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ORIGINAL ARTICLE

Temporal trends, clinical characteristics, and sociodemographic profile of post-neonatally acquired cerebral palsy in Australia, 1973–2012: A population-based observational study

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Abstract

Aim: To describe post-neonatally acquired (PNN) cerebral palsy (CP) in terms of temporal trends in prevalence, clinical and sociodemographic profiles, known causes and associations between causes, and sociodemographic variables.

Method: Numerator data, a count of children with PNN-CP confirmed at 5 years of age (n = 523), was drawn from two Australian state CP registers (birth years 1973–2012). Poisson regression was used to investigate temporal trends in the prevalence of PNN-CP by 5-year intervals, calculated per 10 000 live births. Using data from all state and territory Australian CP registers (n = 469), distributions of clinical characteristics, PNN-CP causes, and sociodemographic factors were tabulated (birth years 1995–2012). χ^2 and logistic regression analyses were used to assess associations between sociodemographic profile, Australian reference data, and known causes.

Results: A significant temporal decline in PNN-CP in Victoria (p = 0.047) and Western Australia (p = 0.033) was observed. The most common proximal causes of PNN-CP were cerebrovascular accidents (34%, n = 158), infection (25%, n = 117), and non-accidental injuries (12%, n = 58). Children born to teenage mothers, Aboriginal and/or Torres Strait Islander mothers, or children born in remote areas were over-represented in this cohort compared with reference data (all $p \leq 0.001$). Infectious

This original article is commented on by Durkin on pages 13–14 of this issue.

Abbreviations: ACPR, Australian Cerebral Palsy Register; ARIA, Accessibility and Remoteness Index of Australia; CVA, Cerebral vascular accident; PNN, post-neonatally acquired; SEIFA, Socio-Economic Indexes for Areas..

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causes were strongly associated with teenage motherhood (odds ratio 3.0 [95% confidence interval 1.1–8.2], $p = 0.028$) and remote living (odds ratio 4.5 [95% confidence interval 2.0–10.2], $p < 0.001$).

Interpretation: Although prevalence of PNN-CP has declined, the over-representation of priority populations, and the relative severity of a condition that is largely preventable, suggest the need for more specific primary preventive measures and support.

Cerebral palsy (CP) describes a group of heterogeneous conditions that result from a diverse range of static lesions/anomalies in the fetal or infant brain.[1] The defining characteristics are abnormal muscle tone, posture, and/or involuntary movements that impact activities of daily living.[2] People with CP may also have intellectual and/or sensory impairments and other conditions such as epilepsy that may also affect function.[3] CP is common, accounting for 1.4 per 1000 live births in Australia, and is lifelong.[4] In most children, the brain injury/anomaly responsible for CP occurs in the pre/perinatal period. However, for a small group, their CP is subsequent to brain injury acquired in the post-neonatal (PNN) period, after 28 days, and before 2 years of age.[3,5]

Emerging evidence suggests that the causal pathways to PNN-CP are complex, with sociodemographic, environmental, and biological factors probably placing some children at increased risk of subsequent PNN injury.[6,7] Although the complete causal pathways to PNN-CP are frequently not fully understood, by definition the proximal causal event (e.g. infection or traumatic brain injury) is known. Identifying potentially modifiable components of the causal pathway to PNN-CP can inform preventive measures aimed at reducing prevalence and severity:[3] for example public health interventions, child protection policies, and legislative change.

In high-income countries, PNN-CP accounts for 3% to 9% of all children with CP,[3] with this group commonly presenting with a severe disability profile.[5] Encouragingly, evidence from population-based studies suggests that prevalence of PNN-CP is declining in high-income countries.[3,5,6] In contrast, recent data from CP registers for low- and middle-income countries have highlighted that, in these settings, PNN-CP can account for between 6% and 47% of all children with CP, with the proximal causes differing considerably to those observed in high-income settings.[8–11] These differences emphasize the importance of understanding the epidemiology of PNN-CP in context, to appropriately inform prevention efforts.

