificity and the ability to differentiate between Bordetella species. PCR protocols and Ct values, including indeterminate results, should be accessible to state health departments, as clusters of high Ct results may alert them to diagnostic problems. Although clinicians are encouraged to familiarize themselves with the potentials and pitfalls of PCR, at the patient level, sensitivity should be maximized so that early treatment prevents complications, shortens the infectious period, and interrupts transmission. Thus, when faced with an indeterminate PCR result in an appropriately tested patient, it may be reasonable to treat as if positive. At the public health level, specificity should be optimized to ensure that public health responses, such as chemoprophylaxis of contacts, exclusion of children from child-care and school settings, and furloughing of adults, are commensurate with the true epidemiologic picture.

CONCLUSIONS

During pertussis outbreaks, heightened physician awareness might lead to overbroad testing, which when combined with clinic contamination of NP specimens by environmentally present DNA, and lack of PCR standardization or meaningful cutoffs among laboratories, may contribute to false-positive results and pseudo-outbreaks. In response to this investigation and other recent data, CDC has published best practices for health care professionals on the use of PCR for pertussis diagnosis to improve its clinical accuracy. The guidance covers who to test, when to test, optimal collection techniques, recommendations for preventing specimen contamination, and understanding PCR results. This investigation reinforces the importance of interpreting PCR results in the context of other clinical parameters and obtaining culture or serologic confirmation. Absent or inconsistent supporting clinical data and negative confirmatory tests in appropriately collected specimens should prompt testing for alternate pathogens. Correctly classifying outbreaks, although challenging with pertussis, is of paramount importance to avoid the considerable health care–associated and societal costs resulting from pseudo-outbreaks.

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REFERENCES


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