*Journal of Developmental Origins of Health and Disease*

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# Growth from birth to 6 months of infants with and without intrauterine preeclampsia exposure

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**Brief Report**

**Cite this article:** Gow ML, Roberts L, Henry A, Davis G, Mangos G, Pettit F, Khouri JM, Dosen A, Brown MA, and Craig ME. (2022) Growth from birth to 6 months of infants with and without intrauterine preeclampsia exposure. *Journal of Developmental Origins of Health and Disease* **13**: 151–155. doi: 10.1017/S2040174421000167

Received: 14 January 2021
Revised: 16 March 2021
Accepted: 18 March 2021
First published online: 12 May 2021

**Keywords:**
Growth; infants; preeclampsia; small for gestational age

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## Abstract

Intrauterine preeclampsia exposure affects the lifelong cardiometabolic health of the child. Our study aimed to compare the growth (from birth to 6 months) of infants exposed to either a normotensive pregnancy or preeclampsia and explore the influence of being born small for gestational age (SGA). Participants were children of women participating in the Post-partum, Physiology, Psychology and Paediatric follow-up cohort study. Birth and 6-month weight and length z-scores were calculated for term and preterm (<37 weeks) babies, and change in weight z-score, rapid weight gain (≥0.67 increase in weight z-score) and conditional weight gain z-score were calculated. Compared with normotensive exposed infants ($n = 298$), preeclampsia exposed infants ($n = 84$) were more likely to be born SGA (7% versus 23%; $P < 0.001$), but weight gain from birth to 6 months, by any measure, did not differ between groups. Infants born SGA, irrespective of pregnancy exposure, were more likely to have rapid weight gain and had greater increases in weight z-score compared with those not born SGA. Preeclampsia exposed infants born SGA may benefit from interventions designed to prevent future cardiometabolic disease.

## Introduction

Preeclampsia, a multisystem disorder of pregnancy characterised by new onset hypertension and evidence of maternal organ dysfunction and/or fetal growth restriction, affects 2–5% of pregnancies worldwide.[1] The effects of preeclampsia on the lifelong cardiovascular health of the mother are well documented,[2,3] and effects on the child are also profound. The associated fetal growth restriction and increased preterm birth rates lead to an estimated 500,000 neonatal deaths each year globally, as well as related morbidity for survivors.[4,5] Cardiometabolic health in particular is impacted with a systematic review in individuals aged 4–30 years demonstrating increased blood pressure and weight in those exposed to intrauterine preeclampsia compared with a normotensive pregnancy.[6] As adults, cardiovascular morbidity is independently associated with intrauterine preeclampsia exposure.[7] Detrimental associations with neuro-development have also been reported including reduced cognitive function[8] and increased risks of autism spectrum disorder,[9] cerebral palsy, epilepsy and obstructive sleep apnoea.[10]

Intrauterine preeclampsia exposure is also associated with increased risk of the baby being born small for gestational age (SGA) due to compromised intrauterine nutrition.[4] Individuals born SGA are more likely to experience rapid 'catch-up' growth in infancy and early childhood which has been implicated in an increased risk of cardiovascular disease as adults.[11,12] However, there are limited data examining the growth trajectories of preeclampsia exposed infants specifically, and the influence of being born SGA on the growth of these infants is unknown. One study reported that children born following preeclampsia exposure had greater height gain from birth to 18 months than those unexposed, irrespective of SGA status.[13]

Therefore, the primary aim of this paper was to compare the growth of infants with or without intrauterine preeclampsia exposure from birth to 6 months of age, including examining how this may differ by SGA status.

## Methods

Infants in this study were offspring of participants in a cohort trial known as the P4 (Post-partum, Physiology, Psychology and Paediatric follow up) study conducted at St George Hospital in Sydney, Australia. The study was approved by the Prince of Wales Hospital Human Research Ethics Committee (HREC/12/POWH395). Written informed consent was obtained from the mother at study enrolment. The trial was registered with the Australian New Zealand Clinical Trial Register (ACTRN12613001260718). A detailed study protocol has been published, as has the primary outcome paper reporting maternal outcomes.[14,15] In brief, eligible women were those who gave birth to a live baby within the previous 6 months and had a good understanding of written and spoken English. Women were excluded if they had diabetes, hypertension, renal or other serious maternal disease prior to pregnancy, a multiple pregnancy, their baby was born with a congenital anomaly or they were pregnant again.

