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The Epidemiology of Pertussis: A Comparison of the Epidemiology of the Disease Pertussis With the Epidemiology of *Bordetella pertussis* Infection

James D. Cherry, MD, MSc

ABSTRACT. In the prevaccine era pertussis epidemics followed a cyclic pattern, with peaks every 2 to 5 years. With the marked reduction of pertussis by vaccination, the same cyclic pattern still occurs. Studies relating to reported pertussis and *Bordetella pertussis* infection have been reviewed and analyzed. The increase in reported pertussis over the last 2 decades is mainly due to a greater awareness of pertussis and perhaps to the use of several less efficacious vaccines.

Studies of prolonged cough illnesses in adolescents and adults reveal that 13% to 20% are a result of *B pertussis* infection. Serologic studies suggest that the rate of *B pertussis* infection in adolescents and adults is ~2.0% per year. The rate of cough illnesses (pertussis) caused by *B pertussis* infection in adolescents and adults is between 370 and 1500 per 100 000 population. These data suggest that there are between ~800 000 and 3.3 million cases per year in the United States.

The coming availability of adolescent- and adult-formulated diphtheria and tetanus toxoids and acellular pertussis vaccines for adolescents and adults and their widespread use should reduce the reservoir of *B pertussis* disease. It is suggested that a universal program of adolescent and adult boosters would decrease the circulation of *B pertussis* in these age groups and possibly could lead to the elimination of the organism from the population. *Pediatrics* 2005;115:1422-1427; *pertussis, Bordetella pertussis, adult pertussis, adolescent pertussis, pertussis, epidemiology.*

ABBREVIATIONS. DTaP, adolescent- and adult-formulated diphtheria and tetanus toxoids and acellular pertussis; ELISA, enzyme-linked immunosorbent assay; PCR, polymerase chain reaction; PT, pertussis toxin; FHA, filamentous hemagglutinin; PRN, pertactin; FIM, fimbriae; DTP, diphtheria and tetanus toxoids and pertussis; APERT, adult acellular pertussis vaccine efficacy trial.

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Conflict of interest: During the last 3 years, Dr Cherry has received honoraria from GlaxoSmithKline and Aventis (both manufacturers of diphtheria and tetanus toxoids and acellular pertussis vaccines) for talks and participation in meetings related to diphtheria and tetanus toxoids and acellular pertussis vaccines.

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In the prevaccine era pertussis epidemics followed a cyclic pattern, with peaks every 2 to 5 years.¹⁻⁴ In the present vaccine era the overall incidence of pertussis has been reduced dramatically, but the cyclic pattern has not changed. This pattern contrasts with other infectious diseases that have been controlled by immunization. For example, with measles, as vaccine use became widely used, there was a lengthening of time intervals between cycles and an overall reduction in disease incidence.^{3,5}

These 2 different patterns suggest that with measles both the disease incidence and the circulation of the virus have been reduced. With pertussis, however, the incidence of reported disease has been reduced, but the circulation of *Bordetella pertussis* has continued in the overall population in a manner similar to that which occurred in the prevaccine era.

Data generated during the last 15 years suggest that the circulation of *B pertussis* is occurring in adolescents and adults and is manifest by prolonged cough illnesses, which most often go unrecognized as pertussis.^{1,2} The understanding of the epidemiology of *B pertussis* infection is critical at the present time, because new adolescent- and adult-formulated diphtheria and tetanus toxoids and acellular pertussis component (DTaP) vaccines will soon be available in the United States.^{6,7} The optimal use of these vaccines will require an understanding of the epidemiology of *B pertussis* infection.

In this article, the epidemiology of reported pertussis (the disease) will be compared with selected aspects of *B pertussis* infections (recognized or unrecognized) in adolescents and adults.

