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The burden of atopic dermatitis and bacterial skin infections among urban-living Indigenous children and young people in high-income countries: A systematic review

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Abstract

Background: A high burden of bacterial skin infections (BSI) is well documented in remote-living Indigenous children and young people (CYP) in high-income countries (HIC). Atopic dermatitis (AD) is the most common chronic inflammatory skin condition seen in CYP and predisposes to BSI. Despite the rate of urbanization for Indigenous people increasing globally, research is lacking on the burden of AD and BSI for urban-living Indigenous CYP in HIC. Indigenous people in HIC share a history of colonization, displacement and subsequent ongoing negative impacts on health.

Objective: To provide a global background on the burden of AD and BSI in urban-living Indigenous CYP in HIC.

Methods: A systematic review of primary observational studies on AD and BSI in English containing epidemiologic data was performed. MEDLINE, EMBASE, EMCARE, Web of Science, and PubMed databases were searched for articles between January 1990 and December 2021.

Results: From 2278 original manuscripts, 16 were included: seven manuscripts documenting eight studies on AD; and nine manuscripts documenting nine studies on BSI. Current and severe symptoms of AD were more common in urban-living Indigenous CYP in HIC compared with their non-Indigenous peers, with children having a higher prevalence than adolescents. Urban-living Indigenous CYP in HIC had a higher incidence of all measures of BSI compared with their non-Indigenous peers, and were over-represented for all measures of BSI compared with their proportion of the background population. Limitations include incomplete representation of all Indigenous populations in HIC.

Conclusion: A significant burden of AD and BSI exists in urban-living Indigenous CYP in HIC.

KEYWORDS
atopic dermatitis, bacterial skin diseases, children, epidemiology, high-income population, indigenous population, urban population

1 | INTRODUCTION

Globally, Indigenous youth face higher rates of health disparities than their non-Indigenous peers.[1] Bacterial skin infections (BSI) including impetigo, cellulitis and abscesses have a high burden in Indigenous children and young people (CYP). In a recent systematic review on the global epidemiology of impetigo, the highest median prevalence was seen in Australian Aboriginal children in remote Australia at 44.5% (IQR 34%–49%).[2] However, in that systematic review of 89 studies, no studies were available for Indigenous CYP in urban settings.

Atopic dermatitis (AD) is the most common chronic inflammatory skin condition in CYP; however, its prevalence varies across countries, geographic regions, and genetically similar populations.[3,4] Since 1990, the prevalence of AD in CYP has been rising.[5] A higher AD prevalence is generally observed in urban settings relative to rural populations.[3,6] This implicates environmental factors (i.e., industrialization and an urban lifestyle) in the pathogenesis of AD.[4]

Both BSI and AD adversely impact general health, school performance and overall quality of life in affected CYP and their families.[7,8] In AD, *S.aureus* colonizes the skin, exacerbating and contributing to more severe disease, as well as increasing the risk of BSI.[9] Untreated BSI can lead to serious complications including sepsis, post-infectious glomerulonephritis, and rheumatic heart disease.[10]

Recently, Asiniwasis et al highlighted AD and BSI to be a poorly documented crisis in Canada's Indigenous CYP.[11] This is consistent with the findings for Australian Aboriginal CYP[2] reflecting a history of colonization, dispossession and subsequent ongoing negative impacts on health shared by Indigenous peoples in high-income countries (HIC). Despite the rate of urbanization for Indigenous people increasing globally, research is lacking on the burden of AD and BSI for Indigenous CYP living in urban areas.[12] We aimed to assess the burden of AD and BSI in Indigenous CYP living on traditional lands that are now urban environments, as well as those who have moved from their traditional lands to urban settings.

2 | METHODS

We have reported according to the PRISMA 2020 statement and checklist.[13] The systematic review protocol is published online, with only two notable amendments to the original information: (1) Exclusion of scabies from the systematic review due to a paucity of published literature; and (2) Extension of the age of CYP from ≤18 to <20 years to better reflect the published literature (Systematic review registration: PROSPERO registration number: CRD42021277288).[14]

2.1 | Eligibility criteria

Primary observational studies with epidemiologic data on AD or BSI in urban-living Indigenous CYP (<20 years) in HIC were eligible for inclusion. This included cross-sectional studies or baseline assessments of longitudinal cohort studies (prospective or retrospective) with both population-based and institutional-based studies considered. Author-reported definitions of AD and BSI were used.

