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Lung disease in indigenous children

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EDUCATIONAL AIMS

The reader will be able to:

• Discuss the common respiratory problems encountered by indigenous children.

• Appreciate the similarities and differences in the aetiology, associations and management of these common respiratory problems.

• Understand the many facets that impact on the clinical outcomes of common respiratory conditions affecting indigenous children.

• Discuss potential intervention targets that can reduce the morbidity and mortality of respiratory diseases in indigenous children.
SUMMARY

Children in indigenous populations have substantially higher respiratory morbidity than non-indigenous children. Indigenous children have more frequent respiratory infections that are, more severe and, associated with long-term sequelae. Post-infectious sequelae such as chronic suppurative lung disease and bronchiectasis are especially prevalent among indigenous groups and have lifelong impact on lung function. Also, although estimates of asthma prevalence among indigenous children are similar to non-indigenous groups the morbidity of asthma is higher in indigenous children. To reduce the morbidity of respiratory illness, best-practice medicine is essential in addition to improving socio-economic factors, (eg household crowding), tobacco smoke exposure, and access to health care and illness prevention programs that likely contribute to these issues. Although each indigenous group may have unique health beliefs and interfaces with modern health care, a culturally sensitive and community-based comprehensive care system of preventive and long term care can improve outcomes for all these conditions. This article focuses on common respiratory conditions encountered by indigenous children living in affluent countries where data is available.

INTRODUCTION

Globally, the disparity in health between indigenous and non-indigenous people is striking. It is most marked and well documented in affluent countries; Aboriginal and Torres Strait Islanders in Australia; Māori in New Zealand (NZ); First Nation, Inuit and Metis People in Canada; and American Indians and Alaska Natives (AI/AN) and Native Hawaiians in USA (here forth referred as indigenous).\textsuperscript{1} Indigenous populations of these countries bear a high burden of ill health from acute and chronic respiratory disease. In indigenous Australians, respiratory disorders are the second most common reason for hospitalisation.\textsuperscript{2} In indigenous populations of the USA, Canada and NZ, respiratory illnesses are also one of the most prevalent acute and chronic illness.\textsuperscript{3,4}
This review focuses on the most common acute and chronic respiratory diseases in indigenous children living in Australia, NZ, Canada and USA. The little data on indigenous populations in other countries with indigenous and colonial/immigrant populations precludes meaningful comparison. This review does not discuss basic treatment principles as the same high quality best practice care is paramount in these settings and readers are referred to evidence based guidelines. Tobacco exposure and its effects are briefly mentioned but not discussed in depth.

In these countries, the disease patterns (frequency, age distribution, severity and/or co-morbidities) in indigenous children have important differences compared with that of non-indigenous children. The severity and morbidity (eg hospitalisations) of asthma, acute and other chronic respiratory diseases in indigenous populations are greater compared to their non-Indigenous counterparts.\textsuperscript{5,6} Across these countries, indigenous children share some similarities but also differences in respiratory diseases.\textsuperscript{1,4} For example, indigenous children with bronchiectasis living in USA, NZ and Australia share similarities in the frequency of household crowding, prematurity and early respiratory infections.\textsuperscript{4} However, there are also differences among these populations with respect to prevalence of wheeze, ear disease and plumed water.\textsuperscript{4}

**ASTHMA**

Asthma is one of the most common chronic respiratory illness in indigenous children, with prevalence rates of asthma of 14.3% in Canadian indigenous children aged 6-14 years,\textsuperscript{7} 14.8% of New Zealand Māori and Pacific children aged 2-14%,\textsuperscript{5} 12-23% in Australian indigenous children (0-17 years)\textsuperscript{8} and 13% in USA AI/AN children.\textsuperscript{9} However, the accuracy of these data is limited by the various definitions of asthma used. Nevertheless, asthma prevalence in indigenous children tends to be lower in rural regions compared to urban centres.\textsuperscript{7}
While the prevalence of asthma in indigenous children, is similar to that of non-indigenous children, the associated chronic morbidity of asthma is higher in indigenous children compared to their non-Indigenous counterparts.\textsuperscript{5,10,11} For example, the hospitalization and mortality rate for asthma is 2-3 times higher in indigenous Australians.\textsuperscript{12} The poorer clinical outcomes have also been documented in indigenous children of New Zealand, Canada and USA as well as other minority groups globally.\textsuperscript{5,13}