There have been two previous state-specific studies on PNN-CP in Australia.[6,7] Ours is the first study using Australian Cerebral Palsy Register (ACPR) data drawn from all Australian states and territories to investigate the causes and risk factors of PNN-CP in Australia. The more recent, expanded cohort enables us to identify at-risk groups and to inform the development of preventive strategies to minimize the risk of children acquiring PNN-CP. Our objectives were to (1) explore temporal trends in prevalence of PNN-CP between 1973 and 2012, (2) describe the clinical profile of children with PNN-CP, and (3) describe the sociodemographic profile of children with PNN-CP and identify potential associations with PNN causes.

What this paper adds

- Prevalence of post-neonatally acquired (PNN) cerebral palsy (CP) in Australia significantly declined between 1973 and 2012.
- Cerebrovascular accidents are the most common proximal cause of PNN-CP.
- Children born in remote areas are at greater risk of PNN-CP.

METHOD

The ACPR

Data were obtained from the ACPR which holds CP data from 1995 onwards. All Australian states and territories contribute data from their respective registers to the ACPR on a biennial basis. The ACPR data are used to (1) monitor the birth cohort prevalence of CP; (2) gain further understanding about the causes of CP; (3) evaluate preventive strategies; and (4) assist in planning services for children and adults who have CP. Registers use multiple ascertainment sources to collect CP data, including hospital records, state-wide data sets, clinician assessments, and family reports. The ACPR inclusion criteria require a diagnosis of CP confirmed at age 5 years. For example, a child born in 2012 will have their clinical details confirmed in 2017. If a child dies before their fifth birthday and their CP diagnosis had been confirmed, their data are still included. A definition of CP is accepted if five key elements are met: 'CP is (1) a group of disorders i.e. it is an umbrella term; (2) it is permanent but not unchanging; (3) it involves a disorder of movement and/or posture and of motor function; (4) it is due to a non-progressive interference/lesion/abnormality; (5) this interference/lesion/abnormality arises in the developing/immature brain'.[12] The timing of brain injury is recorded in the ACPR as PNN-CP when a PNN cause is identified; otherwise the timing is recorded as prenatal/perinatal.

Study cohort

In this study, we included records of children with PNN-CP born in Australia (1995–2012) who acquired their brain injury between 28 days and 2 years of age.[3–5] Also, two long-standing state registers – the Victorian Cerebral Palsy Register and the Western Australian Register of Developmental Anomalies – provided additional historical data from 1973 to 1994.

Study variables

PNN brain injury was categorized into one of four broad causal events as listed in the ACPR (infection, cerebrovascular accident [CVA], head injury, other). Clinical data included gestational age at delivery (<32 weeks, 32–36 weeks, and 37+ weeks), predominant CP subtype at 5 years (spastic unilateral, spastic bilateral [diplegia, triplegia/quadriplegia], ataxia, dyskinesia, hypotonia), sex (male, female), and Gross Motor Function Classification System (GMFCS)[13] level (I and II [mild]; III–V [moderate–severe]). Comorbid conditions were dichotomized: intellectual impairment (no or mild impairment [IQ ≥ 50] versus moderate to severe intellectual impairment [IQ < 50]; speech impairment (no, or some impairment, vs non-verbal); vision impairment (no, mild, or moderate impairment vs functionally blind); epilepsy (no active epilepsy at age 5 years vs active epilepsy at age 5 years); hearing impairment (no, mild, or moderate impairment vs bilateral deafness).

Sociodemographic profile

Sociodemographic factors investigated in this study included maternal age at the time of birth of the child with PNN-CP (<20 years vs ≥20 years), Accessibility and Remoteness Index of Australia$^+$ 2016 (ARIA$^+$ 2016),[14] and Socio-Economic Indexes for Areas (SEIFA).[15] All SEIFA and ARIA$^+$ 2016 data were drawn from postcode at birth; or, if missing, from postcode at age 5 years. ARIA$^+$ 2016 data were sourced from the Hugo Centre for Population and Housing, University of Adelaide, and were categorized as major city, inner regional, outer regional, remote, and very remote. SEIFA 2016 data were sourced from the Australian Bureau of Statistics. The SEIFA index used in this study was the Index of Relative Socio-Economic Advantage and Disadvantage described using SEIFA quintiles (1, least advantaged; 5, most advantaged).

