Pertussis

The post-COVID-19 resurgence in Australia

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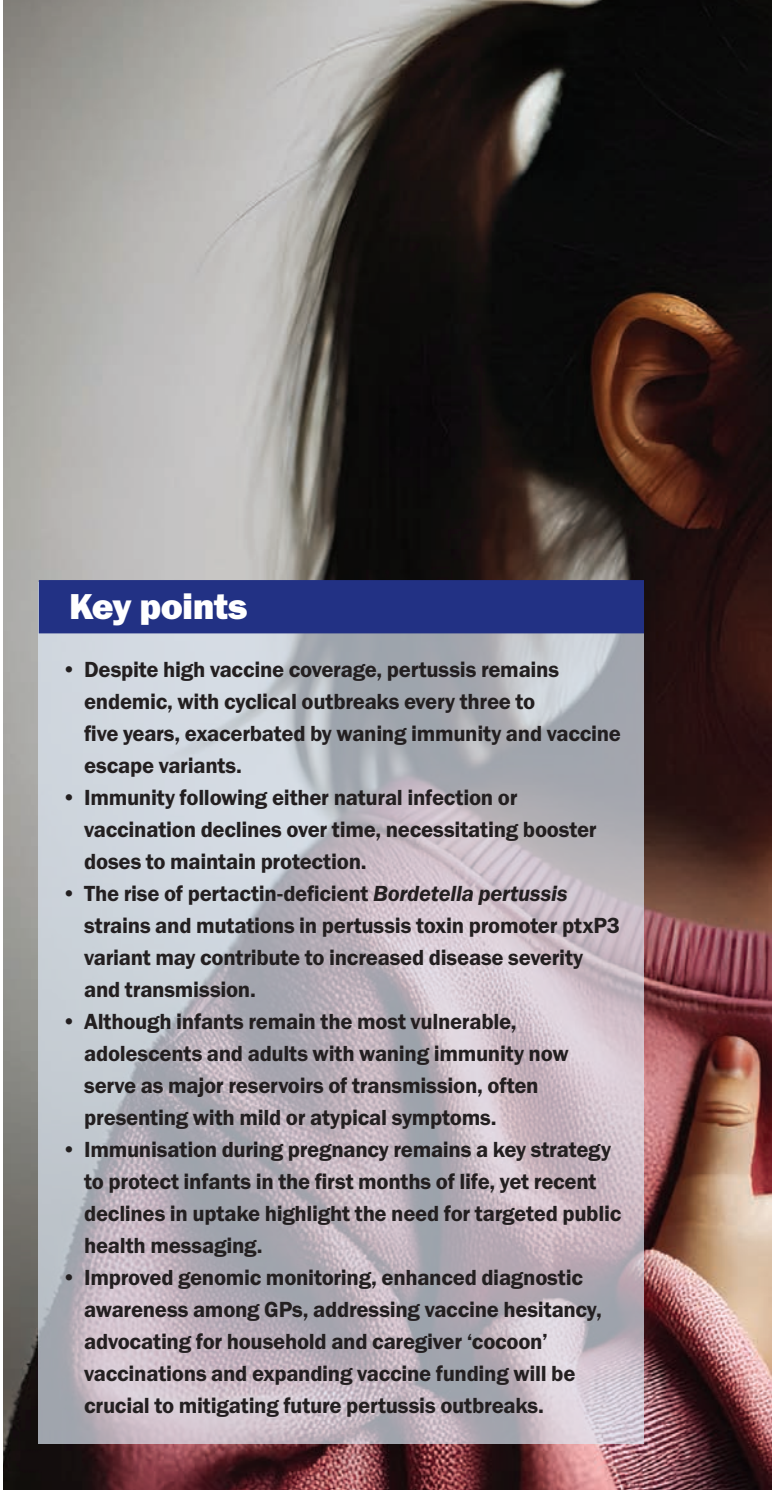
Despite high vaccination coverage, pertussis remains endemic in Australia, with cyclical outbreaks driven by waning immunity and vaccine escape variants. The post-COVID-19 resurgence highlights the need for enhanced surveillance, improved vaccination strategies and strengthened clinical awareness to mitigate transmission and protect vulnerable populations.



Key points

- **Despite high vaccine coverage, pertussis remains endemic, with cyclical outbreaks every three to five years, exacerbated by waning immunity and vaccine escape variants.**
- **Immunity following either natural infection or vaccination declines over time, necessitating booster doses to maintain protection.**
- **The rise of pertactin-deficient *Bordetella pertussis* strains and mutations in pertussis toxin promoter ptxP3 variant may contribute to increased disease severity and transmission.**
- **Although infants remain the most vulnerable, adolescents and adults with waning immunity now serve as major reservoirs of transmission, often presenting with mild or atypical symptoms.**
- **Immunisation during pregnancy remains a key strategy to protect infants in the first months of life, yet recent declines in uptake highlight the need for targeted public health messaging.**
- **Improved genomic monitoring, enhanced diagnostic awareness among GPs, addressing vaccine hesitancy, advocating for household and caregiver 'cocoon' vaccinations and expanding vaccine funding will be crucial to mitigating future pertussis outbreaks.**

Pertussis, or whooping cough, remains a significant public health concern despite widespread vaccination. Caused primarily by the bacterium *Bordetella pertussis*, this highly infectious acute respiratory disease has a unique clinical presentation characterised by severe paroxysmal coughing and a prolonged course of illness. It poses a particularly severe threat to incompletely vaccinated infants (who have received fewer than three doses), in whom complications can lead to hospitalisation or death.

Australia has achieved high vaccine coverage through public health measures, including the National Immunisation Program (NIP), contributing to a marked reduction in pertussis incidence and mortality over decades. However, the resurgence of cases following the coronavirus disease 2019 (COVID-19) pandemic raises important questions about waning immunity, vaccine effectiveness and shifting epidemiology. The stark decline in pertussis notifications observed during the pandemic because of public health actions (e.g. mask wearing and physical distancing) has been followed by a profound rebound in cases as these measures have eased.

This resurgence underscores the need for renewed attention among healthcare professionals to the clinical features, diagnosis, management and prevention of pertussis. As frontline clinicians, GPs play a pivotal role in recognising and managing pertussis cases, particularly given the atypical presentations in adolescents and adults, who are often sources of infection for infants.

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This article provides a review of pertussis with a focus on the Australian context and recent epidemiological trends. Key topics include the microbiology, pathogenesis, clinical manifestations and diagnosis of pertussis, along with therapeutic approaches and preventive strategies including vaccination. The objective is to equip GPs and other healthcare professionals with the knowledge needed to manage pertussis effectively in this evolving landscape.

Microbiology of *Bordetella pertussis* and related species

First isolated in 1906 by Bordet and Gengou, *B. pertussis* is a fastidious, motile Gram-negative, pleomorphic aerobic coccobacillus that exclusively infects humans (although it can be experimentally infected in baboons and mice).[1,2] It is the primary causative agent of pertussis and is highly specialised for colonising the ciliated epithelium of the respiratory tract.[3,4] Of the 16 described *Bordetella* species, a further three are considered medically important – *Bordetella parapertussis, Bordetella bronchiseptica* and *Bordetella holmesii* – each with similar signs and symptoms.