There is little data on whether the risk factors for having asthma in indigenous children are identical to that in non-indigenous children. Factors that have been identified were being male, allergy, obesity, low birth weight, poor housing, tobacco exposure, urban residence and history of bronchitis.\textsuperscript{7,14} Reasons why indigenous children have poorer asthma outcomes is largely unknown and is likely multi-factorial. There is paucity of data that have directly compared outcomes between indigenous with non-indigenous children within a single setting. Through interviews, a NZ study found that Māori and Pacific children were more likely to receive sub-optimal asthma treatment, such as lower rates of the use of inhaled corticosteroids in primary care despite higher rates of chronic symptoms.\textsuperscript{5} An Australian study of 200 children hospitalized for asthma found that intrinsic biological factors are unlikely to account for the poorer asthma outcomes as there was no significant difference between indigenous and non-Indigenous children in the length of hospitalisation and risk factors (eg prematurity), other than environmental tobacco smoke (ETS) exposure.\textsuperscript{12} The retrospective Australian study also identified possible intervention points to improve the management of asthma, particularly in indigenous children. These included better identification, documentation and management of ETS exposure, and improvement of acute management and discharge planning including education and utilisation of asthma action plans.\textsuperscript{12}

Improvement in asthma outcomes for indigenous children will need to focus on better individual management and systems that support such care. Despite major advances in the understanding of
the pathogenesis of asthma (with relatively large amounts of research dollars), the accompanying benefits from public health initiatives have arguably been less than expected. Some have proposed that the priority for asthma research should be improving the use of existing treatments as evidence-based measures to prevent (or reduce) the prevalence of asthma in children are unavailable (other than reducing tobacco smoke exposure).

**Management of asthma**

The principles of managing asthma with respect to evidence-based use of appropriate medications (though a step-wise approach), use of spacers and asthma action plans to control asthma symptoms and reduce exacerbations are no different in indigenous populations compared to non-indigenous children. The key difference is the delivery of service and the framework of care (e.g. inclusion of indigenous healthcare workers, appropriate education resources). The problems of poor access to high-quality health care, affordable medications for indigenous and other minority groups in affluent countries has been documented. Indigenous children have very high rates of ETS exposure, a particular concern in people with asthma. Obesity and diagnostic accuracy (given high relatively high levels of co-morbidities such as chronic suppurative lung disease (CSLD) where wheeze is also common) are also of particular importance. In addition to the specific management issues relevant to asthma, other generic respiratory health measures (see section below) should be embedded in a culturally specific context

**ACUTE LOWER RESPIRATORY INFECTIONS (ALRIs)**

**Epidemiology and context**

Globally, ALRIs including pneumonia represent the largest (18%) single cause of death in children aged <5-years. While there are substantial inter-country and inter-continental differences in the annual incidence of pneumonia globally (0.33 episodes per child-year in Africa, 0.05 in developed countries), there is also wide intra-country variability. For example, in contrast to the rest of
Australia, ALRIs are the commonest cause of preventable deaths in infants, emergency medical retrievals from remote communities, and hospitalizations among indigenous children aged <5-years. The incidence of hospitalized-pneumonia among infants in the Northern Territory of Australia (the region with the highest proportion of indigenous people) is 0.43 per child-year. The same preponderance of ALRI in indigenous children (vs non-indigenous children) is also seen in the USA, New Zealand and Canada. Hospitalisation rates for respiratory infections in AI/AN children were almost double that for the rest of the paediatric population (116.1 versus 63.2/1000 respectively). Hospitalized ALRIs in Australian indigenous children are decreasing, but they are increasing among AN children (39% of all infectious disease hospitalisations and 74% of infant infectious disease hospitalizations). In New Zealand, hospitalised ALRI rates in children aged <2 years are 103/1,000 nationally but reach 177/1,000 in South Auckland Māori and Pacifica children. These higher rates in indigenous populations in affluent countries compared to developing countries is likely related to the invariably better data collection in affluent countries. Bronchiolitis and pneumonia account for the majority of the hospitalized ALRI burden and these are discussed in further details below.