The P4 study was powered on maternal 24 h mean diastolic blood pressure.[14] In total, 302 women who had a normotensive pregnancy, 23 who experienced gestational hypertension and 90 who had preeclampsia during pregnancy were recruited to the trial. This study focuses on paediatric growth and includes infants with a recorded weight at both birth and 6 months born following either a normotensive pregnancy or preeclampsia exposure during pregnancy. Preeclampsia was diagnosed according to the criteria of the International Society for the Study of Hypertension in Pregnancy[16] as new onset hypertension (blood pressure ≥140 mmHg systolic or ≥90 mmHg diastolic) at or after 20 weeks' gestation with one or more of the following: proteinuria, other maternal organ dysfunction and/or uteroplacental dysfunction. The 6-month infant data collection began in 2013 and was complete in August 2019.

### Primary outcome data

Birth weight and length were collected retrospectively by author LR from the mother's maternity medical record. Weight and length at 6 months were measured using standard procedures.

Birth and 6-month weight and length z-scores were calculated for preterm (<37 weeks gestation) babies, corrected for gestational age, using the international INTERGROWTH-21st Preterm Postnatal Growth Standards[17] and for term babies using the British 1990 referenced data, reanalysed in 2009 and the WHO Child Growth Standards.[18,19] Body mass index (BMI) (weight (kg)/length $(m)^2$) was calculated to assess body proportionality. BMI z-score or weight-for-length z-score was not calculated as they cannot be determined using the INTERGROWTH-21st Preterm Postnatal Growth Standards.[17] SGA was defined as a birth weight z-score below −1.28 for sex and gestational age (corresponding to the 10th percentile).[20,21]

Infant weight gain was explored in three ways. First, weight z-score change from birth to 6 months was calculated. Second, rapid weight gain was calculated, defined as an increase in weight z-score above or equal to 0.67 from birth to 6 months, which is interpreted clinically as crossing centile lines on a growth chart.[22] This method is commonly used but subject to bias related to age, length of time between measures and starting weight, especially in babies born SGA who are more likely to experience rapid weight gain as their weight z-score regresses to the mean over time. Therefore, a third measure of weight gain, conditional weight gain z-score, was determined, calculated as the standardised residuals from the linear regression of the 6-month weight z-score on birth weight z-score, with age and sex entered as covariates, as previously described.[23–26] The conditional weight gain z-score calculation considers the potential confounding influence of birth weight z-score, age and sex. A positive value indicates a faster and a negative value a slower rate of weight gain compared with the population mean weight gain.

### Covariates

The following infant, maternal and birth detail data were collected and considered in relation to their association with preeclampsia exposure and infant growth outcomes. Detailed methods are described in the published protocol.[14]

- Infant data: sex, gestational age at birth, parity, length of any neonatal intensive care unit and/or special care nursery stay, feeding at discharge and breastfeeding status at 6 months.
- Prenatal maternal exposures: age, education, ethnicity, smoking history, pre-pregnancy weight and BMI
- Birth details: labour onset, mode of birth

### Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics, version 26.0 (Chicago, IL). Descriptive statistics were conducted to summarise infant growth outcomes and covariates for the two groups (i.e. normotensive pregnancy or preeclampsia exposure). Independent sample *t*-tests (parametric distribution), Mann–Whitney U tests (non-parametric distribution) or Chi-Square tests (categorical data) were performed to estimate whether infant growth outcomes and covariates differed between groups. Additionally, the influence of SGA status on change in growth outcomes by preeclampsia exposure was also investigated using independent sample *t*-tests and Chi-Square tests as appropriate. Simple linear regression was conducted to determine associations between infant weight gain (i.e. weight z-score change, rapid weight gain and conditional weight gain z-score) and birth, infant and maternal variables. In all analyses, a $P$-value $< 0.05$ was considered statistically significant.