Maternal Aboriginal and/or Torres Strait Islander status (Aboriginal and/or Torres Strait Islander, non-Indigenous) was also investigated in this study as a proxy for culturally unsafe health care. Decades of culturally unsafe health care has had detrimental effects on how First Nations families expect to experience the health care system and the subsequent health and well-being of Aboriginal and/or Torres Strait Islander people.[16] It is important to acknowledge that being born to an Aboriginal and/or Torres Strait Islander mother is not considered here as a 'risk factor'; instead, being treated unsafely is what is considered the actual risk factor.

Denominator data for birth years 1995 to 2012 were sourced from the National Perinatal Data Collection, Australian Institute of Health and Welfare,[17] whereas the Victorian and Western Australian denominator data were sourced from The Victorian Perinatal Data Collection for birth years 1973 to 1994 and the Australian Bureau of Statistics Demography (1973–1979) and Midwives Notification System Data, Department of Health, Western Australia (1980–1994).

Statistical analysis

Analysis of temporal trends was limited to data from two long-standing state CP registers (Victoria and Western Australia) that are understood to have near-complete population ascertainment between 1973 and 2012. The birth prevalence for Victoria and Western Australia was calculated in 5-year intervals (1973–1977, 1978–1982, 1983–1987, 1988–1992, 1993–1997, 1998–2002, 2003–2007, 2008–2012) as follows: the numerator was the count of eligible children with CP (as described above) born in these states for each 5-year epoch and included in these registers; the denominator was a count of all live births in these states during each 5-year interval. Birth prevalence for each epoch was calculated as the ratio of the numerator to denominator expressed as number of cases per 10 000 live births, together with the 95% confidence intervals (CI) around each estimate. Poisson regression was used to investigate temporal trends. The linearity of the average trends in the Poisson regression models was checked using added variable plots.

For all other analyses, available data from all states and territories were included for birth years 1995 to 2012. If a state or territory had over 20% missing data for a variable then those state or territory data were excluded from the analysis of that variable. A χ^2 goodness-of-fit test was used to compare PNN-CP clinical and sociodemographic data against Australian population reference data. Logistic regression was performed to assess the strength of association reported as the odds ratio (OR) between causes of PNN-CP and available clinical and sociodemographic variables. Variables were dichotomized owing to small numbers (e.g. ARIA$^+$ 2016 [remote and very remote vs major city, inner regional, and outer regional]). Analyses were performed using IBM SPSS Statistics version 27 (IBM Corp., Armonk, NY, USA).

Ethics

Ethics approval was received from The University of Sydney Human Research Ethics Committee and the Aboriginal

Health and Medical Research Council. Additional approvals were received from individual states and territories, as required. The ethics process and the analyses and interpretation of data, including those pertaining to Aboriginal and Torres Strait Islander people, were overseen by an Aboriginal researcher and listed author (TM). In addition, the authors sought consultation about the interpretation of results from the Community, Aboriginal and Torres Strait Islander Reference Group.

RESULTS

Victoria and Western Australia (1973–2012)

Temporal trends

There were 523 children with PNN-CP born between 1973 and 2012 registered with the Victorian Cerebral Palsy Register and Western Australian Register of Developmental Anomalies. A significant downward trend in prevalence of PNN-CP was evident in both Victoria and Western Australia (n = 523, 1973–2012). PNN-CP in Victoria declined by 7% per interval (95% CI 0.9–1.0, p = 0.047). Similarly, PNN-CP in Western Australia declined from 2.3 per 10 000 live births (1973–77) to 1.6 per 10 000 live births (2008–2012). Although prevalence fluctuated, the overall decline equated to 13% per epoch (95% CI 0.7–1.0, p = 0.033) (Figure 1, Table S1).