In addition to the high disease burden, the importance of ALRIs is also reflected in non-hospitalised morbidity and mortality and long term consequences especially when ALRIs are recurrent. Recurrent ALRIs are an independent risk factor for subsequent bronchiectasis and lower pulmonary function later in life. Not only are low birth weight and pre-existing small lungs important determinants of future lung function, but there is increasing evidence that early events in life are at least equally important determinants of adult pulmonary dysfunction. Early infectious or inflammatory insults in the first few years of life, when postnatal lung development is the most important, are most likely to result in long-term effects.
Bronchiolitis

Bronchiolitis is the most common serious ALRI in infants. The prevalence and severity of bronchiolitis is greater in indigenous children compared to non-Indigenous children.\textsuperscript{6,26,27} In New Zealand, being Māori was as an independent risk factor for hospitalisation from RSV bronchiolitis.\textsuperscript{28} In contrast, a retrospective Australian study found that being indigenous was not an independent risk factor for RSV hospitalisation once socioeconomic risks factors were accounted for in their analysis.\textsuperscript{29}

Indigenous children with bronchiolitis have more severe disease, and poorer clinical outcomes including higher re-hospitalisation rates.\textsuperscript{6,27} In Alaskan children, previous bronchiolitis is a risk factor for the development of chronic productive cough,\textsuperscript{30} which is the most common symptom of CSLD and bronchiectasis (BE). Co-existent clinical pneumonia is common in indigenous children with bronchiolitis and in these setting, many receive antibiotics.\textsuperscript{27} While this may be a diagnostic error, it is also biologically plausible. Concurrent bacterial infection are more common in those with severe bronchiolitis,\textsuperscript{31} and bacteria pneumonia rates may be truly higher for several reasons including the aspiration of nasal secretions which, in these children, contains large bacterial load.\textsuperscript{32} This potentially overwhelms the local lung defences (mucosal and innate immunity) already impaired by the viral infection.\textsuperscript{33} In Australian indigenous children, bacterial (\textit{S. pneumoniae}, \textit{H. influenzae}, \textit{Moraxella catarrhalis}) colonisation of the nasopharynx occurs as early 2 weeks of age and is heavier than in non-Indigenous children.\textsuperscript{34} Some children are susceptible to bacterial infections during, or shortly after, a viral respiratory tract infection and indigenous children have high rates of viral infections. Viral-bacterial interactions are more likely to occur when the upper airway respiratory epithelium is densely colonised with respiratory pathogens or with repeated infections.\textsuperscript{33}
Pneumonia

Pneumonia also has a high preponderance in indigenous children.\textsuperscript{22,35} Obtaining accurate prevalence figures on bacteria pneumonia is problematic as differentiating bacterial from viral or mixed-cause pneumonia is difficult. Even the use of WHO-defined radiological pneumonia (end-point consolidation (EPC)) is fraught with problems especially in the clinical context. In a cohort of indigenous children hospitalized with pneumonia, a paediatric pulmonologist’s blinded assessment of radiological pneumonia had significantly higher positive predicted value for the presence of crackles and elevated white cell counts when compared to WHO-EPC.\textsuperscript{36} Over a 2-year period, viral infection (by PCR) were positive in 90% of AN children hospitalised for ALRI.\textsuperscript{37} Among those with PCRs positive for influenza, parainfluenza, RSV and human metapneumovirus, 60% of children had radiographic evidence of parenchymal densities compatible with pneumonitis or pneumonia.\textsuperscript{37} The many gaps in the diagnosis and management of pneumonia in children have been highlighted.\textsuperscript{38}

In addition, indigenous children with pneumonia have associated poorer clinical outcomes and consequences. Indigenous Australian children hospitalised with pneumonia were 15 times more likely to develop bronchiectasis (OR 15.2; 95% CI 4.4, 52.7) and when pneumonia is recurrent, the risk increases further.\textsuperscript{23} In a cohort study, 25.6% of children hospitalised lobar pneumonia had a newly diagnosed and treatable chronic respiratory illness (18% CSLD) on follow-up.\textsuperscript{39} In a New Zealand study of predominantly (94%) Māori and Pacifica children, 74% of the 81 children followed at 10-14 months after hospitalization for an ALRI, had features of ongoing respiratory morbidity (wet cough, auscultatory chest crackles, CXR abnormalities).\textsuperscript{22}

Several vaccines have reduced the worldwide incidence of pneumonia. However, the benefits from population-based vaccination programs are not universal. Although pneumococcal vaccine (PCV)-7 programs have significantly reduced pneumonia rates, its impact on some populations has been
negligible.\textsuperscript{35,40} Despite achieving high population PCV-7 vaccination coverage rates of \textasciitilde90\%, the incidence of WHO-defined radiological pneumonia in indigenous children living in the Northern Territory (Australia) was not significantly reduced. Among AN children, the serotypes of pneumococcus causing pneumonia transitioned to those uncovered by PCV-7 with no decrease in the frequency of pneumococcal pneumonia related hospitalizations.\textsuperscript{35,40} The effect of PCV-13 remains unknown.

