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Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

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Title: Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

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Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

Abstract

Objectives: To investigate differences in demographic and clinical characteristics of Aboriginal and non-Aboriginal children aged 0-4 years hospitalised for unintentional poisoning in New South Wales (NSW), Australia.

Design and setting: Retrospective whole-of-population cohort analysis of linked hospital and mortality data for 2000-2014

Participants: All children (Aboriginal and non-Aboriginal) under the age of five years who born in a hospital in NSW from 2000 to 2009

Outcomes: The primary outcome was hospitalisation for unintentional poisoning. Logistic regression was used to estimate odds of poisoning hospitalisation for Aboriginal and non-Aboriginal children. Poisoning agents and clinical outcomes were compared by Aboriginality.

Results: The cohort included 767,119 children, including 28,528 (3.7%) Aboriginal children. Aboriginal children had approximately three times higher rates of hospitalised poisoning (1.34%) compared to non-Aboriginal children (0.41%). Poisoning incidence peaked at 2-3 years of age. Male sex, socioeconomic disadvantage and geographic remoteness were associated with higher odds of poisoning hospitalisation for Aboriginal and non-Aboriginal children, but associations with disadvantage and remoteness were statistically significant only for non-Aboriginal children. Most (83%) poisonings were caused by pharmaceutical agents. Few Aboriginal and non-Aboriginal children had repeat admissions for poisoning; most had a length of stay of one day or less. Only 8% of poisoning admissions involved contact with a social worker.

Conclusion: Commonly used medications in the general population contribute to poisonings amongst both Aboriginal and non-Aboriginal preschool aged children. This study highlights a need to develop culturally safe poisoning prevention strategies and policies.

Strengths and limitations of this study

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- This study's strengths include its focus on preschool-aged children, large sample size due whole-of-population design, and minimal recall bias due to the use of routinely collected administrative data.
- However, the use of administrative data limited the available covariates, so risk factors such as parent demographics, supervision and storage practice, and clinical information such as symptoms and treatment details, could not be examined.
- Children were identified as Aboriginal if they had been recorded at least once as Aboriginal ('ever-identified' algorithm), which enhances the proportion of Aboriginal children recorded using hospital data but may introduce bias in that sicker children with more frequent hospitalisations have more opportunities for identification.
- Our analysis of poisoning agents was limited to the International Classification of Diseases 10th edition (ICD-10) taxonomy, and in some circumstances it was not possible to determine specific agents, for example, 'other' categories.

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Introduction

Poisoning is a leading cause of childhood injury worldwide.[1] Preschool children (0-4 years) have a higher risk of poisoning related hospital admissions [2-4] due to their developmental stage [5] and are a priority for prevention. Amongst preschool children, poisoning incidence peaks at two to three years of age,[3, 5-8] reflecting their increased mobility and exploratory skills.[5, 6, 9]

Pharmaceutical substances, such as over-the-counter and prescription medications, cause most unintentional poisonings in Australia.[4] Amongst preschool children in Australia, the most common causes of hospitalised pharmaceutical poisoning include analgesic, anti-epileptic, psychotropic, and other systemic and cardiovascular medications.[3, 4, 7, 10-12]

Various demographic and socioeconomic risk factors contribute to poisoning amongst preschool children. Factors relating to the child (male, higher birth order), parents (younger age, perinatal depression, alcohol misuse), unsafe storage and socioeconomic deprivation are associated with increased poisoning risk.[1-3, 7, 8, 13-16] In Australia, remote areas also have higher poisoning rates.[4, 16]

Poisoning represents one of the largest Indigenous child health inequalities in high-income countries, with rate ratios for hospitalisation and mortality ranging from 1.1 to 3.9 and 1.2 to 15.4 respectively.[17] A recent whole-of-population study in New South Wales, Australia reported that Aboriginal children had nearly three times higher risk of poisoning hospitalisation compared with non-Aboriginal children.[18] Existing studies about poisoning amongst Indigenous children in high-income countries have limitations, such as the lack of a non-Indigenous comparison group and wide age ranges up to 18 years old that do not differentiate younger children.[19, 20] Previous Australian studies of poisoning amongst preschool children in the general population have not focussed on Aboriginal children.[3, 5, 7, 22] The current literature about poisoning amongst Aboriginal children in Australia is limited in its examination of risk factors and clinical outcomes.[11, 12, 18, 21] Specific poisoning agents and clinical outcomes of poisoning for Aboriginal children have not been described.[2, 10, 16, 23]

This is the first study to examine the characteristics of unintentional poisoning amongst preschool-aged Aboriginal Australian children. The study aimed to answer questions that will inform prevention efforts:

1) Are the demographic risk factors for poisoning (sex, socioeconomic status and geographic remoteness) similar for both Aboriginal and non-Aboriginal children?
2) Which agents most frequently result in poisoning hospitalisation and are they different for Aboriginal and non-Aboriginal children?

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3) Are the clinical outcomes from hospitalised poisoning similar for Aboriginal and non-Aboriginal children?

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Methods

Study design and setting

This was a retrospective, whole-of-population cohort study using linked hospital and mortality data for NSW, Australia for the period 2000 to 2014. According to the 2011 Census, NSW had a population of 6,917,658 people, of whom 172,624 (2.5%) were Aboriginal or Torres Strait Islander.[24]

Data Sources

This study was part of the Indigenous Health Outcomes Patient Evaluation (IHOPE) project which compares health outcomes for Aboriginal and non-Aboriginal people in NSW using linked hospital and mortality data **(**Figure 1**)**. Hospital data were sourced from the Admitted Patient Data Collection (APDC) and mortality data from the NSW Register of Births, Deaths and Marriages (RBDM). Probabilistic data linkage was performed by the NSW Centre for Health Record Linkage (http://www.cherel.org.au). De-identified, linked datasets were supplied to the researchers.

The RBDM contains details for all deaths in NSW. The APDC contains records for all inpatient separations (discharges, transfers and deaths) for all public and private hospitals and day procedure centres in NSW. Each separation record contains information about patient demographics, diagnoses, and procedures. Diagnoses are coded according to the International Classification of Diseases and Related Problems Australian Modification, tenth revision (ICD-10-AM). Separations are hereafter referred to as hospital admissions.

Participants

Children born in a NSW hospital between June 1, 2000 and March 31, 2009 were followed from birth until their fifth birthday. Birth records were identified from the APDC dataset using the criteria of 'live born infant' (ICD-10-AM code Z38) in any diagnosis field. This birth cohort has been previously described.[18]

Aboriginal status

Aboriginal status is routinely recorded in the APDC. The main analyses used an 'ever identified' algorithm that coded children as Aboriginal if they were ever identified as Aboriginal, yielding higher proportions of Aboriginal children compared to the most recent record or birth record (Supplement 1).[25] To test the sensitivity of this identification method, all analyses were repeated using Aboriginality defined solely on the child's birth record.

Study variables

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The main outcome was hospitalisation for unintentional poisoning, defined as an acute accidental exposure to, or overdose of, a toxic substance, whether accessed by a child or incorrectly administered by another person (wrong dosage, agent or frequency). The study focussed on ingested poisoning agents likely to require similar prevention approaches, including pharmaceutical (over-the-counter and prescription) and non-pharmaceutical agents, and excluding environmental exposures (Supplement 2). Poisoning cases were identified by a primary diagnosis of injury with an ICD-10-AM code T36-65, excluding toxic effect of metals (T56), carbon monoxide and other gases (T58-59) and noxious substances, venoms and food substances (T61-64).

A poisoning admission was defined from the start date of an episode of care, to the date of separation or death. Multiple hospital admissions within a short time could be due to follow-up visits, transfer between hospitals, or change in care type within a hospital, related to the same poisoning event. Records falling within 75 days after the initial poisoning admission were excluded, based on the published 'clear zone' for poisoning.[26]

Available demographic variables included child's age, sex and area-level variables for socioeconomic disadvantage (Australian Bureau of Statistics Socioeconomic Indices for Areas (SEIFA) Index of Relative Socioeconomic Advantage and Disadvantage (IRSAD) [27]) and remoteness (Accessibility/Remoteness Index of Australia (ARIA) [28]) based on the child's statistical local area of residence at birth. Due to small numbers in some categories, IRSAD was divided into tertiles, and ARIA into four categories 'Major Cities', 'Inner Regional', 'Outer Regional' and 'Remote/Very Remote' as used previously.[18] The age of first poisoning hospitalisation was analysed by single year intervals for Aboriginal and non-Aboriginal children.

Poisoning agents were included if they were recorded in any diagnosis field (up to 50 fields). Clinical outcomes (repeat admissions, length of stay (LOS), procedures) were analysed for all poisoning admissions. LOS included the total number of hospital days related to the initial admission, and any subsequent admissions within 75 days. Procedures were classified at the 'block' level according to the Australian Classification for Health Interventions (ACHI), 8th edition.[29]

Statistical analysis

Differences in poisoning incidence were compared by demographic characteristics using chi squared tests, stratified by Aboriginality. The crude and adjusted odds of poisoning hospitalisation associated with demographic characteristics were estimated using logistic regression. Separate models were built for Aboriginal and non-Aboriginal children. Fully adjusted models included the variables sex, socioeconomic disadvantage and remoteness.

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Amongst children with a poisoning hospitalisation, incidence was examined by year of age. The classes of poisoning agents and the ten most common individual agents were examined across all poisoning admissions. Differences in poisoning agents (both classes and individual agents) by Aboriginality were tested using chi squared tests. The number and types of procedures and LOS were examined for all poisoning admissions, and compared by Aboriginality. Data was prepared with SAS Version 9.4 (SAS Institute) and analysed in Stata Version 14 (StataCorp).

Ethics Approval

Ethics approval was granted by the New South Wales Population & Health Services Research Committee (2009/04/141) and the Aboriginal Health and Medical Research Council Committee (684/09).

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Results

Study Population

The cohort included 767,119 children born in NSW between July 1, 2000 and March 31, 2009, of whom 28,528 (3.7%) were Aboriginal (Figure 2). From 2000-2014, there were 3,757 hospital admissions with a primary diagnosis of poisoning. Of these, 321 fell within the 75-day clear zone and were not analysed as new admissions. This left 3,436 poisoning admissions for 3,385 individual children, including 382 (11%) Aboriginal children. Aboriginal children were significantly overrepresented amongst children who had been poisoned (χ^2=543.6, p<0.001).

Demographic Characteristics

Poisoning admission by demographics characteristics for all children in the cohort are shown in Table 1. More Aboriginal than non-Aboriginal children lived in the most disadvantaged areas and in Remote or Very Remote areas. The proportion of children hospitalised for poisoning was higher for boys compared to girls, and increased with increasing socioeconomic disadvantage and increasing remoteness for both Aboriginal and non-Aboriginal children.

Incidence by one-year age intervals was similar amongst Aboriginal and non-Aboriginal children. Few Aboriginal or non-Aboriginal children had poisoning admissions at under one year of age (6%, n=21 and 7%, n=220 respectively) or older than four years (10%, n=40 and 6%, n=174). Poisoning admissions increased from the age of one (25%, n=96 and 32%, n=952), peaked at age two (40%, n=152 and 40%, n=1,211) and declined at age three years (19%, n=73 and 15%, n=446).

Crude and adjusted odds for poisoning hospitalisation for Aboriginal and non-Aboriginal children are shown in Figure 3. Male sex was significantly associated with increased risk of poisoning for both Aboriginal (adjusted odds ratio [aOR] 1.27, 95% CI 1.03-1.56) and non-Aboriginal children (aOR 1.17, 95% CI 1.09-1.26). Poisoning risk also increased with increasing socioeconomic disadvantage and increasing geographic remoteness. This trend was similar for both Aboriginal and non-Aboriginal children, although confidence intervals were wider for Aboriginal children.

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Table 1: Poisoning hospitalisations by demographic characteristics, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014

	Aboriginal children			Non-Aboriginal children			All children		
	N (column %)	N poisoned (row %)	p	N (column %)	N poisoned (row %)	p	N (column %)	N poisoned (row %)	p
Total	28,528 (100)	382 (1.34)		738,591 (100)	3,003 (0.41)		767,119 (100)	3,385 (0.44)	
Sex			0.03			<0.001			<0.001
Female	13,472 (47.2)	159 (1.18)		358,899 (48.6)	1.342 (0.37)		372,371 (48.5)	1,501 (0.40)	
Male	15,056 (52.8)	223 (1.48)		379,692 (51.4)	1.661 (0.44)		394,748 (51.5)	1,884 (0.48)	
Area-level disadvantage			0.05			<0.001			<0.001
1 - Most disadvantaged	18,857 (66.1)	259 (1.37)		241,835 (32.7)	1,315 (0.54)		260,692 (34.0)	1,574 (0.60)	
2	7,558 (26.5)	107 (1.42)		250,639 (33.9)	1,003 (0.40)		258,197 (33.7)	1,110 (0.43)	
3 - Least Disadvantaged	2,113 (7.4)	16 (0.76)		246,117 (33.3)	685 (0.28)		248,230 (32.4)	701 (0.28)	
Remoteness			0.11			<0.001			<0.001
Major city	9,056 (31.7)	105 (1.16)		492,228 (66.6)	1,659 (0.34)		501,284 (65.3)	1,764 (0.35)	
Inner regional	10,058 (35.3)	132 (1.31)		182,366 (24.7)	880 (0.48)		192,424 (25.1)	1,012 (0.53)	
Outer regional	6,852 (24.0)	110 (1.61)		58,709 (7.9)	419 (0.71)		65,561 (8.5)	529 (0.81)	
Remote/Very Remote	2,562 (9.0)	35 (1.37)		5,288 (0.7)	45 (0.85)		7,850 (1.0)	80 (1.02)	

P-values are from chi-squared tests, testing the null hypothesis that poisoning admissions are distributed equally across the levels of each covariate.

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Poisoning agents

Pharmaceutical agents caused most poisonings for both Aboriginal (83%) and non-Aboriginal (81%) children (Table 2). The leading classes of poisoning agents were similar by Aboriginality: nonopioid analgesic, antipyretic and antirheumatics (including paracetamol and ibuprofen); antiepileptic, sedative-hypnotic and antiparkinsonism medications; psychotropics; and cardiovascular medications (Table 2). Aboriginal children had a higher proportion of poisoning admissions from cardiovascular medications than non-Aboriginal children (14% and 11%, p=0.032), whilst non-Aboriginal children had a higher proportion from nonopioid analgesic, antipyretic and antirheumatics (15% and 8%, p<0.001) and autonomic nervous system medications (8% and 5%, p=0.048).

Benzodiazepines (11%) and 4-aminophenol derivatives (paracetamol) (11%) were the two most common individual poisoning agents (Table 2). Together with other antihypertensives not elsewhere classified, they accounted for one in three poisoning admissions. Benzodiazepines and other antihypertensives not elsewhere classified were the two most common individual agents for Aboriginal children and caused a higher proportion of admissions for Aboriginal than non-Aboriginal children (benzodiazepines: 14% and 10%, p=0.025; other antihypertensives not elsewhere classified: 10% and 6%, p=0.002).

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Table 2: Poisoning agents by class and ten most frequent individual agents, for Aboriginal and Non-Aboriginal children, New South Wales, 2000-2014

ICD-10 Diagnosis Code	Total N (%)	Aboriginal N (%)	Non-Aboriginal N (%)	p
T36-65: All poisoning admissions	3,436 (100)	391 (11)	3,045 (89)	
T36-50: Pharmaceutical[a]	2,859 (83)	318 (81)	2,541 (83)	0.291
T51-65: Non-pharmaceutical[a]	588 (17)	76 (19)	512 (17)	0.195
Pharmaceutical Agents				
T36-37: Antibiotics, anti-infectives, anti-parasitics[b]	47 (1)	7 (2)	40 (1)	0.445
T38: Hormones and synthetic substitutes	141 (4)	22 (6)	119 (4)	0.107
T38.3: Insulin and oral hypoglycemic drugs	96 (3)	17 (4)	79 (3)	0.048
T39: Nonopioid analgesics, antipyretics and antirheumatics	493 (14)	31 (8)	462 (15)	<0.001
T39.1: 4-aminophenol derivatives	374 (11)	24 (6)	350 (11)	0.001
T40 Narcotics and hallucinogens	178 (5)	25 (6)	153 (5)	0.25
T41: Poisoning by anaesthetics and therapeutic gases	14 (0)	0 (0)	14 (0)	0.179
T42: Antiepileptic, sedative-hypnotic and antiparkinsonism	492 (14)	66 (17)	426 (14)	0.125
T42.4: Benzodiazepines	370 (11)	55 (14)	315 (10)	0.025
T43: Psychotropic drugs, not elsewhere classified	394 (11)	55 (14)	339 (11)	0.087
T43.2: Other and unspecified antidepressants	149 (4)	15 (4)	134 (4)	0.606
T44: Autonomic nervous system drugs	251 (7)	19 (5)	232 (8)	0.048
T44.3: Other parasympatholytics and spasmolytics, not elsewhere classified	93 (3)	7 (2)	86 (3)	0.236
T44.7: Beta adrenoreceptor antagonists, not elsewhere classified	105 (3)	7 (2)	98 (3)	0.122
T45: Systemic and haematological agents	286 (8)	23 (6)	263 (9)	0.063
T45.0: Antiallergic and antiemetic drugs	150 (4)	14 (4)	136 (4)	0.42
T45.4: Iron and its compounds	83 (2)	[c]	[c]	
T46: Cardiovascular system drugs	382 (11)	56 (14)	326 (11)	0.032
T46.5: Other antihypertensives, not elsewhere classified	211 (6)	38 (10)	173 (6)	0.002
T47: Gastrointestinal system drugs	80 (2)	8 (2)	72 (2)	0.694
T48: Smooth and skeletal muscle and respiratory system agents	98 (3)	6 (2)	92 (3)	0.096
T49: Topical agents and opthalmological, otorhinolaryngological and dental drugs	143 (4)	12 (3)	131 (4)	0.25
T50: Diuretics and other and unspecified drugs, medicaments and biological	207 (6)	22 (6)	185 (6)	0.725

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substances				
T50.9: Other and unspecified drugs, medicaments and biologicals	94 (3)	10 (3)	84 (3)	0.819
Non-pharmaceutical agents				
T51: Toxic effect of alcohol	35 (1)	5 (1)	30 (1)	0.586
T52: Toxic effect of organic solvents	185 (5)	22 (6)	163 (5)	0.822
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons	10 (0)	0 (0)	10 (0)	0.256
T54-55: Toxic effect of corrosive substances, soaps and detergents[b]	132 (4)	17 (4)	115 (4)	0.58
T57: Toxic effect of other inorganic substances	0 (0)	0 (0)	0 (0)	
T60: Toxic effect of pesticides	106 (3)	12 (3)	94 (3)	0.985
T65: Toxic effect of other and unspecified substances	124 (4)	21 (5)	103 (3)	0.047
ICD-10 Diagnosis Code	**Total N (%)**	**Aboriginal N (%)**	**Non-Aboriginal N (%)**	**p**
T36-65: All poisoning admissions	3,436 (100)	391 (11)	3,045 (89)	
T36-50: Pharmaceutical*	2,859 (83)	318 (81)	2,541 (83)	0.291
T51-65: Non pharmaceutical*	588 (17)	76 (19)	512 (17)	0.195
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T45.4: Iron and its compounds	83 (2)	***	***	
T46: Cardiovascular system drugs	382 (11)	56 (14)	326 (11)	0.032
T46.5: Other antihypertensives, not elsewhere classified	211 (6)	38 (10)	173 (6)	0.002
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T65: Toxic effect of other and unspecified substances	124 (4)	21 (5)	103 (3)	0.047

[a] May exceed total number of poisoning admissions due to multiple agents per admission
[b] Categories combined due to small cell numbers
[c] Suppressed due to small cell numbers

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Clinical outcomes

Aboriginal and non-Aboriginal children had similar clinical outcomes in terms of repeat admissions, LOS and procedures (Table 3). There was one death from poisoning during the study period.