Given heterogeneity in how "urban" is defined globally, we used the definition as applied by the country at the time which the study occurred. The Degree of Urbanization classification was used to further categorize studies into cities, towns/suburbs, and villages.[15] We defined Indigenous peoples as the first occupants of the lands where they live, or from which they have been displaced. The Organization for Economic Co-operation and Development (OECD) was used to identify HIC.[16]

2.2 | Information sources

MEDLINE, EMBASE, EMCARE, Web of Science, and PubMed were searched (final search Dec 13, 2021). Google Scholar was used to identify relevant gray literature. The reference lists of all included articles were hand-searched for additional manuscripts.

2.3 | Search strategy

The pre-defined search strategy adapted for MEDLINE is provided, restricted to English language studies published between January 1990 and December 2021 (Appendix S1).

2.4 | Selection process

Duplicate records and conference abstracts were removed before two investigators (BR and HK) working independently reviewed titles and abstracts for relevance. Discrepancies between investigators were resolved by the senior investigator (AB). The corresponding author was e-mailed to request data relevant to our study population if not included.

2.5 | Data collection process

Independent data extraction was completed (BR and HK) on all papers meeting the inclusion criteria.

2.6 | Data items

Data were extracted for the primary outcome of AD and BSI disease frequency (proportion, prevalence, incidence). Where available, the secondary outcomes of clinical features, risk factors, co-morbidities, complications, and culture-proved etiology were extracted. Data were sought for the following variables—study period/site/design/aim, inclusion/exclusion criteria, diagnostic criteria, qualification of person conducting screening, method of data collection, recruitment and

sampling, number of participants, response rate, age range, sex, ethnicity, name/population/climate of town/city where study conducted, country where study conducted and definition of "urban" in that country.

2.7 | Risk of bias assessment

Where relevant, two authors (BR and HK) independently appraised the studies reporting prevalence data using the Joanna Briggs Institute (JBI) appraisal checklist.[17]

2.8 | Synthesis

The results are described separately under the themes of AD and BSI, synthesized into narrative form and tables. The one-year prevalence of AD was estimated from those reporting AD symptoms currently, or having had AD during the past 12 months (as per the International Study of Asthma and Allergies in Childhood ISAAC questionnaire).[5] The lifetime prevalence of AD was estimated from those reporting a history of "eczema ever." Proportion, prevalence, and/or incidence data were extracted for the BSI manuscripts. As anticipated, there were insufficient data to conduct meta-analysis.