All states and territories (1995–2012)

Study population and clinical profile

There were 469 children with PNN-CP who were born in Australia between 1995 and 2012. Most children were singletons (94%, n = 387), born at term (82%, n = 242), and 176 (40%) children were classified in GMFCS levels III to V, indicating they required assistive equipment for mobility. Comorbid conditions were common, particularly intellectual (29%, n = 118) and speech (32%, n = 134) impairments and active epilepsy at 5 years (50%, n = 219) (Table 1). Spastic unilateral CP was the most common predominant motor subtype.

PNN-CP cause

The recorded proximal causes of PNN-CP are detailed in Table 1. CVAs were the most common cause, constituting one-third of the group, followed by infection and head injury which respectively accounted for one-quarter and one-fifth of the group. Among children who had sustained a PNN head injury, the most common cause was non-accidental injury.

CVAs were positively associated with GMFCS level I and II (OR 3.5 [95% CI 2.3–5.6], p < 0.001). Children with CVAs were significantly less likely to experience comorbid conditions including epilepsy (OR 0.5 [95% CI 0.3–0.7], p < 0.001),

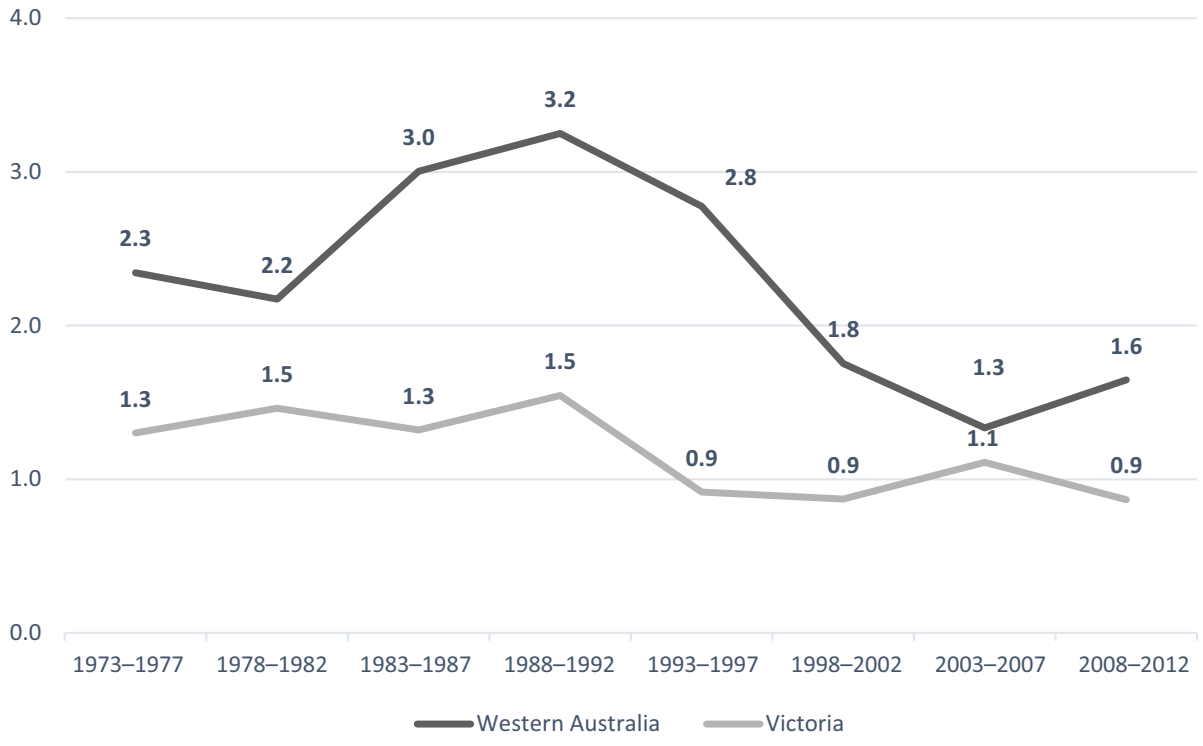

FIGURE 1 Temporal trends in prevalence of post-neonatally acquired cerebral palsy per 10 000 live births with 95% confidence intervals, Victoria and Western Australia, 1973–2012.

functional blindness (OR 0.3 [95% CI 0.1–0.7], $p = 0.004$), intellectual impairment (OR 0.3 [95% CI 0.1–0.4] $p < 0.001$), and non-verbal communication (OR 0.3 [95% CI 0.2–0.5], $p < 0.001$).