Repeat admissions

Fifty one children (1.5%) had a repeat poisoning admission. Of these, 42 were non-Aboriginal and 9 were Aboriginal. Due to small numbers, no further analysis of this group was undertaken.

Length of stay

Most children (91%) hospitalised for poisoning had a short LOS of ≤1 day. Excluding one outlier, LOS ranged from <1 to 18 days. The distribution of LOS was similar for Aboriginal and non-Aboriginal children (Table 3, χ^2=3.4147, p=0.332).

Table 3: Clinical outcomes of Aboriginal and non-Aboriginal children with a poisoning hospitalisation, New South Wales, 2000-2014

	Aboriginal N (%)	Non-Aboriginal N (%)	Total N (%)
Total number of poisoning admissions	391 (100)	3,045 (100)	3,436 (100)
Repeat admissions	9 (2)	42 (1)	51 (1)
Length of stay (days)			
<1	125 (32)	1,085 (36)	1,210 (35)
1	225 (58)	1,707 (56)	1,932 (56)
2	25 (6)	158 (5)	183 (5)
3+	16 (4)	95 (3)	111 (3)
Total number of procedures	39 (10)	335 (11)	374 (11)
Allied health interventions	27 (7)	233 (8)	260 (8)
Airway and ventilation	5 (1)	32 (1)	37 (1)
Other (including diagnostics and pharmacotherapy)[a]	7 (2)	70 (2)	77 (2)

[a]Combined due to small cell numbers

Procedures

Most children hospitalised for poisoning had no recorded procedures (89%, 3,062/3,436) and this proportion was similar for Aboriginal and non-Aboriginal children. Of the 374 hospital admissions with a recorded procedure, the majority were allied health interventions, mostly a social worker consultation (8% of poisoning admissions).

Sensitivity Analyses

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Defining Aboriginal status based solely on the birth record reduced the number of children identified as Aboriginal in the study cohort (n=21,576, 2.8%), and amongst the cases with a poisoning admission (n=246, 7.2%). The other study findings remained similar.

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Discussion

Compared with non-Aboriginal children, preschool Aboriginal children in NSW have almost three times the rate of hospitalised poisoning, but similar patterns of poisoning risk across available demographic factors, poisoning agents and clinical outcomes.

Amongst both Aboriginal and non-Aboriginal children, the incidence of hospitalised poisoning increased from age one, peaked at two to three years, and declined by four years, consistent with previous studies.[3, 5-8, 11] Male sex was a significant risk factor for both Aboriginal and non-Aboriginal children, consistent with findings from the general population [2, 3, 7, 16] and amongst Aboriginal children in NSW.[11, 21] Higher poisoning rates amongst boys may be due to earlier development of motor skills than risk perception.[5]

Poisoning risk increased with socioeconomic disadvantage and geographic remoteness amongst Aboriginal and non-Aboriginal children. Whilst these differences were not statistically significant amongst Aboriginal children, comparisons were limited by small subgroup sizes. Socioeconomic disadvantage is a well-established risk factor for unintentional injury [14, 30] and children from low-income backgrounds have a higher poisoning risk [13, 14, 31] due to more co-morbidities and medication usage, less access to childcare and storage equipment, and overcrowding.[31]

Higher poisoning hospitalisation rates in remote areas may suggest more children are being poisoned, but also that children presenting to rural hospitals are more likely to be admitted [4, 16] for reasons including distance from facilities, limited specialist services and delayed diagnostic tests.[23, 32]

There are several possible reasons for the disparity in hospitalised poisoning between Aboriginal and non-Aboriginal children. Aboriginal children disproportionately grow up in disadvantaged and remote areas. Aboriginal people have a higher burden of chronic disease, potentially exposing children to more medications. Overcrowding and poor housing disproportionally affects Aboriginal people and contributes to stress, poor supervision and health problems.[33] Higher levels of mobility amongst Aboriginal people [33] may expose children to medications belonging to different people in their own or other people's homes.

Pharmaceuticals, including adult and paediatric over-the-counter and prescription medications, are the leading causes of hospitalised unintentional poisonings amongst both Aboriginal and non-Aboriginal children. The leading classes of poisoning agents identified in this study—analgesic; psychotropic, antiepileptic, and sedative; and cardiovascular agents—are commonly prescribed medications in Australia. Benzodiazepines, 4-aminophenol derivatives (paracetamol) and other antihypertensives not

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elsewhere classified (for example clonidine) accounted for almost one-third of all poisoning admissions. The rising use of prescription medications in Australia could be contributing towards unintentional poisonings amongst young children, as has been demonstrated in the United States, where childhood pharmaceutical poisonings have been associated with trends in adult medication prescriptions.[35] For example, benzodiazepines were the most common poisoning agent for Aboriginal children, similar to a previous report amongst Aboriginal and non-Aboriginal people in NSW.[21] Benzodiazepines are widely prescribed in Australia, often for anxiety and insomnia. Aboriginal people experience a higher burden of mental health issues [36] and higher rates of benzodiazepine poisoning amongst Aboriginal children may reflect higher adult usage, although we cannot confirm this using the available data.

The second most common poisoning agent for Aboriginal children in our study was other antihypertensives not elsewhere classified. In Australia, this largely includes clonidine, which is used for child behavioural disorders.[37] Aboriginal children, particularly those from disadvantaged backgrounds, have a higher risk of emotional or behavioural difficulties.[38, 39] Childhood clonidine poisoning has been found in other research to be often caused by a child accidentally ingesting clonidine belonging to another child such as an older sibling.[40]

Most children hospitalised for poisoning had a short LOS of ≤1 day and few required procedures, consistent with previous reports.[3, 23, 32] Allied health interventions were the most common procedure, yet fewer than one in ten poisoning hospitalisations had a recorded social worker consultation. Social workers can provide support to parents during a stressful hospitalisation, as well as injury prevention education when they are more likely to be receptive.[41] Low utilisation of this resource presents a lost opportunity for safety promotion. However, sensitivity is required around addressing parental guilt about their child's poisoning, especially among Aboriginal families, due to the effects of child removal and intergenerational trauma.[42]

Strengths and Limitations

This study's strengths include its focus on preschool-aged children, whole-of-population design and minimal recall bias using routinely collected administrative data. However, covariates were limited, and key risk factors (such as parent demographics, supervision, storage practices) and clinical information (such as time to presentation, symptoms, laboratory results, treatment details) were unavailable. The use of an area-level index for socioeconomic status may introduce ecological bias.

The under-identification of Aboriginal children in Australian public hospital data can affect estimates of health outcomes.[25] This study used an 'ever-identified' algorithm for children recorded at least once as Aboriginal, demonstrated to enhance the proportion of Aboriginal children.[25] This may introduce bias

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because sicker children with more frequent hospitalisations have more opportunities for identification. However, results of the sensitivity analyses indicated that identification did not alter the main conclusions.

Hospital admissions represent only a fraction of poisonings. Our study does not reflect the complete spectrum of early childhood poisoning incidents, but captures the most severe hospitalised cases with the largest impact on children and health services.

Finally, our analysis of poisoning agents was limited to the ICD-10 taxonomy. In some circumstances it was not possible to determine specific agents, for example, 'other' categories.

Implications for practice and prevention

Safe packaging and storage are key to childhood poisoning prevention. Child resistant packaging for medications was introduced in Australia under the *Therapeutic Goods Act 1989*.[43] However, child resistant closures for medications are not always consistently used [32] and many adult medications are kept in non-reclosable packaging that children can access. Limitations in packaging design should be improved for adult medications as well as child medications.

Unsafe storage also increases poisoning risk.[15] Aboriginal families and low-income families may face barriers in promoting safe storage due to costs of safety equipment and prohibitions on home installations in public housing or rental properties.[44, 45] Providing low cost or free equipment can improve household storage practices, particularly with education and fitting assistance.[46] Reducing poisoning rates requires combined strategies of household storage interventions, accessible poisons information centres, and enforcement of packaging legislation.[47]

No known injury prevention programs have specifically addressed unintentional poisoning in Aboriginal communities.[48] The 'Safe Homes Safe Kids' program provided home safety education and devices to Aboriginal families in the Illawarra region of NSW.[47] Future research involving community consultation is needed to address poisoning prevention in Aboriginal communities, considering factors such as poverty, trauma, housing, and family separation.[49]

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Conclusion

Unintentional poisoning is most prevalent amongst preschool-aged children, and disproportionately affects Aboriginal children. This study confirmed the inequity in hospitalised poisonings between Aboriginal and non-Aboriginal children, and demonstrated that adult and child medications commonly used in the general population contribute to poisonings amongst both groups. With a rising prevalence of chronic disease and increases in prescription medication use amongst both Aboriginal people and the wider community, keeping children safe from unintentional poisoning should be a priority. Further research and policy development is needed to investigate culturally safe prevention strategies that include improved packaging, safe storage and education about medication toxicity.

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Contributions

CL, SW, LJ, and MH contributed to the conception and design of the study, including the research questions and the analysis. CL wrote each drafts, and together with MH, produced the tables and figures. LJ, SW and MH provided technical and moral support throughout the study. All authors contributed to the drafting of the work, approval of the final manuscript, and critical interpretation of its findings, with particular contributions from KZ, SW and NL regarding the translation of findings with other stakeholders for prevention.

Acknowledgements

The authors wish to thank Dr Holger Moller who prepared the dataset prior to the study period; this study builds upon knowledge generated by his previous research. The authors also wish to thank Professor Nicholas Buckley, Dr Jane Bell, Dr Ruth Barker and Professor Sallie Pearson for their assistance in the interpretation of poisoning agents; and Professor Rebecca Ivers, Professor Kathleen Clapham, and Dr Kathleen Falster for consultation about translating the research findings for policy and practice.

Competing Interests

None declared

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Data sharing statement: no additional data are available.

Patient and public involvement: patients were not directly involved in the development of the research question, outcome measures or study design. All patient data was de-identified prior to analysis. Results were presented at a public Aboriginal community health forum in La Perouse, Sydney, Australia, attended by health providers and community members. Results were also presented at an international injury prevention conference attended by policymakers and researchers, and to the Board of Kidsafe Australia. A patient information poster will be developed and presented to Kidsafe Board.

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Figure 1: Indigenous Health Outcomes Patient Evaluation (IHOPE) data sources and linkage

Figure 2: Flow diagram showing process of cohort selection

Figure 3: Crude and adjusted odds ratios with 95% confidence intervals for poisoning hospitalisation, by demographic factors, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014. Legend: OR: odds ratio, aOR: adjusted odds ratio, CI: confidence interval. Fully adjusted models included variables sex, area-level disadvantage, and geographic remoteness

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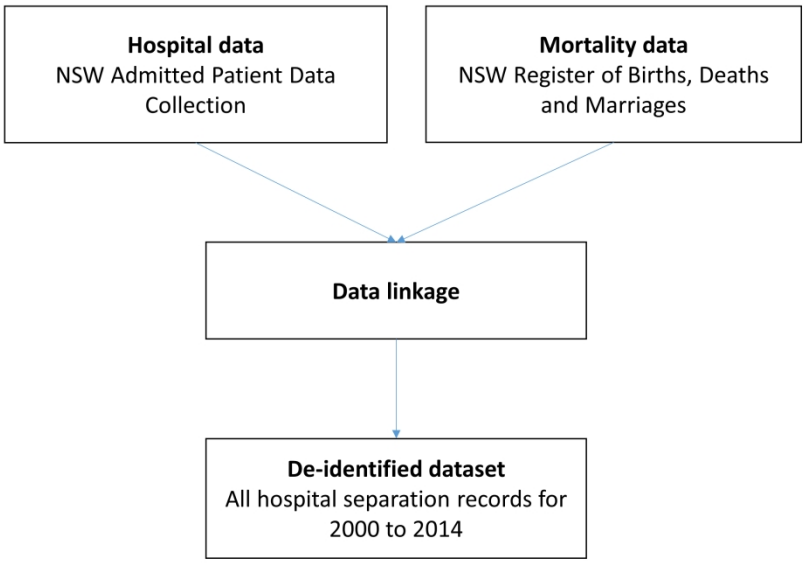


Figure 1: Indigenous Health Outcomes Patient Evaluation (IHOPE) data sources and linkage

254x190mm (300 x 300 DPI)

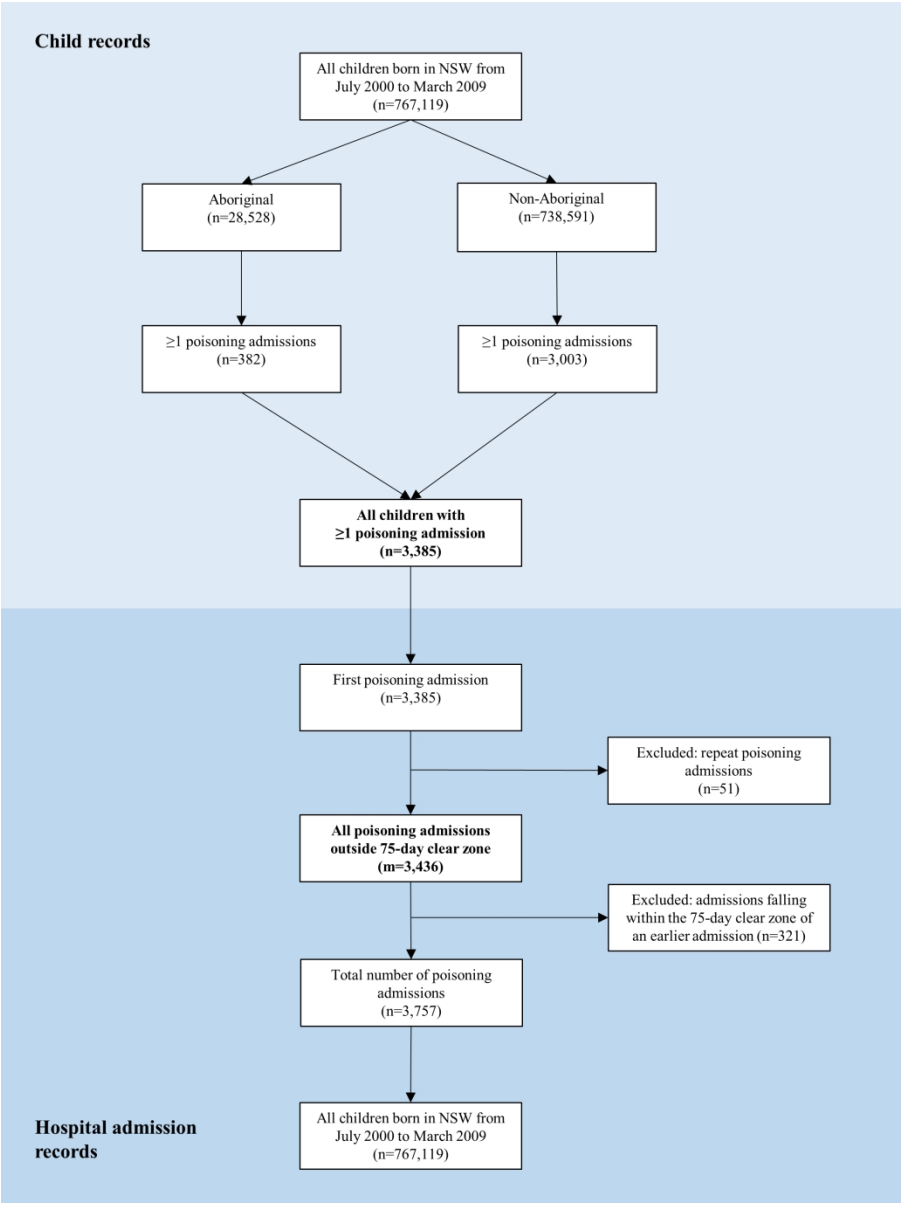


Figure 2: Flow diagram with process of cohort selection

190x254mm (300 x 300 DPI)