## Results

In total, 382 infants (298 exposed to a normotensive pregnancy and 84 with preeclampsia exposure) with both a birth and 6-month weight were included in this analysis. As expected, infants with preeclampsia exposure had a shorter gestation, were more likely to be born preterm, more likely to have had a neonatal intensive care or special nursery admission following birth and more likely to be their mother's first-born child (Table 1). They were also more likely to have been born following non-spontaneous labour (preeclampsia: 91%, normotensive: 42%; $P < 0.001$) and less likely to have been delivered via a normal vaginal birth (preeclampsia: 33%, normotensive: 66%; $P < 0.001$). Compared with mothers who had a normotensive pregnancy, mothers who had preeclampsia were younger (preeclampsia: 31.9 ± 5.0 years, normotensive: 33.3 ± 4.7 years; $P = 0.026$) and had a higher pregnancy booking-in BMI (preeclampsia: 25.8 ± 5.3 $kg/m^2$, normotensive: 24.0 ± 5.0 $kg/m^2$; $P = 0.007$). Ethnicity, smoking history and education of the mother did not differ between groups. Mothers who had preeclampsia were significantly less likely to be breastfeeding at discharge (preeclampsia: 86%, normotensive: 94%; $P = 0.019$) and less likely to be breastfeeding at 6 months (preeclampsia: 60%, normotensive: 82%; $P < 0.001$).

https://doi.org/10.1017/S2040174421000167 Published online by Cambridge University Press

**Table 1.** Birth, feeding growth and developmental outcomes of infants with or without intrauterine exposure to preeclampsia exposure

| | Normotensive pregnancy ($n = 298$) | Preeclampsia ($n = 84$) |
|---|---|---|
| Birth details | | |
| Gestation, week, median [IQR] | 39.7 [1.9] | 37.5 [2.7]* |
| Preterm (<37 weeks), n [%] | 19 [6] | 29 [35]* |
| Neonatal ICU/special care nursery admission, n [%] | 38 [13] | 45 [54]* |
| Male sex, n [%] | 153 [51] | 41 [49] |
| First-born child, n [%] | 148 [50] | 61 [73]* |
| Birth anthropometry | | |
| Weight, kg | 3.37 (0.51) | 2.75 (0.73)* |
| Length, cm[a] | 50.28 (2.62) | 47.63 (4.01)* |
| BMI (kg/m$^2$)[a] | 13.27 (1.33) | 12.16 (1.74)* |
| Weight z-score | 0.05 (0.95) | −0.46 (0.93)* |
| Length z-score[a] | 0.08 (1.09) | −0.43 (1.03)* |
| SGA, n [%] | 22 [7] | 19 [23]* |
| Six-month anthropometry | | |
| Weight, kg | 7.93 (0.96) | 7.61 (0.99)* |
| Length, cm | 68.47 (2.987) | 67.55 (2.68)* |
| BMI (kg/m$^2$) | 16.89 (1.50) | 16.64 (1.46) |
| Weight z-score | 0.05 (0.94) | −0.21 (0.95)* |
| Length z-score | 0.37 (1.20) | 0.12 (0.99) |
| Change in anthropometry, birth to 6 months | | |
| Weight gain, kg | 4.56 (0.94) | 4.86 (0.99)* |
| Length gain, cm[a] | 18.20 (3.26) | 19.94 (3.80)* |
| Change in BMI (kg/m$^2$)[a] | 3.62 (1.84) | 4.48 (2.02)* |
| Change in weight z-score | 0.00 (1.11) | 0.26 (1.10) |
| Change in length z-score[a] | 0.29 (1.33) | 0.56 (1.14) |
| Rapid weight gain, n [%] | 80 [27] | 30 [36] |
| Conditional weight gain z-score | 0.02 (1.00) | −0.06 (0.99) |

All data reported as mean (SD), unless otherwise indicated.
cm, centimetre; ICU, intensive care unit; kg, kilogram; m, metre; n, number; SGA, small for gestational age.
*Indicates significant difference ($P < 0.05$) between groups.
[a]Missing data: NP $n = 296$, PE $n = 80$.

