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# Impact of social disadvantage on cerebral palsy severity

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***See Appendix S1 (online supporting information) for names and affiliations of the members of the Australian Cerebral Palsy Register Group and CP QUEST.**

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**ABBREVIATIONS**

| | |
|---|---|
| ACPR | Australian Cerebral Palsy Register |
| IRSAD | Indices of Relative Socio-economic Advantage and Disadvantage |
| SES | Socio-economic status |

**AIM** To investigate the impact of socio-economic disadvantage on indicators of cerebral palsy (CP) severity – motor impairment, intellectual disability, and the presence of severe comorbidities – in children with CP in Australia.

**METHOD** Data from the Australian Cerebral Palsy Register were analysed. Socio-economic disadvantage was assessed using maternal age, maternal country of birth, and a measure of neighbourhood socio-economic status (SES) at the time of the child's birth. Descriptive

bivariate analysis, trend analysis, risk ratios, and mediation analysis were undertaken to examine the impact of disadvantage on the indicators of CP severity.
**RESULTS** A socio-economic gradient was demonstrated with an increasing proportion of children with non-ambulant status, at least moderate intellectual disability, and the presence of severe comorbidities (having epilepsy, functional blindness, bilateral deafness, and/or no verbal communication) with decreasing neighbourhood SES, teenage motherhood, and maternal minority ethnicity.
**INTERPRETATION** In Australia, socio-economic disadvantage at birth impacts adversely on CP severity at age 5 years. By identifying that socio-economically disadvantaged children with CP are at greater risk of more severe functional outcomes, we can inform targeted interventions at the family and neighbourhood level to reduce these inequities for children with CP.

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Socio-Economic Disadvantage and Severity of CP *Sue Woolfenden et al.*

**What this paper adds**

- Socio-economic disadvantage is associated with increased severity of cerebral palsy functional outcomes.
- This encompasses low neighbourhood socio-economic status, teenage motherhood, and maternal minority ethnicity.

[main text]

Cerebral palsy (CP) is a permanent but not unchanging disorder of movement and/or posture caused by a non-progressive lesion or anomaly originating in the immature brain.[1] It is the

most common physical disability of childhood,[2] with a prevalence of 1.4 to 2.1 per 1000 live births in Australia.[3] Motor impairment in CP ranges from mild to severe; common comorbidities include intellectual impairment, epilepsy, lack of communication, blindness, and deafness.[4,5] In order for children with more severe impairments and comorbidities to fully participate and contribute to society, they require more resources and support to optimize their long-term health, development, and well-being.[6,7]

While congenital anomalies and severe perinatal insults resulting in moderate to severe neonatal encephalopathy are associated with increased severity of CP,[8–12] the role of socio-economic disadvantage is also emerging. In studies from Canada, the USA, the UK, and Taiwan, low neighbourhood and family socio-economic status (SES) and/or low maternal education have been associated with increasing severity of motor impairment and/or intellectual impairment in CP.[13–17] In the USA, African American children have been reported to have more severe functional motor limitations compared to white children with CP.[18,16] If children with CP from socio-economically disadvantaged settings are more likely to have severe functional outcomes, this inequity may be compounded by educational disadvantage and poorer access to early intervention, which has clear implications for policy and practice.[19]

Our aim was to investigate the association between socio-economic disadvantage at birth and indicators of CP severity in Australian children. These indicators were the level of motor and intellectual impairment and presence of severe comorbidities (epilepsy, bilateral deafness, blindness, and no verbal communication) at age 5 years. By identifying if socio-economically disadvantaged children with CP have a greater risk of more severe functional outcomes, we are able to inform targeted multilevel approaches to prevention and early intervention at the individual, family, and neighbourhood level.