Greater limitations in gross motor function (GMFCS levels III–V) were positively associated with non-accidental injury (OR 2.3 [95% CI 1.3–4.1], $p = 0.005$) and infection (OR 1.9 [95% CI 1.2–2.8], $p = 0.007$). Children with non-accidental injury were also more likely to have comorbid conditions, including epilepsy (OR 2.1 [95% CI 1.2–3.8], $p = 0.012$), functional blindness (OR 4.4 [95% CI 2.2–9.0], $p < 0.001$), intellectual disability (OR 3.4 [95% CI 1.9–6.1], $p < 0.001$), and non-verbal communication (OR 2.0 [95% CI 1.1–3.5], $p = 0.029$).

Sociodemographic factors and clinical factors

Compared with Australian population data, children with PNN-CP were more likely to be born preterm (18%, $n = 53$), and/or to Aboriginal and/or Torres Strait Islander mothers (13%, $n = 39$). Drawing on available data from two states, we also found that 18% ($n = 20$) of children were born to teenage mothers, compared with 5% of the Australian population ($p < 0.001$). Most (81%, $n = 236$) children with PNN-CP were born in major cities or inner regional centres. However, compared with the general population, a higher proportion of children with PNN-CP lived in remote areas ($p < 0.001$). Of the remote group, children born to Aboriginal and/or Torres Strait Islander mothers were over-represented (17 out of 26) (OR 21.3 [95% CI 8.4–53.6], $p < 0.001$). PNN-CP was not associated with the child's sex or socioeconomic disadvantage, with children in this study relatively evenly represented across all SEIFA quintiles ($p = 0.169$; see Table 2).

Of children born to teenage mothers, one in two acquired PNN-CP through infectious pathways. These children were three times more likely to have acquired PNN-CP as a result of an infection than children of mothers 20 years old or older (OR 3.0 [95% CI 1.1–8.2], $p = 0.028$).

In addition, children born in remote areas were over four times more likely to have acquired PNN-CP through infectious pathways than children living in more urban areas (OR 4.47 [95% CI 2.0–10.2], $p < 0.001$), regardless of Aboriginal and/or Torres Strait Islander status. There was a lack of strong evidence to suggest that any of the investigated sociodemographic factors increased the risk of PNN-CP from other causes (Table 3).

DISCUSSION

Children with PNN-CP are frequently overlooked in research; however, this group holds potential for primary prevention opportunities that warrant our investigation. Using data from all eight Australian state and territory CP registers that constitute the ACPR, this study identified that 6% of all children with CP born within the study period acquired their condition in the PNN period.[4] This finding mirrors the proportions of PNN-CP observed in Europe.[3] Previous research suggests that children with PNN-CP are more likely to have a severe phenotype than children with pre/perinatally acquired CP.[4,5] In this study, 29% of children had moderate to severe intellectual disability, 32% relied on non-verbal communication, and 50% had epilepsy, all considerably higher proportions than those observed among children with pre/perinatally acquired CP.[4]

A statistically significant decline in the prevalence of PNN-CP was identified between 1973 and 2012 in both Victoria and Western Australia. These findings were consistent with earlier Victorian and European register papers, with birth years ranging from 1975 to 2000.[3,5,6] In high-income countries, there have been many legislative and public health interventions introduced to reduce child fatalities and brain injuries. Vaccinations, mandatory child safety seats, and pool fencing were introduced in this period and are likely to have contributed to this decline.[6,19–21] However, in more recent years (2003–2012) the prevalence of PNN-CP seems to have stabilized. Continued monitoring of PNN-CP prevalence by the CP registers will be required to see whether this low prevalence is maintained in the future.