Variables	Aboriginal (n=28,528)		Non Aboriginal (n=738,591)	
	OR (95% CI)	aOR (95% CI)	OR (95% CI)	aOR (95% CI)
Sex				
Female	1	1	1	1
Male	1.26 (1.03-1.54)	1.27 (1.03-1.56)	1.17 (1.09-1.26)	1.17 (1.09-1.26)
Area-level disadvantage				
1 (Least disadvantaged)	1	1	1	1
2	1.88 (1.11-3.19)	1.84 (1.07-3.15)	1.44 (1.31-1.59)	1.34 (1.21-1.48)
3 (Most disadvantaged)	1.83 (1.10-3.03)	1.57 (0.92-2.69)	1.96 (1.79-2.15)	1.64 (1.48-1.81)
Remoteness				
Major Cities	1	1	1	1
Inner Regional	1.13 (0.88-1.47)	1.06 (0.82-1.39)	1.43 (1.32-1.56)	1.33 (1.22-1.44)
Outer Regional	1.39 (1.06-1.82)	1.39 (1.02-1.88)	2.13 (1.91-2.37)	1.66 (1.47-1.87)
Remote/Very Remote	1.18 (0.80-1.74)	1.18 (0.78-1.79)	2.54 (1.89-3.42)	1.97 (1.46-2.66)

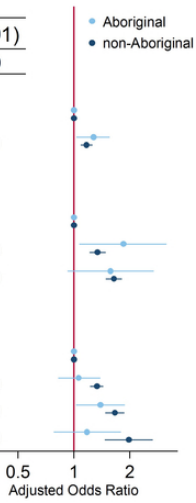
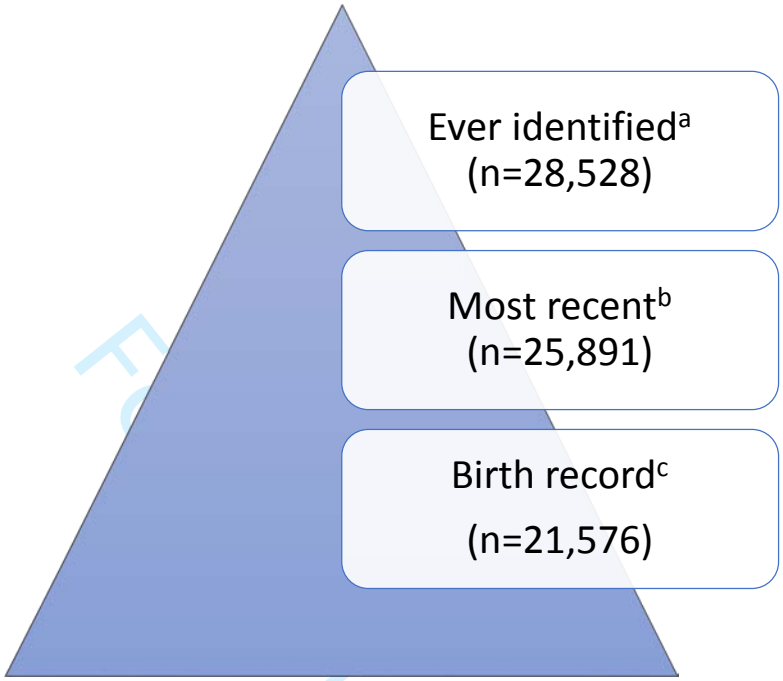


Figure 3: Crude and adjusted odds ratios with 95% confidence intervals for poisoning hospitalisation, by demographic factors, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014. Legend: OR: odds ratio, aOR: adjusted odds ratio, CI: confidence interval. Fully adjusted models included variables sex, area-level disadvantage, and geographic remoteness

38x21mm (600 x 600 DPI)

Supplement 1: Number of Aboriginal children identified, by identification method



[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record

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Supplement 2: Included and excluded poisoning agents, by ICD-10-AM code

Included agents
Pharmaceutical Agents
T36: Poisoning by systemic antibiotics
T37: Poisoning by other systemic anti-infectives and antiparasitics
T38: Poisoning by hormones and their synthetic substitutes and antagonists, not elsewhere classified
T39: Poisoning by nonopioid analgesics, antipyretics and antirheumatics
T40: Poisoning by narcotics and psychodysleptics [hallucinogens]
T41: Poisoning by anaesthetics and therapeutic gases
T42: Poisoning by antiepileptic, sedative-hypnotic and antiparkinsonism drugs
T43: Poisoning by psychotropic drugs, not elsewhere classified
T44: Poisoning by drugs primarily affecting the autonomic nervous system
T45: Poisoning by primarily systemic and haematological agents, not elsewhere classified
T46: Poisoning by agents primarily affecting the cardiovascular system
T47: Poisoning by agents primarily affecting the gastrointestinal system
T48: Poisoning by agents primarily acting on smooth and skeletal muscle and respiratory system
T49: Poisoning by topical agents primarily affecting skin and mucosal membrane and by opthalmological, otorhinolaryngological and dental drugs
T50: Diuretics and other and unspecified drugs, medicaments and biological substances
Non pharmaceutical agents
T51: Toxic effect of alcohol
T52: Toxic effect of organic solvents
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons
T54: Toxic effect of corrosive substances
T55: Toxic effect of soaps and detergents
T57: Toxic effect of other inorganic substances
T60: Toxic effect of pesticides
T65: Toxic effect of other and unspecified substances
Excluded agents
T56: Toxic effect of metals *(includes lead, mercury)*
T58: Toxic effect of carbon monoxide
T59: Toxic effect of other gases, fumes and vapours
T61: Toxic effect of noxious substances eaten as food *(includes fish, shellfish)*
T62: Toxic effect of other noxious substances eaten as food *(includes mushrooms, berries)*
T63: Toxic effect of contact with venomous animals
T64: Toxic effect of aflatoxin and other mycotoxins

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STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation	Completed (page)
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	Yes (2)
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	Yes (2)
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	Yes (4)
Objectives	3	State specific objectives, including any prespecified hypotheses	Yes (4)
Methods			
Study design	4	Present key elements of study design early in the paper	Yes (6)
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	Yes (6)
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	Cohort study: Eligibility criteria and methods of selection included (6)
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	Outcomes, exposures, predictors (logistic regression model, demographic predictor variables) ICD-10 diagnostic codes described (full list of included/excluded codes available in Supplement 2) (7)
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	Yes (6)
Bias	9	Describe any efforts to address potential sources of bias	"Clear zone" method used to reduce potential overcounting of

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			hospital admissions related to same incident presentation (7)
Study size	10	Explain how the study size was arrived at	(9)
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	Yes (7-8)
		(*b*) Describe any methods used to examine subgroups and interactions	
		(*c*) Explain how missing data were addressed	All model covariates were fully observed
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	
		(*e*) Describe any sensitivity analyses	Yes (16)

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Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	Yes (9)
		(b) Give reasons for non-participation at each stage	N/A
		(c) Consider use of a flow diagram	Yes
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	Yes (Table 1) (9)
		(b) Indicate number of participants with missing data for each variable of interest	N/A
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	Yes (9)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	Yes (9)
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	Unadjusted and adjusted odds ratios presented with 95% CI (9)
		(*b*) Report category boundaries when continuous variables were categorized	N/A
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N/A
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	Sensitivity analyses described (16)
Discussion			
Key results	18	Summarise key results with reference to study objectives	Yes (17)
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	Limitations described (18)
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	Yes (17)
Generalisability	21	Discuss the generalisability (external validity) of the study results	Implications of study findings described (19)
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	Yes (21)

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

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Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

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Title: Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

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Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

Abstract

Objectives: To investigate differences in demographic and clinical characteristics of Aboriginal and non-Aboriginal children aged 0-4 years hospitalised for unintentional poisoning in New South Wales (NSW), Australia.

Design and setting: Retrospective whole-of-population cohort analysis of linked hospital and mortality data for 2000-2014

Participants: All children (Aboriginal and non-Aboriginal) under the age of five years who born in a hospital in NSW from 2000 to 2009

Outcomes: The primary outcome was hospitalisation for unintentional poisoning. Logistic regression was used to estimate odds of poisoning hospitalisation for Aboriginal and non-Aboriginal children. Poisoning agents and clinical outcomes were compared by Aboriginality.

Results: The cohort included 767,119 children, including 28,528 (3.7%) Aboriginal children. Aboriginal children had approximately three times higher rates of hospitalised poisoning (1.34%) compared to non-Aboriginal children (0.41%). Poisoning incidence peaked at 2-3 years of age. Male sex, socioeconomic disadvantage and geographic remoteness were associated with higher odds of poisoning hospitalisation for Aboriginal and non-Aboriginal children, but associations with disadvantage and remoteness were statistically significant only for non-Aboriginal children. Most (83%) poisonings were caused by pharmaceutical agents. Few Aboriginal and non-Aboriginal children had repeat admissions for poisoning; most had a length of stay of one day or less. Only 8% of poisoning admissions involved contact with a social worker.

Conclusion: Commonly used medications in the general population contribute to poisonings amongst both Aboriginal and non-Aboriginal preschool aged children. This study highlights a need to develop culturally safe poisoning prevention strategies and policies.

Strengths and limitations of this study

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- This study's strengths include its focus on preschool-aged children, large sample size due whole-of-population design, and minimal recall bias due to the use of routinely collected administrative data.
- However, the use of administrative data limited the available covariates, so risk factors such as parent demographics, supervision and storage practice, and clinical information such as symptoms and treatment details, could not be examined.
- Children were identified as Aboriginal if they had been recorded at least once as Aboriginal ('ever-identified' algorithm), which enhances the proportion of Aboriginal children recorded using hospital data but may introduce bias in that sicker children with more frequent hospitalisations have more opportunities for identification.
- Our analysis of poisoning agents was limited to the International Classification of Diseases 10th edition (ICD-10) taxonomy, and in some circumstances it was not possible to determine specific agents, for example, 'other' categories.

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Introduction

Poisoning is a leading cause of childhood injury worldwide.[1] Preschool children (0-4 years) have a higher risk of poisoning related hospital admissions [2-4] due to their developmental stage [5] and are a priority for prevention. Amongst preschool children, poisoning incidence peaks at two to three years of age,[3, 5-8] reflecting their increased mobility and exploratory skills.[5, 6, 9]

Pharmaceutical substances, such as over-the-counter and prescription medications, cause most unintentional poisonings in Australia.[4] Amongst preschool children in Australia, the most common causes of hospitalised pharmaceutical poisoning include analgesic, anti-epileptic, psychotropic, and other systemic and cardiovascular medications.[3, 4, 7, 10-12]

Various demographic and socioeconomic risk factors contribute to poisoning amongst preschool children. Factors relating to the child (male, higher birth order), parents (younger age, perinatal depression, alcohol misuse), unsafe storage and socioeconomic deprivation are associated with increased poisoning risk.[1-3, 7, 8, 13-16] In Australia, remote areas also have higher poisoning rates.[4, 16]

Poisoning represents one of the largest Indigenous child health inequalities in high-income countries, with rate ratios for hospitalisation and mortality ranging from 1.1 to 3.9 and 1.2 to 15.4 respectively.[17] A recent whole-of-population study in New South Wales, Australia reported that Aboriginal children had nearly three times higher risk of poisoning hospitalisation compared with non-Aboriginal children.[18] Existing studies about poisoning amongst Indigenous children in high-income countries have limitations, such as the lack of a non-Indigenous comparison group and wide age ranges up to 18 years old that do not differentiate younger children.[19, 20] Previous Australian studies of poisoning amongst preschool children in the general population have not focussed on Aboriginal children.[3, 5, 7, 21] The current literature about poisoning amongst Aboriginal children in Australia is limited in its examination of risk factors and clinical outcomes.[11, 12, 18, 22] Specific poisoning agents and clinical outcomes of poisoning for Aboriginal children have not been described.[2, 10, 16, 23]

This is the first study to examine the characteristics of unintentional poisoning amongst preschool-aged Aboriginal Australian children. The study aimed to answer questions that will inform prevention efforts for this at-risk group during a time of peak incidence:

1) Are the demographic risk factors for poisoning (sex, socioeconomic status and geographic remoteness) similar for both Aboriginal and non-Aboriginal children?
2) Which agents most frequently result in poisoning hospitalisation and are they different for Aboriginal and non-Aboriginal children?

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3) Are the clinical outcomes from hospitalised poisoning similar for Aboriginal and non-Aboriginal children?

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Methods

Study design and setting

This was a retrospective, whole-of-population cohort study using linked hospital and mortality data for NSW, Australia for the period 2000 to 2014. In 2011, NSW had a population of 7,218,529 people, of whom an estimated 208,476 (2.9%) were Aboriginal or Torres Strait Islander.[24]

Data Sources

This study was part of the Indigenous Health Outcomes Patient Evaluation (IHOPE) project which compares health outcomes for Aboriginal and non-Aboriginal people in NSW using linked hospital and mortality data **(**Figure 1**)**. Hospital data were sourced from the Admitted Patient Data Collection (APDC) and mortality data from the NSW Register of Births, Deaths and Marriages (RBDM). Probabilistic data linkage was performed by the NSW Centre for Health Record Linkage (http://www.cherel.org.au). De-identified, linked datasets were supplied to the researchers.

The RBDM contains details for all deaths in NSW. The APDC contains records for all inpatient separations (discharges, transfers and deaths) for all public and private hospitals and day procedure centres in NSW. Each separation record contains information about patient demographics, diagnoses, and procedures. Diagnoses are coded according to the International Classification of Diseases and Related Problems Australian Modification, tenth revision (ICD-10-AM). Separations are hereafter referred to as hospital admissions.

Participants

Children born in a NSW hospital between June 1, 2000 and March 31, 2009 were followed from birth until their fifth birthday. Birth records were identified from the APDC dataset using the criteria of 'live born infant' (ICD-10-AM code Z38) in any diagnosis field. This birth cohort has been previously described.[18]

Aboriginal status

Aboriginal status is routinely recorded in the APDC. The main analyses used an 'ever identified' algorithm that coded children as Aboriginal if they were ever identified as Aboriginal, yielding higher proportions of Aboriginal children compared to the most recent record or birth record (Supplement 1).[25] To test the sensitivity of this identification method, all analyses were repeated using Aboriginality defined solely on the child's birth record.

Study variables

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The main outcome was hospitalisation for unintentional poisoning, defined as an acute accidental exposure to, or overdose of, a toxic substance, whether accessed by a child or incorrectly administered by another person (wrong dosage, agent or frequency). The study focussed on ingested poisoning agents likely to require similar prevention approaches, including pharmaceutical (over-the-counter and prescription) and non-pharmaceutical agents, and excluding environmental exposures (Supplement 2). Poisoning cases were identified by a primary diagnosis of injury with an ICD-10-AM code T36-65, excluding toxic effect of metals (T56), carbon monoxide and other gases (T58-59) and noxious substances, venoms and food substances (T61-64).

A poisoning admission was defined from the start date of an episode of care, to the date of separation or death. Multiple hospital admissions within a short time could be due to follow-up visits, transfer between hospitals, or change in care type within a hospital, related to the same poisoning event. Records falling within 75 days after the initial poisoning admission were excluded, based on the published 'clear zone' for poisoning.[26]

Available demographic variables included child's age, sex and area-level variables for socioeconomic disadvantage (Australian Bureau of Statistics Socioeconomic Indices for Areas (SEIFA) Index of Relative Socioeconomic Advantage and Disadvantage (IRSAD) [27]) and remoteness (Accessibility/Remoteness Index of Australia (ARIA) [28]) based on the child's statistical local area of residence at birth. Due to small numbers in some categories, IRSAD was divided into tertiles, and ARIA into four categories 'Major Cities', 'Inner Regional', 'Outer Regional' and 'Remote/Very Remote' as used previously.[18] The age of first poisoning hospitalisation was analysed by single year intervals for Aboriginal and non-Aboriginal children.

Poisoning agents were included if they were recorded in any diagnosis field (up to 50 fields). Clinical outcomes (repeat admissions, length of stay (LOS), procedures) were analysed for all poisoning admissions. LOS included the total number of hospital days related to the initial admission, and any subsequent admissions within 75 days. Procedures were classified at the 'block' level according to the Australian Classification for Health Interventions (ACHI), 8th edition.[29]

Statistical analysis

Differences in poisoning incidence were compared by demographic characteristics using chi squared tests, stratified by Aboriginality (RQ1). The crude and adjusted odds of poisoning hospitalisation associated with demographic characteristics were estimated using logistic regression (RQ1). Separate models were built for Aboriginal and non-Aboriginal children. Fully adjusted models included the variables sex, socioeconomic disadvantage and remoteness.

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Amongst children with a poisoning hospitalisation, incidence was examined by year of age (RQ1). The groups of poisoning agents and the ten most common individual agents were examined across all poisoning admissions (RQ2). Differences in poisoning agents (both groups and individual agents) by Aboriginality were tested using chi squared tests (RQ2). The number and types of procedures and LOS were examined for all poisoning admissions, and compared by Aboriginality, with descriptive statistics and chi squared tests (RQ3). Data was prepared with SAS Version 9.4 (SAS Institute) and analysed in Stata Version 14 (StataCorp).

Ethics Approval

Ethics approval was granted by the New South Wales Population & Health Services Research Committee (2009/04/141) and the Aboriginal Health and Medical Research Council Committee (684/09).