- *B. pertussis* is the quintessential aetiological agent causing the most severe forms of whooping cough. Humans are the only known reservoirs.[5]
- *B. parapertussis* can cause the classic whooping cough-like illness, although it is considered less severe than infection caused by *B. pertussis*. Its ability to circulate within populations and cause sporadic outbreaks has been observed, with children often being the most affected.[6-8]
- *B. bronchiseptica* infection produces respiratory illness in a wide range of mammals, including dogs (kennel cough). Most human cases are seen in immunocompromised hosts.[9-11]
- *B. holmesii* has also been associated with infections in immunocompromised individuals; however, recent Australian data suggest it may be more widespread, cocirculating with *B. pertussis* and accounting for up to 16.8% of pertussis cases.[12]
- There are many other *Bordetella* species and some isolates await further speciation and description.[13]

Codetection with other respiratory pathogens (e.g. respiratory syncytial virus, adenovirus, human rhinovirus) does occur.[14,15]

Genetic and phenotypic features

The four medically important *Bordetella* species (*B. pertussis, B. parapertussis, B. bronchiseptica* and *B. holmesii*) exhibit both genetic similarities and significant differences in pathogenicity, host adaptation, virulence factor expression and epidemiological impact. Although all four species can cause respiratory infections, their distinct genomic structures and phenotypic traits shape their disease presentation and public health significance.[16] For example, only *B. pertussis* expresses pertussis toxin (PT), with the other species causing a milder disease course in most cases.[17]

The genome of *B. pertussis* is smaller compared with other *Bordetella* species, with evidence of significant gene loss. These adaptations reflect its specialisation for human hosts.[18,19] The bacterium's strict requirements for growth in vitro, requiring protective substances such as charcoal or blood in culture, are another testament to its niche specificity.[20]

B. pertussis is distinguished from other *Bordetella* species by specific genetic elements, such as insertion sequences, which are used as targets in diagnostic nucleic acid amplification testing, most commonly polymerase chain reaction (PCR) assays.[21,22]

Epidemiology

Pertussis remains a global health concern, with cases reported across all continents. Although vaccination programs have significantly reduced the disease burden, pertussis continues to cause considerable morbidity and mortality. The historically cited basic reproduction

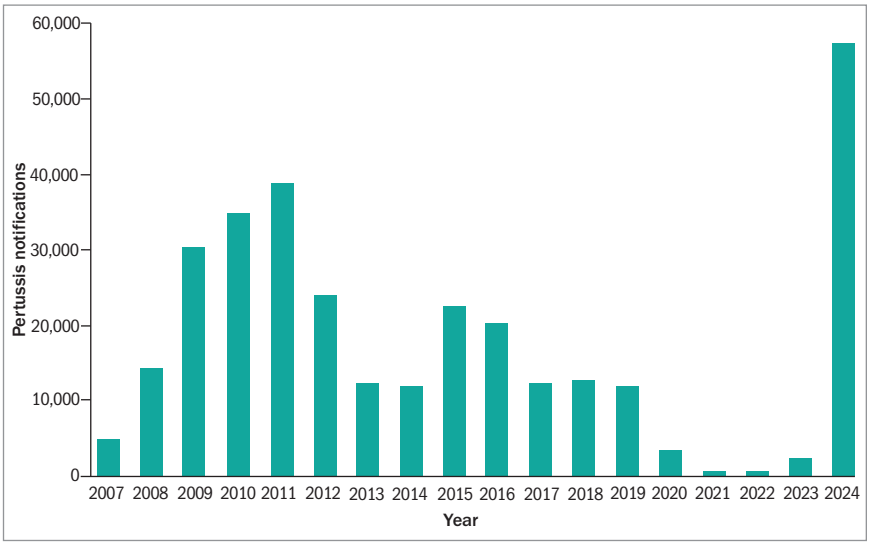

Figure 1. Yearly pertussis notifications to the Australian National Notifiable Disease Surveillance System.

Adapted from data published by the Australian Government Department of Health and Aged Care: National Notifiable Diseases Surveillance System.[33]

number (R_0; the average expected number of infections caused by one case in a susceptible population) for pertussis of 12 to 17, derived from epidemiological data collected between 1908 and 1917 in the United States and 1944 to 1979 in England and Wales, may not accurately reflect current transmission dynamics because of changes in population structure, vaccination coverage and social behaviours.[23,24] More recent analyses indicate that R_0 values for pertussis may be lower, with estimates ranging from 5.5 in Europe to between 9 and 12 in the United States and 10 in Australia.[25-27]

Australian context

Australia has achieved commendable pertussis vaccination coverage in infant, child and adolescent target groups through the NIP. Despite this, pertussis remains endemic throughout Australia, with periodic outbreaks occurring every three to five years.[28,29]

The COVID-19 pandemic had a profound impact on pertussis epidemiology (Figure 1). Public health measures such as physical distancing, mask use and reduced international travel led to an unprecedented decline in reported pertussis cases in Australia and globally.[30] However, with the relaxation of these measures, a resurgence of pertussis has been observed.[31,32] In 2024, Australia recorded 57,146 pertussis notifications to the National Notifiable Disease Surveillance System, more than 47% higher than the previous peak of 38,748 in 2011.[33] For the first three months of 2025 there were 9577 notifications, more than three-fold higher compared with January to March 2024.

Age and risk group dynamics

In Australia, the age distribution of pertussis cases has shifted over time (Figure 2). Historically, the incidence of pertussis was the highest in infants and children younger than 5 years of age but, since the late 1990s, this has shifted toward adolescents. However, infants lacking full immunisation remain at the highest risk for severe disease and complications, including hospitalisation and mortality.[2,34-36] Data from 2013–18 confirm this, showing infants younger than 2 months of age had the highest rates of pertussis-related hospitalisations (143.2 per 100,000), closely followed by those aged 2 to 3 months (137.8 per 100,000).[37] During this period, nine deaths had a causal link with pertussis, of which six were in infants younger than 1 year of age. Siblings, adolescents and adults often serve as reservoirs of infection because of waning immunity, and often present with atypical or mild symptoms.[38,39] These groups play a crucial role in transmitting the disease to vulnerable infants.[40]

Transmission and cyclical patterns

Despite recent advances in animal modelling, much uncertainty remains regarding the mechanism of pertussis transmission.[41-44] Pertussis is thought to be transmitted from an infected patient through aerosolised droplets by direct inhalation and via indirect self-inoculation from surrounding contaminated surfaces.[41,45] Transmission rates of more than 80% have been observed among susceptible household contacts.[46] Bacterium shedding via respiratory secretions is most infectious during the early symptomatic phase, whereas transmission is most efficient once coughing has developed.[47] The cyclical nature of pertussis outbreaks, with inter-epidemic periods of three to five years, persists despite widespread vaccination.[28,29,44,48,49]

Waning immunity and vaccine escape variants

One of the major challenges in pertussis control is waning immunity following either natural infection or vaccination, with ongoing debate regarding the underlying causes and varying impact seen in infants, children, adolescents and adults.[44] Although *B. pertussis* infection induces a robust T helper (Th)1/Th17 immune response, evidence suggests that infection-derived immunity wanes within four to 20 years, whereas vaccine-induced immunity – particularly from acellular pertussis (aP) vaccines – wanes within four to 12 years, necessitating booster doses to maintain protection.[42,50,51] Studies indicate that these aP vaccines induce a primarily Th2-skewed response, which fails to generate strong mucosal immunity, leading to asymptomatic colonisation and continued bacterial transmission despite vaccination.[52] Although whole-cell inactivated pertussis (wP) vaccines generate a more durable Th1/Th17 response, their high reactogenicity – local reactions such as injection site pain, swelling and redness through to systemic reactions including fever, persistent crying and convulsions – led to widespread disapproval and subsequent replacement with aP vaccines in most high-income countries including Australia from the mid-1990s.