At birth, all anthropometric outcomes of infants exposed to preeclampsia were reduced compared with infants exposed to a normotensive pregnancy and preeclampsia exposed infants were more likely to be born SGA (Table 1). At 6 months, weight, weight z-score and length remained significantly reduced in preeclampsia compared with normotensive exposed infants, but length z-score and BMI were no longer significantly different. From birth to 6 months, infants exposed to preeclampsia had significantly greater increases in absolute weight, length and BMI. However, change in weight z-score and length z-score were not different between groups. Similarly, rapid weight gain and conditional weight gain z-score did not differ between groups (Table 1).

Infants with preeclampsia exposure who were born SGA ($n = 19$) had significantly greater weight z-score gain compared with preeclampsia exposed infants who were not born SGA ($n = 65$) (mean difference [SE]: 1.00 [0.266], $P < 0.001$; Fig. 1). Similarly, infants with a normotensive pregnancy exposure who were born SGA ($n = 22$) had significantly greater gain in weight z-score from birth to 6 months than normotensive pregnancy exposed infants who were not born SGA ($n = 276$) (1.19 [0.24], $P < 0.001$) (Fig. 1). Furthermore, 27 of 41 infants born SGA (66%) experienced rapid weight gain, compared with 83 of 341 infants not born SGA (24%; $P < 0.001$). Conditional weight gain z-score did not differ by SGA status (Fig. 1).

In simple linear regression, a greater increase in weight z-score from birth to 6 months was significantly associated with being born SGA, term birth (≥ 37 weeks), being the mother's first-born child, less/no days stay in neonatal intensive care and/or special care nursery and lower birth weight and length z-scores. Infants were significantly more likely to be classified as having rapid weight gain from birth to 6 months if they were born SGA, had lower birth weight and length z-scores and were born following nonspontaneous onset of labour. A faster rate of weight gain, as measured by conditional weight gain z-score, was associated with less/no days stay in neonatal intensive care and/or special care nursery.

## Discussion

This study is the first to report on the growth of babies exposed to preeclampsia compared to a normotensive pregnancy in early infancy (i.e. first 6 months of life). As expected, infants exposed to preeclampsia were smaller at birth than normotensive exposed infants. However, while preeclampsia exposed infants experienced greater absolute increases in weight and length, the proportion of those experiencing rapid weight gain, change in weight z-score and conditional weight gain z-score (i.e. corrected for age, sex and birth weight z-score) were not significantly different between groups.

Our findings support research describing the effects of preeclampsia associated utero-placental dysfunction and fetal growth restriction on birth weight z-score, length z-score and SGA percentage.[4] In our study, 91% of preeclampsia exposed infants were born following non-spontaneous onset of labour, indicating frequent obstetric intervention to relieve deteriorating maternal or fetal condition. This increased rate of active intervention is characteristic of pregnancies affected by preclampsia[4] and results in both an increased preterm birth rate and increased likelihood of being born SGA.

There have been only a few studies to report on the growth trajectories of children exposed to preeclampsia. A 2020 study by Huang *et al.* found increased BMI trajectory from 18 months to 6 years in preeclampsia exposed compared with normotensive pregnancy exposed children.[27] Similarly, we found a significant increase in BMI from birth to 6 months in preeclampsia exposed infants compared with normotensive pregnancy exposed infants. One other study reporting growth of 23,763 Swedish children with or without preeclampsia exposure reported increased height gain from birth to 5 years in children with preeclampsia exposure.[13] Similarly we found significant absolute length gain from birth to 6 months in our cohort, but given the differences in age of the two cohorts, direct comparisons cannot be made.