METHODS

Centers for Disease Control and Prevention data on reported pertussis over time will be presented and compared with data from recent studies on prolonged cough illnesses in adolescents and adults and with the rate of total *B pertussis* infections and the rate of *B pertussis* infections with cough illnesses in defined populations. All prospective, published studies during the last 20 years of prolonged cough illnesses in adolescents and adults in which *B pertussis* infection was looked for by serologic study (enzyme-linked immunosorbent assay [ELISA]) have been analyzed. Rate data over time of *B pertussis* infection in adolescents and adults were determined from studies in which *B pertussis* antibody-titer changes over time were determined. Rate data on *B pertussis* illnesses were determined from the prolonged cough-illnesses studies mentioned above and 1 serologic study with specimens at multiple time points and data collected on respiratory illness.

The *B pertussis* infections have been determined by culture, polymerase chain reaction (PCR), antigen detection (direct fluorescent antibody), and most often by significant antibody-titer rises or high single-serum antibody values to pertussis toxin (PT)

and other *B pertussis* proteins (filamentous hemagglutinin [FHA], pertactin [PRN], and fimbriae [FIM]) by ELISA or agglutination.

RESULTS

The Epidemiology of Reported Pertussis

The number of cases of pertussis reported each year from 1922 to 2003 is presented in Fig 1, and the percent distribution of reported pertussis according to age group is presented in Fig 2. In the prevaccine era pertussis was a universally present disease with cyclic peaks every 2 to 5 years.¹⁻⁴ The average yearly rate of reported pertussis was 157 per 100 000 population.⁴ Significant underreporting of pertussis was known to occur; thus, an estimated corrected yearly attack rate for the United States was 872 cases per 100 000 population.⁴

In the prevaccine era reported cases occurred almost exclusively in children; >93% of reported cases occurred in children <10 years old.^{1,4,8} Pertussis immunization commenced in the United States in the 1940s, and by 1960 there were <10 cases of reported pertussis per 100 000 population.⁴ A decade later (1970) the rate had fallen to <1 per 100 000 population.

In contrast with the prevaccine and early vaccine era, the reporting of pertussis in adolescents and adults has increased dramatically.^{1,8} As noted in Fig 2, ~50% of the reported pertussis cases were in persons >10 years old. Since 1984 there has been a modest but steady increase in reported cases in the United States.⁸ In 2002 there were 9771 reported cases, for a yearly rate of 3.4 per 100 000 population.⁹ As noted in the introduction, in the vaccine era reported pertussis still has a cyclic pattern, and the peaks still occur at 2- to 5-year intervals.

In recent years there has been much concern in the lay and scientific literature about the "resurgence of pertussis." This resurgence was discussed recently in a *Pediatrics* commentary.⁸ To summarize, there are 5 possible causes for the increased reporting of pertussis: (1) genetic changes in *B pertussis* making vaccines less effective; (2) lessened potency of pertussis vaccines; (3) waning of vaccine-induced immunity; (4)

greater awareness of pertussis; and (5) the general availability of better laboratory tests in some areas of the country. Of these possible causes, it was felt that the most significant factor was a general greater awareness of pertussis that has occurred in recent years. However, the observation of multiple outbreaks of pertussis in preadolescent and adolescent school populations suggest that, in my opinion, waning vaccine-induced immunity caused by vaccines that were less efficacious than in the past has also been a contributor.

Specifically, before the introduction of and universal use of DTaP vaccines in the United States there were 2 whole-cell pertussis component diphtheria and tetanus toxoids and pertussis (DTP) vaccines available. Both of these vaccines in the mid 1980s were immunogenic, but 1 of the 2 subsequently was found to be poorly immunogenic and had poor efficacy.¹⁰⁻¹² In contrast, the other DTP vaccine remained highly immunogenic and efficacious.^{13,14} In conjunction with the DTaP-vaccine efficacy trials in the early 1990s, it was found that all DTP vaccines except the Connaught USA vaccine were more efficacious than the comparative DTaP vaccines. Of the 3 DTaP vaccines in use in the United States today, 2 had less efficacy than a comparative DTP vaccine.^{15,16} The use of lesser potency vaccines from the late 1980s until the present may well be a contributing factor in the rise in preadolescent and adolescent pertussis.