The genomic evolution of *B. pertussis* has

resulted in antigenic divergence from vaccine strains, raising concerns about vaccine-driven selection pressure. Key studies have identified an increase in pertactin (PRN)-deficient visolates, which have been increasingly reported in Australia and other countries using aP vaccines, potentially reducing their effectiveness.[27] The loss of PRN, an autotransporter protein, is hypothesised to confer a selective advantage in aP-immunised populations, allowing PRN-deficient strains to persist and spread. Furthermore, mutations in the pertussis toxin promoter ptxP3 variant have been associated with higher toxin production, potentially contributing to increased disease severity.[53,54]

Demographic disparities

Indigenous Australians face unique challenges regarding pertussis incidence and vaccination coverage. Historically, notification and hospitalisation rates for pertussis in Indigenous children have been three to eight times higher than those in non-Indigenous children across all age groups younger than 5 years of age.[37]

Vaccination coverage among Indigenous children has shown both progress and areas of concern. In 2022, the immunisation rate for Indigenous 1-year-olds was 91.1%, compared with 94.0% for non-Indigenous children. For 2-year-olds, the coverage was 89.1% among Indigenous children versus 92.2% among their non-Indigenous counterparts. However, at 5 years of age, Indigenous children had a higher coverage rate of 96.1%, surpassing the 94.1% observed in non-Indigenous children.[55]

The adolescent dose is seen as a crucial tool at reducing pertussis transmission to vulnerable infants. Despite its importance, the overall national coverage rate for adolescents turning 13 years of age who had received a booster dose declined from 76.2% in 2022 to 73.9% in 2023.[56]

Maternal vaccination plays a crucial role in protecting infants, who are at the highest risk for severe pertussis.[57] However, vaccination rates among pregnant women have declined in Queensland, for instance, with vaccination rates dropping from 77.2% in 2020 to 70.7% in 2023.[58]

Pathogenesis and pathophysiology

B. pertussis is highly adapted to the human respiratory tract. Infection is typically characterised by a prolonged illness, starting with an incubation period and progressing through three distinct clinical stages:

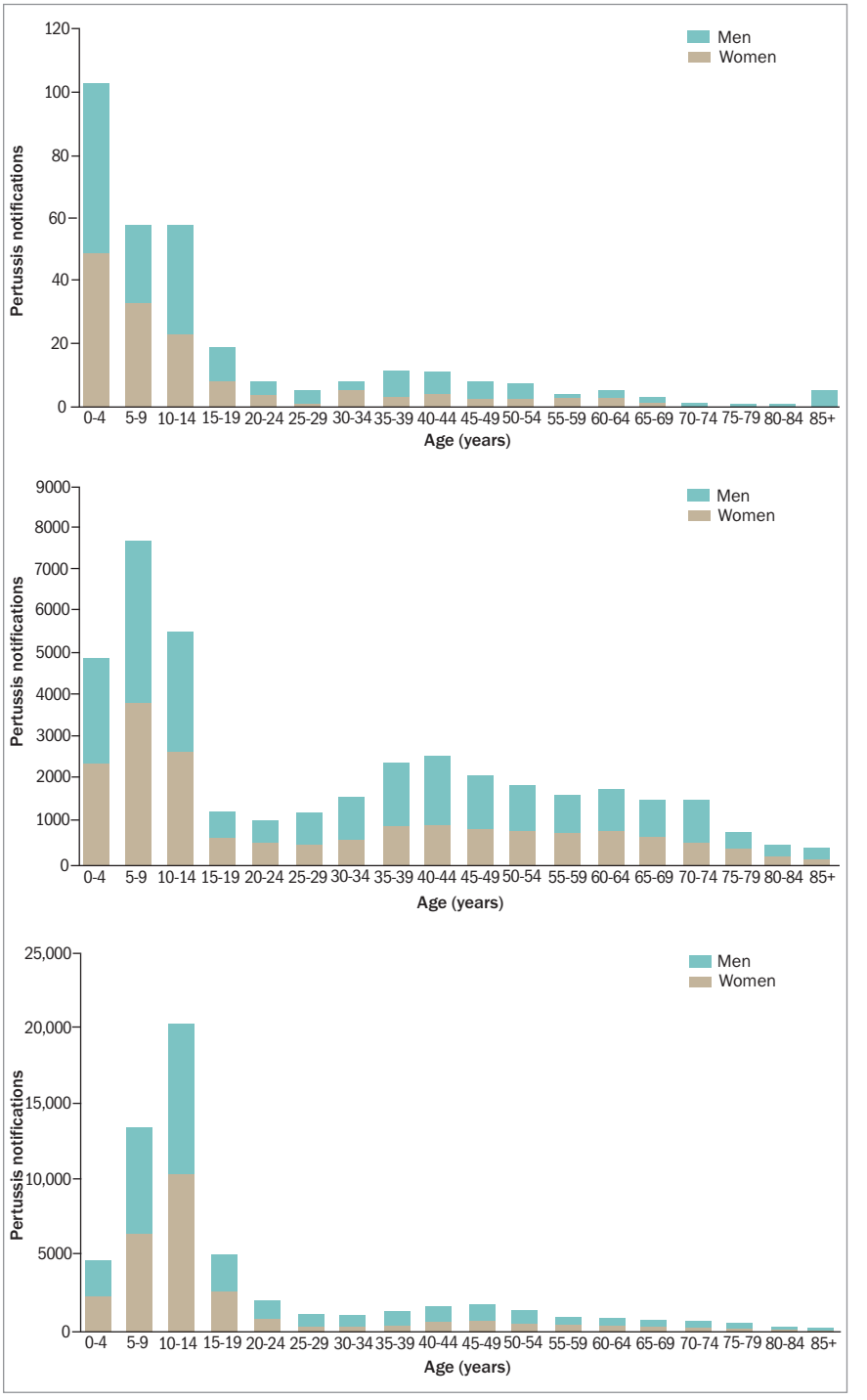

Figure 2. Pertussis notifications by age and sex in Australia – 1991 (top), 2011 (middle), 2024 (bottom).

Adapted from data published by the Australian Government Department of Health and Aged Care: National Notifiable Diseases Surveillance System.[33]

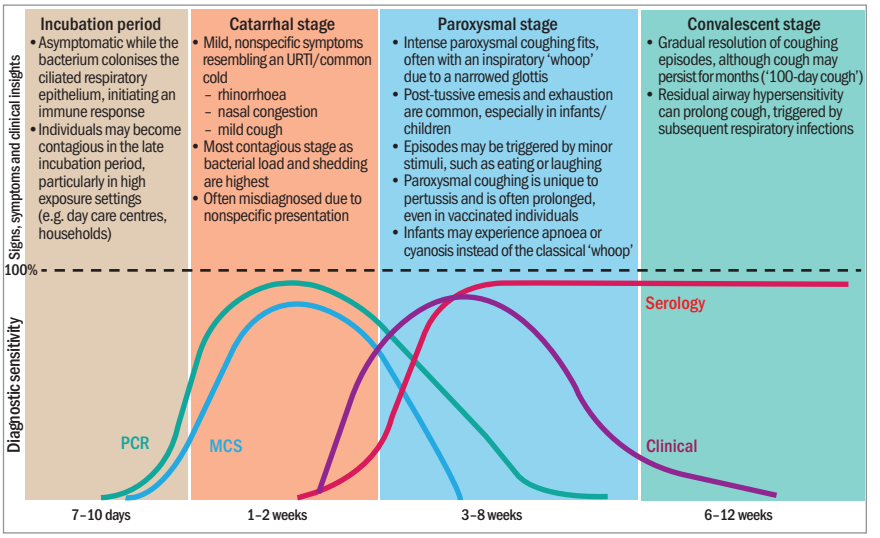

Figure 3. Classic stages of pertussis infection including the incubation period, with relative diagnostic sensitivities of laboratory tests and clinical diagnosis during the various stages.

Abbreviations: MCS = microscopy, culture and sensitivity testing; PCR = polymerase chain reaction; URTI = upper respiratory tract infection.