## METHOD

### Setting and participants

This study used data from the Australian Cerebral Palsy Register (ACPR), which holds non-identifiable data that is collected every 3 years from each of the eight Australian states and territories. These registers record the demographic, perinatal, diagnostic, and clinical characteristics of individuals described as having CP.[3] Data are confirmed at 5 years of age from either the child's medical record or a clinical assessment undertaken by staff from the child's home state or territory register.[4] Data from birth years 1993 to 2009 (the most recent ACPR birth cohort with data verified) were included. While the ACPR accepts data from all

state and territory CP registers, case ascertainment differs between the contributing registers. Each participating register uses active surveillance methods and multiple sources of ascertainment. At the time of data extraction, based on international prevalence figures, the ACPR required that registers meet a minimum ascertainment birth prevalence of 1.5 per 1000 live births within the state/territory boundaries excluding those with a known postneonatal cause of CP. Secondly, there should be no known bias in ascertainment of birth prevalence in the region as reported by CP register staff. At the time of data extraction, three states with long-standing registers met this requirement: Western Australia, South Australia, and Victoria. The remaining states and territories have more recently established registers and, although ascertainment is increasing rapidly, they were under ascertained at the time of data extraction.[3]

**Data collection**

Data items were selected based on available register data that have been demonstrated in the literature to have an impact on indicators of CP severity.[13–16,18,20,21] These factors were categorized as perinatal, child, individual level, and neighbourhood level SES in keeping with the bioecological framework of early childhood development.[22]

Perinatal factors were gestational age at birth (<37wks vs ≥37wks), birthweight (<2500g vs ≥2500g), plurality (singleton vs multiple birth), and number of previous births (≤3 vs ≥4). Child level factors were sex and timing of CP (presumed prenatal/perinatal vs postneonatal). Individual level SES factors were maternal age (<20 years vs ≥20 years), and maternal country of birth, which was classified according to the Australian Standard Classification of Cultural and Ethnic Groups based on the Australian Bureau of Statistics listing.[23] We then further classified these into country groups that represent minority ethnicities from low-income and middle-income settings (Africa, the Middle East, the Pacific, and Asia excluding Japan/Korea). There was insufficient register data available on maternal education and paternal occupation, therefore we did not use these two variables.

Neighbourhood SES was estimated using birth postcode which was then linked to the related Socio-Economic Indexes for Areas and Indices of Relative Socio-economic Advantage and Disadvantage (IRSAD) decile published by the Australian Bureau of Statistics.[24] The IRSAD is a composite of indicators that rank geographic areas according to their socio-economic characteristics based on 5-yearly census data of people, families, and dwellings across Australia, including education levels, income, and employment.[24] The lower the Socio-Economic Indexes for Areas decile, the higher the level of neighbourhood

disadvantage, that is the greater the number of households with low incomes, low education levels, and/or unskilled occupations.[24] Socio-Economic Indexes for Areas and IRSAD deciles were then combined into quintiles with quintile 1 being the most disadvantaged (deciles 1–2, described in this paper as 'low SES') and quintile 5 being the most advantaged (deciles 9–10, described in this paper as 'high SES').

**Indicators of CP severity**

Indicators of CP severity were level of motor impairment, intellectual impairment, and the presence of other severe comorbidities in children with CP at age 5 years. To categorize level of motor impairment, Gross Motor Function Classification System (GMFCS) levels were dichotomized to levels I to III (ambulant) and levels IV to V (non-ambulant).[25] Intellectual impairment was dichotomized as an IQ under 50 (at least moderate intellectual impairment) and IQ of 50 or more (no more than mild impairment). The presence of an additional severe comorbidity was defined as having epilepsy, functional blindness, bilateral deafness, and/or no verbal communication (reference category having no additional severe comorbidities).

**Statistical analysis**

Descriptive bivariate analysis and comparisons among perinatal, child, individual level, and neighbourhood SES IRSAD quintiles and indicators of CP severity were conducted using $\chi^2$ tests. The $\chi^2$ test for linear trend was used to test for strength of associations between perinatal, child, individual level, and neighbourhood SES across quintiles. The distribution (and 95% confidence intervals) of CP cases within each measure of severity was calculated and compared across neighbourhood SES quintiles. Crude risk ratios using log-binomial models were calculated to further examine the relationship between indicators of CP severity and maternal age, minority ethnicity, and low SES neighbourhood, stratified by gestational age (preterm <37wks, term ≥37wks). Mediation analysis was conducted to separate the effect of maternal age and minority ethnicity from socio-economic disadvantage using paramed STATA 14 (StataCorp, College Station, TX, USA). The controlled direct effect was estimated for socio-economic disadvantage that was not mediated.[26] Missing data were not imputed because the outcomes of interest were clinically assigned categorical variables and all missing percentage values are stated for each variable. Statistical analysis was performed in STATA 14. A sensitivity analysis was undertaken to examine the impact of ascertainment level.