The Epidemiology of *B pertussis* Infections

During the last 2 decades a number of studies have provided data that are useful in the delineation of *B pertussis* infection from reported pertussis. These studies include the study of prolonged cough illnesses in adolescents and adults, serologic studies over time used to determine infection rates in populations, and studies in defined populations to determine rates of symptomatic infections in adolescents and adults.

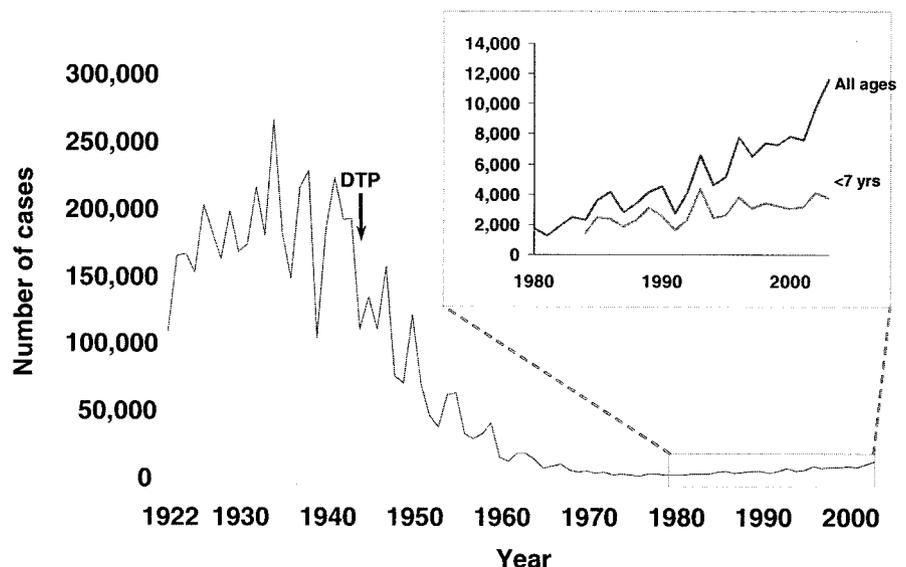
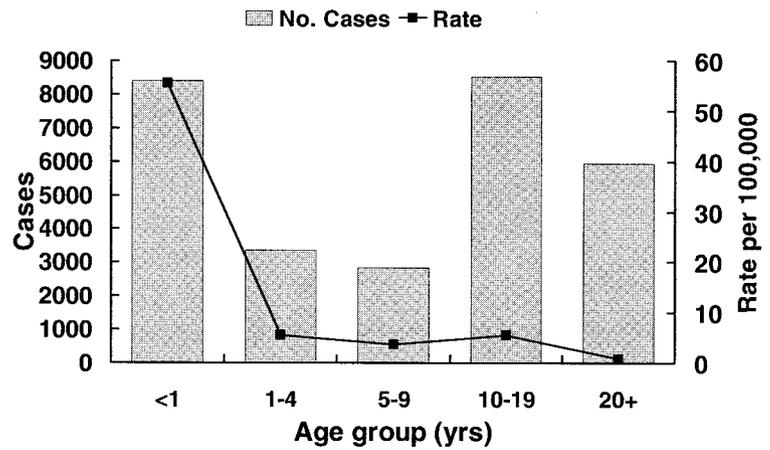


Fig 1. Reported pertussis cases: United States, 1922–2003. Source: Trudy V. Murphy, MD, Centers for Disease Control and Prevention, written communication, November 2004.

Fig 2. Age distribution and incidence of reported pertussis cases: United States, 1997–2000. Source: the Centers for Disease Control and Prevention, www.cdc.gov/nip/ed/slides/pertussis8p.ppt.



Percentage of Prolonged Cough Illnesses in Adolescents and Adults Caused by B pertussis Infection

Since 1987 there have been 13 published studies of adolescents and adults with prolonged cough illnesses in which the rates of *B pertussis* infections could be evaluated.^{3,17–28} The breakthrough that allowed these investigations was the development of quantitative ELISA to determine antibodies to *B pertussis* and selected *B pertussis* antigens.²⁹ The ELISA, as developed by Manclark et al,²⁹ allows a high level of precision so that twofold changes in ELISA antibodies between acute-phase and convalescent-phase sera are diagnostic. Furthermore, *B pertussis* infection in adolescents and adults can be reliably diagnosed by high single-serum ELISA antibody titers.^{3,30} This is an important breakthrough, because adolescents and adults often do not come to medical attention until the 3rd or 4th week of illness, a time at which the peak antibody titer has already occurred.^{3,19} Because of this, titer changes between acute-phase and convalescent-phase sera frequently do not occur.

