

Socioeconomic Disadvantage in Infancy and Academic and Self-Regulation Outcomes

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abstract

OBJECTIVES: A comprehensive understanding of how timing of exposure to disadvantage affects long-term developmental risk is needed for greater precision in child health policy. We investigated whether socioeconomic disadvantage in infancy (age 0–1 years) directly affects academic and self-regulation problems in late childhood (age 10–12 years), independent of disadvantage at school entry (age 4–6 years).

METHODS: Analyses were replicated in 2 population-based cohorts: the Australian Temperament Project (ATP; $N = 2443$) and the Longitudinal Study of Australian Children (LSAC; $N = 5107$). Generalized linear models were used to estimate the crude and adjusted effects. Marginal structural models were used to estimate the controlled direct effect of socioeconomic disadvantage in infancy on academic and self-regulation outcomes in late childhood, independent of disadvantage at school entry.

RESULTS: In both cohorts, socioeconomic disadvantage in infancy and at school entry was associated with poorer academic and self-regulation outcomes. Socioeconomic disadvantage in infancy had a direct effect on academic outcomes not mediated by disadvantage at school entry (ATP: risk ratio [RR] = 1.42; 95% confidence interval [CI]: 1.09–1.86; LSAC: RR = 1.87; 95% CI: 1.52–2.31). Little evidence was found for a direct effect of disadvantage in infancy on self-regulation (ATP: RR = 1.22; 95% CI: 0.89–1.65; LSAC: RR = 1.19; 95% CI: 0.95–1.49).

CONCLUSIONS: Socioeconomic disadvantage in infancy had a direct effect on academic but not self-regulation outcomes in late childhood. More precise public policy responses are needed that consider both the timing of children's exposure to disadvantage and the specific developmental domain impacted.



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WHAT'S KNOWN ON THIS SUBJECT: Exposure to socioeconomic disadvantage impacts children's development across multiple domains, including their academic learning and self-regulation skills.

WHAT THIS STUDY ADDS: We suggest that exposure to socioeconomic disadvantage in infancy has a direct effect on academic skills in late childhood, whereas the effect on self-regulation outcomes is mediated by disadvantage experienced more proximally in time.

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Exposure to socioeconomic disadvantage impacts children's development, which tracks forward to unequal rates of mortality and physical, social, and cognitive impairment in adulthood.¹ These inequities exist in relative degrees in all countries and perpetuate cycles of disadvantage across generations.¹ The World Health Organization's Commission on Social Determinants of Health has called for the elimination of inequitable health outcomes within a generation.¹ The success of policy efforts depends on having a robust evidence base that can be used to more precisely guide intervention strategies to achieve maximum impact.

In building this evidence, the commission has called for a nuanced approach to consider how disadvantage might affect different aspects of child development.¹ In the cognitive domain, academic skills such as literacy and numeracy reflect a major opportunity for changing the life chances of disadvantaged children, with strong links between educational pathways and future employment and earnings² as well as health status.³ Increasingly, the importance of "noncognitive skills" such as self-regulation (or ability to control behaviors), emotion, and attention are also recognized as critical in shaping health and economic pathways.⁴

Although the inverse association between exposure to disadvantage and these aspects of children's development is now well documented,⁵ a gap remains in our understanding of the implications of the timing of this exposure.⁶ Disadvantage is not a single event, and parents' economic status can fluctuate across the early child-rearing years. For example, household income typically drops around the time of childbirth before increasing with mothers' return to work. The career progression of mothers can slow as they balance caregiving and

paid work, and many move into roles that, although "mother friendly," are also lower status.⁷

The timing of children's exposure to disadvantage is a potentially important determinant of the severity and duration of its impact on development. The sensitive periods hypothesis suggests the importance of adversity experienced during the infant years.⁸ Early exposure to stressors associated with deprivation, including reduced stimulation and sensitivity in infant-caregiver interactions, may cause changes in physiologic function and brain structure.⁹ These changes are expected to track forward given that the capacity for increasing complexity in skills is built on those laid down earlier in life.⁴