CVAs can occur spontaneously or they may be associated with congenital cardiac anomalies, surgery, infection, and hypoxia.[22,23] CVAs were the most common cause of PNN-CP in our study. This is a shift from more historic findings reporting infection as the most common cause of PNN-CP,[6,7] possibly because of continued increase of infant vaccination coverage (such as measles–mumps–rubella) in Australia, especially in priority populations.[19,24] Bacterial infection accounted for most (84%) of all known infectious causes, with a much smaller proportion of children with viral infections. The specific causes of non-accidental injury were not specified in this paper; however, it is evident that, overall, non-accidental injury as a cause of CP commonly results in severe disability.

There are additional complexities to consider beyond the known proximal cause of PNN-CP. In some instances, risk factors existed before the defining PNN insult. In our cohort, over 10% of children acquired CP from a CVA associated with surgery that was undertaken to treat a pre-existing health condition. Children in our cohort were also significantly more likely to be born preterm than those in Australian reference data. A recent international data linkage study reported over 25% of children with PNN-CP had a major congenital anomaly.[25] These findings suggest that pre-existing health conditions, some of which may be genetic in origin, play an important, and as yet undefined, role in brain injury acquisition and subsequent PNN-CP.

Aboriginal and/or Torres Strait Islander children were disproportionately represented in PNN-CP. This finding is similar to a 2016 study that found that Aboriginal and/or Torres Strait Islander children had an almost five-fold increased risk of PNN-CP.[26] In this current study, Aboriginal and/or Torres Strait Islander children with PNN-CP were more likely to live in remote areas, which

TABLE 1 Clinical characteristics and causes of post-neonatally acquired (PNN) cerebral palsy (CP) in children born 1995–2012

Clinical characteristics	*n* (%)
Total PNN-CP	469
Predominant subtype	443
Spastic unilateral	233 (52.6)
Spastic bilateral (diplegia)	33 (7.4)
Spastic bilateral (triplegia/quadriplegia)	133 (30.0)
Ataxic	9 (2.0)
Dyskinetic	24 (5.4)
Hypotonic	11 (2.5)
Missing	26 (5.5)
GMFCS level	445
I and II	269 (60.4)
III–V	176 (39.6)
Missing	24 (5.3)
Comorbid conditions	
Intellect	414
No/mild intellectual impairment	296 (71.5)
Moderate/severe intellectual impairment	118 (28.5)
Missing	55 (11.7)
Speech	419
No/mild/moderate speech impairment	285 (68.0)
Non-verbal	134 (32.0)
Missing	50 (10.7)
Vision	413
No/mild/moderate impairment	370 (89.6)
Functional blindness	43 (10.4)
Missing	56 (11.9)
Epilepsy	436
No active epilepsy	217 (49.8)
Active epilepsy	219(50.2)
Missing	33 (7.0)
Hearing	396
No/mild/moderate impairment	378 (95.5)
Bilateral deafness	18 (4.5)
Missing	73 (15.6)
PNN-CP cause	***n* (%)**
Total	469
Infection total	117 (24.9)
Bacterial infection	81 (17.3)
Viral infection	16 (3.4)
Infection unknown	20 (4.3)
Cerebrovascular accidents total	158 (33.7)
Associated with surgery	48 (10.2)
Cardiac complications	18 (3.8)
Spontaneous/unknown	92 (19.6)
Head injury total	101 (21.5)
Non-accidental injury	58 (12.4)
Fall	15 (3.2)
Motor vehicle accident	10 (2.1)
Other head injury not specified	18 (3.8)
Other PNN-CP cause total	93 (19.8)
Post-seizure	22 (4.9)
Near-drowning	16 (3.4)
Perioperative hypoxia	10 (2.1)
Apparent life-threatening event	13 (2.7)
Other post-neonatal event	32 (6.8)

Missing numbers/percentages are based on the full count of 469 children.

was itself an independent risk factor. Urbanization typically accelerates population health; however, it can also emphasize inequities between major cities and remote priority populations.[27] These inequities include a lack of access to culturally safe health care and services, educational programmes, and maternal programmes, and limited employment opportunities in remote areas.[18,28,29] Children born in remote areas, regardless of Aboriginal and/or Torres Strait Islander status, were at greater risk of CP from PNN infection. Although vaccine uptake is generally equitable in remote communities[19] there is an increased risk of exposure to non-vaccine preventable infections such as Murray Valley encephalitis.[26] Future research into the causality of PNN-CP will investigate this. Inadequate access to health care services in rural and remote areas may also play a role in delayed treatment of the infection. This highlights the importance of equitable health care provision to families living in remote regions.