**Management of ALRI**

Outside of vaccine trials, most RCTs on pneumonia in the last 2 decades were based in resource-poor areas\textsuperscript{41} where the risk factors, aetiologies, patient factors and settings are different to resource-rich countries. Meta-analysis of studies have shown discrepancies in results between studies conducted in resource-poor vs. -rich countries.\textsuperscript{42} Those conducted in resource-poor settings have significantly more favourable treatment effects than in resource-rich settings.\textsuperscript{42} Extrapolating data from resource-poor settings to that in resource-rich countries may not be prudent. For example, the an Australian-based RCT evaluating the use of zinc and/or vitamin A as an adjunctive treatment of indigenous children hospitalized with pneumonia described likely increased harm which is in contrast to data in developing countries\textsuperscript{43}. This likely relates to the low prevalence of micronutrient deficiency in Australia. Further, there is little data on long term consequences of pneumonia\textsuperscript{38} and indigenous children have a high risk of CSLD and bronchiectasis.\textsuperscript{4}

Given the link between pneumonia and bronchiectasis,\textsuperscript{23,39} as well as duration of chronic cough with CT bronchiectasis score and lung function decline,\textsuperscript{44} it is good clinical practice to follow up children 3–4 weeks after hospitalisation. They should be screened for the presence of chronic cough and persistence of other respiratory symptoms and signs (wheeze and crackles in chest auscultation). If the cough persists, evidence-based management of chronic cough in children should be instituted.\textsuperscript{45}
**PROTRACTED BACTERIAL BRONCHITIS (PBB)**

PBB is a condition clinically characterised by (a) chronic (> 4 weeks) wet cough, (b) resolution of cough when treated with 2 weeks of appropriate antibiotics, and (c) absence of other symptoms and signs suggestive of other known aetiologies for cough. Clinicians should be cognisant that in some indigenous settings, parental reporting of cough is poor and serial observations of the child over multiple clinic visits are often required to determine chronicity. PBB is characterised by intense airway neutrophilia (40-44%) and elevated markers of neutrophilic inflammation (IL-8, MMP-9) accompanying the bacteria infection with upregulation of innate immunity markers.

PBB is likely linked with CSLD and bronchiectasis, both of which have a high prevalence among Indigenous children. Early treatment of endobronchial infection can halt on-going infection and inflammation that predispose to chronic airway injury. Based on the increased frequency of chronic wet cough among different groups of indigenous children, it is likely that PBB is also more common in indigenous children. Reasons for this include the dense bacteria colonisation of the nasopharynx of indigenous children early in life, post-viral endobronchial bacterial infection, and ongoing exposure to indoor irritants such as tobacco smoke.

**Management of PBB**

The treatment of PBB with is based a 2-week course of antibiotics is based on common bacteria pathogens (*S. pneumoniae*, non-typable *H. influenzae*, *M. catarrhalis*) cultured from the lower airways. Children should be re-evaluated and if the cough persists or recurs, a repeat 2 week course of antibiotics should be prescribed. If the wet cough does not resolve, the child should be referred for further investigation including a chest HRCT scan. In a retrospective study of 144 children, those whose cough failed to respond to 4-weeks of antibiotics were 21 times (95%CI 5.4-
more likely to have bronchiectasis, especially if the child is Indigenous (OR 5.9; 95% CI 1.2-28.5).51

CHRONIC SUPPURATIVE LUNG DISEASE (CSLD) AND BRONCHIECTASIS (BE)
We include CSLD with bronchiectasis for several reasons.52,53 These conditions overlap and children with CSLD experience similar clinical disease patterns as children with CT-confirmed BE.49 They also respond similarly to therapies used to treat children with BE.52,53 Also thirdly, indigenous children living in non-urban centres and children in developing countries have limited access to CT scanning to confirm BE and defining ‘irreversibility’ requires two CT scans, which is neither safe (increased radiation) nor feasible. Finally, there are limitations in the radiographic definitions of BE in children because HRCT definitions are derived from adult studies and are not necessarily equivalent to those in children. In particular, the key diagnostic criterion of bronchiectasis is increased bronchoarterial ratio, which is clearly influenced by age (r=0.77, p<0.0001).54 Also the sensitivity of determining bronchiectasis radiologically is dependent upon the modality used as scans obtained on a multi-detector CT scan are more sensitive than those from a HRCT scan.55 Thus in children, the term CSLD is used to describe a diagnosis where there are clinical symptoms of BE without HRCT confirmation.