Patient and public involvement

Patients were not directly involved in the development of the research question, outcome measures or study design. All patient data was de-identified prior to analysis. Results were presented at a public Aboriginal community health forum in La Perouse, Sydney, Australia, attended by health providers and community members. Results were also presented at an international injury prevention conference attended by policymakers and researchers, and to the Board of Kidsafe Australia. A patient information poster will be developed and presented to Kidsafe Board.

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Results

Study Population

The cohort included 767,119 children born in NSW between July 1, 2000 and March 31, 2009, of whom 28,528 (3.7%) were Aboriginal (Figure 1). From 2000-2014, there were 3,757 hospital admissions with a primary diagnosis of poisoning. Of these, 321 fell within the 75-day clear zone and were not analysed as new admissions. This left 3,436 poisoning admissions for 3,385 individual children, including 382 (11%) Aboriginal children. Aboriginal children were significantly overrepresented amongst children who had been poisoned (χ^2=543.6, p<0.001).

Demographic Characteristics

Poisoning admission by demographics characteristics for all children in the cohort are shown in Table 1. More Aboriginal than non-Aboriginal children lived in the most disadvantaged areas and in Remote or Very Remote areas. The proportion of children hospitalised for poisoning was higher for boys compared to girls, and increased with increasing socioeconomic disadvantage and increasing remoteness for both Aboriginal and non-Aboriginal children.

Crude and adjusted odds for poisoning hospitalisation for Aboriginal and non-Aboriginal children are shown in Figure 2. Male sex was significantly associated with increased risk of poisoning for both Aboriginal (adjusted odds ratio [aOR] 1.27, 95% CI 1.03-1.56) and non-Aboriginal children (aOR 1.17, 95% CI 1.09-1.26). For non-Aboriginal children, poisoning risk increased with increasing socioeconomic disadvantage and increasing geographic remoteness. For Aboriginal children, increasing socioeconomic disadvantage was also associated with higher poisoning risk, although confidence intervals were wider. However, geographic remoteness was not significantly associated with an increasing poisoning risk.

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Table 1: Poisoning hospitalisations by demographic characteristics, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014

	Aboriginal children			Non-Aboriginal children			All children		
	N (column %)	N poisoned (row %)	p	N (column %)	N poisoned (row %)	p	N (column %)	N poisoned (row %)	p
Total (row %)	28,528 (100)	382 (1.34)		738,591 (100)	3,003 (0.41)		767,119 (100)	3,385 (0.44)	
Sex			0.03			<0.001			<0.001
Female	13,472 (47.2)	159 (1.18)		358,899 (48.6)	1.342 (0.37)		372,371 (48.5)	1,501 (0.40)	
Male	15,056 (52.8)	223 (1.48)		379,692 (51.4)	1.661 (0.44)		394,748 (51.5)	1,884 (0.48)	
Area-level disadvantage			0.05			<0.001			<0.001
1 - Least Disadvantaged	2,113 (7.4)	16 (0.76)		246,117 (33.3)	685 (0.28)		248,230 (32.4)	701 (0.28)	
2	7,558 (26.5)	107 (1.42)		250,639 (33.9)	1,003 (0.40)		258,197 (33.7)	1,110 (0.43)	
3 - Most disadvantaged	18,857 (66.1)	259 (1.37)		241,835 (32.7)	1,315 (0.54)		260,692 (34.0)	1,574 (0.60)	
Remoteness			0.11			<0.001			<0.001
Major city	9,056 (31.7)	105 (1.16)		492,228 (66.6)	1,659 (0.34)		501,284 (65.3)	1,764 (0.35)	
Inner regional	10,058 (35.3)	132 (1.31)		182,366 (24.7)	880 (0.48)		192,424 (25.1)	1,012 (0.53)	
Outer regional	6,852 (24.0)	110 (1.61)		58,709 (7.9)	419 (0.71)		65,561 (8.5)	529 (0.81)	
Remote/Very Remote	2,562 (9.0)	35 (1.37)		5,288 (0.7)	45 (0.85)		7,850 (1.0)	80 (1.02)	

P-values are from chi-squared tests, testing the null hypothesis that poisoning admissions are distributed equally across the levels of each covariate.

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Incidence by one-year age intervals was similar amongst Aboriginal and non-Aboriginal children (Table 2). Poisoning admissions increased from one year, were most frequent between two to three years, and were least frequent at under one year and over four years of age.

Table 2: Age at poisoning admission, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014

Age (years)	**Aboriginal children** *N* (column %)	**Non-Aboriginal children** *N* (column %)
<1	21 (6)	220 (7)
1-2	96 (25)	952 (32)
2-3	152 (40)	1211 (40)
3-4	73 (19)	446 (15)
4-5	40 (10)	174 (6)
Total	382	3,003

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Poisoning agents

Pharmaceutical agents caused most poisonings for both Aboriginal (83%) and non-Aboriginal (81%) children (Table 3).

The most frequent groups of poisoning agents were similar for both Aboriginal and non-Aboriginal children: nonopioid analgesic, antipyretic and antirheumatics (including paracetamol and ibuprofen); antiepileptic, sedative-hypnotic and antiparkinsonism medications; psychotropics; and cardiovascular medications (Table 3). Aboriginal children had a higher proportion of poisoning admissions from cardiovascular medications than non-Aboriginal children (14% and 11%, p=0.032), whilst non-Aboriginal children had a higher proportion from nonopioid analgesic, antipyretic and antirheumatics (15% and 8%, p<0.001) and autonomic nervous system medications (8% and 5%, p=0.048).

The two most frequent individual poisoning agents were benzodiazepines (11%) and 4-aminophenol derivatives (paracetamol) (11%) (Table 3). Together with other antihypertensives not elsewhere classified, they accounted for one in three poisoning admissions. Benzodiazepines and other antihypertensives not elsewhere classified were the two most common individual agents for Aboriginal children and caused a higher proportion of admissions for Aboriginal than non-Aboriginal children (benzodiazepines: 14% and 10%, p=0.025; other antihypertensives not elsewhere classified: 10% and 6%, p=0.002).

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Table 3: Poisoning agents by class and ten most frequent individual agents, for Aboriginal and Non-Aboriginal children, New South Wales, 2000-2014

ICD-10 Diagnosis Code	Total N (%)	Aboriginal N (%)	Non-Aboriginal N (%)	p
T36-65: All poisoning admissions	3,436 (100)	391 (11)	3,045 (89)	
T36-50: Pharmaceutical[a]	2,859 (83)	318 (81)	2,541 (83)	0.291
T51-65: Non-pharmaceutical[a]	588 (17)	76 (19)	512 (17)	0.195
Pharmaceutical Agents				
T36-37: Antibiotics, anti-infectives, anti-parasitics[b]	47 (1)	7 (2)	40 (1)	0.445
T38: Hormones and synthetic substitutes	141 (4)	22 (6)	119 (4)	0.107
T38.3: Insulin and oral hypoglycemic drugs	**96 (3)**	**17 (4)**	**79 (3)**	**0.048**
T39: Nonopioid analgesics, antipyretics and antirheumatics	493 (14)	31 (8)	462 (15)	<0.001
T39.1: 4-aminophenol derivatives	**374 (11)**	**24 (6)**	**350 (11)**	**0.001**
T40 Narcotics and hallucinogens	178 (5)	25 (6)	153 (5)	0.25
T41: Poisoning by anaesthetics and therapeutic gases	14 (0)	0 (0)	14 (0)	0.179
T42: Antiepileptic, sedative-hypnotic and antiparkinsonism	492 (14)	66 (17)	426 (14)	0.125
T42.4: Benzodiazepines	**370 (11)**	**55 (14)**	**315 (10)**	**0.025**
T43: Psychotropic drugs, not elsewhere classified	394 (11)	55 (14)	339 (11)	0.087
T43.2: Other and unspecified antidepressants	**149 (4)**	**15 (4)**	**134 (4)**	**0.606**
T44: Autonomic nervous system drugs	251 (7)	19 (5)	232 (8)	0.048
T44.3: Other parasympatholytics and spasmolytics, not elsewhere classified	**93 (3)**	**7 (2)**	**86 (3)**	**0.236**
T44.7: Beta adrenoreceptor antagonists, not elsewhere classified	**105 (3)**	**7 (2)**	**98 (3)**	**0.122**
T45: Systemic and haematological agents	286 (8)	23 (6)	263 (9)	0.063
T45.0: Antiallergic and antiemetic drugs	**150 (4)**	**14 (4)**	**136 (4)**	**0.42**
T45.4: Iron and its compounds	**83 (2)**	[c]	[c]	
T46: Cardiovascular system drugs	382 (11)	56 (14)	326 (11)	0.032
T46.5: Other antihypertensives, not elsewhere classified	**211 (6)**	**38 (10)**	**173 (6)**	**0.002**
T47: Gastrointestinal system drugs	80 (2)	8 (2)	72 (2)	0.694
T48: Smooth and skeletal muscle and respiratory system agents	98 (3)	6 (2)	92 (3)	0.096
T49: Topical agents and opthalmological, otorhinolaryngological and dental drugs	143 (4)	12 (3)	131 (4)	0.25

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T50: Diuretics and other and unspecified drugs, medicaments and biological substances	207 (6)	22 (6)	185 (6)	0.725
T50.9: Other and unspecified drugs, medicaments and biologicals	**94 (3)**	**10 (3)**	**84 (3)**	**0.819**
Non-pharmaceutical agents				
T51: Toxic effect of alcohol	35 (1)	5 (1)	30 (1)	0.586
T52: Toxic effect of organic solvents	185 (5)	22 (6)	163 (5)	0.822
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons	10 (0)	0 (0)	10 (0)	0.256
T54-55: Toxic effect of corrosive substances, soaps and detergents[b]	132 (4)	17 (4)	115 (4)	0.58
T57: Toxic effect of other inorganic substances	0 (0)	0 (0)	0 (0)	
T60: Toxic effect of pesticides	106 (3)	12 (3)	94 (3)	0.985
T65: Toxic effect of other and unspecified substances	124 (4)	21 (5)	103 (3)	0.047

[a] May exceed total number of poisoning admissions due to multiple agents per admission
[b] Categories combined due to small cell numbers
[c] Suppressed due to small cell numbers

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Clinical outcomes

Aboriginal and non-Aboriginal children had similar clinical outcomes in terms of repeat admissions, LOS and procedures (Table 4). There was one death from poisoning during the study period.

Repeat admissions

There were 51 repeat admissions (1% of poisoning admissions). Of these, 42 were non-Aboriginal and 9 were Aboriginal. Due to small numbers, no further analysis of this group was undertaken.

Length of stay

Most children (91%) hospitalised for poisoning had a short LOS of ≤1 day. Excluding one outlier, LOS ranged from <1 to 18 days. The distribution of LOS was similar for Aboriginal and non-Aboriginal children (Table 3, χ^2=3.4147, p=0.332).

Table 4: Clinical outcomes of Aboriginal and non-Aboriginal children with a poisoning hospitalisation, New South Wales, 2000-2014

	Aboriginal N (%)	Non-Aboriginal N (%)	Total N (%)
Total number of poisoning admissions	391 (100)	3,045 (100)	3,436 (100)
Repeat admissions	9 (2)	42 (1)	51 (1)
Length of stay (days)			
<1	125 (32)	1,085 (36)	1,210 (35)
1	225 (58)	1,707 (56)	1,932 (56)
2	25 (6)	158 (5)	183 (5)
3+	16 (4)	95 (3)	111 (3)
Total number of procedures	39 (10)	335 (11)	374 (11)
Allied health interventions	27 (7)	233 (8)	260 (8)
Airway and ventilation	5 (1)	32 (1)	37 (1)
Other (including diagnostics and pharmacotherapy)[a]	7 (2)	70 (2)	77 (2)

[a]Combined due to small cell numbers

Procedures

Most children hospitalised for poisoning had no recorded procedures (89%, 3,062/3,436) and this proportion was similar for Aboriginal and non-Aboriginal children. Of the 374 hospital admissions with a recorded procedure, the majority were allied health interventions, mostly a social worker consultation (8% of poisoning admissions).

Sensitivity Analyses

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Compared with the ever-identified method, defining Aboriginal status by most recent record and birth record reduced the numbers of children in the cohort to 25,891 and 21,576 respectively, and the number of children poisoned to 319 and 246 respectively (Supplement 1). Other findings remained similar.

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Discussion

Compared with non-Aboriginal children, preschool Aboriginal children in NSW have almost three times the rate of hospitalised poisoning, but similar patterns of poisoning risk across available demographic factors, poisoning agents and clinical outcomes.

Amongst both Aboriginal and non-Aboriginal children, the incidence of hospitalised poisoning increased from age one, peaked at two to three years, and declined by four years, consistent with previous studies.[3, 5-8, 11] Male sex was a significant risk factor for both Aboriginal and non-Aboriginal children, consistent with findings from the general population [2, 3, 7, 16] and amongst Aboriginal children in NSW.[11, 21] Higher poisoning rates amongst boys may be due to earlier development of motor skills than risk perception.[5]

Poisoning risk increased with socioeconomic disadvantage amongst Aboriginal and non-Aboriginal children, and increased with geographic remoteness amongst non-Aboriginal children. Socioeconomic disadvantage is a well-established risk factor for unintentional injury [14, 30] and children from low-income backgrounds have a higher poisoning risk [13, 14, 31] due to more co-morbidities and medication usage, less access to childcare and storage equipment, and overcrowding.[31]

Geographic remoteness was associated with significantly increased poisoning risk amongst non-Aboriginal children. In contrast, no significant trend between poisoning and geographic remoteness was evident amongst Aboriginal children, possibly reflecting small subgroup sizes and wider confidence intervals. This may also reflect additional barriers in access to health services, or differences in housing, medication availability, or other social factors experienced by Aboriginal people in remote areas.

Broadly, higher poisoning hospitalisations in remote areas may suggest more children are being poisoned, but also that children presenting to rural hospitals are more likely to be admitted [4, 16] for reasons including distance from facilities, limited specialist services and delayed diagnostic tests.[23, 32] Differences in poisoning agents by geographic region may also contribute. Some have suggested children in rural areas are more likely to be poisoned by chemical such as pesticides. However, evidence is conflicting, with this hypothesis supported by a Queensland study [2] but not a subsequent NSW study which did not find higher rates of chemical poisoning in rural areas.[10]

Several reasons may contribute to the disparity in hospitalised poisoning between Aboriginal and non-Aboriginal children. Aboriginal people experience a higher burden of socioeconomic disadvantage and chronic disease, potentially exposing children to more medications. Overcrowding and poor housing disproportionally affects Aboriginal people and contributes to stress, poor supervision and health

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problems.[33] Higher levels of mobility amongst Aboriginal people [33] may expose children to medications belonging to different people in their own or other people's homes.

Pharmaceuticals, including adult and paediatric over-the-counter and prescription medications, are the leading causes of hospitalised unintentional poisonings amongst both Aboriginal and non-Aboriginal children. The most frequent groups of poisoning agents identified in this study—analgesic; psychotropic, antiepileptic, and sedative; and cardiovascular agents—are commonly prescribed medications in Australia. Of these, the most frequent individual agents benzodiazepines, 4-aminophenol derivatives (paracetamol) and other antihypertensives not elsewhere classified (for example clonidine) accounted for almost one-third of all poisoning admissions.

Chronic diseases are highly prevalent, affecting 11 million Australians. In 2014-15, 117 million medications were prescribed by a GP, a 17% increase since 2010-11.[34] This rising medication use in Australia could be contributing towards unintentional childhood poisonings, as has been demonstrated in the United States, where pharmaceutical poisonings in children have been associated with trends in adult medication prescriptions.[35]

Benzodiazepines were the most common poisoning agent for Aboriginal children, similar to a previous report amongst Aboriginal and non-Aboriginal people in NSW.[22] Benzodiazepines are widely prescribed in Australia, often for anxiety and insomnia. Aboriginal people experience a higher burden of mental health issues [36] and higher rates of benzodiazepine poisoning amongst Aboriginal children may reflect higher adult usage, although we cannot confirm this using the available data.

Antihypertensives not elsewhere classified were the second most frequent poisoning agent for Aboriginal children in our study. In Australia, this largely includes clonidine, which is used for child behavioural disorders.[37] Aboriginal children, particularly those from disadvantaged backgrounds, have a higher risk of emotional or behavioural difficulties.[38, 39] Childhood clonidine poisoning has been found in other research to be often caused by a child accidentally ingesting clonidine belonging to another child such as an older sibling.[40]

Most children hospitalised for poisoning had a short LOS of ≤1 day and few required procedures, consistent with previous reports.[3, 23, 32] Allied health interventions were the most common procedure, yet fewer than one in ten poisoning hospitalisations had a recorded social worker consultation. Social workers can support parents during a stressful hospitalisation, and provide injury prevention education when they are more likely to be receptive.[41] Low utilisation of this resource presents a lost opportunity for safety promotion. However, cultural sensitivity is required to promote safety and not instigate blame or fear, given the intergenerational trauma around child removal for Aboriginal families.[42]

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Strengths and Limitations

This study's strengths include its focus on preschool-aged children, whole-of-population design and minimal recall bias using routinely collected administrative data. However, covariates were limited, and risk factors previously demonstrated to affect poisoning risk were unavailable, such as perinatal depression [13, 14], younger maternal age [8], parental alcohol misuse [8, 13], poor storage and supervision [21], household dwelling and socioeconomic deprivation [13, 14]. Clinical information (such as time to presentation, symptoms, laboratory results, treatment, complications [7]) were also unavailable. The use of an area-level index for socioeconomic status may introduce ecological bias.