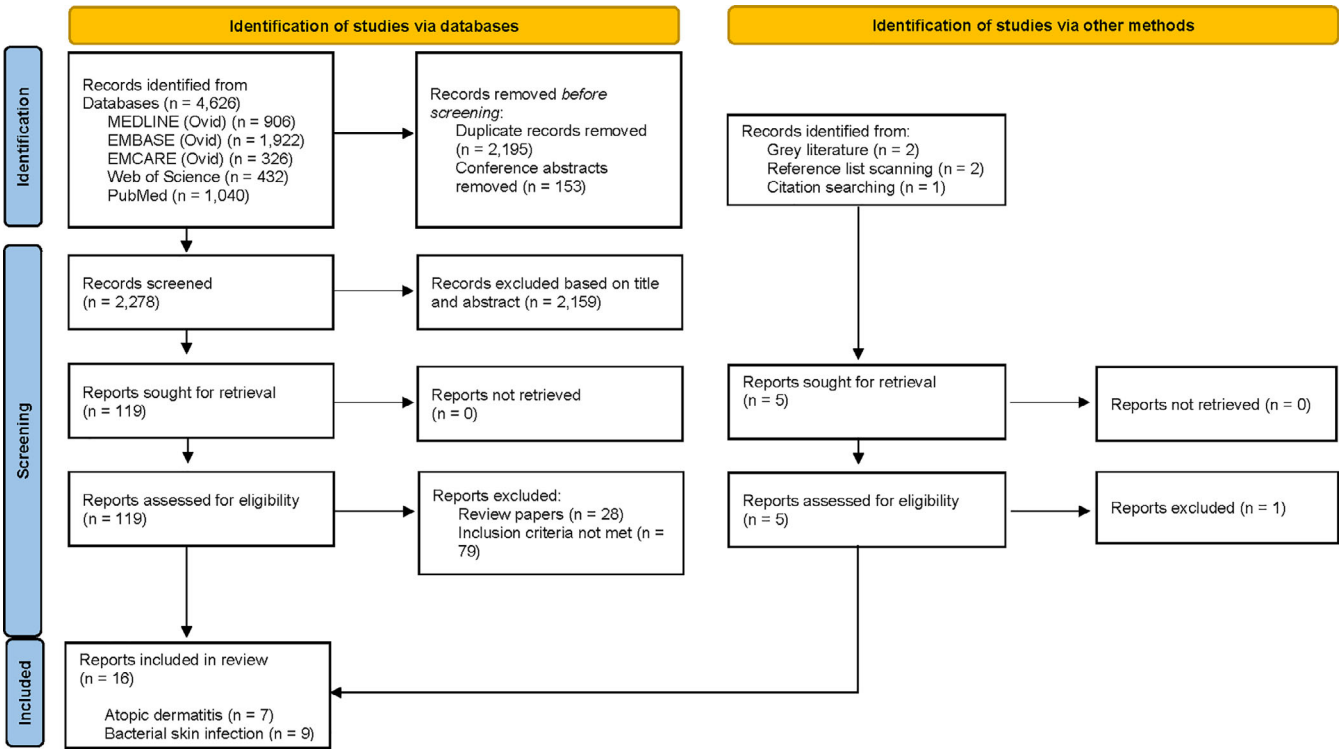

FIGURE 1 Flowchart of systematic review according to the PRISMA 2020 statement

TABLE 1 Number of articles on atopic dermatitis and bacterial skin infections by country, Indigenous population, degree of urbanization, region and Koppen climate classification

Number of articles	Country	Indigenous population	Degree of urbanization	Region[a]	Climate[b]
Atopic dermatitis					
2	Australia	Aboriginal and/or Torres Strait Islander	Cities[2]	Oceania	Temperate
1 (ISAAC-1 & ISAAC-3 data)	New Zealand	Māori	Cities[2]	Oceania	Temperate
2	Canada	Inuit	Towns/suburbs[2]	North America	Polar
2	Greenland	Inuit	Towns/suburbs,[2] villages[1]	North America	Polar
Bacterial skin infections					
6	Australia	Aboriginal and/or Torres Strait Islander	Cities,[5] unclassifiable[1]	Oceania	Temperate,[5] Arid[1]
3	New Zealand	Māori	Cities,[2] towns/suburbs[1]	Oceania	Temperate

[a]Region as defined by United Nations Statistics Division.
[b]Climate as defined by Koppen Climate Classification.