In the last century, CSLD/BE has declined. However in the recent two decades, CSLD/BE are increasingly recognised as an important contributor to chronic respiratory morbidity in both indigenous and non-indigenous children and adults.4 Given the requirement of a HRCT for diagnosis of BE, it is not surprising that there is little data on its prevalence; available figures will invariably be under-representative of the true prevalence. In an Australian state with the highest proportion of indigenous people, the incidence in indigenous infants (first year of life) is 1.18 per 1000 child-years (95% CI, 0.60-2.16)18 and the prevalence is one in every 68 children aged <15
years.\textsuperscript{56} In Alaskan AI/AN children, the prevalence is one in every 63 children\textsuperscript{57} and that in Māori children of New Zealand is 1 in 1428.\textsuperscript{4}

Hospitalisation rates for CSLD/BE is increasing; the rate in Indigenous people living in Queensland, Australia was 2.7 times that of non-Indigenous Queenslanders in 2009.\textsuperscript{58} There is no data in urban Australia but among 346 children newly presenting with chronic cough to a respiratory service, indigenous children had a significantly higher incidence of radiological BE compared to non-indigenous children (29.4\% vs 6.7\% respectively).\textsuperscript{59}

**Management of CSLD**

There are evidence-based guidelines for the management of CSLD/BE specific for indigenous people,\textsuperscript{52} which are largely the same as for non-indigenous people. Although the majority of CSLD and BE in indigenous children is related to previous respiratory infections, these children merit evaluations for serious underlying conditions such as immunodeficiencies. The principles of treatment include the following: (a) control of symptoms, (b) prevention and prompt treatment of exacerbations, (c) preservation of lung function, and (d) prevent secondary complications, such as haemoptysis. Treatments include combination of antibiotics, bronchodilators, airway clearance techniques, and regular evaluations.\textsuperscript{52} within an appropriate cultural framework. This may include altering the frequency of medications, such as weekly azithromycin, which we have shown is feasible for up to 2 years.\textsuperscript{53}

**CONTRIBUTING FACTORS TO ALRIs AND CSLD/BE**

In indigenous settings in Australia and USA, a combination of factors may explain why the young children living in remote communities experience a high burden of pneumonia. Canadian,\textsuperscript{19} Alaskan,\textsuperscript{60} and New Zealand studies have linked housing issues (e.g. overcrowding, lack of running water, reduced ventilation) to excess ARLIs. In an Alaskan\textsuperscript{60} study, medically high-risk status
(prematurity, congenital heart disease, chronic lung disease), a woodstove in the house, bottle feeding, and vomiting after feeding were additional risk factors for hospitalisations for ALRIs. A Canadian study on pneumonia and influenza identified that low education, being Aboriginal, daily smoking, heavy drinking, environmental factors (passive smoking, poor housing, temperature), and health care factors (influenza vaccination) as significantly associated with increased rates of ALRIs requiring medical care. However, in some indigenous settings housing upgrades were insufficient to improve health outcomes in children. Also, whether the lung microbiome differs (and/or influence disease manifestation) among indigenous and non-indigenous children within the same setting is unknown.

These same risk factors for ALRIs pertain to CSLD-BE. A combined (Australia, NZ and Alaska) study of indigenous children with CSLD/BE found that the indigenous children in all regions experienced substantial disparities in poverty indices and risk factors (ETS exposure, low education levels, etc) common (similar rates) with their respective regional Indigenous populations (Table 1). However, household crowding, prematurity and a high frequency and early onset of ALRIs were associated with development of bronchiectasis.

**GENERIC APPROACH TO MANAGEMENT**

In addressing prevention of respiratory illness, social determinants of health are undeniably important which cannot be adequately addressed here. Major advances in indigenous child health will follow improvements in education and housing, and reductions in poverty, and improved access to high quality culturally-sensitive health care. Provision of high quality care requires the application of the best evidence on prevention and management of common illnesses.
Prevention

The respiratory conditions discussed above share some common risk factors. Risk factors for
development of these conditions (e.g. in utero smoke exposure, passive and active smoking, low
birth weight, obesity) and factors associated with increased severity (e.g. poor psychosocial well-
being, passive and active smoking) are higher in indigenous people compared to their non-
indigenous counterparts. Generic and disease-specific documented risk factors are summarised in
Tables 2 and 3.