The under-identification of Aboriginal children in Australian public hospital data can affect estimates of health outcomes.[25] This study used an 'ever-identified' algorithm for children recorded at least once as Aboriginal in order to maximise the number of poisoning events in Aboriginal children for analysis. Although this method may introduce bias in comparison of event rates between Aboriginal and non-Aboriginal people due to differential increase in identification among sicker individuals with more hospital records, such comparisons were not the main purpose of our study. Also, results of the sensitivity analysis indicated that the methods of identification did not alter the main conclusions (Supplement 1).

Hospital admissions represent only a fraction of poisonings. Our study does not reflect the complete spectrum of early childhood poisoning incidents, but captures the most severe hospitalised cases with the largest impact on children and health services.

Finally, our analysis of poisoning agents was limited to the ICD-10 taxonomy. In some circumstances it was not possible to determine specific agents, for example, 'other' categories.

Implications for practice and prevention

Safe packaging and storage are key to childhood poisoning prevention. Child resistant packaging for medications was introduced in Australia under the *Therapeutic Goods Act 1989*.[43] However, child resistant closures for medications are not always consistently used [32] and many adult medications are kept in non-reclosable packaging that children can access. Limitations in packaging design should be improved for adult medications as well as child medications.

Unsafe storage also increases poisoning risk.[15] Aboriginal families and low-income families may face barriers in promoting safe storage due to costs of safety equipment and prohibitions on home installations in public housing or rental properties.[44, 45] Providing low cost or free equipment can improve household storage practices, particularly with education and fitting assistance.[46] Reducing poisoning

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rates requires combined strategies of household storage interventions, accessible poisons information centres, and enforcement of packaging legislation.[47]

No known injury prevention programs have specifically addressed unintentional poisoning in Aboriginal communities.[48] The 'Safe Homes Safe Kids' program provided home safety education and devices to Aboriginal families in the Illawarra region of NSW.[47] Future research involving community consultation is needed to address poisoning prevention in Aboriginal communities, considering factors such as poverty, trauma, housing, and family separation.[49]

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Conclusion

Unintentional poisoning is most prevalent amongst preschool-aged children, and disproportionately affects Aboriginal children. This study confirmed the inequity in hospitalised poisonings between Aboriginal and non-Aboriginal children, and demonstrated that adult and child medications commonly used in the general population contribute to poisonings amongst both groups. With a rising prevalence of chronic disease and increases in prescription medication use amongst both Aboriginal people and the wider community, keeping children safe from unintentional poisoning should be a priority. Further research and policy development is needed to investigate culturally safe prevention strategies that include improved packaging, safe storage and education.

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Contributions

CL, SW, LJ, and MH contributed to the conception and design of the study, including the research questions and the analysis. CL wrote each drafts, and together with MH, produced the tables and figures. LJ, SW and MH provided technical and moral support throughout the study. All authors contributed to the drafting of the work, approval of the final manuscript, and critical interpretation of its findings, with particular contributions from KZ, SW and NL regarding the translation of findings with other stakeholders for prevention.

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Competing Interests

None declared

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Data sharing statement: no additional data are available.

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Figure 1: Indigenous Health Outcomes Patient Evaluation (IHOPE) data sources and study cohort selection

Figure 2: Crude and adjusted odds ratios with 95% confidence intervals for poisoning hospitalisation, by demographic factors, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014. Legend: OR: odds ratio, aOR: adjusted odds ratio, CI: confidence interval. Fully adjusted models included variables sex, area-level disadvantage, and geographic remoteness

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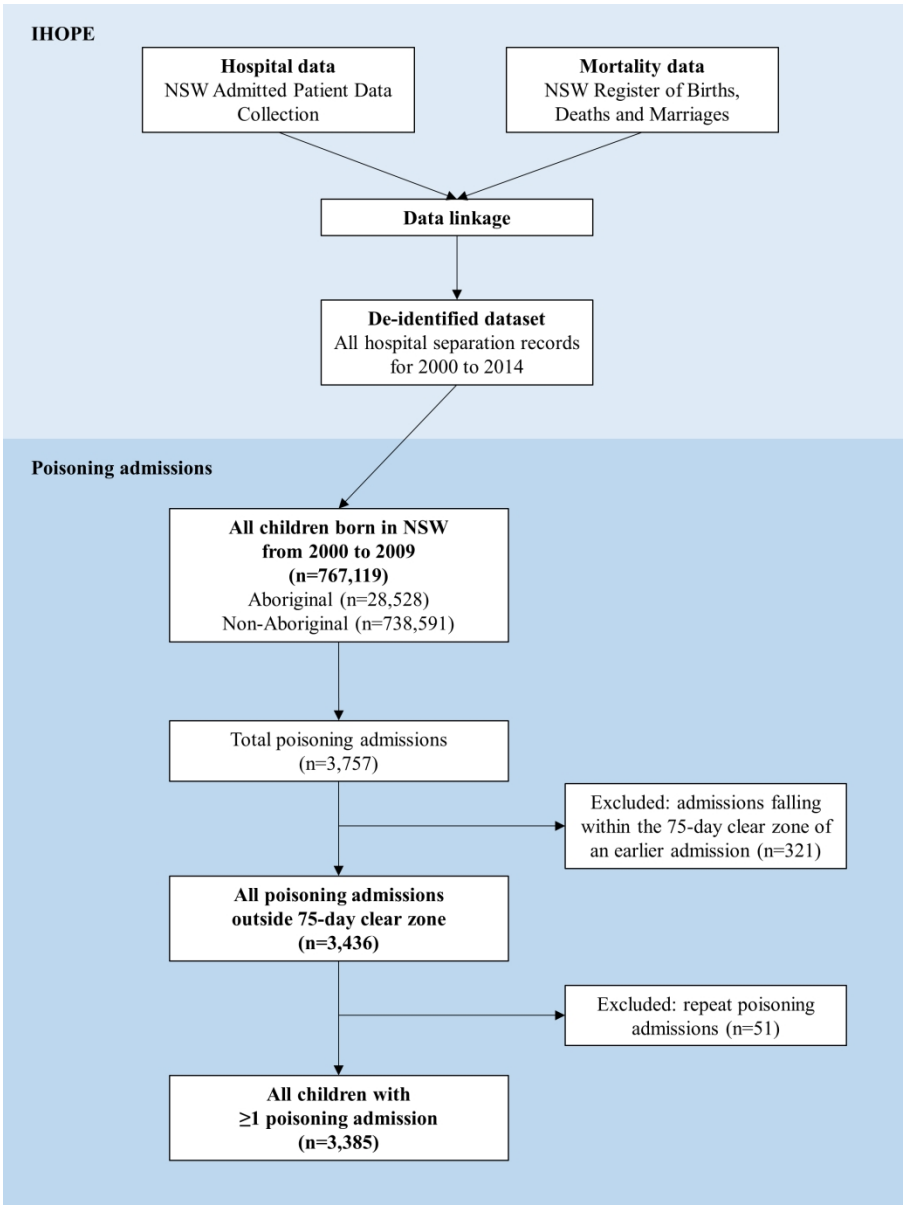

Figure 1: Indigenous Health Outcomes Patient Evaluation (IHOPE) data sources and study cohort selection

190x254mm (300 x 300 DPI)

Variables	Aboriginal (n=28,528)		Non Aboriginal (n=738,591)	
	OR (95% CI)	aOR (95% CI)	OR (95% CI)	aOR (95% CI)
Sex				
Female	1	1	1	1
Male	1.26 (1.03-1.54)	1.27 (1.03-1.56)	1.17 (1.09-1.26)	1.17 (1.09-1.26)
Area-level disadvantage				
1 (Least disadvantaged)	1	1	1	1
2	1.88 (1.11-3.19)	1.84 (1.07-3.15)	1.44 (1.31-1.59)	1.34 (1.21-1.48)
3 (Most disadvantaged)	1.83 (1.10-3.03)	1.57 (0.92-2.69)	1.96 (1.79-2.15)	1.64 (1.48-1.81)
Remoteness				
Major Cities	1	1	1	1
Inner Regional	1.13 (0.88-1.47)	1.06 (0.82-1.39)	1.43 (1.32-1.56)	1.33 (1.22-1.44)
Outer Regional	1.39 (1.06-1.82)	1.39 (1.02-1.88)	2.13 (1.91-2.37)	1.66 (1.47-1.87)
Remote/Very Remote	1.18 (0.80-1.74)	1.18 (0.78-1.79)	2.54 (1.89-3.42)	1.97 (1.46-2.66)

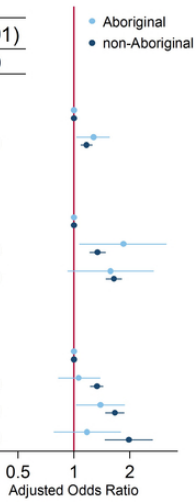

Figure 2: Crude and adjusted odds ratios with 95% confidence intervals for poisoning hospitalisation, by demographic factors, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014. Legend: OR: odds ratio, aOR: adjusted odds ratio, CI: confidence interval. Fully adjusted models included variables sex, area-level disadvantage, and geographic remoteness

38x21mm (600 x 600 DPI)

Supplement 1: Comparison of results by Aboriginal identification method

Number of Aboriginal children, by identification method

Identification method	Number of Aboriginal children identified	
	Cohort	Poisoned (% total cohort)
Ever identified[a]	28,528	382 (1.34)
Most recent[b]	25,891	319 (1.23)
Birth record[c]	21,576	246 (1.14)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record

RQ1: Adjusted odds ratio of poisoning admission by demographic characteristics, Aboriginal and non-Aboriginal children, by identification method

Variables	Non-Aboriginal children (n=738,591)	Aboriginal children		
		Ever-identified[c] (n=28,528)	Most recent[d] (n=25,891)	Birth record[e] (n=21,576)
Logistic regression[a]	**aOR[b] (95% CI)**	**aOR (95% CI)**	**aOR (95% CI)**	**aOR (95% CI)**
Sex				
Female	1	1	1	1
Male	1.17 (1.09-1.26)	1.27 (1.03-1.56)	1.34 (1.07-1.67)	1.44 (1.12-1.87)
Area-level disadvantage				
1 (Least disadvantaged)	1	1	1	1
2	1.34 (1.21-1.48)	1.84 (1.07-3.15)	1.95 (1.00-3.82)	1.90 (0.89-4.05)
3 (Most disadvantaged)	1.64 (1.48-1.81)	1.57 (0.92-2.69)	1.71 (0.88-3.34)	2.02 (0.96-4.27)
Remoteness				
Major cities	1	1	1	1
Inner regional	1.33 (1.22-1.44)	1.06 (0.82-1.39)	1.32 (0.97-1.79)	1.01 (0.72-1.41)
Outer regional	1.66 (1.47-1.87)	1.39 (1.02-1.88)	1.65 (1.17-2.34)	1.10 (0.75-1.62)
Remote/Very remote	1.97 (1.46-2.66)	1.18 (0.78-1.79)	1.38 (0.87-2.17)	1.10 (0.68-1.77)

[a] Full model included all demographic variables (sex, disadvantage, remoteness)
[b] aOR: adjusted odds ratio
[c] Child identified as Aboriginal in any APDC record between their birth and fifth birthday

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[d] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[e] Child identified as Aboriginal in their APDC birth record

RQ1: Age at admission, Aboriginal children, by identification method

Age (years)	Total	Aboriginal children		
		Ever-identified[a]	Most recent[b]	Birth record[c]
	N (column %)	*N* (column %)	*N* (column %)	*N* (column %)
<1	241 (7)	21 (6)	18 (6)	13 (5)
1-2	1048 (31)	96 (25)	80 (25)	62 (25)
2-3	1363 (40)	152 (40)	127 (40)	95 (39)
3-4	519 (15)	73 (19)	59 (19)	46 (19)
4-5	214 (6)	40 (10)	35 (11)	30 (12)
Total number of children poisoned	3385 (100)	382 (100)	319 (100)	246 (100)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record

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RQ2: Poisoning agents, Aboriginal children, by identification method

ICD-10 Diagnosis Code	Total N (%)	Aboriginal children N (%)		
		Ever-identified[a]	Most recent[b]	Birth record[c]
T36-65: All poisoning admissions	3,436 (100)	391 (11)	327 (10)	251 (7)
T36-50: Pharmaceutical[d]	2,859 (83)	318 (81)	261 (80)	207 (82)
T51-65: Non-pharmaceutical[d]	588 (17)	76 (19)	69 (21)	46 (18)
Pharmaceutical Agents				
T36-37: Antibiotics, anti-infectives, anti-parasitics[e]	47 (1)	7 (2)	4 (1)	6 (2)
T38: Hormones and synthetic substitutes	141 (4)	22 (6)	18 (6)	14 (7)
T38.3: Insulin and oral hypoglycemic drugs	**96 (3)**	**17 (4)**	**15 (5)**	**11 (4)**
T39: Nonopioid analgesics, antipyretics and antirheumatics	493 (14)	31 (8)	23 (7)	18 (7)
T39.1: 4-aminophenol derivatives	**374 (11)**	**24 (6)**	**19 (6)**	**16 (6)**
T40 Narcotics and hallucinogens	178 (5)	25 (6)	20 (6)	19 (8)
T41: Poisoning by anaesthetics and therapeutic gases	14 (0)	0 (0)	0 (0)	0 (0)
T42: Antiepileptic, sedative-hypnotic and antiparkinsonism	492 (14)	66 (17)	57 (17)	46 (18)
T42.4: Benzodiazepines	**370 (11)**	**55 (14)**	**48 (15)**	**38 (15)**
T43: Psychotropic drugs, not elsewhere classified	394 (11)	55 (14)	47 (14)	36 (14)
T43.2: Other and unspecified antidepressants	**149 (4)**	**15 (4)**	**11 (3)**	**9 (4)**
T44: Autonomic nervous system drugs	251 (7)	19 (5)	12 (4)	12 (5)
T44.3: Other parasympatholytics and spasmolytics, not elsewhere classified	**93 (3)**	**7 (2)**	**5 (2)**	**5 (2)**
T44.7: Beta adrenoreceptor antagonists, not elsewhere classified	**105 (3)**	**7 (2)**	**3 (1)**	**4 (2)**
T45: Systemic and haematological agents	286 (8)	23 (6)	14 (4)	12 (5)
T45.0: Antiallergic and antiemetic drugs	**150 (4)**	**14 (4)**	**10 (3)**	**8 (3)**
T45.4: Iron and its compounds	**83 (2)**	[f]	[f]	[f]
T46: Cardiovascular system drugs	382 (11)	56 (14)	52 (16)	34 (14)
T46.5: Other antihypertensives, not elsewhere classified	**211 (6)**	**38 (10)**	**35 (11)**	**21 (8)**
T47: Gastrointestinal system drugs	80 (2)	8 (2)	6 (2)	6 (2)
T48: Smooth and skeletal muscle and respiratory system agents	98 (3)	6 (2)	4 (1)	5 (2)
T49: Topical agents and opthalmological, otorhinolaryngological and dental drugs	143 (4)	12 (3)	9 (3)	8 (3)
T50: Diuretics and other and unspecified drugs, medicaments and biological substances	207 (6)	22 (6)	17 (5)	15 (6)
T50.9: Other and unspecified drugs, medicaments and biologicals	**94 (3)**	**10 (3)**	**10 (3)**	**5 (2)**

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Non-pharmaceutical agents				
T51: Toxic effect of alcohol	35 (1)	5 (1)	4 (1)	2 (1)
T52: Toxic effect of organic solvents	185 (5)	22 (6)	19 (6)	14 (6)
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons	10 (0)	0 (0)	0 (0)	0 (0)
T54-55: Toxic effect of corrosive substances, soaps and detergents[e]	132 (4)	17 (4)	15 (5)	9 (4)
T57: Toxic effect of other inorganic substances	0 (0)	0 (0)	0 (0)	0 (0)
T60: Toxic effect of pesticides	106 (3)	12 (3)	12 (4)	5 (2)
T65: Toxic effect of other and unspecified substances	124 (4)	21 (5)	20 (6)	16 (6)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record
[d] May exceed total number of poisoning admissions due to multiple agents per admission
[e] Categories combined due to small cell numbers
[f] Suppressed due to small cell numbers

RQ3: Clinical outcomes, Aboriginal children, by identification method

Clinical outcomes	**Total N (%)**	**Aboriginal children N (%)**		
		Ever-identified[a]	**Most recent[b]**	**Birth record[c]**
Total number of poisoning admissions	3,436 (100)	391 (100)	327 (100)	251 (100)
Repeat admissions	51 (1)	9 (2)	8 (2)	5 (2)
Length of stay (days)				
<1	1,210 (35)	125 (32)	102 (31)	75 (30)
1	1,932 (56)	225 (58)	193 (59)	150 (60)
2	183 (5)	25 (6)	19 (6)	17 (7)
3+	111 (3)	16 (4)	13 (4)	9 (4)
Total number of procedures	374 (11)	39 (10)	33 (10)	26 (10)
Allied health interventions	260 (8)	27 (7)	24 (7)	20 (8)
Airway and ventilation	37 (1)	5 (1)	3 (1)	3 (1)
Other (including diagnostics and pharmacotherapy)[d]	77 (2)	7 (2)	6 (2)	3 (1)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record
[d] Combined due to small cell numbers

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Supplement 2: Included and excluded poisoning agents, by ICD-10-AM code