An unresolved question, however, is the extent to which the effects of these early experiences are mediated by children's subsequent exposure to disadvantage. The recency hypothesis alternatively suggests that the effects of adversity can be time limited, and so developmental outcomes may be more strongly influenced by proximal rather than distal events.⁸ The recency hypothesis has received some empirical support, particularly in relation to long-term social and emotional outcomes.⁸

We investigate whether socioeconomic disadvantage in infancy has a direct effect on academic and self-regulation skills at 10 to 12 years of age, independent of children's subsequent exposure to disadvantage. In seeking to understand causal pathways in child health, innovations in methodologic design can be used to help further develop the evidence base.¹⁰ We use marginal structural models (MSMs) to estimate the controlled direct effect (CDE) of infant disadvantage on developmental outcomes.¹¹ The CDE is the effect of a given exposure (in this case, socioeconomic disadvantage in infancy) on an outcome (academic

and self-regulation skills) when setting the mediator (socioeconomic disadvantage at school entry) to a specific value for all individuals in the population.¹¹

METHODS

Data Sources

Analyses were replicated in independent data from 2 high-quality longitudinal Australian population cohort studies: the Australian Temperament Project (ATP) and Longitudinal Study of Australian Children (LSAC). Use of the ATP and LSAC cohorts is ideal for the purpose of replication because of the similarity in the domains measured and the ages at which assessments were taken, as well as coverage of 2 different historical epochs. Children in the LSAC were born 21 years after the ATP was established, during which time there have been substantial shifts in population demographics and government policies. The data collection procedures for both of these studies was approved by the Australian Institute of Family Studies Human Research Ethics Review Board.

ATP

The ATP is one of Australia's longest-running studies of social and emotional development. The sample was recruited from Maternal and Child Health Centers in 1983 and comprised a representative sample of 2443 infants (mean age 6.3 months; SD 3.45) and their parents from urban and rural areas in the state of Victoria, Australia. Details of methods and procedures are provided elsewhere.¹² Briefly, families have been surveyed by mail every 1 to 2 years until 19 years of age, and every 4 years thereafter (15 survey waves).