Disease specific primary prevention measures for respiratory disease are categorised into (a
promoting normal lung development and (b removing risk factors for development of respiratory
disease. Both share the common intervention features in Table 2 and disease-specific interventions
in Table 3. Strategies for secondary prevention (the early detection and treatment) and tertiary
prevention (reduction of further complications, morbidity and mortality) are also summarised in
Tables 2 and 3.

EQUITY AND CONSEQUENCES OF POOR RESPIRATORY HEALTH IN CHILDHOOD

In the context of indigenous health, ALRIs is an important indicator of the health disparities that
persist between indigenous and non-Indigenous children in developed countries.63 Health is
intertwined with every aspect of life - education, human rights, social justice, the environment,
economy and employment. Redressing health equity through action on the social determinants of
health for indigenous children has to occur through indigenous models of health that respect and
understand indigenous beliefs and world iews.64 Racism, subtle and overt, remains a challenge in
indigenous health care.64

Addressing acute and chronic childhood respiratory disease is also important as the antecedents of
chronic respiratory disease in adults are likely to occur in childhood. Sub-optimally treated people
with bronchiectasis die early and Australian Indigenous adults with BE die in their 30s and 40s. Many of the common respiratory conditions discussed in this article are potentially modifiable or preventable through clinically based interventions. Further, chronic respiratory disease is an independent risk factor for cardiovascular disease, another common chronic illness leading to considerable mortality in indigenous adult populations.

Therapeutic studies that address the severity and sequelae of common respiratory problems in young indigenous children are needed. These conditions are also common in resource-limited countries. Not surprisingly, on a global scale as well as within affluent countries, disproportionately fewer research dollars are spent on these common conditions. Collectively, in collaboration with indigenous leaders, researchers and communities, we must strive to make clinical service and research relevant to indigenous populations.

**RESEARCH DIRECTIONS**

The respiratory illnesses that have preponderance among indigenous children also affect non-indigenous children particularly those living in developing countries. Thus, research directions on the many unanswered knowledge and clinical questions on asthma, pneumonia, bronchiolitis, PBB and bronchiectasis are relevant and not discussed here. Some suggested specific research directions are listed below but these need to occur concurrently with research (including evidence-based policies) that addresses ways to improve the social determinants of health, education and training of indigenous people.

- Determine reasons why asthma outcomes for indigenous children are poorer so as to develop evidence-based culture-appropriate interventions that improve asthma care and outcomes;
- Define intervention points for the prevention of ALRIs in indigenous children, including reduction and delay of nasopharyngeal colonisation by respiratory pathogens in the first few months of life;
• Determine whether the airway microbiota of indigenous children with chronic suppurative lung disease differs from their non-indigenous counterparts;

• Develop culturally safe environments where educators, both indigenous and non-indigenous work collaboratively within the framework of lung health;

• Understand how the indigenous world view can be incorporated within a model that improves respiratory-specific evidence-based clinical service and practice;

• Evaluate ways to best provide education for parents regarding lung health that impacts care of children at home;

• Reduce the incidence of low gestation age and birth weight of indigenous newborns;

• Conduct clinical trials that prevent and/or reduce the uptake of tobacco smoking among indigenous school-aged children.
Table 1
Demographic characteristics and known risk factors for respiratory infection in Indigenous Bronchiectasis Study children from Australia, Alaska United States, and New Zealand, compared with their respective Indigenous populations and general populations