**Ethics**

The ACPR has ethics approval through the Cerebral Palsy Alliance Human Research Ethics Committee (NHMRC HREC: EC00402). An ethical waiver was granted for use of ACPR data in this research study (project waiver number: 20170501).

**RESULTS**

At the time of data extraction, there was a total of 7241 Australian-born children with CP with birth years 1993 to 2009.[3] Postcode at birth, and therefore Socio-Economic Indexes for Areas, was available for 6289 (87%) children. Characteristics of this CP cohort are outlined in Table I. The CP cohort included relatively more families at the higher end of the neighbourhood SES spectrum (quintiles 4 and 5, 43%) compared with the wider Australian population of live births (quintiles 4 and 5, 38%); however comparisons of these two sets of data are mathematically limited because they compare different durations of time. Of the mothers of children with CP, 359 (6%) were aged under 20 years when they gave birth and 534 (9%) were of a minority ethnicity (born in low-income and middle-income countries). The proportion of mothers aged under 20 years, of minority ethnicity, or who had four or more previous births in low SES neighbourhoods was higher (for each $p<0.001$, $p=0.001$, $p<0.001$ respectively). There was a significant association between high SES neighbourhoods and multiple birth ($p=0.002$), first-born child ($p<0.001$), and a mother aged 40 years or more at the time of their birth ($p<0.001$).

In a univariate analysis of the association between perinatal and child risk factors and severity of CP indicators there were greater proportions of children with CP at age 5 years in GMFCS levels IV to V, with an IQ under 50, and/or severe comorbidity who had a postneonatal onset type of CP, birthweight of 2500g or lower, comorbidity, and term gestation (for each $p<0.001$) (Table SI, online supporting information).

There were significant positive linear trends between IRSAD quintiles and the three indicators of severe functional outcomes in CP (Fig. 1). In quintile 1 (most disadvantaged – low SES), 32 per cent of children had a GMFCS level of IV to V, 32 per cent had an IQ under 50, and 48 per cent had a severe comorbidity, compared with 26 per cent, 26 per cent, and 40 per cent of children in quintile 5 (most advantaged – high SES) respectively.

The crude and stratified risk ratios of indicators of CP severity at 5 years of age are shown in Table II. The increased risk of GMFCS levels IV to V, an IQ under 50, and presence of a severe comorbidity in people with CP born in low SES neighbourhoods (quintle 1) was seen regardless of gestational age (preterm vs term). For teenage motherhood (aged

<20y) the increased risk for all severity indicators was present except in infants born at term in GMFCS levels IV to V. For maternal minority ethnicity, the increased risk for all severity indicators were recorded, with the exception of the presence of severe comorbidities for infants born preterm.

In our mediation analysis in STATA, when the mediator was set to mother aged under 20 years or maternal minority ethnicity there was a minimal direct effect of low neighbourhood SES on GMFCS levels IV to V (risk ratio 1.03, 95% CI 1.02–1.05 [<20 y]/risk ratio 1.07 95% CI 1.05–1.08 [minority]), an IQ under 50 (risk ratio 1.03, 95% CI 1.02–1.04 [<20y]/risk ratio 1.07 95% CI 1.05–1.08 [minority]), and the presence of severe comorbidities (risk ratio 1.03, 95% CI 1.02–1.04 [<20y]/risk ratio 1.05, 95% CI 1.04–1.07 [minority]). This would suggest that the increased risk of being non-ambulant, having at least a moderate intellectual impairment, and the presence of severe comorbidities in the children of teenage and/or minority ethnicity mothers is not further modified by living in a low SES neighbourhood. However, when the mediator was set to mothers aged more than 20 years, or not from a minority ethnicity, there was a strong direct effect of low neighbourhood SES on GMFCS levels IV to V (risk ratio 2.9, 95% CI 2.6–3.3 [≥20y]/risk ratio 2.7, 95% CI 2.4–3.0 [not minority]), an IQ under 50 (risk ratio 3.6, 95% CI 3.0–4.3 [≥20y]/risk ratio 3.2, 95% CI 2.8–3.9 [not minority]), and the presence of severe comorbidities (risk ratio 3.0, 95% CI 2.6–3.4 [≥20y]/risk ratio 2.9, 95% CI 2.5–3.3 [not minority]). This suggests that living in a low SES neighbourhood is associated with an additional risk of being non-ambulant, having at least a moderate intellectual impairment, and the presence of severe comorbidities in the offspring of mothers who are older than 20 years of age and/or who were not from a minority ethnicity.