Presented in Table 1 are the details and results of the 13 studies in which the percentage of prolonged cough illnesses resulting from *B pertussis* infection are presented. As noted, the rates vary from 12% to 52%. The extreme variation in rates can be explained by multiple factors such as the number of *B pertussis* antigens for which antibody was determined, the

biases of the study physicians, and the precision of the ELISA studies.

The studies done in Germany, Chicago, Illinois, Denmark, and Paris, France, were all done during known regional outbreaks of pertussis so that physician bias (selecting patients who they think have pertussis) led to the selection of more typical cases rather than all persons with prolonged coughs. This approach will reveal a greater percentage of positives than if a protocol that dictates the study of all persons with prolonged cough is followed.

The other studies in Table 1 (Seattle, WA; New South Wales, Australia; Los Angeles, CA; Nashville, TN; San Diego, CA; San Francisco, CA; Minneapolis/St Paul, MN; and Korea) were investigations of cough illnesses in populations during times at which pertussis was not recognized as an outbreak. As seen in Table 1, the determined rate in these 8 studies still varies from 12% to 50%. These differences can be explained largely by the number of antigens for which antibody was determined. In general, the studies in which antibodies to FHA, FHA and PRN, or FHA, PRN, and FIM were analyzed in addition to antibody to PT found a greater percentage of positives.

It is now recognized that infection with *Bordetella parapertussis* commonly elicits an antibody response to *B pertussis* FHA and PRN and also occasionally to

TABLE 1. Percentage of Prolonged Cough Illnesses in Adolescents and Adults Caused by Infections With Serologic, PCR, or Culture Evidence of *Bordetella* Infections

Source	Location	Study Performed During Pertussis Outbreak?	Year	Percent
Jackson et al ¹⁷	Seattle, WA	No	1983–1987	15
Robertson et al ¹⁸	New South Wales, Australia	No	1985–1986	26
Mink et al ³	Los Angeles, CA	No	1986–1989	26
Schmitt-Grohé et al ¹⁹	Germany	Yes	1992–1994	32
Wright et al ²⁰	Nashville, TN	No	1992–1994	21
Wirsing von König et al ²¹	Germany	Yes	1992–1994	31
Rosenthal et al ²²	Chicago, IL	Yes	1992–1994	26
Jansen et al ²³	San Diego, CA	No	1993–1994	17
Nennig et al ²⁴	San Francisco, CA	No	1994–1995	12
Strebel et al ²⁵	Minneapolis/St Paul, MN	No	1995–1996	13
Birkebaek et al ²⁶	Denmark	No	1995–1997	17
Vincent et al ²⁷	Korea	No	1997–1998	50
Gilberg et al ²⁸	Paris, France	Yes	1999	52

Significant ELISA titer rise or high titer to PT, FHA, PRN, or FIM or significant agglutinin titer or titer rise.

TABLE 2. Percentage of Prolonged Cough Illnesses in Adolescents and Adults Caused by Infections With Serologic, PCR, or Culture Evidence of *B pertussis* Infections

Source	Location	Years	Percent
Mink et al ³	Los Angeles, CA	1986–1989	13
Wright et al ²⁰	Nashville, TN	1992–1994	16
Jansen et al ²³	San Diego, CA	1993–1994	1
Nennig et al ²⁴	San Francisco, CA	1994–1995	12
Strebel et al ²⁵	Minneapolis/St Paul, MN	1995–1996	13
Birkebaek et al ²⁶	Denmark	1995–1997	17
Vincent et al ²⁷	Korea	1997–1998	7

Significant immunoglobulin A or G antibody-titer rise or high titer to PT or culture or PCR positive.