This is the first study to examine the impact of preeclampsia on growth during childhood using robust measures of growth. Use of the international INTERGROWTH-21$^{st}$ Preterm Postnatal Growth Standards allowed us to accurately assess and combine

https://doi.org/10.1017/S2040174421000167 Published online by Cambridge University Press

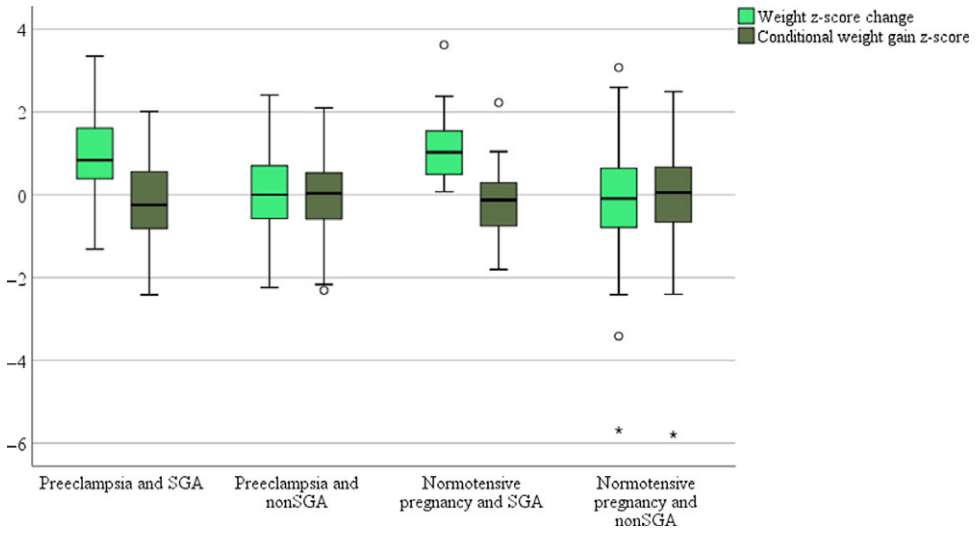


**Fig. 1.** Weight z-score change and conditional weight gain z-score for preeclampsia and normotensive pregnancy exposed infants by small for gestational age status from birth to 6 months.

data of all infants in our cohort and gave us a mechanism with which we could accurately track change in weight z-score over time. Furthermore, conditional weight gain z-score has been suggested to reflect weight gain more accurately as it considers the tendency of children born small or large to experience regression to the mean postpartum.[28] Despite this, few studies report conditional weight gain z-score but report rapid weight gain as a $\geq 0.67$ change in weight z-score between measurements.[28] This is subject to bias due to variation in time between measures (i.e. 6 months versus 12 months), age of cohort and starting weight z-score. However, due to its common use our decision was to report both measures in this paper. Indeed, it is likely that, without the use of correct standards, and reporting of conditional weight gain z-score, we may have observed differences in weight gain from birth to 6 months between groups.

In line with growth not differing between exposed and non-exposed infants from birth to 6 months, preeclampsia exposed infants remained smaller than normotensive exposed infants at 6 months in our study. However, if the trend of greater absolute weight gain continues, anthropometric outcomes of preeclampsia exposed children will surpass those exposed to a normotensive pregnancy. This has been described in the study by Byberg et al who reported that weight z-scores were lower in preeclampsia exposed infants until preschool age, from which time weight z-scores were higher in preeclampsia exposed children up to 13 years of age.[29] This is supported by systematic reviews in children and adolescents that report increased BMI and/or rates of obesity in preeclampsia exposed children/adolescents compared with their peers.[6,30] Further research is required to determine the pattern and timing of increased growth trajectories in children exposed to preeclampsia.