**Sensitivity analysis**

When only population ascertained states (Western Australia, Victoria, South Australia, *n*=3628 [58%]) were examined, the impact of neighbourhood low SES was only seen when IQ was under 50. The impact of teenage motherhood was unchanged and the impact of maternal minority ethnicity remained for GMFCS level and IQ under 50 (Table SII, online supporting information).

## DISCUSSION

The aim of the study was to investigate the association between socio-economic disadvantage and CP severity in Australian children. CP is a condition which is traditionally associated with biological mechanisms and risk factors.[8,9,20,21] In this study of Australian CP data we have identified a social gradient where with decreasing neighbourhood SES at birth there were increasing proportions of children who: were non-ambulant; had at least a moderate intellectual impairment; and/or had a severe comorbidity in terms of functional blindness, deafness, epilepsy, and/or no verbal communication at age 5 years. In addition to low neighbourhood SES, individual level measures of socio-economic disadvantage, teenage motherhood, and/or maternal minority ethnicity, were associated with more severe functional outcomes associated with CP. Of interest, when the distribution of quintiles in our study was compared with those of live births in the 1996, 2001, and 2006 Australian censuses, there appear to be more children with CP born in postcodes in higher quintiles.[13] This fact makes the finding of a social gradient with severe/complex CP even more striking, given this pattern was not seen in CP overall. Further study on how SES affects risk of CP is needed to better understand this phenomenon.

Our findings concur with international research that has demonstrated that low neighbourhood SES is associated with increased risk of severe intellectual impairment[14] and greater motor impairment.[15–17] Our study is the first to show a relationship between low neighbourhood SES and the presence of severe comorbidities other than intellectual impairment. In Canada, the relationship between low neighbourhood SES and reduced mobility in 5-year-old children with CP was only seen in those who were born preterm.[13] However, in our study, the impact of low neighbourhood SES on indicators of CP severity was seen consistently in the infants born at term for GMFCS levels IV to V, presence of severe comorbidities, and in all gestational groups for an IQ under 50.

The relationship between low neighbourhood SES and an increased risk of being non-ambulant, having at least a moderate intellectual disability, and/or a severe comorbidity may reflect increased exposure to prenatal/perinatal and postneonatal injury such as poor nutrition, stress, infection, and teratogens.[8,12,27] In addition, there may be a compounding issue with the fact that families from low SES environments may have more difficulty accessing timely antenatal and early childhood intervention in their low SES neighbourhoods that may improve child health and developmental outcomes.[19]

To date no research has demonstrated an association between teenage motherhood and increased motor and intellectual impairment and severity of comorbidities. In Australia, it

is in this group that there has been no decline in CP prevalence, despite there being an overall decline in CP prevalence, which makes the finding even more concerning.[3] This group is potentially one of the most difficult to engage in health promotion. Our mediation analysis indicated that being a teenage mother conferred its own risk, which may include increased risk of congenital anomalies,[28] risky behaviour during pregnancy, a lack of support, and barriers to accessing antenatal services.