LSAC

The LSAC is a nationally representative sample of 2 cohorts of

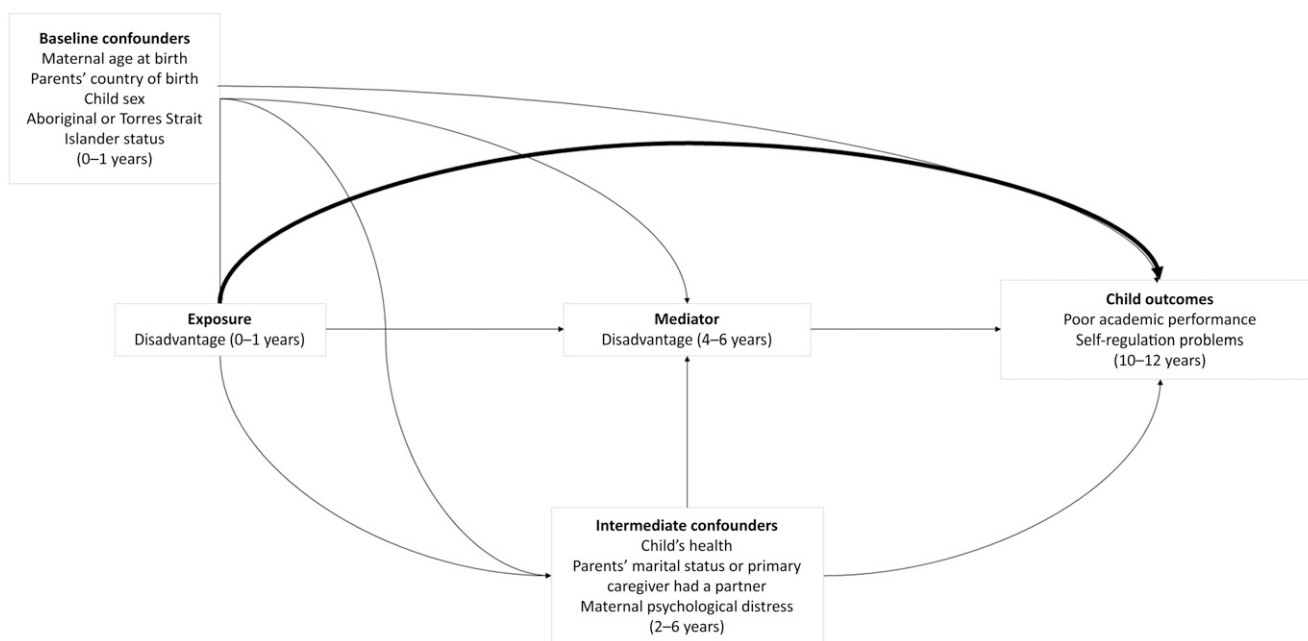


FIGURE 1

Directed acyclic graph depicting the relationship between early disadvantage and academic and self-regulation outcomes in late childhood.

Australian children: a birth cohort of infants ($N = 5107$) and a kindergarten cohort of 4-year-olds ($N = 4983$). The study commenced in May 2004, with 7 waves of data collected to date. We draw on data from the birth cohort, with a mean age of 8.8 months at recruitment (SD 2.57). The LSAC sample selection is documented elsewhere.¹³ A complex survey design was used to select a sample that is broadly representative of all Australian children with the exception of the small proportion of children living in highly remote geographic areas.¹³

Measures

Directed acyclic graphs are graphical tools that are used to visually represent causal processes that lead to the development of child health outcomes.¹⁴ A directed acyclic graph was drawn to visually represent the current research question and inform the selection of variables, including potential confounders, on the basis of current knowledge from the extant literature (Fig 1). Measures were selected to be as similar as possible across the 2 cohorts, and key

variables were dichotomized for interpretability.

Outcomes: Poor Academic Performance and Self-Regulation Problems at 10 to 12 Years of Age

To assess academic skills, in the ATP, 6 items from the teacher-rated Academic Competence subscale in the Social Skills Rating System¹⁵ were used (eg, “compared with other children in classroom, this child’s intellectual functioning is...”), with responses from 1 “lowest” (0%–10%) to 5 “highest” (91%–100%). In the LSAC, teachers reported on the literacy and numeracy subscales of the Academic Rating Scale.¹⁶ Teachers rated the child’s skills in literacy (9 items [eg, “reads fluently”]) and numeracy (10 items [eg, “subtracts numbers that require regrouping”]) as 1 “not yet proficient” to 5 “proficient,” and these items were averaged to create a continuous score (mean [SD] = 3.71 [0.91] in the ATP; 3.59 [0.81] in the LSAC). Children scoring in the bottom 20% were categorized as having poor academic performance to capture children demonstrating

lower-than-average ability in the competencies measured in that domain. This cut point was used to align with Australian data estimating that ~20% of children require additional support in the classroom setting because of developmental difficulties.¹⁷

To assess self-regulation skills in the ATP, the persistence and negative reactivity subscales of the School-Age Temperament Inventory were used.¹⁸ Persistence included 11 items (eg, “switches from one activity to another before finishing the first”); negative reactivity included 9 items (eg, “reacts strongly to disappointment”) rated by parents from 1 “Never” to 5 “Always,” and scores for both scales were averaged (mean [SD] = 2.74 [0.64] in the ATP; 2.56 [0.64] in the LSAC). As for the academic domain, the top 20% were defined as having poor self-regulation to capture those with relatively lower scores in that domain. The same approach was used in the LSAC, employing the parent-reported short form of the School-Age Temperament Inventory.

