

Comparing risks of cerebral palsy in births to Australian Indigenous and non-Indigenous mothers

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Children born to Aboriginal and Torres Strait Islander (Indigenous) women tend to have less favourable health outcomes than those born to non-Indigenous women^{1, 2}. However, Indigenous births form only a small proportion of all Australian births (~5%) and cerebral palsy (CP) is a reasonably rare outcome (~2-2.5/1000 live births). Data from CP registers in the Northern Territory (NT) and Queensland (QLD), both jurisdictions with higher proportions of people with Indigenous heritage, are now available to add to that of the longer standing CP register in Western Australia (WA). This has enabled comparisons of the frequency and characteristics of CP between the children of Indigenous and non-Indigenous women in a larger population than has previously been possible.

Data from statutory birth records (for numbers of neonatal survivors) and CP registers for the 1996-2005 birth cohorts in NT, QLD and WA were stratified by Indigenous status and whether the CP was acquired pre/perinatally or postneonatally. To be considered postneonatally acquired a recorded event with brain damaging potential, considered by medical caregivers to be the cause of their CP, had to have occurred between the ages of 28 days and 2 years. Relative risks associated with Indigenous status were estimated overall and separately for pre/perinatal and postneonatal acquisition. The distributions of birth characteristics, of impairments and of causes of postneonatal CP were compared between those born to Indigenous and non-Indigenous mothers.

In the three jurisdictions 1,469 cases of CP met inclusion criteria: 161 (11%) were born to Indigenous women and 83 (5.7%) were postneonatally acquired. The proportion postneonatally acquired was higher in those born to Indigenous women (14.3%) than to non-Indigenous women (4.6%). The relative risk of postneonatal CP with Indigenous status was 4.9 (95% confidence interval 3.0, 7.9) but only 1.42 (95% CI 1.2, 1.7) for pre/perinatal CP. The relative risks were highest in the NT where the proportion of the Indigenous community living in very remote areas is much higher than in QLD or WA. Almost half of postneonatal CP in Indigenous infants resulted from infection, and a further 35% from head injury, with significantly increased risks relative to the non-Indigenous: relative risks of 9.9 (95% CI 4.3, 23) and 11.4 (95% CI 4.4, 30) respectively. For non-Indigenous infants the most frequent cause was cerebrovascular accident, a cause which WA data suggests has increased over time with the increased survival of births with congenital heart defects. Within time of acquisition strata, the impairments of those with CP born to Indigenous women tended to be more numerous and more severe than those born to non-Indigenous women and within ethnic strata, the impairments postneonatally acquired were more numerous and severe than those pre/perinatally acquired.

Particularly in view of a concern shared by all three registers that CP in those of Indigenous heritage may be the subject of greater under-ascertainment than those without Indigenous heritage, we conclude that Indigenous children are at significantly greater risk of CP, particularly postneonatal CP, and their impairments tend to be more severe. The predominant cause of postneonatal CP in non-Indigenous children has shifted to cerebrovascular accident over time; however the historical causes of cerebral infections followed by head injury are still the most frequent causes in infants born to Indigenous women.

References

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Presenter

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