Our finding of increased severity of CP with maternal minority ethnicity is of interest. The results of our mediation analysis suggest that minority ethnicity confers its own risk in terms of severity of CP. This may be due to an increased biological risk and/or factors such as cultural acceptability and discrimination being barriers to accessing services.[29]

An alternative explanation is that teenage motherhood, minority ethnicity, and low neighbourhood SES are all markers for an unmeasured latent causal variable that increases severity of CP. These variables may include genetic predisposition and other psychosocial risk factors that have independent and cumulative effects on CP. Further detailed analysis of causal pathways is required to investigate this in future studies

**Strengths and limitations**

While a strength of this study is the use of register-based data, it is also a limitation. The ACPR minimum data set does not include data on family wealth or language spoken at home and there was missing data on maternal education and paternal occupation resulting in these factors being excluded from the analysis. There are limitations to the accuracy of interpretation of neighbourhood SES data applied retrospectively on the ACPR population as the IRSAD are designed for comparison at one point in time rather than longitudinally.[24] In addition, in the newer not yet fully ascertained state/territory registers there is the possibility of bias in data collection as illustrated by the sensitivity analysis with the impact of neighbourhood SES not seen in population ascertained states for motor impairment or presence of a severity comorbidity; however the impact on intellectual impairment remained.

Although we have demonstrated a clear association between socio-economic disadvantage and indicators of CP severity, we are unable to further investigate causal pathways with the current level of non-identifiable data in the CP registers. Of note we did not demonstrate a link between postneonatal causes of CP and socio-economic disadvantage. This may reflect our relatively small sample size of children with postneonatal onset in the

ACPR compared to the preneonatal/perineonatal sample. In addition, we are unable to tell how much of the associations we have demonstrated related to the type and severity of brain injury or, alternatively, to development after birth, or both. It is likely that the causal pathways involve cumulative exposure to both biological and psychosocial risk factors preneonatally/perineonatally and postneonatally.[22] Future research with predictive modelling of the impact of these factors is required.

**Conclusion**

We have shown that in Australia, socio-economic disadvantage at birth is associated with severe and complex CP and this in part is mediated by low neighbourhood SES, teenage motherhood, and minority ethnicity. It is important that this is examined in detail in future research to better understand why socio-economic disadvantage confers an increased risk of having a child with severe CP at age 5 years, so that the risk might be modified through appropriate prevention strategies. Potentially, this also means that the children with greatest need for early intervention services are the most likely to have the least financial resources available to them and to live in neighbourhoods with fewer available services.[13] We thus need to ensure that our family and neighbourhood level early interventions for CP have the reach and intensity to support families of children with CP who are socio-economically disadvantaged. This includes innovative engagement approaches such as digital strategies for teenage mothers and culturally safe services for mothers from minority ethnicities. CP is a lifelong condition; the greater we can help reduce this inequity in the severity of its impairments, the greater the ease with which all children with CP can fully participate in society.

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**Supporting information**

The following additional material may be found online:

**Appendix S1:** Names and affiliations of the members of the Australian Cerebral Palsy Register Group and CP QUEST.

**Table SI:** Associations between perinatal, child, and individual factors and indicators of severity CP at age 5 years

**Table SII:** Sensitivity analysis of impact ascertainment level



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**Table I**: Associations between perinatal, child, and individual factors and neighbourhood socio-economic status (SES)