<table>
<thead>
<tr>
<th>Demographic Characteristics</th>
<th>Study</th>
<th>Local Indigenous Population</th>
<th>National Population</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Household</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overcrowding</td>
<td>84.3</td>
<td>58</td>
<td>2.6</td>
</tr>
<tr>
<td><strong>Caregiver Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school graduate</td>
<td>12.4</td>
<td>31</td>
<td>83</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>0.0</td>
<td>3.6 (remote)</td>
<td>25</td>
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<tr>
<td><strong>Risk Factors</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Breast feeding</td>
<td>86.8</td>
<td>84.1 (urban)</td>
<td>88</td>
</tr>
<tr>
<td>Tobacco exposure in utero</td>
<td>58.9</td>
<td>35.4</td>
<td>51 (Indigenous)</td>
</tr>
<tr>
<td>Smoke exposure (any)</td>
<td>84.5</td>
<td>98</td>
<td>65 (Indigenous)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>32 (general)</td>
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<tr>
<td>Premature</td>
<td>37.1</td>
<td>14</td>
<td>14 (Indigenous)</td>
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<td></td>
<td></td>
<td></td>
<td>8 (general)</td>
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<tr>
<td>Low birth weight (&lt;2500g)</td>
<td>37.4</td>
<td>13</td>
<td>13.1 (Indigenous)</td>
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<td></td>
<td></td>
<td></td>
<td>6.1 (general)</td>
</tr>
<tr>
<td>Proportion with hospitalized ARLI</td>
<td>87.6</td>
<td>49.2</td>
<td>4</td>
</tr>
<tr>
<td>Age at first hospitalized pneumonia (median)</td>
<td>6.1 months</td>
<td>15 months</td>
<td>N/A</td>
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<table>
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<tr>
<th>Demographic Characteristics</th>
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<th>Local Indigenous Population</th>
<th>National Population</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Household</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overcrowding (&gt;1 person/room)</td>
<td>92.7</td>
<td>39</td>
<td>5.8</td>
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<tr>
<td>No running water (%)</td>
<td>43.9</td>
<td>47</td>
<td>0.6</td>
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<tr>
<td>Wood burning in house</td>
<td>47.5</td>
<td>37</td>
<td>1.7</td>
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<td><strong>Educational Attainment</strong></td>
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<td></td>
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<tr>
<td>High school graduate (female)</td>
<td>80.5</td>
<td>80 (both sexes)</td>
<td>85.6</td>
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</tbody>
</table>
Bachelor’s degree  0.0  13  27.3  

**Risk Factors**

- Breast feeding  63.4  79  75  
- Tobacco exposure in utero  87.5  43.4  23  
- Smoke exposure (any)  73.2  58  12.2 (national)  
- Premature  23.3  12.8  9.6% (Alaska)  
- Low birth weight (<2500g)  12.5  5.9  8.2 (national)  
- Hospitalized ALRI (%)  95.1  41.2 per 1000 (Alaska Native)  13.8 per 1000  

<table>
<thead>
<tr>
<th>Demographic Characteristics</th>
<th>Study</th>
<th>Indigenous Population</th>
<th>National Population</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Household</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overcrowded**</td>
<td>50.0</td>
<td>23 (Maori)</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>43 (Pacific)</td>
<td></td>
</tr>
<tr>
<td><strong>Caregiver Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school graduate</td>
<td>50 (female)</td>
<td>52.0 (Pacific)</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td></td>
<td>65.4 (Maori)</td>
<td></td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>7 (female)</td>
<td>8.3 (Pacific)</td>
<td>22</td>
</tr>
<tr>
<td><strong>Risk Factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast feeding</td>
<td>73.8</td>
<td>58 (Maori)</td>
<td>65</td>
</tr>
<tr>
<td>Tobacco exposure (any)</td>
<td>33.3</td>
<td>59.3 (Maori)</td>
<td>35.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>48.1% (Pacific)</td>
<td></td>
</tr>
<tr>
<td>Premature</td>
<td>21.4</td>
<td>6.6 (Maori)</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.6 (Pacific)</td>
<td></td>
</tr>
<tr>
<td>Low birth weight (&lt;2500g)</td>
<td>23.1</td>
<td>6.8 (Maori)</td>
<td>5.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.6% (Pacific)</td>
<td></td>
</tr>
<tr>
<td>Hospitalized ALRI</td>
<td>92.9</td>
<td>6.7/1000 (Maori)</td>
<td>5.0 per 1000</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14.0/1000 (Pacific)</td>
<td></td>
</tr>
</tbody>
</table>

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# Table 2

**Generic primary, secondary and tertiary prevention for asthma, acute respiratory infections and bronchiectasis**

<table>
<thead>
<tr>
<th>PRIMARY PREVENTION</th>
<th>SECONDARY PREVENTION</th>
<th>TERTIARY PREVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>[Normal lung development and/or Remove modifiable risk factors]</td>
<td>[Early detection and appropriate treatment]</td>
<td>[Reduce morbidity, mortality, secondary complications and adverse events from medications]</td>
</tr>
</tbody>
</table>