TABLE 2 Atopic dermatitis: Study characteristics

Author site, Country	Age range (y)	Study period	Study design	Participants (*% Indigenous*)	Response rate (%)	F/M (%)	Diagnostic method	One-year AD prevalence in urban Indigenous CYP (*vs. non-Indigenous*)	Current severe AD prevalence in urban Indigenous CYP (*vs. non-Indigenous*)	Lifetime AD prevalence in urban Indigenous CYP (*vs. non-Indigenous*)	Cumulative incidence of AD in urban Indigenous CYP (*vs. non-Indigenous*)
Hall, K. et al *Caboolture Community Medical*, Australia	0–5	2013–2015	Cross-sectional analysis of baseline data in prospective cohort	180 (*100% Aboriginal*)	44.7	49/51	Parent-reported or clinical record-"history of eczema ever"	-	-	13.3%	-
Glasgow, N. et al *Schools in Australian Capital Territory*, Australia	4–6	1999–2001	Cross-sectional questionnaire survey	10,821 (*2% Aboriginal*)	80	50/50	Parent-reported-"history of eczema ever"	-	-	25% (vs. *32% non-Indigenous*)	-
Clayton, T. et al (ISAAC-1) *Schools in Auckland, Christchurch, Nelson, Bay of Plenty*, New Zealand	6–7	1992–1993	Cross-sectional questionnaire survey	11,393 (*20% Māori*)	91.4	51/49	Parent-reported-ISAAC 1 questionnaire	17.9% (vs. *12.6% European*)	3.9% (vs. *0.9% European*)	25.6% (vs. *26.5% European*)	-
Clayton, T. et al (ISAAC-3) *Schools in Auckland, Christchurch, Nelson, Bay of Plenty*, New Zealand	6–7	2001–2003	Cross-sectional questionnaire survey	10,873 (*24% Māori*)	85.2	50/50	Parent-reported-ISAAC 3 questionnaire	17.2% (vs. *13.8% European*)	3% (vs. *0.8% European*)	32.1% (vs. *34.3% European*)	-
Clayton, T. et al (ISAAC-1) *Schools in Auckland, Christchurch, Nelson, Bay of Plenty, Wellington*, New Zealand	13–14	1992–1993	Cross-sectional questionnaire survey	15,460 (*19% Māori*)	92	54/46	Self-reported-ISAAC 1 questionnaire	16.9% (vs. *11.7% European*)	3.7% (vs. *1.3% European*)	26.1% (vs. *27.4% European*)	-
Clayton, T. et al (ISAAC-3) *Schools in Auckland, Christchurch, Nelson, Bay of Plenty, Wellington*, New Zealand	13–14	2001–2003	Cross-sectional questionnaire survey	13,317 (*19% Māori*)	89.2	52/48	Self-reported-ISAAC 3 questionnaire	10.4% (vs. *7.3% European*)	2.1% (vs. *0.8% European*)	26.4% (vs. *28.8% European*)	-
Ahmed, A. et al *Schools in Iqaluit*, Canada	6–7	2015–2016	Cross-sectional questionnaire survey	44 (*55% Inuit*)	33.8	41/59	Parent-reported-modified ISAAC questionnaire	25% (vs. *14.3% non-Inuit*)	-	20.8% (vs. *28.6% non-Inuit*)	-
Ahmed, A. et al *School in Iqaluit*, Canada	13–14	2016–2017	Cross-sectional questionnaire survey	58 (*59% Inuit*)	53.7	52/48	Self-reported-modified ISAAC questionnaire	14.7% (vs. *50% non-Inuit*)	-	17.6% (vs. *66.7% non-Inuit*)	-
Tamsmark, T. et al *Sisimiut Health Centre*, Greenland	0–2	1996–1998	Prospective open cohort study	143 (*83% Inuit*)	82.2	52/48	Clinical exam-Hanifin & Rajka criteria	-	-	-	16.4% (vs. *27.3% mixed/ Caucasian*)
Schultz Larson, F. et al *Schools in Nuuk, Sisimiut, Ilulisat, Uammannaq, Tasiilaq*, Greenland	7–8	2000	Cross-sectional questionnaire survey	622 (*88% Inuit*)	65	-	Parent-reported-validated questionnaire	-	-	13.9% (vs. *14.5% Danes*)	-
Median								17.05% (vs. *13.2%*)	3.35% (vs. *0.85%*)	25% (vs. *28.7%*)	

TABLE 3 Bacterial skin infections: Study characteristics

Author site, Country	Age range (y)	Study period	Study design	Participants	F/M (%)	Skin infection(s) assessed	Proportion data in urban Indigenous CYP	Prevalence data in urban Indigenous CYP	Incidence data in urban Indigenous CYP
Zubrick, S. et al *Western Australia*, Australia	<18	2000–2002	Cross-sectional questionnaire survey completed by carer.	5289	52/48	Recurring skin infections; incl. School sores, scabies.	-	Prevalence of recurring skin infections = 7.1% (720/10,200)	-
Abdalla, T. et al *Western Australian birth cohort*, Australia	<16	1996–2012	Retrospective population-based cohort study of live-births, with data linkage to hospitalizations for ALL skin infections.	469,589	59/51	ALL skin infections; incl. Scabies, impetigo, pyoderma, cellulitis, abscess, fungal, lice, other.	Proportion of hospitalizations for ALL skin infections = 24.5% (11,864/7586)	-	Hospital admission rates for ALL skin infections'' in: • <16 year = 20.8/1000 child-years (95% CI 19.8–21.7). • <1 year = 46.6/1000 child-years (95% CI 42.7–50.9).
Whitehall, J. et al *The Base Hospital, Mt Isa*, Australia	<14	2006–2010	Retrospective audit of "scabies" admissions to a regional hospital.	85 *(68 urban admissions)*	50/50	'Scabies'; incl. Pyoderma, infected scabies.	Proportion of hospital admissions for "scabies" = 100% (68/68)	-	-
Salleo, E. et al *Perth Children's Hospital*, Australia	<16	2018	Retrospective audit of cellulitis cases at a tertiary pediatric hospital.	302 *(282 urban cases)*	40/60	Cellulitis.	Proportion of cellulitis cases = 10.6% (30/282)	-	-
Haysom, L. et al *New South Wales Juvenile Custody*, Australia	12–20	2013–2015	Prospective cohort study of incarcerated youth presenting to custodial health centers with a SSTI.	72 *(77 total SSTIs, 33 MRSA SSTIs)*	8/92	SSTI; incl. Boil, abscess, impetigo, cellulitis, surgical wound or other.	Proportion of: • SSTIs = 59.7% (46/77) • MRSA SSTIs = 81.8% (27/33).	-	Incidence rate of SSTI = 13 SSTI per 1000 Aboriginal custodial admissions.
Thomas, Susan. et al *Hunter New England Local Health District (HNELHD)*, Australia	<20	2008–2014	Linked data–presentations to ED and skin/wound swabs within 2-days.	1222 *(327 urban swabs)*	43/57	CA-MRSA skin infections.	Proportion of CA-MRSA positive skin swabs = 23.9% (78/327)	-	-
O'Sullivan, C. et al Gisborne Hospital, New Zealand[a]	<15	2006–2007	Retrospective audit of "serious skin infections" overnight admissions to a secondary referral center hospital.	161 *(163 serious skin infection admissions)*	46/54	Serious skin infections; incl. Cellulitis, abscess, others.	Proportion of hospital admissions for serious skin infections = 84% (135/161)	-	-