| Characteristic<br>Total *n*=6289 (100%) | Quintile 1 (low SES)<br>*n*=1110, (17.7%) | Quintile 2<br>*n*=1101 (17.5%) | Quintile 3<br>*n*=1370 (21.8%) | Quintile 4<br>*n*=1228 (19.5%) | Quintile 5 (high SES)<br>*n*=1480 (23.5%) | *p* |
|---|---|---|---|---|---|---|
| Australian Bureau of Statistics census live births comparison (1996, 2001, 2006) | 21% | 20% | 20% | 20% | 18% | |
| Sex (*n*=6289) | | | | | | 0.790 |
| Male *n*=3591 | 17.9 | 17.7 | 21.8 | 19.0 | 23.6 | |
| Female *n*=2698 | 17.3 | 17.3 | 21.7 | 20.2 | 23.5 | |
| Onset (*n*=6289) | | | | | | 0.183 |
| Preneonatal/perineonatal *n*=5909 | 21.0 | 17.4 | 22.9 | 15.5 | 23.2 | |
| Postneonatal *n*=380 | 17.4 | 17.5 | 21.7 | 19.8 | 23.6 | |
| Gestation (*n*=6189) | | | | | | 0.203 |
| <37wks *n*=2571 | 17.1 | 18.1 | 20.4 | 20.1 | 24.3 | |
| ≥37wks *n*=3618 | 17.8 | 17.2 | 22.6 | 19.3 | 23.1 | |
| Birthweight (*n*=6031) | | | | | | 0.423 |
| <2500g (low birthweight) *n*=2519 | 17.1 | 18.3 | 21.4 | 18.8 | 24.4 | |
| ≥2500g (birthweight) *n*=3512 | 17.8 | 17.1 | 22.0 | 20.1 | 23.0 | |
| Plurality (*n*=6138) | | | | | | 0.002 |
| Multiple *n*=711 | 13.8 | 16.1 | 20.8 | 20.7 | 28.6 | |
| Singleton *n*=5427 | 18.0 | 17.6 | 22.0 | 19.5 | 22.9 | |
| Parity (*n*=5779) | | | | | | <0.001 |
| Multiparous *n*=3015 | 19.5 | 18.6 | 21.6 | 19.0 | 21.3 | |
| Primiparous *n*=2764 | 15.6 | 16.2 | 22.3 | 20.9 | 25.0 | |
| Previous births (*n*=5779) | | | | | | <0.001 |
| ≥4 *n*=264 | 31.5 | 17.8 | 22.7 | 14.4 | 13.6 | |
| ≤3 *n*=5515 | 17.0 | 17.5 | 21.9 | 20.1 | 23.5 | |
| Maternal age (*n*=5905) | | | | | | <0.001 |
| <20y *n*=359 | 26.5 | 18.1 | 25.4 | 15.3 | 14.8 | |
| ≥20y–<40y *n*=5366 | 17.2 | 17.9 | 21.7 | 19.8 | 23.4 | |
| ≥40y *n*=180 | 10.6 | 10.6 | 18.3 | 17.8 | 42.8 | |
| Mother ethnicity (*n*=5913) | | | | | | 0.001 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Minority *n*=534 | 26.8 | 10.9 | 18.5 | 20.4 | 23.4 | |
| Non-minority *n*=5379 | 17.0 | 18.4 | 22.1 | 19.3 | 23.2 | |

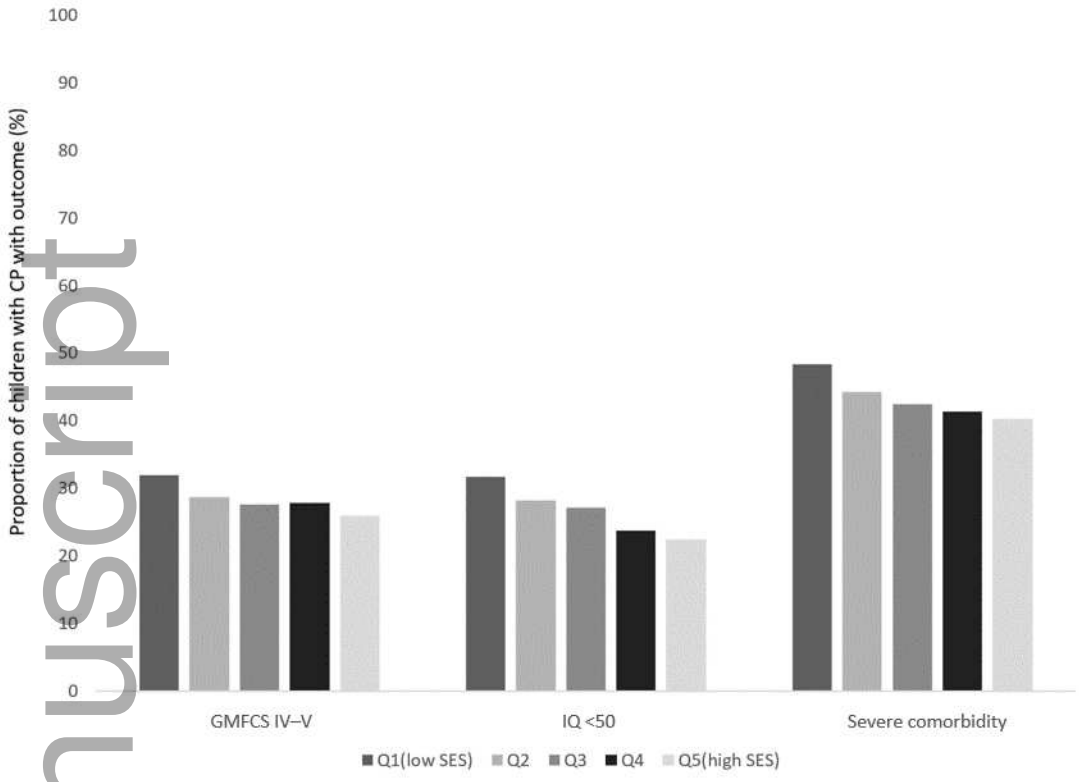
**Table II**: Crude risk ratios of indicators of severity of cerebral palsy by measures of family and neighbourhood social disadvantage, stratified by gestational age