B pertussis FIM.^{1,13} It is likely that human infection with *Bordetella bronchiseptica* and perhaps *Bordetella holmesii* also elicit similar antibody responses. In addition, it is clear that other infectious agents have proteins that, with infection, result in antibodies to FHA.²⁷ Therefore, the studies shown in Table 1 in which FHA, PRN, and FIM antibodies contributed to the percentage of positives overestimate *B pertussis* infections.

In contrast with the above, antibody to PT is *B pertussis* specific.¹ Therefore, the use of PT alone in ELISA-antibody studies gives a better indication of the rate of prolonged cough illnesses that are caused by *B pertussis* infection.

In Table 2 the data from the 7 studies done at nonoutbreak periods in which antibody to only PT was determined are presented. The rate in these 7 analyses varied from 1% to 17% (median: 13%). The 1% value in the San Diego study is difficult to explain, because there was a high rate of positives noted by FIM analysis.²³ It seems most likely to me that the low percentage of PT antibody was an artifact caused by the care of the sera before assay.^{17,31}

The data in Table 2 suggest that ~13% of prolonged cough illnesses in adolescents and adults are a result of *B pertussis* infection. However, because all persons infected with *B pertussis* do not have an antibody response to PT, this percentage is an underestimate. In children, as many as 24% of those with culture-proven pertussis do not have a PT-antibody response.¹³ In adults, the percentage is ~10% (ref 26; unpublished data).

It is important to note that all 13 studies of adolescents and adults with prolonged cough illnesses have found evidence of *B pertussis* infection. These studies have been conducted in 6 countries and 7 geographic areas of the United States over a 16-year period. These data suggest that *B pertussis* infection in adolescents and adults is endemic and independent of the cyclic pattern observed with reported pertussis.

Rate of *B pertussis* Infections in Adolescents and Adults

There have been 5 studies performed in the United States in which data on rates of *B pertussis* infection have been presented^{32–36} (Table 3). In all of these studies the rates have been determined by the demonstration of significant ELISA antibody-titer rises in sera collected from the same individuals over an extended time period. In 4 of the studies the sera were collected for other purposes, but in 1 study the sera were collected from controls in an adolescent

and adult acellular pertussis vaccine efficacy trial (APERT). As can be seen in Table 3, the rates vary from ~1% to 8% (median: 2.2%).

Rate of Cough Illnesses (*Pertussis*) Caused by *B pertussis* Infection

Attempts to determine the annual rate of pertussis resulting from *B pertussis* infection were made in 4 of the cough-illness studies presented in Table 1 and 2 of the studies presented in Table 3.^{3,19,24,25,34,36} These data are presented in Table 4. As noted, the estimates vary by almost 10-fold. The relatively low rates noted in the Los Angeles, San Francisco, and Germany studies may be due to an overestimate of the whole population.^{3,19,24} In contrast, the study by Strebel et al²⁵ was specifically designed to obtain rate data, and the APERT data³⁶ were obtained from controls in the vaccine efficacy trial. They were a defined population that had been monitored closely.

The Cleveland, OH, study with the highest rate involved 100 adults who were monitored for respiratory illness and had serum assayed for PT antibody every 4 months for 3 years.³⁴ The rate of 1500 per 100 000 assumes that all the respiratory illnesses in the specific time periods were a result of *B pertussis* infection.

The data from the APERT, Minneapolis/St Paul, and Cleveland studies indicate a total yearly adolescent and adult pertussis burden of between ~ 800 000 and 3.3 million in the United States.^{25,34,36}

DISCUSSION

Today there is a preoccupation with the resurgence of pertussis without a clear understanding of the epidemiology of *B pertussis* infection.^{1,37} One problem in this regard has been the opinion that

TABLE 3. Rate of *B pertussis* Infection in Adolescents and Adults

Source	Location	Years	Annual Rate, %
Deville et al ³²	Los Angeles, CA	1984–1989	8
Cromer et al ³³	Columbus, OH	1985–1990	~1
Hodder et al ³⁴	Cleveland, OH	1989–1992	3
Wright et al ²⁰	Nashville, TN	1992–1994	2.2
Ward et al*	8 US cities	1997–1999	1.3

Infections were determined by the demonstration of a significant serum antibody-titer rise to PT in successive serum samples.