(Continues)

TABLE 3 (Continued)

Author site, Country	Age range (y)	Study period	Study design	Participants	F/M (%)	Skin infection(s) assessed	Proportion data in urban Indigenous CYP	Prevalence data in urban Indigenous CYP	Incidence data in urban Indigenous CYP
Williamson, D. et al *Auckland District Health Board*, New Zealand[a]	<15	2007–2010	Retrospective audit of CA *S.aureus* SSTI admissions to a tertiary-level university-affiliated institution.	1860	47/53	CA-*S.aureus* SSTI	Proportion of CA-*S. aureus* SSTI = 27.8% (517/1860)	-	Annual incidence of CA-*S. aureus* SSTI in urban Maori youth = 1488/100,000 population
Thomas, Sally. et al *Auckland region*, New Zealand[a]	<20	2010–2016	Cross-sectional study of skin swab data in a large urban region. Data linked to first known hospitalizations for ARF.	239,494 (*377,410 total swabs*)	-	Wound and/or skin swabs. First known hospitalization for ARF.	-	-	Incidence rates for: • GAS detection in skin swabs = 9.7/1000 p-y • ARF initial hospitalization = 12.4/100,000 p-y

[a]Study setting is an urban center, however participant place of residence not reported.

3 | RESULTS

3.1 | Study selection

In total, 4626 records were identified with 2348 duplicates and conference abstracts removed. An additional 2159 were excluded for irrelevance on title and abstract screening. Following full text review of 119 papers, 107 were excluded. An additional five records were identified; four of which met inclusion criteria. From this, 16 manuscripts (seven manuscripts documenting eight studies on AD and nine manuscripts documenting nine studies on BSI) from four countries over a 26-year period were included (Figure 1, Table 1).

3.2 | Atopic dermatitis (AD)

Two studies were conducted in each of the following HIC: Australia,[18,19] New Zealand (ISAAC phase 1 and ISAAC phase 3),[5] Canada[20,21] and Greenland[22,23] (Table 2). Of the 62,900 CYP, 81.1% (51,043) were from New Zealand, 17.5% (11,001) from Australia, 1.2% (754) from Greenland, and 0.2% (102) from Canada. Indigenous participants accounted for 18.1% (11,372) with the proportion of Indigenous CYP in individual studies ranging from 2%[19] to 100%.[18]

Six studies were cross-sectional questionnaire surveys distributed to families through schools,[5,19–22] and two were prospective open cohort studies conducted in clinical settings.[18,23] In seven studies, AD diagnosis was based on survey responses and in one study, diagnosis was based on clinical examination.[23] Prevalence data (1-year and/or lifetime) were available in seven studies[5,18–22] and cumulative incidence data in the eighth.[23] No study contained data on the secondary outcomes.