Adapted from Fry NK, et al. J Med Microbiol 2021; 70(10): 001442.[59]

catarrhal, paroxysmal and convalescent (Figure 3).[59] Unlike many respiratory infections, pertussis is largely noninflammatory and does not typically induce fever, a feature that may help distinguish it from viral upper respiratory tract infections.[2,60]

The pathogenicity of the medically important *Bordetella* species is mainly attributed to the arsenal of virulence factors that facilitate attachment, immune evasion and damage to the host's respiratory epithelium (Table 1).[61-63]

Attachment and colonisation

B. pertussis attaches to ciliated epithelial cells lining the respiratory tract. Adhesins such as PT, filamentous haemagglutinin (FHA), and fimbriae 2/3 (FIM2/3) facilitate this process. PT, an exotoxin unique to *B. pertussis*, also plays a crucial role in disease severity, particularly in infants.[64,65] Antibodies to FIM protect against colonisation, and vaccines containing FIM2/3 antigens are associated with enhanced immunogenicity.[66,67]

Immune evasion

After attachment, *Bordetella* species deploy virulence factors to impair host defences. For *B. pertussis*, PT modifies G-protein signalling in host cells, leading to a cascade of effects, including lymphocytosis, impaired immune cell migration and altered cytokine responses.[17] Adenylate cyclase toxin intoxicates neutrophils and macrophages by increasing intracellular cyclic adenosine monophosphate, disrupting their immune function.[2] This helps *B. pertussis*, *B. parapertussis* and *B. bronchiseptica* evade immune clearance. PRN also confers resistance to neutrophil-mediated clearance.[68] PRN-specific antibodies are highly protective, but the emergence of PRN-deficient strains caused by aP vaccine-driven selection pressure and antigenic variations poses a challenge for vaccine efficacy.[27,68]

Local and systemic effects

Tracheal cytotoxin damages ciliated epithelial cells, impairing mucociliary clearance.[61] This contributes to, but does not cause, the characteristic paroxysmal cough. PT induces lymphocytosis by inhibiting lymphocyte migration and function, a hallmark of pertussis in infants and young children.[17,64] In severe cases, leucocytosis may obstruct pulmonary vasculature, leading to pulmonary hypertension.[70]

Paroxysmal cough and 'whoop'

The hallmark paroxysmal cough arises from persistent stimulation of sensory nerves in the respiratory tract, likely mediated by prolonged exposure to bacterial toxins and inflammatory mediators including bradykinin.[71] The characteristic whoop occurs during the inspiratory phase following a prolonged coughing spell and results from forced inhalation through narrowed airways.

Complications in severe cases

Infants are particularly vulnerable to complications such as apnoea, cyanosis and secondary bacterial pneumonia. Severe pulmonary hypertension, often mediated by extreme leucocytosis, can lead to cardiac failure in young infants.[70]

Clinical features

Classic stages

B. pertussis infection follows a distinct clinical progression (Figure 3). The illness typically has a seven- to 10-day incubation period (range: six to 20 days). However, household exposure studies have shown that 22% of secondary cases can occur more than 28 days after symptom onset in the index case, especially when the infector is the father or sibling.[72,73]

Clinical manifestations usually last six to 12 weeks, although the timeline and severity may vary by age, vaccination status and immune response.[2]

Atypical presentations

Deviations from the classic pertussis presentation are seen throughout the age spectrum, especially when caused by *B. parapertussis*, *B. bronchiseptica* and *B. holmesii*.[74]

Infants (younger than 6 months of age)

Infants often present with atypical features compared with older children, making early diagnosis challenging. Rather than the characteristic paroxysmal cough with a whoop, infants, especially those younger than 3 months of age, often exhibit apnoea, cyanosis and feeding difficulties as their primary symptoms.[2,75] These manifestations occur because of immature respiratory control mechanisms and increased susceptibility to PT-induced immune dysregulation. In some cases, the absence of an overt cough can further delay recognition of the disease.

Table 1. Pertussis virulence factor expression for four *Bordetella* species

Virulence factor	*B. pertussis*	*B. parapertussis*	*B. bronchiseptica*	*B. holmesii*
PT	✓	×	×	×
FHA	✓	✓	✓	✓
FIM	✓	✓	✓	×
ACT	✓	✓	✓	×
PRN	✓*	✓	✓	×
TCT	✓	✓	✓	?
T6SS	×	×	✓	?

Abbreviations: ACT = adenylate cyclase toxin; FHA = filamentous haemagglutinin; FIM 2/3 = fimbriae 2 and 3; PRN = pertactin; PT = pertussis toxin; TCT = tracheal cytotoxin; T6SS = type VI secretion system.
* But vaccine-driven PRN-deficit strains emerging.
Key: ? = uncertain.
Adapted from Belcher T, et al. Virulence 2021; 12: 2608-2632[61] and Linz B, et al. BMC Genomics 2016; 17: 767.[63]

Infants are at the highest risk of severe complications, including pneumonia, seizures, encephalopathy and respiratory failure, often requiring hospitalisation.[76] The high mortality rate in this age group underscores the importance of early diagnosis, prompt antibiotic treatment and maternal immunisation during pregnancy to provide passive immunity.

Adolescents and adults

Adolescents and adults with pertussis often develop a prolonged cough rather than the classic whooping cough.[77] The illness may present as a persistent dry cough lasting weeks to months, often leading to misdiagnosis as post-viral cough, asthma exacerbation or gastro-oesophageal reflux disease. Although the paroxysmal coughing episodes characteristic of pertussis may still occur, the inspiratory whoop is typically absent in these age groups.

Because of waning immunity, older children, adolescents and adults serve as reservoirs for transmission, particularly to vulnerable populations such as infants and immunocompromised individuals.[38,39] Although complications are generally less severe than in infants, rib fractures, pneumothorax, subconjunctival haemorrhage, hernias and urinary incontinence can occur because of the forceful nature of paroxysmal coughing.[2,47] The lack of awareness regarding pertussis in this age group can contribute to delayed presentation and diagnosis with increased transmission.

Vaccinated individuals

Individuals who have been previously vaccinated against pertussis can still become infected and transmit pertussis, often experiencing modified or milder disease.[5] Although the classic paroxysmal cough and inspiratory whoop may still be present, the duration and intensity of symptoms tend to be less severe than in unvaccinated individuals.[78] Post-tussive vomiting and complications occur less frequently.

Complications

Pertussis can lead to a range of complications, particularly in infants and in older people.

Respiratory complications

Pneumonia is a common and serious respiratory complication, caused by *B. pertussis* itself or secondary infection with other respiratory pathogens including *Streptococcus pneumoniae, Haemophilus influenzae*, respiratory syncytial virus and influenza A virus.[20,79] In infants, pneumonia is a major contributor to pertussis-related morbidity and mortality, often requiring hospitalisation and respiratory support.

Another significant respiratory complication is atelectasis, which results from airway obstruction caused by excessive mucus production and impaired mucociliary clearance.[2] In severe cases, respiratory distress and respiratory failure can develop, particularly in young infants who may require mechanical ventilation. Prolonged paroxysmal coughing episodes can also lead to pneumothorax or pneumomediastinum, particularly in older children and adults, because of increased intrathoracic pressure during coughing spasms.[80]

Neurological complications

Neurological complications of pertussis occur primarily in infants and are often secondary to hypoxaemia caused by prolonged coughing episodes or apnoea.[4] Seizures are a common manifestation, occurring in about 4% of hospitalised infants with severe disease. Hypoxic encephalopathy and cerebral haemorrhage, both resulting from intense coughing bouts, can cause long-term neurodevelopmental impairments, including cognitive and motor deficits.[81]

Cardiovascular complications

Although cardiovascular complications are rare, they can occur in severe cases of pertussis, particularly in young infants with profound leucocytosis.[70] Severe leucocytosis can lead to pulmonary hypertension, a potentially fatal complication resulting from vascular obstruction by excessive white blood cells.[65] This condition can cause right heart failure and circulatory collapse, necessitating aggressive interventions such as exchange transfusion.

Older children and adults with pre-existing cardiovascular disease may experience arrhythmias or exacerbation of underlying heart conditions because of the physiological stress caused by prolonged coughing fits and oxygen desaturation.