Historically, teenage pregnancy has been found to be a risk factor for CP regardless of timing of brain injury[30] and our cohort was no different. As shown in Table 2, children born to teenage mothers were over-represented in this cohort compared with Australian reference data. Children born to teenage mothers were another group at increased risk of infection and subsequent PNN-CP. Recent research has found that maternal age younger than 20 years is a predictor of delayed or lower uptake of infant vaccinations in Australia;[24,31] this will be explored in a future PNN-CP study. Youth-focused antenatal services need to be considered as a safe and accessible intervention. In addition, prevention opportunities such as sex education, provision of contraceptives, and keeping females in school should also be considered.[32]

This study had several limitations. It was an ACPR study using population data; however, the numbers were small. Once the data were stratified, the numbers became further reduced and confidence intervals were wide. Data pertaining to maternal age were limited to two states. For PNN-CP this information can be difficult to collect owing to the acquired injury occurring outside

TABLE 2 Clinical, sociodemographic, and maternal Aboriginal and/or Torres Strait Islander status details of children with post-neonatally acquired cerebral palsy born 1995–2012 and comparison with Australian reference data

Clinical and sociodemographic factors	*n* (%)	Australian reference data[a] (%)	*p*
Gestational age[b]	*n* = 295		<0.001
<32 weeks	10 (3.4)	(1)	
32–36 weeks	43 (14.6)	(6)	
37 weeks+	242 (82.0)	(93)	
Missing	16 (5.1)		
Sex	*n* = 469		0.100
Female	212 (45.2)	(49)	
Male	257 (54.8)	(51)	
Accessibility and remoteness[b]	*n* = 291		<0.001
Major city	190 (65.3)	(72)	
Inner regional	46 (15.8)	(18)	
Outer regional	29 (10.0)	(8)	
Remote	7 (2.4)	(1)	
Very remote	19 (6.5)	(1)	
Missing	20 (6.4)		
Socioeconomic advantage and disadvantage[b]	*n* = 291		0.169
Q1 (low socioeconomic status)	67 (23.0)	(19)	
Q2	48 (16.5)	(20)	
Q3	68 (23.4)	(20)	
Q4	54(18.6)	(20)	
Q5 (high socioeconomic status)	54 (18.6)	(20)	
Missing	20 (6.4)		
Maternal age, years[c]	*n* = 114		<0.001
<20	20 (18)		
20–34	77 (68)	(5)	
35+	17 (15)	(76)	
Missing	8 (6.6)	(19)	
Maternal Aboriginal and/or Torres Strait Islander status[b]	*n* = 302		<0.001
Aboriginal and/or Torres Strait Islander	39 (12.9)		
Missing	9 (2.9)	(4)	

[a]Australian reference data were sourced from the Australian Institute of Health and Welfare (1995–2012), Australian Bureau of Statistics Socio-Economic Indexes for Areas (2016), and Accessibility and Remoteness Index of Australia+ 2016 (2016).[15,17,18] [b]Victoria, Queensland, Western Australia, South Australia, and the Northern Territory data (total *n* = 311). [c]Western Australia and South Australia (total *n* = 122).

the pre/perinatal period, and therefore not necessarily documented in hospital records. Additionally, specific proximal cause data were limited, especially about the type of infection and what was classified as 'other PNN-injury'. A future study is planned which will source and analyse more detailed data on cause and timing of injury. A more recent cohort was used as a reference group for ARIA+ 2016 and the Index of Relative Socio-Economic Advantage and Disadvantage; it is unclear whether reference data were equally valid for earlier intervals. We are aware that there may be some under-ascertainment of children with acquired brain injury and CP. The ACPR Group is currently exploring opportunities to improve our ascertainment of CP records that involve an acquired brain injury. There are probably some children who have an acquired brain injury who would also meet the inclusion criteria for CP but who may not have received the clinical description/diagnosis of CP. As they have not been formally described as having CP, some of these children may not be known to the CP registers. This will be investigated in a future study.