**Antenatal**
- Avoidance of in-utero tobacco smoke exposure
- Antenatal care- prevent low birth weight

**Post natal**
- Breast feeding
- Nutrition (avoid growth faltering and later obesity)
- Avoidance of ETS, biomass combustion, petrol sniffing
- Socio-economic factors (poverty alleviation, sufficient housing, etc)
- Vaccinations (with correct timing)

- Make correct diagnosis
- Smoking cessation
- Poverty alleviation
- Optimisation of management of other conditions eg malnutrition, obesity, anaemia, diabetes
- Optimisation of management of other conditions eg malnutrition, anaemia, diabetes

- Adequate follow up
- Minimisation of medications for optimal control
- Yearly influenza
- Avoidance of ETS, smoke, biomass combustion, air pollution and respiratory toxicants (e.g. cannabis, solvents)
### Table 3: Primary, secondary and tertiary prevention specific for asthma, acute respiratory infections and bronchiectasis

<table>
<thead>
<tr>
<th>PRIMARY PREVENTION</th>
<th>SECONDARY PREVENTION</th>
<th>TERTIARY PREVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal lung development</td>
<td>Early detection and appropriate treatment</td>
<td>Reduce morbidity, mortality, secondary complications and adverse events from medications</td>
</tr>
<tr>
<td>Remove modifiable risk factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARIs, CSLD and bronchiectasis</td>
<td>Good follow up of all acute respiratory episodes</td>
<td>Assessment for treatable causes</td>
</tr>
<tr>
<td>Environmental health (hygiene, sanitation, reduce pollution)</td>
<td>Early diagnosis of bronchiectasis</td>
<td>Prevent exacerbations through optimization of treatment such as antibiotics, corticosteroids, bronchodilators, mucolytics, physiotherapy and airway clearance methods</td>
</tr>
<tr>
<td>Identifying predispositions to recurrent ARIs and CSLD (eg primary immunodeficiency states like hypogamma-globulinaemia)</td>
<td>Early treatment of exacerbations</td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td>Obtain good asthma control and prevent exacerbations though step-up and step-down therapies concordant with disease severity</td>
<td>Targeted asthma education and programs</td>
</tr>
</tbody>
</table>
CME SECTION

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(B) Complete the answers online, and receive your final score upon completion of the test.

(C) Should you successfully complete the test, you may download your accreditation certificate (subject to an administrative charge).

QUESTIONS

1. Which of the following statements is true about Indigenous children with asthma;

   (You can choose more than one item as correct)

   a. They have similar prevalence of asthma, compared to non-indigenous children.
   b. They are less likely to receive optimal treatment.
   c. They have a higher exposure to tobacco smoke.
   d. They have lower hospitalisation rates.
   e. They have better outcomes when culture-specific management approaches are used.

2. Which of the following statements is true about respiratory infections among indigenous children?

   a. Indigenous children have higher rates of hospitalised pneumonia than that documented in developing countries.
   b. Prevalence of severe bronchiolitis is lower but morbidity is higher among indigenous children than non-indigenous children.
   c. Chronic cough is uncommon and is generally over-reported in indigenous children.
   d. Respiratory infections early in life do not impact on future lung function as adults.
   e. Indigenous children share common factors of increased severity and poorer outcomes.
3. Which of the following is true of indigenous children with chronic suppurative lung disease (CSLD)?
   a. The same approach for diagnosis and management as non-indigenous children with bronchiectasis should be undertaken.
   b. They do not require further investigations as recurrent pneumonia is often the cause.
   c. Poor outcomes are related to being indigenous (as opposed to sub-optimal treatment).
   d. They may be misdiagnosed as having asthma.
   e. They often have had an antecedent pneumonia.

4. Prematurity and/or low birth weight of indigenous newborns is a known risk factor for increased severity of which of the following conditions? (Choose all options that are correct)
   a. Bronchiolitis.
   b. Bronchiectasis.
   c. Asthma.
   d. Protracted bacterial bronchitis.
   e. Recurrent pneumonia.

5. Indigenous children with an episode of hospitalised pneumonia;
   a. Have the same long term prognosis as non-indigenous children.
   b. Do not require follow-up.
   c. Are at increased risk of having CSLD/bronchiectasis.
   d. Should receive a short course of antibiotics (3 days).
   e. Is often caused by environmental tobacco smoke exposure
Reference List


(42) Panagiotou OA, Contopoulos-Ioannidis DG, Ioannidis JP. Comparative effect sizes in randomised trials from less developed and more developed countries: meta-epidemiological assessment. *BMJ* 2013;346:f707.