The prevalence of AD varied across the urban-living Indigenous populations. Overall, AD prevalence was higher in the studies set in "cities" (as defined by the Degree of Urbanization),[5,18,19] compared with those in "towns/suburbs" and "villages."[20–23] Similarly, a higher AD prevalence was noted in the Oceania region with a temperate climate,[5,18,19] compared with the polar climate of the North America region.[20–23]

Across all studies with comparison data, lifetime prevalence of AD in urban-living CYP was slightly higher in the non-Indigenous participants (median prevalence 28.7%), compared with Indigenous (median prevalence 25%). However, with the exception of one study of small sample size,[21] one-year AD prevalence and current severe AD prevalence were greater in Indigenous CYP (median prevalence 17.05% and 3.35%, respectively) compared with non-Indigenous (median prevalence 13.2% and 0.85%, respectively). Within the Indigenous CYP, children (6–7 years) displayed a higher one-year AD prevalence than adolescents (13–14 years).[5,20]

AD studies were generally of good quality with two of seven manuscripts rated as high across all matrices,[5,23] and the remaining five manuscripts rated as poor across two domains. Two manuscripts had an inadequate sample size,[20,21] poor details on setting (2),[19,22] non-validated diagnostic method (2),[18,19] and inadequate response/participation rate (4).[18,20–22]

TABLE 4 Bacterial skin infections: Comparison of study participants to the background urban Indigenous CYP population

Author	Study period	BSI	Proportion of urban-living Indigenous CYP in study population	Proportion of urban-living Indigenous CYP in background population at time of study [reference source]	Rate Ratio
Abdalla, T. et al	1996–2012	All skin infections	24.5% (1864/7586)	3.6% (15,402/444,281) [2011 Australian census data]	6.8
Whitehall, J. et al	2006–2010	Scabies/pyoderma	100% (68/68)	25% (1273/5015) [2006 Australian census data]	4.0
Salleo, E. et al	2018	Cellulitis	10.6% (30/282)	3% (12,558/415,332) [2016 Australian census data]	3.5
Thomas, Susan. et al	2008–2014	CA-MRSA skin swabs	23.9% (78/327)	4.8% (1663/34,360) [2011 Australian census data]	5.0
O'Sullivan, C. et al[a]	2006–2007	Serious skin infections	84% (135/161)	58% [O'Sullivan C & Baker MG, *N Z Med J 2012*; 125:70–79]	1.5
Williamson, D. et al[a]	2007–2010	CA-MRSA SSTI	27.8% (517/1860)	14% [Data from manuscript]	2.0

[a]Study setting is an urban center, however participant place of residence not reported.

3.3 | Bacterial skin infections (BSI)

All studies were set in Oceania with a predominant temperate climate—six from Australia and three from New Zealand (Table 3). Using the Degree of Urbanization, seven studies were conducted in "cities" and one in "towns/suburbs."[24] The ninth study was set across nine juvenile custodial centers, considered to be a simulated urban environment (based on population size/density and infrastructure) for the purpose of this SR.[25] Epidemiologic data on BSI specific for urban-living Indigenous CYP was available in the six included Australian studies (unpublished data for one[26]). While the three included New Zealand studies were set in an urban environment, place of residence was not reported; hence, it is estimated a proportion (up to 25%) of these participants may be rural-living.[24,27,28]

Across the nine manuscripts, there was significant heterogeneity in both study design and skin infection assessed. One study was a cross-sectional questionnaire survey capturing prevalence data on recurring skin infections.[29] Four studies were retrospective audits of hospital admission data on cellulitis,[26] scabies/pyoderma,[30] community-associated (CA) *S.aureus* skin and soft tissue infection (SSTI),[28] and serious skin infections.[24] Three studies used data linkage to identify "all" skin infection-associated hospitalizations,[31] CA methicillin-resistant *S.aureus* (CA-MRSA) skin infections,[32] group A streptococcal (GAS) detection in skin swabs and primary acute rheumatic fever (ARF) hospitalizations. The final study investigated a prospective cohort of incarcerated youth presenting to custodial health centers with SSTI.[25]

Proportion data were available in six studies:[24,26–28,30,32] prevalence data in one,[29] and incidence data in four.[25,27,28,31] Proportion data are summarized in Table 4, revealing urban-living Indigenous CYP to be over-represented for all measures of BSI compared with their proportion of the background population. For urban-living Australian Aboriginal CYP, the median rate ratio for BSI incidence was 4.5 times higher than non-Aboriginal CYP. While for urban-living Māori CYP, it was 1.75 times higher than non-Māori CYP.