Included agents
Pharmaceutical Agents
T36: Poisoning by systemic antibiotics
T37: Poisoning by other systemic anti-infectives and antiparasitics
T38: Poisoning by hormones and their synthetic substitutes and antagonists, not elsewhere classified
T39: Poisoning by nonopioid analgesics, antipyretics and antirheumatics
T40: Poisoning by narcotics and psychodysleptics [hallucinogens]
T41: Poisoning by anaesthetics and therapeutic gases
T42: Poisoning by antiepileptic, sedative-hypnotic and antiparkinsonism drugs
T43: Poisoning by psychotropic drugs, not elsewhere classified
T44: Poisoning by drugs primarily affecting the autonomic nervous system
T45: Poisoning by primarily systemic and haematological agents, not elsewhere classified
T46: Poisoning by agents primarily affecting the cardiovascular system
T47: Poisoning by agents primarily affecting the gastrointestinal system
T48: Poisoning by agents primarily acting on smooth and skeletal muscle and respiratory system
T49: Poisoning by topical agents primarily affecting skin and mucosal membrane and by opthalmological, otorhinolaryngological and dental drugs
T50: Diuretics and other and unspecified drugs, medicaments and biological substances
Non pharmaceutical agents
T51: Toxic effect of alcohol
T52: Toxic effect of organic solvents
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons
T54: Toxic effect of corrosive substances
T55: Toxic effect of soaps and detergents
T57: Toxic effect of other inorganic substances
T60: Toxic effect of pesticides
T65: Toxic effect of other and unspecified substances
Excluded agents
T56: Toxic effect of metals *(includes lead, mercury)*
T58: Toxic effect of carbon monoxide
T59: Toxic effect of other gases, fumes and vapours
T61: Toxic effect of noxious substances eaten as food *(includes fish, shellfish)*
T62: Toxic effect of other noxious substances eaten as food *(includes mushrooms, berries)*
T63: Toxic effect of contact with venomous animals
T64: Toxic effect of aflatoxin and other mycotoxins

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STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation	Completed (page)
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	Yes (2)
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	Yes (2)
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	Yes (4)
Objectives	3	State specific objectives, including any prespecified hypotheses	Yes (4)
Methods			
Study design	4	Present key elements of study design early in the paper	Yes (6)
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	Yes (6)
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	Cohort study: Eligibility criteria and methods of selection included (6)
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	Outcomes, exposures, predictors (logistic regression model, demographic predictor variables) ICD-10 diagnostic codes described (full list of included/excluded codes available in Supplement 2) (7)
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	Yes (6)
Bias	9	Describe any efforts to address potential sources of bias	"Clear zone" method used to reduce potential overcounting of hospital admissions related to same incident

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			presentation (7) Use of ever-identified identification method may introduce bias in that children who are more frequently sick, and present to hospital more times, are more likely to be identified.
Study size	10	Explain how the study size was arrived at	(9)
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	Yes (7-8)
		(*b*) Describe any methods used to examine subgroups and interactions	
		(*c*) Explain how missing data were addressed	All model covariates were fully observed
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	
		(*e*) Describe any sensitivity analyses	Yes (16)

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Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	Yes (9)
		(b) Give reasons for non-participation at each stage	N/A
		(c) Consider use of a flow diagram	Yes
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	Yes (Table 1) (9)
		(b) Indicate number of participants with missing data for each variable of interest	N/A
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	Yes (9)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	Yes (9)
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	Unadjusted and adjusted odds ratios presented with 95% CI (9)
		(*b*) Report category boundaries when continuous variables were categorized	N/A
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N/A
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	Sensitivity analyses described (16)
Discussion			
Key results	18	Summarise key results with reference to study objectives	Yes (17)
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	Limitations described (18)
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	Yes (17)
Generalisability	21	Discuss the generalisability (external validity) of the study results	Implications of study findings described (19)
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	Yes (21)

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

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Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

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Title: Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

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Demographic and clinical characteristics of hospitalised unintentional poisoning in Aboriginal and non-Aboriginal preschool children in New South Wales, Australia: a population data linkage study

Abstract

Objectives: To investigate differences in demographic and clinical characteristics of Aboriginal and non-Aboriginal children aged 0-4 years hospitalised for unintentional poisoning in New South Wales (NSW), Australia.

Design and setting: Retrospective whole-of-population cohort analysis of linked hospital and mortality data for 2000-2014

Participants: All children (Aboriginal and non-Aboriginal) under the age of five years who born in a hospital in NSW from 2000 to 2009

Outcomes: The primary outcome was hospitalisation for unintentional poisoning. Logistic regression was used to estimate odds of poisoning hospitalisation for Aboriginal and non-Aboriginal children. Poisoning agents and clinical outcomes were compared by Aboriginality.

Results: The cohort included 767,119 children, including 28,528 (3.7%) Aboriginal children. Aboriginal children had approximately three times higher rates of hospitalised poisoning (1.34%) compared to non-Aboriginal children (0.41%). Poisoning incidence peaked at 2-3 years of age. Male sex, socioeconomic disadvantage and geographic remoteness were associated with higher odds of poisoning hospitalisation for Aboriginal and non-Aboriginal children, but associations with disadvantage and remoteness were statistically significant only for non-Aboriginal children. Most (83%) poisonings were caused by pharmaceutical agents. Few Aboriginal and non-Aboriginal children had repeat admissions for poisoning; most had a length of stay of one day or less. Only 8% of poisoning admissions involved contact with a social worker.

Conclusion: Commonly used medications in the general population contribute to poisonings amongst both Aboriginal and non-Aboriginal preschool aged children. This study highlights a need to develop culturally safe poisoning prevention strategies and policies.

Strengths and limitations of this study

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- This study's strengths include its focus on preschool-aged children, large sample size due to the whole-of-population design, and minimal recall bias due to the use of routinely collected administrative data.
- However, the use of administrative data limited the available covariates, so risk factors such as parent demographics, supervision and storage practice, and clinical information such as symptoms and treatment details, could not be examined.
- Children were identified as Aboriginal if they had been recorded at least once as Aboriginal ('ever-identified' algorithm), which enhances the proportion of Aboriginal children recorded using hospital data but may introduce bias in that sicker children with more frequent hospitalisations have more opportunities for identification.
- Our analysis of poisoning agents was limited to the International Classification of Diseases 10th edition (ICD-10) taxonomy, and in some circumstances it was not possible to determine specific agents, for example, 'other' categories.

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Introduction

Poisoning is a leading cause of childhood injury worldwide.[1] Preschool children (0-4 years) have a higher risk of poisoning related hospital admissions [2-4] due to their developmental stage [5] and are a priority for prevention. Amongst preschool children, poisoning incidence peaks at two to three years of age,[3, 5-8] reflecting their increased mobility and exploratory skills.[5, 6, 9]

Pharmaceutical substances, such as over-the-counter and prescription medications, cause most unintentional poisonings in Australia.[4] Amongst preschool children in Australia, the most common causes of hospitalised pharmaceutical poisoning include analgesic, anti-epileptic, psychotropic, and other systemic and cardiovascular medications.[3, 4, 7, 10-12]

Various demographic and socioeconomic risk factors contribute to poisoning amongst preschool children. Factors relating to the child (male, higher birth order), parents (younger age, perinatal depression, alcohol misuse), unsafe storage and socioeconomic deprivation are associated with increased poisoning risk.[1-3, 7, 8, 13-16] In Australia, remote areas also have higher poisoning rates.[4, 16]

Poisoning represents one of the largest Indigenous child health inequalities in high-income countries, with rate ratios for hospitalisation and mortality ranging from 1.1 to 3.9 and 1.2 to 15.4 respectively.[17] A recent whole-of-population study in New South Wales, Australia reported that Aboriginal children had nearly three times higher risk of poisoning hospitalisation compared with non-Aboriginal children.[18] Existing studies about poisoning amongst Indigenous children in high-income countries have limitations, such as the lack of a non-Indigenous comparison group and wide age ranges up to 18 years old that do not differentiate younger children.[19, 20] Previous Australian studies of poisoning amongst preschool children in the general population have not focussed on Aboriginal children.[3, 5, 7, 21] The current literature about poisoning amongst Aboriginal children in Australia is limited in its examination of risk factors and clinical outcomes.[11, 12, 18, 22] Specific poisoning agents and clinical outcomes of poisoning for Aboriginal children have not been described.[2, 10, 16, 23]

This is the first study to examine the characteristics of unintentional poisoning amongst preschool-aged Aboriginal Australian children. The study aimed to answer questions that will inform prevention efforts for this at-risk group during a time of peak incidence:

1) Are the demographic risk factors for poisoning (sex, socioeconomic status and geographic remoteness) similar for both Aboriginal and non-Aboriginal children?
2) Which agents most frequently result in poisoning hospitalisation and are they different for Aboriginal and non-Aboriginal children?

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3) Are the clinical outcomes from hospitalised poisoning similar for Aboriginal and non-Aboriginal children?

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Methods

Study design and setting

This was a retrospective, whole-of-population cohort study using linked hospital and mortality data for NSW, Australia for the period 2000 to 2014. In 2011, NSW had a population of 7,218,529 people, of whom an estimated 208,476 (2.9%) were Aboriginal or Torres Strait Islander.[24]

Data Sources

This study was part of the Indigenous Health Outcomes Patient Evaluation (IHOPE) project which compares health outcomes for Aboriginal and non-Aboriginal people in NSW using linked hospital and mortality data **(**Figure 1**)**. Hospital data were sourced from the Admitted Patient Data Collection (APDC) and mortality data from the NSW Register of Births, Deaths and Marriages (RBDM). Probabilistic data linkage was performed by the NSW Centre for Health Record Linkage (http://www.cherel.org.au). De-identified, linked datasets were supplied to the researchers.

The RBDM contains details for all deaths in NSW. The APDC contains records for all inpatient separations (discharges, transfers and deaths) for all public and private hospitals and day procedure centres in NSW. Each separation record contains information about patient demographics, diagnoses, and procedures. Diagnoses are coded according to the International Classification of Diseases and Related Problems Australian Modification, tenth revision (ICD-10-AM). Separations are hereafter referred to as hospital admissions.

Participants

Children born in a NSW hospital between June 1, 2000 and March 31, 2009 were followed from birth until their fifth birthday. Birth records were identified from the APDC dataset using the criteria of 'live born infant' (ICD-10-AM code Z38) in any diagnosis field. This birth cohort has been previously described.[18]

Aboriginal status

Aboriginal status is routinely recorded in the APDC. The main analyses used an 'ever identified' algorithm that coded children as Aboriginal if they were ever identified as Aboriginal, yielding higher proportions of Aboriginal children compared to the most recent record or birth record (Supplement 1).[25] To test the sensitivity of this identification method, all analyses were repeated using Aboriginality defined by the most recent record or birth record.

Study variables

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The main outcome was hospitalisation for unintentional poisoning, defined as an acute accidental exposure to, or overdose of, a toxic substance, whether accessed by a child or incorrectly administered by another person (wrong dosage, agent or frequency). The study focussed on ingested poisoning agents likely to require similar prevention approaches, including pharmaceutical (over-the-counter and prescription) and non-pharmaceutical agents, and excluding environmental exposures (Supplement 2). Poisoning cases were identified by a primary diagnosis of injury with an ICD-10-AM code T36-65, excluding toxic effect of metals (T56), carbon monoxide and other gases (T58-59) and noxious substances, venoms and food substances (T61-64).

A poisoning admission was defined from the start date of an episode of care, to the date of separation or death. Multiple hospital admissions within a short time could be due to follow-up visits, transfer between hospitals, or change in care type within a hospital, related to the same poisoning event. Records falling within 75 days after the initial poisoning admission were excluded, based on the published 'clear zone' for poisoning.[26]

Available demographic variables included child's age, sex and area-level variables for socioeconomic disadvantage (Australian Bureau of Statistics Socioeconomic Indices for Areas (SEIFA) Index of Relative Socioeconomic Advantage and Disadvantage (IRSAD) [27]) and remoteness (Accessibility/Remoteness Index of Australia (ARIA) [28]) based on the child's statistical local area of residence at birth. Due to small numbers in some categories, IRSAD was divided into tertiles, and ARIA into four categories 'Major Cities', 'Inner Regional', 'Outer Regional' and 'Remote/Very Remote' as used previously.[18] The age of first poisoning hospitalisation was analysed by single year intervals for Aboriginal and non-Aboriginal children.

Poisoning agents were included if they were recorded in any diagnosis field (up to 50 fields). Clinical outcomes (repeat admissions, length of stay (LOS), procedures) were analysed for all poisoning admissions. LOS included the total number of hospital days related to the initial admission, and any subsequent admissions within 75 days. Procedures were classified at the 'block' level according to the Australian Classification for Health Interventions (ACHI), 8th edition.[29]

Statistical analysis

Differences in poisoning incidence were compared by demographic characteristics using chi squared tests, stratified by Aboriginality (RQ1). The crude and adjusted odds of poisoning hospitalisation associated with demographic characteristics were estimated using logistic regression (RQ1). Separate models were built for Aboriginal and non-Aboriginal children. Fully adjusted models included the variables sex, socioeconomic disadvantage and remoteness.

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Amongst children with a poisoning hospitalisation, incidence was examined by year of age (RQ1). The groups of poisoning agents and the ten most common individual agents were examined across all poisoning admissions (RQ2). Differences in poisoning agents (both groups and individual agents) by Aboriginality were tested using chi squared tests (RQ2). The number and types of procedures and LOS were examined for all poisoning admissions, and compared by Aboriginality, with descriptive statistics and chi squared tests (RQ3). Data was prepared with SAS Version 9.4 (SAS Institute) and analysed in Stata Version 14 (StataCorp).

Ethics Approval

Ethics approval was granted by the New South Wales Population & Health Services Research Committee (2009/04/141) and the Aboriginal Health and Medical Research Council Committee (684/09).

Patient and public involvement

Patients were not directly involved in the development of the research question, outcome measures or study design. All patient data was de-identified prior to analysis. Results were presented at a public Aboriginal community health forum in La Perouse, Sydney, Australia, attended by health providers and community members. Results were also presented at an international injury prevention conference attended by policymakers and researchers, and to the Board of Kidsafe Australia. A patient information poster will be developed and presented to Kidsafe Board.

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Results

Study Population

The cohort included 767,119 children born in NSW between July 1, 2000 and March 31, 2009, of whom 28,528 (3.7%) were Aboriginal (Figure 1). From 2000-2014, there were 3,757 hospital admissions with a primary diagnosis of poisoning. Of these, 321 fell within the 75-day clear zone and were not analysed as new admissions. This left 3,436 poisoning admissions for 3,385 individual children, including 382 (11%) Aboriginal children. Aboriginal children were significantly overrepresented amongst children who had been poisoned (χ^2=543.6, p<0.001).

Demographic Characteristics

Poisoning admission by demographics characteristics for all children in the cohort are shown in Table 1. More Aboriginal than non-Aboriginal children lived in the most disadvantaged areas and in Remote or Very Remote areas. The proportion of children hospitalised for poisoning was higher for boys compared to girls, and increased with increasing socioeconomic disadvantage and increasing remoteness for both Aboriginal and non-Aboriginal children.

Crude and adjusted odds for poisoning hospitalisation for Aboriginal and non-Aboriginal children are shown in Figure 2. Male sex was significantly associated with increased risk of poisoning for both Aboriginal (adjusted odds ratio [aOR] 1.27, 95% CI 1.03-1.56) and non-Aboriginal children (aOR 1.17, 95% CI 1.09-1.26). For non-Aboriginal children, poisoning risk increased with increasing socioeconomic disadvantage and increasing geographic remoteness. For Aboriginal children, increasing socioeconomic disadvantage was also associated with higher poisoning risk, although confidence intervals were wider. However, geographic remoteness was not significantly associated with an increasing poisoning risk.

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Table 1: Poisoning hospitalisations by demographic characteristics, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014

	Aboriginal children			Non-Aboriginal children			All children		
	N (column %)	N poisoned (row %)	p	N (column %)	N poisoned (row %)	p	N (column %)	N poisoned (row %)	p
Total (row %)	28,528 (100)	382 (1.34)		738,591 (100)	3,003 (0.41)		767,119 (100)	3,385 (0.44)	
Sex			0.03			<0.001			<0.001
Female	13,472 (47.2)	159 (1.18)		358,899 (48.6)	1.342 (0.37)		372,371 (48.5)	1,501 (0.40)	
Male	15,056 (52.8)	223 (1.48)		379,692 (51.4)	1.661 (0.44)		394,748 (51.5)	1,884 (0.48)	
Area-level disadvantage			0.05			<0.001			<0.001
1 - Least Disadvantaged	2,113 (7.4)	16 (0.76)		246,117 (33.3)	685 (0.28)		248,230 (32.4)	701 (0.28)	
2	7,558 (26.5)	107 (1.42)		250,639 (33.9)	1,003 (0.40)		258,197 (33.7)	1,110 (0.43)	
3 - Most disadvantaged	18,857 (66.1)	259 (1.37)		241,835 (32.7)	1,315 (0.54)		260,692 (34.0)	1,574 (0.60)	
Remoteness			0.11			<0.001			<0.001
Major city	9,056 (31.7)	105 (1.16)		492,228 (66.6)	1,659 (0.34)		501,284 (65.3)	1,764 (0.35)	
Inner regional	10,058 (35.3)	132 (1.31)		182,366 (24.7)	880 (0.48)		192,424 (25.1)	1,012 (0.53)	
Outer regional	6,852 (24.0)	110 (1.61)		58,709 (7.9)	419 (0.71)		65,561 (8.5)	529 (0.81)	
Remote/Very Remote	2,562 (9.0)	35 (1.37)		5,288 (0.7)	45 (0.85)		7,850 (1.0)	80 (1.02)	

P-values are from chi-squared tests, testing the null hypothesis that poisoning admissions are distributed equally across the levels of each covariate.

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Incidence by one-year age intervals was similar amongst Aboriginal and non-Aboriginal children (Table 2). Poisoning admissions increased from one year, were most frequent between two to three years, and were least frequent at under one year and over four years of age.

Table 2: Age at poisoning admission, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014

Age (years)	**Aboriginal children** *N* (column %)	**Non-Aboriginal children** *N* (column %)
<1	21 (6)	220 (7)
1-2	96 (25)	952 (32)
2-3	152 (40)	1211 (40)
3-4	73 (19)	446 (15)
4-5	40 (10)	174 (6)
Total	382	3,003

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Poisoning agents

Pharmaceutical agents caused most poisonings for both Aboriginal (83%) and non-Aboriginal (81%) children (Table 3).