Our analysis suggests that infants born SGA are more likely to experience rapid weight gain than those not born SGA. Rapid growth during early infancy in babies born SGA has potentially lifelong cardiometabolic health implications and, given the high proportion of preeclampsia exposed babies born SGA (23% in our cohort), provides a plausible mechanism for the observed effect of preeclampsia exposure on cardiometabolic health through adolescence and adult years. The Barker Hypothesis stipulates that an intrauterine nutritionally depleted environment, such as in preeclampsia and in infants born SGA, causes epigenetic changes in the infant that predisposes the infant to early rapid weight gain, increased BMI from childhood and compromised cardiometabolic health in the future.[12] As preeclampsia is associated with increased risk of obesity in the offspring, it is possible that increasing rates of preeclampsia may be one of many factors driving the increasing rates of obesity in youth worldwide. Furthermore, obesity itself is a risk factor for preeclampsia, presenting a vicious cycle with transgenerational cardiometabolic health implications.

Although we did not observe a difference in growth from birth to 6 months between preeclampsia exposed and non-exposed infants, our findings nevertheless highlight an opportunity for lifestyle intervention and education for mothers following a pregnancy affected by preeclampsia, especially those who have a baby born SGA. In addition to educating around the importance of good maternal nutrition, this education should also focus on infant nutrition. This should include encouraging exclusive breastfeeding for the first 6 months of life – especially important considering more than one-third of preeclampsia exposed infants were no longer receiving breastmilk at 6 months – and informing appropriate preparation of formula for when breastfeeding is not possible. Education should also include recommendations for later feeding and lifestyle practices that support appropriate infant weight gain, including the introduction of nutrient-rich solids, fussy eating strategies, working towards a diet that includes all five food groups, tips around keeping their child physically active and limiting sedentary behaviour and parents as role models of healthy lifestyle behaviours. In this way, a lifestyle intervention targeting mothers and their infants following a pregnancy affected by preeclampsia may help to mitigate the effects of preeclampsia exposure on the child.

Strengths of this study include the large sample size and the use of appropriate standards to calculate weight and length z-scores, corrected for gestational age, that could be tracked over time. Furthermore, reporting conditional weight gain z-score strengthens the study findings by correcting for regression to the mean, which was highly relevant to this cohort with a high proportion of those born preterm and SGA. Limitations include bias

https://doi.org/10.1017/S2040174421000167 Published online by Cambridge University Press

associated with recruitment of healthy volunteers, which is common in cohort research, and, albeit ethnically diverse, the P4 study is a single-centre trial. Additionally, preeclampsia represents a highly heterogeneous condition with variable maternal and fetal effects which we have not accounted for in our analysis, therefore findings should be interpreted with caution. Furthermore, this paper presents secondary data analysis of the P4 study which was not powered to detect differences in paediatric outcomes between the exposed and the unexposed infants.

## Conclusion

In conclusion, this study is the first to report on the growth of infants from birth to 6 months following exposure to intrauterine preeclampsia, finding no difference in growth outcomes compared to infants exposed to a normotensive pregnancy. However, preeclampsia exposure is associated with greater cardiometabolic risk long term for the child, including increased BMI, suggesting that further research examining the pattern and timing of growth trajectories throughout childhood in children exposed to preeclampsia is warranted. Furthermore, identification of a pregnancy affected by preeclampsia may present an opportunity to provide a postpartum intervention for both mothers and babies designed to optimise healthy lifestyle behaviours throughout the life course and possibly redirect their future cardiometabolic health trajectory.

**Acknowledgements.** The authors wish to thank the mothers/carers and infants who participated in this study, Prof Caroline Homer and the following students for their assistance with the P4 study: Gemma Bylos, Rose Kennedy, Annie Lu, Sarah McLennan, Melissa Ojurovic, Sai Sankare Siritharan, Sathia Sushil, Jaime Worboys, Amanda Yao and Lily Xu.

**Financial Support.** This study was supported by The St George and Sutherland Medical Research Foundation and by a philanthropic donation from Emeritus Professor Richard Henry. MLG and AH are supported by NHMRC Early Career Fellowships (APP1158876 and APP1141570, respectively), and MEC is supported by a NHMRC practitioner fellowship (APP1136735). The funders did not participate in the work.

**Conflicts of Interest.** None.

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