| Outcome | GMFCS IV–V (RR 95% CI) | | | IQ<50 (RR 95% CI) | | | Severe comorbidity (RR 95% CI) | | |
|---|---|---|---|---|---|---|---|---|---|
| Sample | Total sample | Preterm group (<37wks) | Term group (≥37wks) | Total sample | Preterm group (<37wks) | Term group (≥37wks) | Total sample | Preterm group (<37wks) | Term group (≥37wks) |
| Neighbourhood SES | | | | | | | | | |
| Quintile 1 (low SES) | 1.2 (1.1–1.4) | 1.3 (1.0–1.5) | 1.2 (1.0–1.4) | 1.4 (1.2–1.6) | 1.6 (1.2–2.1) | 1.3 (1.1–1.6) | 1.2 (1.1–1.3) | 1.2 (1.0–1.5) | 1.2 (1.1–1.3) |
| Quintile 2 | 1.1 (0.97–1.3) | 1.2 (0.9–1.4) | 1.1 (0.9–1.3) | 1.3 (1.1–1.5) | 1.4 (1.1–1.8) | 1.2 (0.99–1.4) | 1.1 (0.99–1.2) | 1.2 (0.97–1.4) | 1.1 (0.97–1.2) |
| Quintile 3 | 1.1 (0.9–1.2) | 1.0 (0.8–1.3) | 1.1 (0.9–1.3) | 1.2 (1.0–1.4) | 1.1 (0.9–1.5) | 1.2 (1.0–1.4) | 1.1 (0.96–1.2) | 1.1 (0.9–1.3) | 1.0 (0.9–1.1) |
| Quintile 4 | 1.1 (0.9–1.2) | 1.10 (0.9–1.4) | 1.1 (0.9–1.3) | 1.1 (0.9–1.2) | 1.0 (0.8–1.4) | 1.1 (0.9–1.3) | 1.0 (0.9–1.1) | 1.1 (0.9–1.3) | 0.99 (0.9–1.1) |
| Quintile 5 (high SES) | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| Maternal age <20y[a] | 1.2 (1.0–1.4) | 1.3 (1.0–1.6) | 1.1 (0.9–1.4) | 1.4 (1.2–1.6) | 1.6 (1.3–2.1) | 1.3 (1.0–1.5) | 1.3 (1.1–1.4) | 1.5 (1.3–1.8) | 1.1 (1.0–1.3) |
| Maternal minority ethnicity[b] | 1.3 (1.2–1.4) | 1.32 (1.1–1.6) | 1.3 (1.1–1.5) | 1.4 (1.3–1.6) | 1.3 (1.0–1.7) | 1.5 (1.3–1.7) | 1.1 (1.0–1.2) | 1.1 (0.9–1.4) | 1.1 (1.0–1.2) |

[a]Reference category maternal age 20 years and over. [b]Reference category non-minority. GMFCS, Gross Motor Function Classification System; RR, risk ratio; SES, socio-economic status.

[Figure legend]

**Figure 1**: Severity of outcomes (%) by neighbourhood socio-economic status (SES) (Indices of Relative Socio-economic Advantage and Disadvantage [IRSAD] quintiles [Qs]). CP, cerebral palsy; GMFCS, Gross Motor Function Classification System.

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