The most frequent groups of poisoning agents were similar for both Aboriginal and non-Aboriginal children: nonopioid analgesic, antipyretic and antirheumatics (including paracetamol and ibuprofen); antiepileptic, sedative-hypnotic and antiparkinsonism medications; psychotropics; and cardiovascular medications (Table 3). Aboriginal children had a higher proportion of poisoning admissions from cardiovascular medications than non-Aboriginal children (14% and 11%, p=0.032), whilst non-Aboriginal children had a higher proportion from nonopioid analgesic, antipyretic and antirheumatics (15% and 8%, p<0.001) and autonomic nervous system medications (8% and 5%, p=0.048).

The two most frequent individual poisoning agents were benzodiazepines (11%) and 4-aminophenol derivatives (paracetamol) (11%) (Table 3). Together with other antihypertensives not elsewhere classified, they accounted for one in three poisoning admissions. Benzodiazepines and other antihypertensives not elsewhere classified were the two most common individual agents for Aboriginal children and caused a higher proportion of admissions for Aboriginal than non-Aboriginal children (benzodiazepines: 14% and 10%, p=0.025; other antihypertensives not elsewhere classified: 10% and 6%, p=0.002).

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Table 3: Poisoning agents by class and ten most frequent individual agents, for Aboriginal and Non-Aboriginal children, New South Wales, 2000-2014

ICD-10 Diagnosis Code	Total N (%)	Aboriginal N (%)	Non-Aboriginal N (%)	p
T36-65: All poisoning admissions	3,436 (100)	391 (11)	3,045 (89)	
T36-50: Pharmaceutical[a]	2,859 (83)	318 (81)	2,541 (83)	0.291
T51-65: Non-pharmaceutical[a]	588 (17)	76 (19)	512 (17)	0.195
Pharmaceutical Agents				
T36-37: Antibiotics, anti-infectives, anti-parasitics[b]	47 (1)	7 (2)	40 (1)	0.445
T38: Hormones and synthetic substitutes	141 (4)	22 (6)	119 (4)	0.107
T38.3: Insulin and oral hypoglycemic drugs	**96 (3)**	**17 (4)**	**79 (3)**	**0.048**
T39: Nonopioid analgesics, antipyretics and antirheumatics	493 (14)	31 (8)	462 (15)	<0.001
T39.1: 4-aminophenol derivatives	**374 (11)**	**24 (6)**	**350 (11)**	**0.001**
T40 Narcotics and hallucinogens	178 (5)	25 (6)	153 (5)	0.25
T41: Poisoning by anaesthetics and therapeutic gases	14 (0)	0 (0)	14 (0)	0.179
T42: Antiepileptic, sedative-hypnotic and antiparkinsonism	492 (14)	66 (17)	426 (14)	0.125
T42.4: Benzodiazepines	**370 (11)**	**55 (14)**	**315 (10)**	**0.025**
T43: Psychotropic drugs, not elsewhere classified	394 (11)	55 (14)	339 (11)	0.087
T43.2: Other and unspecified antidepressants	**149 (4)**	**15 (4)**	**134 (4)**	**0.606**
T44: Autonomic nervous system drugs	251 (7)	19 (5)	232 (8)	0.048
T44.3: Other parasympatholytics and spasmolytics, not elsewhere classified	**93 (3)**	**7 (2)**	**86 (3)**	**0.236**
T44.7: Beta adrenoreceptor antagonists, not elsewhere classified	**105 (3)**	**7 (2)**	**98 (3)**	**0.122**
T45: Systemic and haematological agents	286 (8)	23 (6)	263 (9)	0.063
T45.0: Antiallergic and antiemetic drugs	**150 (4)**	**14 (4)**	**136 (4)**	**0.42**
T45.4: Iron and its compounds	**83 (2)**	[c]	[c]	
T46: Cardiovascular system drugs	382 (11)	56 (14)	326 (11)	0.032
T46.5: Other antihypertensives, not elsewhere classified	**211 (6)**	**38 (10)**	**173 (6)**	**0.002**
T47: Gastrointestinal system drugs	80 (2)	8 (2)	72 (2)	0.694
T48: Smooth and skeletal muscle and respiratory system agents	98 (3)	6 (2)	92 (3)	0.096
T49: Topical agents and opthalmological, otorhinolaryngological and dental drugs	143 (4)	12 (3)	131 (4)	0.25

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T50: Diuretics and other and unspecified drugs, medicaments and biological substances	207 (6)	22 (6)	185 (6)	0.725
T50.9: Other and unspecified drugs, medicaments and biologicals	**94 (3)**	**10 (3)**	**84 (3)**	**0.819**
Non-pharmaceutical agents				
T51: Toxic effect of alcohol	35 (1)	5 (1)	30 (1)	0.586
T52: Toxic effect of organic solvents	185 (5)	22 (6)	163 (5)	0.822
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons	10 (0)	0 (0)	10 (0)	0.256
T54-55: Toxic effect of corrosive substances, soaps and detergents[b]	132 (4)	17 (4)	115 (4)	0.58
T57: Toxic effect of other inorganic substances	0 (0)	0 (0)	0 (0)	
T60: Toxic effect of pesticides	106 (3)	12 (3)	94 (3)	0.985
T65: Toxic effect of other and unspecified substances	124 (4)	21 (5)	103 (3)	0.047

[a] May exceed total number of poisoning admissions due to multiple agents per admission
[b] Categories combined due to small cell numbers
[c] Suppressed due to small cell numbers

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Clinical outcomes

Aboriginal and non-Aboriginal children had similar clinical outcomes in terms of repeat admissions, LOS and procedures (Table 4). There was one death from poisoning during the study period.

Repeat admissions

There were 51 repeat admissions (1% of poisoning admissions). Of these, 42 were non-Aboriginal and 9 were Aboriginal. Due to small numbers, no further analysis of this group was undertaken.

Length of stay

Most children (91%) hospitalised for poisoning had a short LOS of ≤1 day. Excluding one outlier, LOS ranged from <1 to 18 days. The distribution of LOS was similar for Aboriginal and non-Aboriginal children (Table 4, χ^2=3.4147, p=0.332).

Table 4: Clinical outcomes of Aboriginal and non-Aboriginal children with a poisoning hospitalisation, New South Wales, 2000-2014

	Aboriginal N (%)	Non-Aboriginal N (%)	Total N (%)
Total number of poisoning admissions	391 (100)	3,045 (100)	3,436 (100)
Repeat admissions	9 (2)	42 (1)	51 (1)
Length of stay (days)			
<1	125 (32)	1,085 (36)	1,210 (35)
1	225 (58)	1,707 (56)	1,932 (56)
2	25 (6)	158 (5)	183 (5)
3+	16 (4)	95 (3)	111 (3)
Total number of procedures	39 (10)	335 (11)	374 (11)
Allied health interventions	27 (7)	233 (8)	260 (8)
Airway and ventilation	5 (1)	32 (1)	37 (1)
Other (including diagnostics and pharmacotherapy)[a]	7 (2)	70 (2)	77 (2)

[a]Combined due to small cell numbers

Procedures

Most children hospitalised for poisoning had no recorded procedures (89%, 3,062/3,436) and this proportion was similar for Aboriginal and non-Aboriginal children. Of the 374 hospital admissions with a recorded procedure, the majority were allied health interventions, mostly a social worker consultation (8% of poisoning admissions).

Sensitivity Analyses

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Compared with the ever-identified method, defining Aboriginal status by most recent record and birth record reduced the numbers of children in the cohort to 25,891 and 21,576 respectively, and the number of children poisoned to 319 and 246 respectively (Supplement 1, Table 1). Other findings remained similar (Supplement 1, Tables 2-5). For example, the magnitude of the association between poisoning admission and sex, area-level disadvantage and remoteness were similar with overlapping confidence intervals. The profile of age at admission, common poisoning agents and clinical outcomes were also consistent across all three identification methods.

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Discussion

Compared with non-Aboriginal children, preschool Aboriginal children in NSW have about three times the rate of hospitalised poisoning, but similar patterns of poisoning risk across available demographic factors, poisoning agents and clinical outcomes.

Amongst both Aboriginal and non-Aboriginal children, the incidence of hospitalised poisoning increased from age one, peaked at two to three years, and declined by four years, consistent with previous studies.[3, 5-8, 11] Male sex was a significant risk factor for both Aboriginal and non-Aboriginal children, consistent with findings from the general population [2, 3, 7, 16] and amongst Aboriginal children in NSW.[11, 21] Higher poisoning rates amongst boys may be due to earlier development of motor skills than risk perception.[5]

Poisoning risk increased with socioeconomic disadvantage amongst Aboriginal and non-Aboriginal children, and increased with geographic remoteness amongst non-Aboriginal children. Socioeconomic disadvantage is a well-established risk factor for unintentional injury [14, 30] and children from low-income backgrounds have a higher poisoning risk [13, 14, 31] due to more co-morbidities and medication usage, less access to childcare and storage equipment, and overcrowding.[31]

Geographic remoteness was associated with significantly increased poisoning risk amongst non-Aboriginal children. In contrast, no significant trend between poisoning and geographic remoteness was evident amongst Aboriginal children, possibly reflecting small subgroup sizes and wider confidence intervals. This may also reflect additional barriers in access to health services, or differences in housing, medication availability, or other social factors experienced by Aboriginal people in remote areas.

Broadly, higher poisoning hospitalisations in remote areas may suggest more children are being poisoned, but also that children presenting to rural hospitals are more likely to be admitted [4, 16] for reasons including distance from facilities, limited specialist services and delayed diagnostic tests.[23, 32] Differences in poisoning agents by geographic region may also contribute. Some have suggested children in rural areas are more likely to be poisoned by chemical such as pesticides. However, evidence is conflicting, with this hypothesis supported by a Queensland study [2] but not a subsequent NSW study which did not find higher rates of chemical poisoning in rural areas.[10]

Several reasons may contribute to the disparity in hospitalised poisoning between Aboriginal and non-Aboriginal children. Aboriginal people experience a higher burden of socioeconomic disadvantage and chronic disease, potentially exposing children to more medications. Overcrowding and poor housing disproportionally affects Aboriginal people and contributes to stress, poor supervision and health

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problems.[33] Higher levels of mobility amongst Aboriginal people [33] may expose children to medications belonging to different people in their own or other people's homes.

Pharmaceuticals, including adult and paediatric over-the-counter and prescription medications, are the leading causes of hospitalised unintentional poisonings amongst both Aboriginal and non-Aboriginal children. The most frequent groups of poisoning agents identified in this study—analgesic; psychotropic, antiepileptic, and sedative; and cardiovascular agents—are commonly prescribed medications in Australia. Of these, the most frequent individual agents benzodiazepines, 4-aminophenol derivatives (paracetamol) and other antihypertensives not elsewhere classified (for example clonidine) accounted for almost one-third of all poisoning admissions.

Chronic diseases are highly prevalent, affecting 11 million Australians. In 2014-15, 117 million medications were prescribed by a GP, a 17% increase since 2010-11.[34] This rising medication use in Australia could be contributing towards unintentional childhood poisonings, as has been demonstrated in the United States, where pharmaceutical poisonings in children have been associated with trends in adult medication prescriptions.[35]

Benzodiazepines were the most common poisoning agent for Aboriginal children, similar to a previous report amongst Aboriginal and non-Aboriginal people in NSW.[22] Benzodiazepines are widely prescribed in Australia, often for anxiety and insomnia. Aboriginal people experience a higher burden of mental health issues [36] and higher rates of benzodiazepine poisoning amongst Aboriginal children may reflect higher adult usage, although we cannot confirm this using the available data.

Antihypertensives not elsewhere classified were the second most frequent poisoning agent for Aboriginal children in our study. In Australia, this largely includes clonidine, which is used for child behavioural disorders.[37] Aboriginal children, particularly those from disadvantaged backgrounds, have a higher risk of emotional or behavioural difficulties.[38, 39] Childhood clonidine poisoning has been found in other research to be often caused by a child accidentally ingesting clonidine belonging to another child such as an older sibling.[40]

Most children hospitalised for poisoning had a short LOS of ≤1 day and few required procedures, consistent with previous reports.[3, 23, 32] Allied health interventions were the most common procedure, yet fewer than one in ten poisoning hospitalisations had a recorded social worker consultation. Social workers can support parents during a stressful hospitalisation, and provide injury prevention education when they are more likely to be receptive.[41] Low utilisation of this resource presents a lost opportunity for safety promotion. However, cultural sensitivity is required to promote safety and not instigate blame or fear, given the intergenerational trauma around child removal for Aboriginal families.[42]

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Strengths and Limitations

This study's strengths include its focus on preschool-aged children, whole-of-population design and minimal recall bias using routinely collected administrative data. However, covariates were limited, and risk factors previously demonstrated to affect poisoning risk were unavailable, such as perinatal depression [13, 14], younger maternal age [8], parental alcohol misuse [8, 13], poor storage and supervision [21], household dwelling and socioeconomic deprivation [13, 14]. Clinical information (such as time to presentation, symptoms, laboratory results, treatment, complications [7]) were also unavailable. The use of an area-level index for socioeconomic status may introduce ecological bias. For example, individuals from low socioeconomic households who live in relatively advantaged areas will be misclassified.

The under-identification of Aboriginal children in Australian public hospital data can affect estimates of health outcomes.[25] This study used an 'ever-identified' algorithm for children recorded at least once as Aboriginal in order to maximise the number of poisoning events in Aboriginal children for analysis. Although this method may introduce bias in comparison of event rates between Aboriginal and non-Aboriginal people due to differential increase in identification among sicker individuals with more hospital records, such comparisons were not the main purpose of our study. Also, results of the sensitivity analysis indicated that the methods of identification did not alter the main conclusions (Supplement 1).

Hospital admissions represent only a fraction of poisonings. Our study does not reflect the complete spectrum of early childhood poisoning incidents, but captures the most severe hospitalised cases with the largest impact on children and health services.

Finally, our analysis of poisoning agents was limited to the ICD-10 taxonomy. In some circumstances it was not possible to determine specific agents, for example, 'other' categories.

Implications for practice and prevention

Reducing the poisoning rate among Aboriginal children requires combined strategies of household storage interventions, accessible poisons information centres, and enforcement of packaging legislation.[43] No known injury prevention programs have specifically addressed unintentional poisoning in Aboriginal communities [44], however, safe storage of medications is known to be key to childhood poisoning prevention.[15] Aboriginal families and low-income families may face barriers in promoting safe storage due to costs of safety equipment and prohibitions on home installations in public housing or rental properties.[45, 46] Providing low cost or free equipment can improve household storage practices, particularly with education and fitting assistance.[47] The 'Safe Homes Safe Kids' program provided

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home safety education and devices to Aboriginal families in the Illawarra region of NSW.[47] Future research involving community consultation is needed to address poisoning prevention in Aboriginal communities, considering factors such as poverty, trauma, housing, and family separation.[48]

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Conclusion

Unintentional poisoning is most prevalent amongst preschool-aged children, and disproportionately affects Aboriginal children. This study confirmed the inequity in hospitalised poisonings between Aboriginal and non-Aboriginal children, and demonstrated that adult and child medications commonly used in the general population contribute to poisonings amongst both groups. With a rising prevalence of chronic disease and increases in prescription medication use amongst both Aboriginal people and the wider community, keeping children safe from unintentional poisoning should be a priority. Further research and policy development is needed to investigate culturally safe prevention strategies that include improved packaging, safe storage and education.

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Contributions

CL, SW, LJ, and MH contributed to the conception and design of the study, including the research questions and the analysis. CL wrote each drafts, and together with MH, produced the tables and figures. LJ, SW and MH provided technical support and supervision throughout the study. All authors contributed to the drafting of the work, approval of the final manuscript, and critical interpretation of its findings, with particular contributions from KZ, SW and NL regarding the translation of findings with other stakeholders for prevention.

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Competing Interests

None declared

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Data sharing statement: no additional data are available.