CONCLUSION

This population register study recognized a temporal decline in PNN-CP. CVAs were the most common cause of PNN-CP, a shift from historic findings reporting infection

TABLE 3 Post-neonatally acquired cerebral palsy sociodemographic, clinical factors, and maternal Aboriginal and/or Torres Strait Islander status by cause, 1995–2012

Clinical and sociodemographic factors	Infection		Cerebrovascular accident		Head injury		Other post-neonatal cause	
	n (%)	OR (CI)	*n* (%)	OR (CI)	*n* (%)	OR (CI)	*n* (%)	OR (CI)
Gestational age[a]								
<37 weeks	17 (22)	1.37 (0.7, 2.6)	14 (15)	0.78 (0.4, 1.5)	12 (20)	1.2 (0.6, 2.4)	10 (16)	0.83 (0.4, 1.8)
37 weeks+	62 (78)	Reference	78 (85)	Reference	49 (80)	Reference	53 (84)	Reference
Sex								
Female	56 (48)	1.2 (0.6, 1.3)	76 (48)	1.2 (0.8, 1.8)	37 (37)	0.64 (0.4, 1.0)	43 (46)	0.95 (0.6, 1.5)
Male	61 (52)	Reference	82 (52)	Reference	64 (64)	Reference	50 (54)	Reference
Accessibility and remoteness[a]								
Remote/very remote	15 (20)	**4.47 (2.0, 10.2)**	6 (7)	0.61 (0.2, 1.6)	[a]	0.68 (0.2, 2.0)	[a]	0.14 (0.0, 1.0)
Major city/inner regional/outer regional	62 (80)	Reference	87 (93)	Reference	56 (93)	Reference	60 (98)	Reference
Socioeconomic advantage and disadvantage[a]								
Quintile 1	20 (26)	1.25 (0.7, 2.3)	23 (25)	1.15 (0.7, 2.0)	10 (17)	0.61 (0.3, 1.3)	14 (23)	0.99 (0.5, 2.0)
Quintiles 2–5	57 (74)	Reference	70 (75)	Reference	50 (83)	Reference	47 (77)	Reference
Maternal age, years[b]								
<20	10 (28)	**3.0 (1.1, 8.2)**	[a]	0.52 (0.2, 1.7)	[a]	0.91 (0.3, 3.0)	[a]	0.43 (0.1, 2.0)
20+	26 (72)	Reference	33 (89)	Reference	22 (85)	Reference	21 (91)	Reference
Maternal Aboriginal and/or Torres Strait Islander status[a]								
Aboriginal and/or Torres Strait Islander	15 (19)	1.94 (1.0, 3.9)	12 (13)	0.96 (0.5, 2.0)	10 (16)	1.3 (0.6, 3.0)	[a]	0.18 (0.0, 0.8)
Non-Indigenous	64 (81)	Reference	83 (87)	Reference	54 (84)	Reference	62 (97)	Reference

Bold type signifies a positive statistically significant association. Abbreviations: CI, confidence interval; OR, odds ratio.

[a]Victoria, Queensland, Western Australia, South Australia, and Northern Territory data (total *n* = 311).

[b]Western Australia and South Australia (total *n* = 122).

as the most common cause.[6,7] This suggests historic and current health intervention programmes (e.g. vaccinations) have been somewhat successful. However, it is clear that for children born in remote areas, born to teenage mothers, and born to Aboriginal and/or Torres Strait Islander mothers, more specific preventive measures and support are required. The over-representation of priority populations and the severity of the condition give cause for concern about this largely preventable disorder. A greater focus on strategies to support these groups for primary prevention of CP is recommended for future research.

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DATA AVAILABILITY STATEMENT

Research data are not shared.

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SUPPORTING INFORMATION

The following additional material may be found online:
Table S1: Temporal trends in prevalence of PNN-CP per 10 000 live births with 95% confidence intervals Victoria and Western Australia, 1973–2012.

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