Diagnosis

Early diagnosis of pertussis is crucial to mitigate disease transmission, initiate timely treatment and prevent complications, particularly in high-risk populations such as infants and in older people. However, pertussis can be challenging to diagnose because of its nonspecific early signs and symptoms,

such as the common cold, and atypical presentations in vaccinated individuals. The establishment of pertussis surveillance and laboratory case definitions in Australia (Box 1) has provided a standardised framework for case identification, improving diagnostic consistency, outbreak detection and epidemiological tracking across diverse healthcare settings.[82,83]

Clinical diagnosis

High index of suspicion

A history of a coughing illness lasting two weeks or more, paroxysmal coughing spells, post-tussive vomiting or retching or inspiratory whoop strongly supports the clinical diagnosis of pertussis. Furthermore, recent contact with a confirmed or suspected pertussis case should be ascertained.

Limitations of clinical diagnosis

The reliance on clinical diagnosis alone, particularly in its early stages, can lead to delayed recognition, misdiagnosis and missed opportunities for timely intervention. In infants, adolescents and adults, classic features may be absent or muted, complicating clinical recognition. Furthermore, differentiation from other respiratory illnesses such as viral infections, asthma or bronchitis requires additional diagnostic investigation.

Laboratory diagnosis

The duration of signs and symptoms at the time of presentation can help determine the optimal laboratory test (or combination) for diagnosis (Figure 3 and Box 2).[59,84,85]

Collection of specimens

Bordetella species can be isolated from posterior nasopharyngeal swabs, nasopharyngeal aspirates, throat swabs or induced sputa.[86] Nasopharyngeal aspirates have shown a 15% gain in the isolation rate compared with nasopharyngeal swabs in infants.[87] Given *Bordetella* species predominantly colonise and replicate in the ciliated epithelium of the nasopharynx, throat swabs or anterior nares (nasal)-only swabs are considered suboptimal and are not recommended.[5,84]

1. Australian case definitions for pertussis[82,83]

Confirmed case*
- Laboratory definitive evidence OR
- Laboratory suggestive evidence AND clinical evidence

Probable case*
- A probable case requires clinical evidence AND epidemiological evidence

Laboratory definitive evidence
- Isolation of *Bordetella pertussis* by culture OR
- Detection of *B. pertussis* by NAAT OR
- Seroconversion in paired sera for *B. pertussis* using whole-cell or specific *B. pertussis* antigen(s) in the absence of recent pertussis vaccination

Laboratory suggestive evidence†
- Significant change (increase or decrease) in antibody levels (IgG, IgA) to whole-cell or specific *B. pertussis* antigen(s) OR
- Single high IgG and/or IgA titre to PT OR
- Single high IgA titre to whole-cell *B. pertussis* antigen

Clinical evidence†
- A coughing illness lasting ≥2 weeks OR
- Paroxysms of cough OR
- Inspiratory whoop OR
- Post-tussive vomiting

Epidemiological evidence
- Contact between two people involving a plausible mode of transmission at a time when:
  - one of them is likely to be infectious (from the catarrhal stage, about one week before to three weeks after the onset of cough) AND
  - the other has an illness that starts within 6 to 20 days after this contact AND
  - at least one case in the chain of epidemiologically linked cases (which may involve many cases) is a confirmed case with either laboratory definitive OR laboratory suggestive evidence

Special considerations
- Although *B. pertussis* PCR is regarded as definitive evidence of infection, many laboratories cannot reliably distinguish *B. pertussis* from *B. holmesii* unless dual targets are used; this practice is recommended
- Whole-cell *B. pertussis* IgG is not useful; positive results should not be considered evidence of current infection

Abbreviations: IgA = immunoglobulin A; IgG = immunoglobulin G; NAAT = nucleic acid amplification testing; PCR = polymerase chain reaction; PT = pertussis toxin.

*Both confirmed and probable cases must be reported to the local public health authority; †In the absence of recent vaccination. Adapted from the Communicable Diseases Network Australia (CDNA) Pertussis – Surveillance Case Definition and the Public Health Laboratory Network Whooping Cough (Bordetella pertussis) – Laboratory Case Definition.[82,83]

Polymerase chain reaction

Nucleic acid amplification testing by PCR is the preferred method for pertussis diagnosis during the first three to four weeks of illness.[85,88] It detects specific DNA sequences for *Bordetella* species. Advantages include high sensitivity and specificity, rapid results and continued effectiveness even after antimicrobial initiation (although sensitivity may decrease).[89] A potential limitation includes *B. pertussis* false-positives caused by cross-reactivity with other *Bordetella* species. For example, gene targets IS481 and IS1002 used for detecting *B. pertussis* show cross-reactivity with *B. holmesii* and certain *B. bronchiseptica* strains, and *B. parapertussis*, respectively.[21,90] Reduced sensitivity is experienced later in the disease course. Furthermore, polyester, rayon or flocked nylon swabs are optimal for posterior nasopharyngeal collection, whereas cotton-tipped and calcium alginate swabs must not be used because they inhibit PCR assays.[22,85]

Microscopy, culture and sensitivity

Historically, culturing *Bordetella* species was considered the gold standard for diagnosis, offering 100% specificity for definitive confirmation and enabling antibiotic susceptibility testing; however, its routine use is limited because of several practical challenges.[91-93] These include: a time-consuming process requiring seven to 12 days for results; the need for specialised media such as Regan-Lowe or Bordet-Gengou agar; difficulties in culturing *B. holmesii* because of the presence of cephalexin in selective media; and its low sensitivity.[89] A flexible, flocked nylon swab with charcoal-containing media is the preferred posterior nasopharyngeal specimen to maximise yield.

Serology

Routine serological testing for the diagnosis of pertussis infection is not recommended as previous infection (including asymptomatic) and recent vaccination (past 12 months) can lead to inaccurate results.[88] However, serological testing may be useful when the optimal timing for PCR and/or microscopy, culture and sensitivity testing has elapsed. Quantifying immunoglobulin G (IgG) antibodies to PT is the most used assay, and titres usually peak two to eight weeks after cough onset.[22] Recent Australian data demonstrate that quantifying immunoglobulin A (IgA) antibodies to PT appears to contribute little diagnostic value to an accurate PT IgG assay.[94] Advantages of serological testing include high sensitivity and specificity, rapid results and effectiveness even after antimicrobial initiation (although sensitivity may decrease).[89] Important limitations include interpretation challenges because of prior vaccination and limited use in infants and young children.[22]

2. Laboratory tests for diagnosing pertussis infection[84,85]

Nucleic acid amplification testing

- Often referred to as PCR testing
- Widely considered best practice for diagnosing pertussis
- More sensitive (73 to 100% depending on specific assay) than culture testing
- Optimal sensitivity during the first three weeks, although infected people can remain positive for 5 weeks or longer
- High specificity of 88 to 97%
- NAAT testing after five days of appropriate antimicrobials is unlikely to be of benefit and is generally not recommended
- Posterior nasopharyngeal aspirates, or swabs with polyester-, rayon- or nylon-flocked tips, should be used; cotton-tipped or calcium alginate are not suitable as they inhibit PCR assays; transport medium are usually not required; sputum samples may also be utilised
- Throat swabs alone are not recommended because of poor sensitivity
- Test results are usually available within 1 to 2 days
- Some PCR assays can detect several *Bordetella* species (e.g. *Bordetella pertussis*, *B. parapertussis*, *B. bronchiseptica*)

Culture

- The only 100% specific method for species identification; PPV is 100%; NPV is variable, but highest in young, unvaccinated children early in disease; NPV also low for sporadic cases in adults
- Traditionally considered the gold standard for diagnosing pertussis
- Sensitivity varies widely (20 to 80%), rapidly decreases after cough onset and is highly dependent on specimen quality
- Rarely positive after 2 weeks from catarrhal stage onset, or one week after paroxysmal cough
- Posterior nasopharyngeal aspirates or swabs (as per NAAT requirements above); swabs should be immediately inoculated directly onto specialised pertussis culture media or placed into transport media, according to local laboratory instructions.
- Throat swabs (alone), anterior nasal swabs and sputum are unacceptable specimens
- Prior antimicrobial use decreases sensitivity
- Test results may take 7 to 14 days, especially if extended incubation is required

Serology

- The predominant diagnostic test until recent decades
- Lower sensitivity and specificity; serology may be useful if a clinically compatible illness has been present for more than 14 days
- Paired sera (7 to 10 days apart) preferred
- Not recommended for children <2 years of age because of poor IgA response and problematic venepuncture
- Improved standardisation with the use of purified pertussis antigen assays for PT, FHA and PRN
- Serological interpretation is challenging in those with a history of recent vaccination (up to 2 years)

Abbreviations: FHA = filamentous haemagglutinin; IgA = immunoglobulin A; NAAT = nucleic acid amplification testing; NPV = negative predictive value; PCR = polymerase chain reaction; PPV = positive predictive value; PRN = pertactin; PT = pertussis toxin.