Exposure: Socioeconomic Disadvantage in Infancy (0–1 Years of Age)

In the ATP and LSAC, socioeconomic disadvantage in infancy was measured as a composite of each parent's self-reported educational attainment and occupation. These factors were consistently measured across cohorts and provide key dimensions of socioeconomic position.¹⁹ Occupation level was categorized according to the criteria developed by Broom et al²⁰ (ATP) and the Australian Bureau of Statistics²¹ (LSAC). The standardization approach outlined by Blakemore et al¹⁹ was used to create a continuous score, with the exception that income, which was not available in the ATP, was not included. Values for each parent's education and occupation variables were standardized to have a mean of 0 and an SD of 1 (ie, converted to a z score). A mean score was created by averaging the standardized scores, which was then restandardized to have a mean of 0 and an SD of 1. The bottom 25% were categorized as "disadvantaged" and the top 75% as "not disadvantaged." This cut point was selected to capture relative socioeconomic disadvantage (a relatively lower position in the social hierarchy), which, along with absolute levels of resources, contributes to health inequities.²² A threshold of 25% was selected for consistency with other concurrent investigations of child inequities.²³

Mediator: Socioeconomic Disadvantage at School Entry (4–5 Years of Age)

Disadvantage at the time of school entry (4–5 years of age) was measured in the same way as described for infant disadvantage in both the ATP and LSAC.

Potential Confounders

The baseline confounders in the causal relationship between early disadvantage and developmental outcomes included the mother's age

at birth (continuous), parents' country of birth (English-speaking countries or non-English-speaking countries), child's sex (boy or girl), and indigenous status (Aboriginal or Torres Strait Islander background or not) (Fig 1). Each of these was measured in infancy in both the ATP and LSAC, with the exception of indigenous status, which was only available in the LSAC.

Intermediate confounders refer to other factors that could influence both the mediator and outcome and that may themselves be affected by the initial exposure. Potential intermediate confounders were assessed at 2 to 6 years of age and included the child's health (ATP: the child has been seriously ill [yes or no]; LSAC: the child has a medical condition or illness that lasts for 6 months or more [yes or no]), mother's psychological distress (continuous; only available in the LSAC data), and 2-parent household (yes or no).

Statistical Analyses

First, descriptive analyses were used to characterize the cohorts. The distribution of academic and self-regulation problems was then examined according to socioeconomic disadvantage in infancy and at school entry.

To estimate the effect of disadvantage in infancy on developmental outcomes, we first used generalized linear models with log-link function to estimate the unadjusted associations, and the associations adjusted for baseline confounders. We then used MSMs to estimate the CDE of early disadvantage on developmental outcomes, independent of disadvantage at school entry.¹¹ Further details of how the MSM was applied to estimate the CDE is provided in the Supplemental Information. Three models were run: (1) using data from the ATP, (2) using data from the LSAC to directly replicate this, and (3) using data from

the LSAC incorporating additional confounders that were available only in that data set, to understand how sensitive the findings were to the inclusion of these additional potential confounders.

In the LSAC, there are weighting variables that can be used to adjust for nonresponse and sample attrition to estimate a more representative population. These weights are not available for the ATP. Multiple imputation was therefore used to provide a consistent approach to accounting for nonresponse and attrition over waves and thereby reduce selection bias due to missing data. Multiple imputation by chained equations was used to impute 20 data sets under the missing-at-random assumption.²⁴ The imputation model included all variables in the analysis model and auxiliary variables (stressful life events, persistence, and negative reactivity at age 6–7 years) that were not part of the analyses but used to help predict the missing values. Results were combined using Rubin's rules. Results from imputed data are reported; see Supplemental Table 5 for characteristics of the observed data. All analyses were performed with Stata SE version 14.0 (Stata Corp, College Station, TX).