Urban-living Aboriginal CYP in Australia had a 7.1% prevalence of "recurring skin infections, such as school sores or scabies"[29] and a ten times higher hospitalization rate for "all skin infections" (20.8/1000 child-years; 95% confidence interval [CI] 19.8–21.7) compared with their non-Aboriginal peers (2.2/1000 child-years; 95% CI 2.1–2.2).[31] Hospitalizations were mostly due to abscesses, cellulitis, and scabies with the highest rates seen in infants and during the summer months. In the custodial setting, the incidence rate of SSTI was 13 per 1000 Aboriginal custodial admissions, compared with nine per 1000 non-Aboriginal custodial admissions; with SSTIs in Aboriginal youth almost six times more likely to be due to MRSA than in non-Aboriginal youth (AOR 5.92, 95% CI 2.03–17.21).[25]

Urban-living Māori CYP in New Zealand had a three times higher annual incidence of CA-*S.aureus* SSTI (1488/100,000 population) compared with 552/100,000 for "all youth,"[28] and four times greater GAS detection in swabs compared with European/other ethnicities (RR 4.0; 95% CI 3.9–4.2)—highest in those under five years and most socio-economically deprived.[27] Linking GAS skin swab data with primary ARF hospitalization data, urban Māori youth were 30 times more likely to be

hospitalized for ARF than European/other ethnicities (RR 30.3, 95% CI 19.5–46.9).

BSI studies were generally of good quality. However, given the heterogeneity of study design, no single quality appraisal instrument could be applied to all records.

4 | DISCUSSION

This is the first systematic review to investigate the burden of AD and BSI among urban-living Indigenous children in HIC. We found:

i. Current and severe symptoms of AD were more common in urban-living Indigenous CYP in HIC compared with their non-Indigenous peers, with children having a higher prevalence than adolescents.
ii. Urban-living Indigenous CYP in HIC had a higher incidence of all measures of BSI compared with their non-Indigenous peers, and were over-represented for all measures of BSI compared with their proportion of the background population, although these data were available only for Australia and New Zealand.

We confirm AD is common among urban-living Indigenous children in HIC with current symptoms and current severe symptoms higher than their non-Indigenous peers. This may suggest under-treatment of AD, reflecting the socioeconomic disadvantage that disproportionately affects Indigenous people, creating financial barriers to primary and dermatologic care, prescription treatments, and costly skin care regimens. Secondary BSI of AD may also give rise to the higher prevalence of current (and severe) AD seen in urban-living Indigenous youth with environmental factors such as poverty, close-living, and reduced access to household health hardware contributing.[11] The higher prevalence of current AD seen in urban-living Indigenous children compared with adolescents may reflect the natural history of AD or differences in study methodology (parental reporting for children vs. self-reporting for adolescents).[5,21]

In our SR, the search strategy for BSI was kept broad to include all bacterial SSTIs, including epidemiologic manuscripts focusing on clinical presentation as well as those highlighting diagnostic methods. There was significant heterogeneity in the methodology and results of these papers; however, the overriding conclusion from the nine included records is urban-living Indigenous CYP have a higher incidence of all measures of BSI compared with their non-Indigenous peers and are over-represented for all measures of BSI compared with their proportion of the background population. Again, poverty and the social determinants of health are likely to influence this high burden.

There were limitations to our study. Firstly, heterogeneity in the global definition of "urban" created challenges in screening for suitable manuscripts. Secondly, for studies based in city hospitals, it was not always clear whether participants were urban-living. Thirdly, the included BSI records had significant heterogeneity in study design; precluding the use of a quality appraisal tool. Finally, there was incomplete representation of all Indigenous populations in HIC; specifically, no records from the United States, Norway, Sweden, Finland, or Denmark met our inclusion criteria. Further, the only BSI studies identified were from Australia and New Zealand, impacting on generalizability of these results.

5 | CONCLUSION

This SR highlights a significant burden of AD and BSI among urban-living Indigenous CYP in HIC. We echo the call to action for more studies to better understand the impact of AD and BSI in urban-living Indigenous CYP, noting specifically the poor representation of North American Indigenous CYP in the published literature.[11] Co-designed research with Indigenous communities will help identify sustainable community-wide strategies for the prevention and treatment of AD and BSI in urban-living Indigenous youth.

AUTHOR CONTRIBUTIONS

Design and conceptualization: Bernadette M Ricciardo, Asha C. Bowen. Data extraction: Bernadette M Ricciardo, Heather-Lynn Kessaris, Asha C. Bowen. Manuscript drafting and review: all authors.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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