Adapted from the Pertussis CDNA National Guidelines for Public Health Units, and the Best Practices for Use of Polymerase Chain Reaction for Diagnosing Pertussis.[84,85]

Differential diagnosis

Pertussis should be differentiated from other causes of prolonged cough and paroxysmal episodes, including:

- viral respiratory infections such as adenovirus, parainfluenza viruses and respiratory syncytial virus
- *Mycoplasma pneumoniae, Chlamydia pneumoniae* and *Legionella* species
- asthma exacerbation
- gastro-oesophageal reflux disease, which can mimic post-tussive vomiting
- *Chlamydia trachomatis* in neonates presenting with chronic cough without fever.

Utilising precollected nasopharyngeal swabs or nasopharyngeal aspirates, requesting a multiplex respiratory virus pathogen PCR panel (which typically includes influenza A and B, respiratory syncytial virus A and B, coronavirus [seasonal], severe acute respiratory syndrome coronavirus 2, parainfluenza viruses 1, 2, 3, and 4, human rhinovirus, enterovirus, adenovirus and human metapneumovirus), together with *M. pneumoniae, C. pneumoniae* and *Legionella pneumophila* PCR tests, may help to exclude other causes of acute respiratory illness.

Table 2. Recommended antibiotic treatment and prophylaxis* for pertussis by age group[97]

Age group and PBS/TGA status	Macrolides		Nonmacrolide alternative
	Azithromycin (preferred)	Clarithromycin	Trimethoprim + sulfamethoxazole†
TGA pregnancy category	B1‡	B3‡	C‡
PBS funded	RPBS tablets and liquid only	Tablets and liquid	Tablets and liquid
<1 month	10 mg/kg orally, daily for 5 days§	Not recommended	Not recommended
1 to <6 months	10 mg/kg orally, daily for 5 days	7.5 mg/kg orally (max 500 mg), 12-hourly for 7 days	4 + 20 mg/kg orally (max 160 + 800 mg), 12-hourly for 7 days
Children ≥6 months	Day 1: 10 mg/kg orally, daily (max 500 mg)	7.5 mg/kg orally (max 500 mg), 12-hourly for 7 days	4 + 20 mg/kg orally (max 160 + 800 mg), 12-hourly for 7 days
	Days 2 to 5: 5 mg/kg orally, daily (max 250 mg)		
Adults	Day 1: 500 mg orally, daily	500 mg orally, 12-hourly for 7 days	160 + 800 mg orally, 12-hourly for 7 days
	Days 2 to 5: 250 mg orally, daily		

Abbreviations: PBS = Pharmaceutical Benefits Scheme; RPBS = Repatriation Pharmaceutical Benefits Scheme; TGA = Therapeutic Goods Administration.

* Antibiotic prophylaxis in selected contacts of patients with pertussis.

† Avoid if patient has glucose-6-phosphate dehydrogenase (G6PD) deficiency; use with caution if infant is younger than 1 month old, or is premature, ill or jaundiced – may increase risk of haemolysis, bilirubin displacement and kernicterus. Monitor infant for diarrhoea, vomiting, rash and candidiasis; compatible if infant is older than 1 month of age and healthy – monitor infant for diarrhoea, vomiting, rash and candidiasis.

‡ Compatible with breastfeeding: monitor infant for diarrhoea, vomiting, rash and candidiasis.

§ Oral azithromycin increases the risk of infantile hypertrophic pyloric stenosis during the first six weeks of life, with the highest risk during the first two weeks. Administration of antibiotics to neonates should be done in consultation with specialist infectious diseases, neonatal or paediatric services and may require a risk-benefit assessment.

Adapted from Therapeutic Guidelines: Antibiotics.[97]

Additionally, a full blood count may reveal a lymphocytosis, particularly in infants during the early paroxysmal stage.[95]

Diagnostic implications for public health

Notification, reporting and surveillance

Pertussis, both confirmed and probable cases, is a notifiable disease in all Australian states and territories (Box 1 and Box 2).[84] Prompt reporting ensures public health measures, including contact tracing and prophylaxis for exposed individuals, can be implemented. Accurate diagnosis supports epidemiological monitoring and informs vaccination strategies.[88]

Treatment in primary care

The primary objectives of pertussis treatment are to:

- prevent disease transmission, especially to vulnerable populations
- reduce the severity and duration of symptoms if commenced in the catarrhal stage, and
- minimise complications, such as pneumonia and acute respiratory distress syndrome.[96]

Antimicrobial therapy

Early initiation, ideally during the catarrhal stage when bacterial replication and shedding peaks, significantly reduces transmissibility.[96] Antimicrobials are less effective in altering the disease course during the paroxysmal stage but are still recommended to prevent further spread.

Macrolides as first-line agents

Patients infected with *B. pertussis, B. parapertussis* and *B. holmesii* should be treated with a macrolide antibiotic.[2] Azithromycin is preferred because of its shorter course (five days) and better tolerability (Table 2).[97] Clarithromycin is a suitable alternative, administered for seven days. Erythromycin is an effective agent but is associated with significant gastrointestinal side effects, limiting its use and is not normally recommended in Australia. There is no evidence to support the use of roxithromycin for the treatment of pertussis. Administration of antibiotics to neonates should be done in consultation with specialist infectious diseases, neonatal or paediatric services, especially given that macrolides are associated with infantile hypertrophic pyloric stenosis in this age group.[84,98]

Alternative antimicrobials

Trimethoprim-sulfamethoxazole is an option for patients who are intolerant to macrolides, have known or suspected macrolide resistance or are infected with *B. bronchiseptica*.[2,97]

Supportive care

Supportive measures are essential, particularly for infants and others with severe disease.

Symptom management

Cough suppressants are not recommended, as they do not alleviate the underlying cause of paroxysmal cough and may have adverse effects in children.[99,100] Any form of sedation is reserved for inpatient management only.

Nutritional and hydration support

Frequent small feeds or nasogastric feeding may be necessary for infants and young

Table 3. Pertussis-containing vaccines available in Australia

Vaccine brand	Formulation	TGA registration (age range)	NIP (recommended age)	Pertussis antigens			
				PT mcg	FHA mcg	FIM 2/3 mcg	PRN mcg
Infanrix hexa	DTPa-hepB-IPV-Hib	≥6 weeks	2, 4 and 6 months	25	25	–	8
Vaxelis	DTPa-hepB-IPV-Hib	≥6 weeks	2, 4 and 6 months	20	20	5	3
Infanrix	DTPa	2 to 12 months and 15 months to 6 years	18 months	25	25	–	8
Tripacel	DTPa	2 to 12 months and 15 months to 8 years	18 months	10	5	5	3
Infanrix IPV	DTPa-IPV	≥6 weeks and ≤6 years	4 years	25	25	–	8
Quadracel	DTPa-IPV	2 to 12 months and 15 months to 6 years	4 years	20	20	5	3
Adacel Polio	dTpa-IPV	≥4 years	–	2.5	5	5	3
Boostrix IPV	dTpa-IPV	≥4 years	–	8	8	–	2.5
Adacel	dTpa	≥10 years	10 years to <20 years, in pregnancy	2.5	5	5	3
Boostrix	dTpa	≥4 years	10 years to <20 years, in pregnancy	8	8	–	2.5

Abbreviations: DTPa = infant and child formulation of diphtheria-tetanus-acellular pertussis vaccine; dTpa = adolescent and adult formulation of diphtheria-tetanus-acellular pertussis vaccine; FHA = filamentous haemagglutinin; FIM 2/3 = fimbriae 2 and 3; hepB = hepatitis B; Hib = Haemophilus influenzae type b; IPV = inactivated polio virus; NIP = National Immunisation Program; PRN = pertactin; PT = pertussis toxin; TGA = Therapeutic Goods Administration.