RESULTS

Comparison of Cohort Characteristics

The proportion of parents with postgraduate qualifications was substantially higher in the LSAC cohort, reflecting an increase in educational levels in the Australian population during the 20-year interval between recruitments (Table 1). The proportion of mothers with missing or unknown occupation was higher in the LSAC, potentially because of mothers being on maternity leave at the time of assessment. A larger proportion of parents were born in non-English-

TABLE 1 Characteristics of the ATP and LSAC Cohorts

Variable	ATP (N = 2443)	LSAC (N = 5107)
Child development outcomes, %		
Academic performance (10–12 y)		
High performance	70.8	79.9
Low performance	29.2	20.1
Self-regulation problems (10–12 y)		
Low problems	80.0	80.4
High problems	20.0	19.6
Socioeconomic position, %		
0–1 y		
High	76.6	75.9
Low	23.4	24.1
4–6 y		
High	73.2	73.2
Low	26.8	26.8
Socioeconomic indicators, %		
Mother education (0–1 y)		
Postgraduate	1.6	13.3
Bachelor's degree	9.9	20.0
Technical diploma and/or trade apprenticeship	16.3	35.6
11 or 12 y	38.0	19.8
≤10 y	34.1	11.8
Mother occupation (0–1 y)		
Professional and/or managerial	26.2	25.1
Nonprofessional	71.6	24.8
Housewife or not working	2.1	50.0
Father education (0–1 y)		
Postgraduate	3.4	12.5
Bachelor's degree	13.9	16.3
Technical diploma and/or trade apprenticeship	30.3	46.7
11 or 12 y	25.8	14.3
≤10 y	26.6	10.1
Father occupation (0–1 y)		
Professional and/or managerial	39.6	42.1
Nonprofessional	59.6	42.6
Pensioner, retired, and/or not working	0.8	15.3
Mother education (4–6 y)		
Postgraduate	3.7	15.1
Bachelor's degree	8.1	20.4
Technical diploma and/or trade apprenticeship	22.1	39.7
11 or 12 y	34.6	16.2
≤10 y	31.4	8.5
Mother occupation (4–6 y)		
Professional and/or managerial	32.9	33.7
Nonprofessional	57.1	31.7
Housewife, pensioner, student, or not working	10.0	34.6
Father education (4–6 y)		
Postgraduate	7.3	15.1
Bachelor's degree	13.4	16.2
Technical diploma and/or trade apprenticeship	37.6	49.0
11 or 12 y	20.7	11.9
≤10 y	21.0	7.9
Father occupation (4–6 y)		
Professional and/or managerial	46.1	45.7
Nonprofessional	51.2	38.8
Househusband, pensioner, student, or not working	2.8	15.5
Baseline confounders (0–1 y)		
Both parents born in non-English-speaking countries, %		
Yes	3.0	12.7
No	97.0	87.3
Mother age, mean (SD)	27.9 (0.1)	31.0 (0.1)

speaking countries in the LSAC cohort (12%) than in the ATP cohort (3%).

Distribution of Outcomes According to Socioeconomic Disadvantage

A higher proportion of socioeconomically disadvantaged children had academic and self-regulation problems (Table 2). For example, in the ATP and LSAC, respectively, 25% and 15.3% of children who were not disadvantaged in infancy had poor academic outcomes compared with 43% and 35% of children who were disadvantaged in infancy.

Associations Between Socioeconomic Disadvantage and Developmental Outcomes

Academic Performance

The unadjusted model (model 1; Table 3) shows that socioeconomic disadvantage in infancy and at school entry were associated with poor academic performance at 10 to 12 years of age in both cohorts. Effects remained after adjusting for baseline confounders (model 2). Model 3 shows the CDE of infant disadvantage on academic outcomes. In both cohorts, those children who were exposed to disadvantage in infancy had a higher risk of poor academic performance at 10 to 12 years of age than those who were not, independent of exposure to disadvantage at school entry (ATP: risk ratio [RR] = 1.43; 95% confidence interval [CI]: 1.09–1.86; LSAC: RR = 1.87; 95% CI: 1.52–2.31).