* J.I. Ward, MD, J.D.C., S.J. Chang, MS, S. Partridge, MBA, W. Keitel, MD, K. Edwards, MD, M. Lee, PhD, J. Treanor, MD, D.P. Greenberg, MD, S. Barenkamp, MD, D. Bernstein, MD, R. Edelman, MD, R. Rabinovich, PhD, on behalf of the APERT study group, unpublished data, March 2005.

TABLE 4. Rate of *B pertussis* Infections in Adolescents and Adults

Source	Location	Years	Annual Rate, per 100 000 Population
Strebel et al ²⁵	Minneapolis/St Paul, MN	1995–1996	500
Ward ³⁶	8 US cities	1997–1999	370
Hodder et al ³⁴	Cleveland, OH	1989–1992	1500
Mink et al ³	Los Angeles, CA	1986–1989	69
Schmitt-Grohé et al ¹⁹	Germany	1992–1994	133
Nennig et al ²⁴	San Francisco, CA	1994–1995	176

B pertussis infection diagnosed by culture, PCR, or serologic study.

immunity after *B pertussis* infection is lifelong, whereas that after immunization is relatively short-lived.^{1,2} This has led to the prevalent idea today that the increase in the observation of pertussis in adolescents and adults is because of waning vaccine-induced immunity. Although this is technically true, it is also apparent that waning immunity after infection was a problem in the prevaccine era in the United States and more recently in countries such as Germany, where immunization was infrequent until the very recent present.^{1,2,19,21,38}

Data presently exist that suggest that vaccine-induced immunity is actually better than that induced by *B pertussis* infection.^{1,2,38} It is clear that pertussis in adults is not new, and in Germany in the early 1990s when pertussis was still epidemic, symptomatic adults were a common source for infections in young children.^{19,21}

The data presented here suggest that *B pertussis* infection is common and endemic in adolescents and adults. All 9 studies done in the United States, New South Wales, Denmark, and Korea conducted at non-outbreak times found cases in adolescents and adults.^{3,17,18,20,23–27} Recent serologic data in adults in 8 geographic regions of the United States note the prevalence of antibodies to 4 *B pertussis* proteins (PT, FHA, PRN, and FIM).³⁹ There are few differences by either geographic area or age. These data suggest that adults in Los Angeles have the same susceptibility possibilities as those in the 7 other regions studied. Similarly, 30-year-olds have the same potential risk of infection as all other adult age groups.

Household-contact studies have indicated that infection without illness is common.^{40,41} These data suggest that frequent exposures maintain antibody levels in many persons at protective levels. Illness only occurs when antibody values have fallen below critical values, and then exposure occurs.

The results of this study as well as a careful literature review lead me to suggest the following conclusions:

1. The epidemiology of reported pertussis is different from the epidemiology of *B pertussis* infection.
2. The modest increase in reported pertussis in the United States since 1984 is mainly a result of an increased awareness of *B pertussis* illness and also the use of many vaccines that were less efficacious than DTP vaccines of the past.
3. *B pertussis* infections in adolescents and adults are common and endemic.
4. Immunity after infection or vaccination is not long-lasting.

5. The outcome of an exposure depends on the time since vaccination or a previous infection.
6. Endemic adolescent and adult disease is responsible for the cyclic pattern in unvaccinated children.
7. *B pertussis* circulation cannot be controlled by present immunization programs.
8. Acellular vaccines make adolescent and adult booster immunizations possible.
9. A program with adolescent and adult boosters will decrease the circulation of *B pertussis* in these age groups and could lead to the elimination of the organism from the population.

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Mathis-Lilley B. *New York Magazine*. March 7, 2005

Noted by JFL, MD

The Epidemiology of Pertussis: A Comparison of the Epidemiology of the Disease Pertussis With the Epidemiology of *Bordetella pertussis* Infection

James D. Cherry

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