Adapted from the NIP Schedule and data from the TGA Product and Consumer Medicine Information Repository.[111,104]

children with feeding difficulties caused by paroxysmal coughing and vomiting. Intravenous fluids may be required in cases of significant dehydration or poor oral intake.

Respiratory support

Supplemental oxygen therapy is indicated for hypoxia or cyanotic episodes. Mechanical ventilation is required in cases of severe respiratory distress or apnoea, particularly in infants.

Postexposure prophylaxis

Indications and regimen

Timely initiation of postexposure prophylactic antimicrobials reduces the likelihood of spread to close contacts of confirmed pertussis cases, particularly household members (especially infants and pregnant women) and healthcare workers exposed to infectious patients.[101] The same antimicrobials used for treatment are recommended for postexposure prophylaxis within 21 days of exposure to be effective in preventing secondary cases.[97]

Prevention

The primary aim of pertussis prevention is to reduce disease incidence, severity and transmission, especially among vulnerable populations such as infants. Strategies involve widespread vaccination, public health measures and education.[88,102]

Vaccination

Whole-cell pertussis and acellular pertussis vaccines

The wP vaccine was first introduced in the 1940s and consists of detoxified and heat-killed bacteria. It provides robust and long-lasting immunity to *B. pertussis* by inducing a strong Th1/Th17-driven immune response to more than 3000 antigens, mimicking natural infection.[2] However, concerns over reactogenicity, including high rates of fever and injection site reactions, led to the development of the aP vaccine, subsequently replacing wP vaccination in most developed countries through the 1990s and 2000s.[103]

In Australia, the aP vaccine contains purified antigens to *B. pertussis*, typically including PT, FHA, PRN and FIM2/3 proteins (Table 3).[104] These are combined with an aluminium-based adjuvant to enhance immune responses. Although the aP vaccine significantly decreases pertussis incidence, it induces a predominantly Th2-skewed immune response, which has been associated with reduced long-term protection compared with wP vaccines.[42,50-52]

Neither the wP nor the aP vaccines confer substantial cross-protection against *B. parapertussis, B. bronchiseptica* or *B. holmesii*.[105,106] Studies suggest that prior vaccination with the aP vaccine may even facilitate *B. parapertussis* infection because of immune modulation that fails to provide sufficient clearance.[107]

Australian Immunisation Handbook

The *Australian Immunisation Handbook* (see: https://immunisationhandbook.health.gov.au) provides clinical advice on the safest and most effective use of vaccines.[108] The handbook recommendations are developed by the

Table 4. Pertussis vaccination recommendations by target group[109,111,116]

Target group	Recommendation	Formulation	NIP funded
Infants and children*	Three-dose primary schedule at 2†, 4 and 6 months	DTPa-HepB-IPV-Hib	Yes
	AND one booster dose at 18 months of age	DTPa	Yes
	AND one booster dose at 4 years of age	DTPa-IPV	Yes
Adolescents* (11 to 13 years)	One booster dose during adolescence	dTpa	Yes
Adults	One booster dose if aged ≥65 years and last pertussis-containing vaccine dose >10 years ago	dTpa	No
	One booster dose every 10 years for any adult who wants to reduce their likelihood of becoming unwell with pertussis	dTpa	No
Women who are pregnant or postpartum	One booster dose in each pregnancy	dTpa	Yes
	If not vaccinated during pregnancy (from 20 weeks), a single booster dose is recommended immediately postpartum‡	dTpa	No
Adult household contacts and carers of infants ≤6 months	One booster dose at least 2 weeks before they have close contact with the infant, if their last dose was more than 10 years ago	dTpa	No
Healthcare workers and early childhood educators/carers	One booster dose every 10 years	dTpa	No
Travellers	One booster dose if the last pertussis-containing vaccine dose was more than 10 years ago, or five years ago for high-risk travel	dTpa	No
People with a history of pertussis infection	Infants, children, adolescents and adults who have had laboratory-confirmed pertussis infection should receive all routinely scheduled pertussis-containing vaccines because natural immunity does not provide lifelong protection	As per target group recommendations	As per schedule
Refugees and humanitarian entrants aged ≥20 years*	People ≥10 years of age who did not receive all the pertussis vaccine doses recommended before the age of 10 years only need one dose to be considered up to date (regardless of the number of previous doses they have received)	dTpa	Yes
People who have completed cancer therapy	Single dose of DTPa-IPV if <10 years of age	DTPa-IPV	No
	Single dose of dTpa-IPV if ≥10 years of age	dTpa-IPV	No
Solid organ transplant recipients	Recipients <10 years of age and not previously vaccinated should receive at least 3 primary doses as DTPa-containing vaccine, then follow routine schedule for age	DTPa-HepB-IPV-Hib	Yes
	Recipients ≥10 years of age and not previously vaccinated should receive the first dose as dTpa-IPV. Complete vaccination course with dTpa	dTpa-IPV dTpa	No
	Adults who have received at least three primary doses of a diphtheria-tetanus-pertussis-containing vaccine should receive one dose of dTpa-IPV after transplant if their last dose was >10 years ago	dTpa-IPV	No
Post-HSCT in children and adults	For recipients <10 years of age, give all three primary doses as DTPa-IPV§ vaccine at 6, 8 and 12 months post-HSCT	DTPa-IPV	No
	For recipients ≥10 years of age, give all three primary dose as dTpa-IPV§ vaccine at 6, 8 and 12 months post-HSCT	dTpa-IPV	No
	Young children should complete the recommended age-based vaccination schedule after receiving the three primary post-HSCT vaccination doses	As per schedule	Yes

Abbreviations: DTPa = infant and child formulation; dTpa = adolescent and adult formulation with reduced pertussis and diphtheria antigens; HepB = hepatitis B; Hib = *Haemophilus influenzae* type b; HSCT = haematopoietic stem cell transplantation; IPV = inactivated polio vaccine; NIP = National Immunisation Program.

*Missed dose(s) of pertussis-containing vaccine requires catch-up vaccination; † First dose may be given from 6 weeks of age; ‡ If the mother was not vaccinated immediately postpartum, there may still be some benefit in her being vaccinated at any time until the infant is 6 months of age. § If IPV not required, use DTPa vaccine for <10 years of age or dTpa vaccine for ≥10 years of age.

Infanrix hexa rule: when DTPa / IPV / Hi-b / Hep B are due and recommended within 4 weeks of each other, the antigens may be grouped together and administered on the latest date that any of the 6 antigens are recommended. (Note: DTP is always administered as a combination). Infanrix hexa will be presented as the singular choice when all 6 antigens are due (irrespective of the vaccination history, i.e. whether the antigens were received as a combination or monovalent vaccines including overseas variants i.e. 'generic/other'). No other combinations to be presented. If one or more of the 6 Infanrix hexa antigens are NOT due together then other vaccine brand names can be provided (DTPa only, IPV only, Hep B only, Hib only, DTP-IPV).