Self-Regulation Problems

In the unadjusted model, exposure to disadvantage in infancy and at school entry were associated with poor self-regulation at age 10 to 12 years in both cohorts (model 1; Table 4). This effect was attenuated after adjusting for baseline confounders (model 2). Model 3 shows the CDE of infant disadvantage on self-regulation outcomes. Little evidence was found of a direct effect of infant disadvantage on self-regulation

TABLE 1 Continued

Variable	ATP (N = 2443)	LSAC (N = 5107)
Child sex, %		
Girl	48.0	48.9
Boy	52.0	51.1
Aboriginal or Torres Strait Islander status, %		
Yes	N/A	3.2
No	N/A	96.8
Intermediate confounders (2–6 y), %		
Child is sick		
Yes	9.2	8.0
No	90.8	92.0
Parents are married or partnered		
Yes	92.8	89.1
No	7.2	10.9
Mother psychological distress, mean (SD)	N/A	4.5 (0.01)

N/A, not measured.

problems in late childhood in either the ATP or LSAC cohorts (RR = 1.22; 95% CI: 0.90–1.65; and RR = 1.21; 95% CI: 0.97–1.49, respectively).

DISCUSSION

In this study, we found strong evidence of a direct effect of exposure to disadvantage in infancy on academic skills at 10 to 12 years of age. That is, exposure to disadvantage in infancy increased the risk of poor academic performance at age 10 to 12 years, independent of disadvantage at the start of schooling. For self-regulation problems, a different picture emerged; the effect of exposure to disadvantage in infancy on self-regulation appeared to be mediated by disadvantage

experienced more proximally in time. Results were consistent across the ATP and LSAC cohorts despite a 20-year temporal separation in recruitment periods.

This evidence of a direct effect of exposure to disadvantage in infancy on later academic problems aligns with the sensitive periods hypothesis and the current understanding of brain development that emphasizes the importance of early foundations for learning.⁹ It is also consistent with the findings of previous researchers examining other potential mediators of the relationship between early disadvantage and developmental outcomes.²⁵ It is notable that this finding was observed in data from 2 different historical periods using 2

different measures of academic performance and in analyses including additional potential confounders. Early exposure to disadvantage could impact later academic problems through many points along the developmental pathway that are highly amenable to intervention and amelioration, such as parents' capacity to provide sensitive and stimulating caregiving,²⁶ access to resources that promote learning in the home,²⁷ and access to high-quality early education and care.²⁸

A different pattern was observed in relation to self-regulation. Aligning with existing evidence,²⁹ exposure to disadvantage in infancy was associated with poor self-regulation outcomes. However, we found little evidence of a direct effect of infant disadvantage on self-regulation problems in late childhood. Rather, this effect appeared to occur via exposure to disadvantage at school entry. This result is more consistent with the recency hypothesis, whereby the capacity for self-regulation may be more strongly influenced by proximal, rather than temporally distal, circumstances.⁸ It has been argued that although childhood is a sensitive period, aspects of self-regulation are surprisingly plastic and responsive to environmental circumstances.³⁰

TABLE 2 Distribution of Academic Performance and Self-Regulation Problems According to Socioeconomic Position