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Figure 1: Indigenous Health Outcomes Patient Evaluation (IHOPE) data sources and study cohort selection

Figure 2: Crude and adjusted odds ratios with 95% confidence intervals for poisoning hospitalisation, by demographic factors, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014. Legend: OR: odds ratio, aOR: adjusted odds ratio, CI: confidence interval. Fully adjusted models included variables sex, area-level disadvantage, and geographic remoteness

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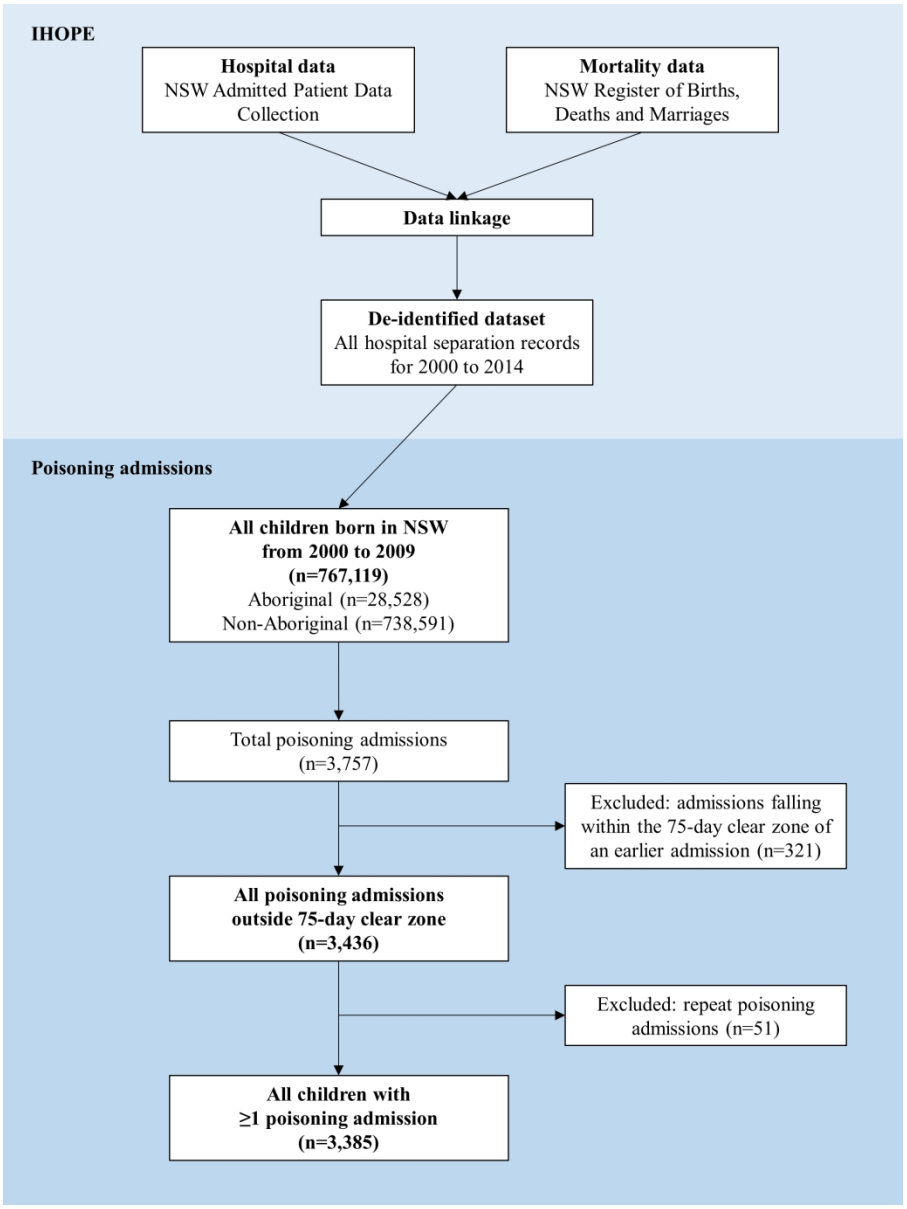

Figure 1: Indigenous Health Outcomes Patient Evaluation (IHOPE) data sources and study cohort selection

190x254mm (300 x 300 DPI)

Variables	Aboriginal (n=28,528)		Non Aboriginal (n=738,591)	
	OR (95% CI)	aOR (95% CI)	OR (95% CI)	aOR (95% CI)
Sex				
Female	1	1	1	1
Male	1.26 (1.03-1.54)	1.27 (1.03-1.56)	1.17 (1.09-1.26)	1.17 (1.09-1.26)
Area-level disadvantage				
1 (Least disadvantaged)	1	1	1	1
2	1.88 (1.11-3.19)	1.84 (1.07-3.15)	1.44 (1.31-1.59)	1.34 (1.21-1.48)
3 (Most disadvantaged)	1.83 (1.10-3.03)	1.57 (0.92-2.69)	1.96 (1.79-2.15)	1.64 (1.48-1.81)
Remoteness				
Major Cities	1	1	1	1
Inner Regional	1.13 (0.88-1.47)	1.06 (0.82-1.39)	1.43 (1.32-1.56)	1.33 (1.22-1.44)
Outer Regional	1.39 (1.06-1.82)	1.39 (1.02-1.88)	2.13 (1.91-2.37)	1.66 (1.47-1.87)
Remote/Very Remote	1.18 (0.80-1.74)	1.18 (0.78-1.79)	2.54 (1.89-3.42)	1.97 (1.46-2.66)

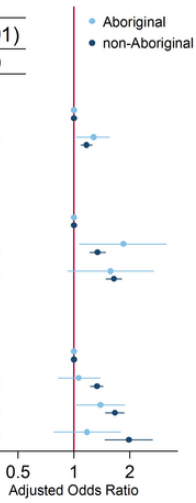

Figure 2: Crude and adjusted odds ratios with 95% confidence intervals for poisoning hospitalisation, by demographic factors, Aboriginal and non-Aboriginal children, New South Wales, 2000-2014. Legend: OR: odds ratio, aOR: adjusted odds ratio, CI: confidence interval. Fully adjusted models included variables sex, area-level disadvantage, and geographic remoteness

38x21mm (600 x 600 DPI)

Supplement 1: Comparison of results by Aboriginal identification method

List of content:

Supplementary Table 1: Number of Aboriginal children, by identification method

Supplementary Table 2: Adjusted odds ratio of poisoning admission by demographic characteristics, Aboriginal and non-Aboriginal children, by identification method (Answers RQ1)

Supplementary Table 3: Age at admission, Aboriginal children, by identification method (Answers RQ1)

Supplementary Table 4: Poisoning agents, Aboriginal children, by identification method (Answers RQ2)

Supplementary Table 5: Clinical outcomes, Aboriginal children, by identification method (Answers RQ3)

Research Questions (RQ):

1) Are the demographic risk factors for poisoning (sex, socioeconomic status and geographic remoteness) similar for both Aboriginal and non-Aboriginal children?
2) Which agents most frequently result in poisoning hospitalisation and are they different for Aboriginal and non-Aboriginal children?
3) Are the clinical outcomes from hospitalised poisoning similar for Aboriginal and non-Aboriginal children?

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Supplementary Table 1: Number of Aboriginal children, by identification method

Identification method	Number of Aboriginal children identified	
	Cohort	Poisoned (% total cohort)
Ever identified[a]	28,528	382 (1.34)
Most recent[b]	25,891	319 (1.23)
Birth record[c]	21,576	246 (1.14)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record

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Supplementary Table 2: Adjusted odds ratio of poisoning admission by demographic characteristics, Aboriginal and non-Aboriginal children, by identification method

Variables	Non-Aboriginal children (n=738,591)	Aboriginal children		
		Ever-identified[c] (n=28,528)	Most recent[d] (n=25,891)	Birth record[e] (n=21,576)
Logistic regression[a]	**aOR[b] (95% CI)**	**aOR (95% CI)**	**aOR (95% CI)**	**aOR (95% CI)**
Sex				
Female	1	1	1	1
Male	1.17 (1.09-1.26)	1.27 (1.03-1.56)	1.34 (1.07-1.67)	1.44 (1.12-1.87)
Area-level disadvantage				
1 (Least disadvantaged)	1	1	1	1
2	1.34 (1.21-1.48)	1.84 (1.07-3.15)	1.95 (1.00-3.82)	1.90 (0.89-4.05)
3 (Most disadvantaged)	1.64 (1.48-1.81)	1.57 (0.92-2.69)	1.71 (0.88-3.34)	2.02 (0.96-4.27)
Remoteness				
Major cities	1	1	1	1
Inner regional	1.33 (1.22-1.44)	1.06 (0.82-1.39)	1.32 (0.97-1.79)	1.01 (0.72-1.41)
Outer regional	1.66 (1.47-1.87)	1.39 (1.02-1.88)	1.65 (1.17-2.34)	1.10 (0.75-1.62)
Remote/Very remote	1.97 (1.46-2.66)	1.18 (0.78-1.79)	1.38 (0.87-2.17)	1.10 (0.68-1.77)

[a] Full model included all demographic variables (sex, disadvantage, remoteness)
[b] aOR: adjusted odds ratio
[c] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[d] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[e] Child identified as Aboriginal in their APDC birth record

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Supplementary Table 3: Age at admission, Aboriginal children, by identification method

Age (years)	Total	Aboriginal children		
		Ever-identified[a]	Most recent[b]	Birth record[c]
	N (column %)	*N* (column %)	*N* (column %)	*N* (column %)
<1	241 (7)	21 (6)	18 (6)	13 (5)
1-2	1048 (31)	96 (25)	80 (25)	62 (25)
2-3	1363 (40)	152 (40)	127 (40)	95 (39)
3-4	519 (15)	73 (19)	59 (19)	46 (19)
4-5	214 (6)	40 (10)	35 (11)	30 (12)
Total number of children poisoned	3385 (100)	382 (100)	319 (100)	246 (100)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record

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Supplementary Table 4: Poisoning agents, Aboriginal children, by identification method

ICD-10 Diagnosis Code	Total N (%)	Aboriginal children N (%)		
		Ever-identified[a]	Most recent[b]	Birth record[c]
T36-65: All poisoning admissions	3,436 (100)	391 (11)	327 (10)	251 (7)
T36-50: Pharmaceutical[d]	2,859 (83)	318 (81)	261 (80)	207 (82)
T51-65: Non-pharmaceutical[d]	588 (17)	76 (19)	69 (21)	46 (18)
Pharmaceutical Agents				
T36-37: Antibiotics, anti-infectives, anti-parasitics[e]	47 (1)	7 (2)	4 (1)	6 (2)
T38: Hormones and synthetic substitutes	141 (4)	22 (6)	18 (6)	14 (7)
T38.3: Insulin and oral hypoglycemic drugs	**96 (3)**	**17 (4)**	**15 (5)**	**11 (4)**
T39: Nonopioid analgesics, antipyretics and antirheumatics	493 (14)	31 (8)	23 (7)	18 (7)
T39.1: 4-aminophenol derivatives	**374 (11)**	**24 (6)**	**19 (6)**	**16 (6)**
T40 Narcotics and hallucinogens	178 (5)	25 (6)	20 (6)	19 (8)
T41: Poisoning by anaesthetics and therapeutic gases	14 (0)	0 (0)	0 (0)	0 (0)
T42: Antiepileptic, sedative-hypnotic and antiparkinsonism	492 (14)	66 (17)	57 (17)	46 (18)
T42.4: Benzodiazepines	**370 (11)**	**55 (14)**	**48 (15)**	**38 (15)**
T43: Psychotropic drugs, not elsewhere classified	394 (11)	55 (14)	47 (14)	36 (14)
T43.2: Other and unspecified antidepressants	**149 (4)**	**15 (4)**	**11 (3)**	**9 (4)**
T44: Autonomic nervous system drugs	251 (7)	19 (5)	12 (4)	12 (5)
T44.3: Other parasympatholytics and spasmolytics, not elsewhere classified	**93 (3)**	**7 (2)**	**5 (2)**	**5 (2)**
T44.7: Beta adrenoreceptor antagonists, not elsewhere classified	**105 (3)**	**7 (2)**	**3 (1)**	**4 (2)**
T45: Systemic and haematological agents	286 (8)	23 (6)	14 (4)	12 (5)
T45.0: Antiallergic and antiemetic drugs	**150 (4)**	**14 (4)**	**10 (3)**	**8 (3)**
T45.4: Iron and its compounds	**83 (2)**	[f]	[f]	[f]
T46: Cardiovascular system drugs	382 (11)	56 (14)	52 (16)	34 (14)
T46.5: Other antihypertensives, not elsewhere classified	**211 (6)**	**38 (10)**	**35 (11)**	**21 (8)**
T47: Gastrointestinal system drugs	80 (2)	8 (2)	6 (2)	6 (2)
T48: Smooth and skeletal muscle and respiratory system agents	98 (3)	6 (2)	4 (1)	5 (2)
T49: Topical agents and opthalmological, otorhinolaryngological and dental drugs	143 (4)	12 (3)	9 (3)	8 (3)
T50: Diuretics and other and unspecified drugs, medicaments and biological substances	207 (6)	22 (6)	17 (5)	15 (6)
T50.9: Other and unspecified drugs, medicaments and biologicals	**94 (3)**	**10 (3)**	**10 (3)**	**5 (2)**

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Non-pharmaceutical agents				
T51: Toxic effect of alcohol	35 (1)	5 (1)	4 (1)	2 (1)
T52: Toxic effect of organic solvents	185 (5)	22 (6)	19 (6)	14 (6)
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons	10 (0)	0 (0)	0 (0)	0 (0)
T54-55: Toxic effect of corrosive substances, soaps and detergents[e]	132 (4)	17 (4)	15 (5)	9 (4)
T57: Toxic effect of other inorganic substances	0 (0)	0 (0)	0 (0)	0 (0)
T60: Toxic effect of pesticides	106 (3)	12 (3)	12 (4)	5 (2)
T65: Toxic effect of other and unspecified substances	124 (4)	21 (5)	20 (6)	16 (6)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record
[d] May exceed total number of poisoning admissions due to multiple agents per admission
[e] Categories combined due to small cell numbers
[f] Suppressed due to small cell numbers

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Supplementary Table 5: Clinical outcomes, Aboriginal children, by identification method

Clinical outcomes	Total N (%)	Aboriginal children N (%)		
		Ever-identified[a]	Most recent[b]	Birth record[c]
Total number of poisoning admissions	3,436 (100)	391 (100)	327 (100)	251 (100)
Repeat admissions	51 (1)	9 (2)	8 (2)	5 (2)
Length of stay (days)				
<1	1,210 (35)	125 (32)	102 (31)	75 (30)
1	1,932 (56)	225 (58)	193 (59)	150 (60)
2	183 (5)	25 (6)	19 (6)	17 (7)
3+	111 (3)	16 (4)	13 (4)	9 (4)
Total number of procedures	374 (11)	39 (10)	33 (10)	26 (10)
Allied health interventions	260 (8)	27 (7)	24 (7)	20 (8)
Airway and ventilation	37 (1)	5 (1)	3 (1)	3 (1)
Other (including diagnostics and pharmacotherapy)[d]	77 (2)	7 (2)	6 (2)	3 (1)

[a] Child identified as Aboriginal in any APDC record between their birth and fifth birthday
[b] Child identified as Aboriginal in the most recent APDC record between their birth and fifth birthday
[c] Child identified as Aboriginal in their APDC birth record
[d] Combined due to small cell numbers

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Supplement 2: Included and excluded poisoning agents, by ICD-10-AM code

Included agents
Pharmaceutical Agents
T36: Poisoning by systemic antibiotics
T37: Poisoning by other systemic anti-infectives and antiparasitics
T38: Poisoning by hormones and their synthetic substitutes and antagonists, not elsewhere classified
T39: Poisoning by nonopioid analgesics, antipyretics and antirheumatics
T40: Poisoning by narcotics and psychodysleptics [hallucinogens]
T41: Poisoning by anaesthetics and therapeutic gases
T42: Poisoning by antiepileptic, sedative-hypnotic and antiparkinsonism drugs
T43: Poisoning by psychotropic drugs, not elsewhere classified
T44: Poisoning by drugs primarily affecting the autonomic nervous system
T45: Poisoning by primarily systemic and haematological agents, not elsewhere classified
T46: Poisoning by agents primarily affecting the cardiovascular system
T47: Poisoning by agents primarily affecting the gastrointestinal system
T48: Poisoning by agents primarily acting on smooth and skeletal muscle and respiratory system
T49: Poisoning by topical agents primarily affecting skin and mucosal membrane and by opthalmological, otorhinolaryngological and dental drugs
T50: Diuretics and other and unspecified drugs, medicaments and biological substances
Non pharmaceutical agents
T51: Toxic effect of alcohol
T52: Toxic effect of organic solvents
T53: Toxic effect of halogen derivatives of aliphatic and aromatic hydrocarbons
T54: Toxic effect of corrosive substances
T55: Toxic effect of soaps and detergents
T57: Toxic effect of other inorganic substances
T60: Toxic effect of pesticides
T65: Toxic effect of other and unspecified substances
Excluded agents
T56: Toxic effect of metals *(includes lead, mercury)*
T58: Toxic effect of carbon monoxide
T59: Toxic effect of other gases, fumes and vapours
T61: Toxic effect of noxious substances eaten as food *(includes fish, shellfish)*
T62: Toxic effect of other noxious substances eaten as food *(includes mushrooms, berries)*
T63: Toxic effect of contact with venomous animals
T64: Toxic effect of aflatoxin and other mycotoxins

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STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation	Completed (page)
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	Yes (2)
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	Yes (2)
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	Yes (4)
Objectives	3	State specific objectives, including any prespecified hypotheses	Yes (4)
Methods			
Study design	4	Present key elements of study design early in the paper	Yes (6)
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	Yes (6)
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	Cohort study: Eligibility criteria and methods of selection included (6)
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	Outcomes, exposures, predictors (logistic regression model, demographic predictor variables) ICD-10 diagnostic codes described (full list of included/excluded codes available in Supplement 2) (7)
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	Yes (6)
Bias	9	Describe any efforts to address potential sources of bias	"Clear zone" method used to reduce potential overcounting of hospital admissions related to same incident

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			presentation (7) Use of ever-identified identification method may introduce bias in that children who are more frequently sick, and present to hospital more times, are more likely to be identified.
Study size	10	Explain how the study size was arrived at	(9)
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	Yes (7-8)
		(*b*) Describe any methods used to examine subgroups and interactions	
		(*c*) Explain how missing data were addressed	All model covariates were fully observed
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	
		(*e*) Describe any sensitivity analyses	Yes (16)

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Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	Yes (9)
		(b) Give reasons for non-participation at each stage	N/A
		(c) Consider use of a flow diagram	Yes
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	Yes (Table 1) (9)
		(b) Indicate number of participants with missing data for each variable of interest	N/A
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	Yes (9)
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	Yes (9)
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	Unadjusted and adjusted odds ratios presented with 95% CI (9)
		(*b*) Report category boundaries when continuous variables were categorized	N/A
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N/A
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	Sensitivity analyses described (16)
Discussion			
Key results	18	Summarise key results with reference to study objectives	Yes (17)
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	Limitations described (18)
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	Yes (17)
Generalisability	21	Discuss the generalisability (external validity) of the study results	Implications of study findings described (19)
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	Yes (21)

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

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Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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