Adapted from the Australian Immunisation Handbook, the National Immunisation Program Schedule and the National Immunisation Program Business Rules.[109,111,116]

Australian Technical Advisory Group on Immunisation and endorsed by the National Health and Medical Research Council. This online resource details information on vaccine preventable diseases and vaccinations for special risk groups.

The NIP funds many, but not all, of the vaccines. Individuals may receive, or be recommended to receive, vaccines described in the handbook that are not part of the routine immunisation schedule, such as those at occupational risk of disease, traveling overseas or who have a medical condition that puts them at increased risk of contracting a vaccine-preventable disease. The pertussis section within the handbook extensively references the NIP and the NIP Schedule.[109-111]

National Immunisation Program schedule

The NIP is a free, Commonwealth government-funded, state and territory government-delivered, program for certain immunisations administered at specific times from birth to adulthood for Medicare eligible individuals (Table 4).[111] In Australia, vaccines containing pertussis are only available as combination vaccines (Table 3).

For pertussis, a primary three-dose schedule is administered at 2, 4 and 6 months of age, with single booster doses given at 18 months of age, and again at 4 years of age. Reduced-dose diphtheria-tetanus-acellular pertussis (dTpa) boosters are available for adolescents and pregnant women. When administered between 20 and 32 weeks' gestation to optimise the transfer of maternal antibodies to the fetus, pertussis-containing immunisation provides passive immunity to infants during their first few months of life when they are most vulnerable. Importantly, recent Australian and international data confirm the effectiveness of this strategy, significantly reducing the risk of laboratory-confirmed disease among infants younger than 2 months of age.[37,57,112-114]

Refugees and humanitarian entrants aged 20 years or older can also get certain vaccines for free if they did not receive them in childhood. If a person has a record of vaccination from overseas, consider any previous doses when planning a catch-up vaccination schedule. Check the dosing intervals as some doses may be invalid because the interval between doses was too short. Check all vaccination records before vaccination as well because people may have visited multiple immunisation providers after they arrived in Australia.

There are no additional recommendations for Aboriginal and Torres Strait Islander children and adolescents.

Catch-up immunisation

Any immunisation given after the recommended age is a catch-up immunisation.[115] Catch-up immunisations give patients their recommended vaccinations and help protect against disease by giving the best protection as fast as possible. This also includes individuals who have completed cancer therapy, have had a haemopoietic stem cell transplant, or are having/have had a solid organ transplant.[116] The handbook includes a comprehensive National Immunisation Catch-up Calculator (available at: https://immunisationhandbook.health.gov.au/catch-up-calculator/calculator), enabling the production of an individualised catch-up vaccination schedule. People aged 20 years or older who have not received all vaccines may still benefit from a catch-up schedule. These are not funded under the NIP.

Cocooning strategy

Ensuring vaccination of close contacts (e.g. parents, household members, grandparents, caregivers) to create a protective 'cocoon' around infants is safe and effective in reducing transmission risk.[117] Recent data demonstrated regular incomplete coverage, highlighting an area of focus when counselling expectant parents, especially fathers.[118,119]

Vaccination for healthcare workers

Healthcare workers can minimise transmission to vulnerable patients by receiving a pertussis-containing vaccine booster dose every 10 years.[109]

Vaccination for immunosuppressed patients

People with HIV, including pregnant women, can safely receive DTPa and dTpa (including when combined with *H. influenzae* type b and inactivated polio virus) vaccines according to routine recommendations.[120-124] Because of immune suppression, the use of hepatitis B virus antigen-containing DTPa vaccines (DTPa-hepB-IPV-Hib) is not recommended, as higher strength hepatitis B formulations or increased doses may be required.

Herd immunity

High community vaccine coverage helps to reduce *B. pertussis* circulation and may indirectly protect unvaccinated individuals. Given that immunity to pertussis is not lifelong, regardless of natural infection or immunisation, regular booster dose coverage must remain consistently high to maintain herd immunity.[52]

Addressing vaccine hesitancy

Honest and transparent communication on vaccine benefits and safety profiles is a cornerstone of good medical practice. The complex issue of vaccine hesitancy, brought to the fore by COVID-19, may be approached through tailored messaging addressing specific concerns, including misinformation amplified by social media, to create a trusting environment for healthcare providers to advocate for vaccination.[125-127]

Public health measures

Surveillance, reporting and contact tracing

Enhanced surveillance ensures timely outbreak detection, case investigation, and implementation of control measures.[84] With laboratory and clinician reporting, contact tracing can identify individuals exposed to confirmed cases and offer postexposure prophylaxis to limit secondary transmission, especially in high-risk community settings such as childcare facilities.

Education and awareness

Strategic community education campaigns help promote timely vaccination and recognition of pertussis symptoms.[128,129] Frequent messaging for parents may further reinforce the importance of adhering to vaccination schedules.

Challenges to overcome

The resurgence of pertussis in Australia in the post-COVID-19 era highlights important challenges in controlling this largely

vaccine-preventable disease. Despite high vaccination coverage achieved through the NIP, pertussis remains endemic, with periodic outbreaks occurring every three to five years pre-COVID-19. The decline in notifications observed during the pandemic because of widespread public health measures has been followed by a sharp rebound as these interventions have been lifted, underscoring the ongoing vulnerability of the population. This resurgence may be attributed, at least in part, to several factors, including epidemiological shifts such as genomic reshuffling, asymptomatic infection, waning immunity, declining immunisation uptake in some target groups, and increased transmission as social interactions normalised.

Given these challenges, a multifaceted approach is required to strengthen pertussis control in Australia. Enhancing vaccine strategies, including optimising booster schedules and investigating next-generation vaccines that elicit more durable immunity, is crucial. Maternal vaccination remains a highly effective strategy for protecting highly vulnerable infants, yet recent declines in uptake emphasise the need for continued public health messaging and provider advocacy. Additionally, improving access to vaccines among Indigenous and other high-risk populations must be prioritised to reduce health disparities.

Diagnostic vigilance is essential, particularly in recognising atypical presentations across age groups, including older individuals and vaccinated populations. Early identification through PCR-based laboratory testing remains the preferred method, given its high sensitivity in the first few weeks of illness. Given the current unprecedented levels of pertussis circulating throughout the Australian community, it is imperative that GPs and other frontline healthcare workers maintain a high index of suspicion for pertussis, particularly in cases of prolonged cough or exposure to infected individuals. This improved clinical awareness, combined with prompt reporting and contact tracing, is vital for timely intervention and outbreak mitigation.

Public health efforts should also focus on addressing vaccine hesitancy, which has been exacerbated by misinformation and pandemic-related disruptions. Strengthening communication strategies to reinforce vaccine safety and effectiveness are important to improve public confidence in immunisation programs. Enhanced surveillance systems, including genomic monitoring of circulating *B. pertussis* strains and improving diagnostic data collection, will aid in assessing vaccine effectiveness, improve outbreak response and inform future immunisation policies.

Conclusion

The unique pathophysiology of pertussis explains its protracted course and characteristic clinical features. Understanding the mechanisms of immune evasion and the role of toxin-mediated damage is essential for timely diagnosis, effective treatment and targeted prevention strategies. Recognising the pathophysiological underpinnings can help clinicians anticipate complications, particularly in high-risk groups such as infants and immunocompromised individuals.

The role of GPs in recognising, treating and preventing pertussis cannot be overstated. By staying informed of evolving epidemiological trends and clinical guidelines, healthcare providers can play a pivotal role in reducing the burden of pertussis and protecting vulnerable populations.

As Australia transitions into the post-COVID-19 era, the lessons learned during the pandemic, such as the importance of public health infrastructure and community engagement, must be applied to strengthen pertussis control efforts. With sustained vigilance and innovation, the goal of reducing pertussis-related morbidity and mortality remains achievable. **RMT**

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A list of references is included in the online version of this article (www.respiratorymedicinetoday.com.au).

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Pertussis

The post-COVID-19 resurgence in Australia

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