	ATP				LSAC			
	Academic Performance, % (95% CI)		Self-Regulation Problems, % (95% CI)		Academic Performance, % (95% CI)		Self-Regulation Problems, % (95% CI)	
	High Performance	Low Performance	Low Problems	High Problems	High Performance	Low Performance	Low Problems	High Problems
Socioeconomic position at 0–1 y								
High	75.0 (72.6–77.4)	25.0 (22.6–27.4)	82.1 (79.8–84.5)	17.8 (15.5–20.2)	84.7 (83.3–86.1)	15.3 (13.9–16.7)	81.5 (80.2–82.9)	18.5 (17.1–19.8)
Low	57.0 (51.3–62.8)	43.0 (37.2–48.7)	72.9 (67.9–77.9)	27.1 (22.1–32.1)	65.0 (61.5–68.5)	35.0 (31.5–38.5)	76.6 (73.6–79.7)	23.4 (20.3–26.4)
Socioeconomic position at 4–6 y								
High	75.0 (72.8–77.3)	25.0 (22.7–27.2)	82.8 (80.3–85.3)	17.2 (14.7–19.7)	84.0 (82.6–85.4)	16.0 (14.6–17.4)	81.4 (80.0–82.8)	18.6 (17.2–20.0)
Low	59.2 (53.7–64.8)	40.8 (35.2–46.3)	72.3 (67.1–77.6)	27.7 (22.4–32.9)	68.9 (65.6–72.2)	31.1 (27.8–34.4)	77.5 (74.7–80.3)	22.5 (19.7–25.3)

TABLE 3 Effect Estimates for the Association Between Disadvantage and Poor Academic Outcomes at 10–12 Years

	Model 1: Regression Model, Unadjusted	Model 2: Regression Model, Adjusted for Baseline Confounders	Model 3: MSM, CDE
	RR (95% CI)	RR (95% CI)	RR (95% CI)
ATP (<i>N</i> = 2443)			
Disadvantage 0–1 y	1.72 (1.44–2.04)	1.73 (1.44–2.07)	1.43 (1.09–1.86)
Disadvantage 5–6 y	1.63 (1.37–1.94)	1.63 (1.37–1.95)	—
LSAC replication of ATP (<i>N</i> = 5107)			
Disadvantage 0–1 y	2.28 (2.01–2.59)	2.19 (1.91–2.50)	1.87 (1.52–2.31)
Disadvantage 4–5 y	1.94 (1.71–2.21)	1.85 (1.62–2.10)	—
LSAC adjusted for additional confounders ^a (<i>N</i> = 5107)			
Disadvantage 0–1 y	—	2.10 (1.83–2.40)	1.87 (1.52–2.31)
Disadvantage 4–5 y	—	1.77 (1.56–2.02)	—

Baseline confounders, mother age and ethnicity, and child's sex. —, not applicable.

^a Additionally adjusted for Aboriginal or Torres Strait Islander status at 0–1 y and mother psychological distress at 2–3 y.

These findings are relevant to the consideration of when efforts to reduce inequities in children's development can be maximally effective. In relation to academic skills, many current approaches that are focused on remediating the effects of disadvantage appear limited by the relatively late developmental window at which they are targeted. Our most intensive such intervention is schooling itself, which typically begins at ~5 years of age. Although schools represent a potent public health opportunity,³ even the best efforts in a school setting are unlikely to fully ameliorate the legacy of early

exposure to disadvantage for academic learning. Effective interventions that can be initiated much earlier in the infancy period include, for example, unconditional cash transfers,³¹ interventions on parenting through services such as nurse home visiting,³² and interventions used to promote rich home learning environments.³³ Gains achieved from such early efforts can then be built on and reinforced over time, including once children enter the school environment. In contrast, the current results suggest that the preschool and early elementary years remain an important period for

interventions that are focused on minimizing the consequences of disadvantage for children's self-regulation abilities. For example, high-quality early childhood education and care in the preschool years³⁴ and social and emotional learning programs³⁵ in the early years of school may be well timed to reduce inequities in this domain.

Replication in 2 data sets collected 2 decades apart helps to reduce the potential for false-positive findings.³⁶ We employed the analytic method MSM, which can better account for potential confounding than conventional regression approaches.³⁷ Nevertheless, some limitations should be borne in mind. Within the confines of the data available and what was comparable across both cohorts, we defined disadvantage narrowly using parents' education and occupation. Self-regulation was not a core domain assessed in the ATP or LSAC, with temperament measures used as proxies to indicate this construct. It is possible that results might have differed if self-regulation was measured in a different way (eg, through structured observations in the school or family setting) or captured other dimensions like cognitive flexibility. Parent and teacher reports of child development outcomes are not as robust as individualized psychometric testing, albeit a pragmatic requirement in large cohort studies. For interpretability, measures were dichotomized to capture children from relatively lower socioeconomic status backgrounds and with relatively poorer developmental outcomes, as opposed to absolute differences according to resources or skills. It would be of interest to explore other cut points in future studies as well as across the full distribution of the exposure and outcomes. The possibility of residual confounding can never be fully eliminated. Although we did not have

TABLE 4 Effect Estimates for the Association Between Disadvantage and Poor Self-Regulation at 10–12 Years

	Model 1: Regression Model, Unadjusted	Model 2: Regression Model, Adjusted for Baseline Confounders	Model 3: MSM, CDE
	RR (95% CI)	RR (95% CI)	RR (95% CI)
ATP (<i>N</i> = 2443)			
Disadvantage at 0–1 y	1.52 (1.22–1.88)	1.39 (1.12–1.74)	1.22 (0.90–1.65)
Disadvantage at 5–6 y	1.61 (1.27–2.03)	1.51 (1.20–1.91)	—
LSAC replication of ATP (<i>N</i> = 5107)			
Disadvantage at 0–1 y	1.26 (1.09–1.46)	1.19 (1.02–1.38)	1.21 (0.97–1.50)
Disadvantage at 4–5 y	1.21 (1.05–1.39)	1.15 (1.01–1.33)	—
LSAC adjusted for additional confounders ^a (<i>N</i> = 5107)			
Disadvantage at 0–1 y	—	1.17 (1.00–1.37)	1.19 (0.95–1.49)
Disadvantage at 4–5 y	—	1.14 (1.00–1.31)	—

Baseline confounders, mother age and ethnicity, and child's sex. —, not applicable.

^a Additionally adjusted for Aboriginal or Torres Strait Islander status at 0–1 y and mother psychological distress at 2–3 y.

any specific confounders that were identified but could not be accounted for, and the baseline confounders that were examined did not appear to account for much of the relationship between socioeconomic disadvantage and children's developmental outcomes, we cannot rule out the possibility that such factors exist.

Findings from this study raise a number of important questions that warrant further exploration. These include the effect of exposure to disadvantage in infancy on other developmental domains, such as the extent to which disadvantage lays the foundations for adult chronic disease. Exposure to disadvantage during other key developmental periods (eg, adolescence) could similarly have a direct effect on specific developmental outcomes, and this is also critical to explore. In addition, we examined 1 aspect of disadvantage (ie, low socioeconomic position) and it would be of interest to explore additional indicators of socioeconomic position (eg, household income) and whether results are similar when considering disadvantage from a social determinants perspective (eg, according to geographic characteristics like rural location and risk factors like parental smoking⁵). Also warranting further exploration is whether similar effects might be observed in other countries despite differing policy levers and incentives and a range of welfare benefit structures. These questions have important implications for policies

and interventions: they can be used to help identify particularly important time points to intensify interventions (or combinations of periods), which could create a cumulative benefit for later outcomes and offer opportunities for targeted and rational policy spending.

CONCLUSIONS

Results replicated in 2 longitudinal cohorts support a direct effect of socioeconomic disadvantage in infancy on academic problems in late childhood. For self-regulation, the effect of exposure to disadvantage in infancy appeared to be mediated by disadvantage experienced more proximally in time. These findings suggest the need for more precise public policy responses that consider both the specific developmental domain and the timing of disadvantage exposure. This requires a longitudinal perspective on children's exposure to disadvantage, including the potentially lasting effects of early exposure to disadvantage and the resources needed to redress associated developmental sequelae of those early experiences.

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ABBREVIATIONS

ATP: Australian Temperament Project
CDE: controlled direct effect
CI: confidence interval
LSAC: Longitudinal Study of Australian Children
MSM: marginal structural model
RR: risk ratio

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