BACKGROUND AND OBJECTIVES: Early pubertal timing is linked with a range of adverse health outcomes later. Given recent trends of earlier pubertal maturation, there is growing interest in the factors influencing pubertal timing. Socioeconomic disadvantage has been previously linked with reproductive strategies later in life. In this study, we aim to determine the association between cumulative social disadvantages in early life and early puberty in a population-based birth cohort.

METHODS: Data are from the B (baby) cohort of The Longitudinal Study of Australian Children. Children ($n = 5107$) were aged 0 to 1 years when recruited in 2004 and 10 to 11 years ($n = 3764$) at Wave 6 in 2015. Household socioeconomic position (SEP) and neighborhood socioeconomic disadvantage were collected at all 6 waves. Trajectories of disadvantage were identified through latent class models. Early puberty at Wave 6 was assessed from parental reports using an adaptation of the Pubertal Development Scale.

RESULTS: Cumulative exposure to extremely unfavorable household SEP in boys independently predicted a fourfold increase (odds ratio = 4.22, 95% confidence interval 2.27–7.86) in the rate of early puberty. In girls, the increase was twofold (odds ratio = 1.96, 95% confidence interval 1.08–3.56). We found no effect from neighborhood disadvantage once family SEP was taken into account.

CONCLUSIONS: Cumulative exposure to household socioeconomic disadvantage in early life predicts earlier pubertal timing in both boys and girls. This may represent 1 mechanism underpinning associations between early life disadvantage and poor health in later life.
Early maturation has demonstrable links to a broad range of emotional, behavioral, and social problems during adolescence, including depressive disorders, substance disorders, eating disorders, and precocious sexuality.\textsuperscript{1–3} Early pubertal timing also carries risks for the development of reproductive tract cancer and cardiometabolic diseases in later life.\textsuperscript{4,5} Given the recent trend toward earlier pubertal maturation in many countries, a clearer understanding of the factors influencing pubertal timing is important.\textsuperscript{5,6}

Life history theory is a branch of evolutionary biology that deals with the strategies that organisms use to allocate their limited time and energy to shaping the various stages of their life cycles. According to life history theory, humans have evolved to be sensitive to specific features of early childhood environments, and this exposure to different environments biases children toward different reproductive strategies that may include early pubertal timing.\textsuperscript{7} Results prove generally consistent with these propositions, with factors like childhood neglect, paternal absence, and family conflict associated with earlier pubertal development in girls.\textsuperscript{8,9} However, fewer data exist on the key social determinants of pubertal timing. Krieger et al\textsuperscript{10} have shown that although average age at menarche has declined in US-born women over the last 50 years, these patterns vary by socioeconomic position (SEP). An earlier, inverse socioeconomic gradient (lower SEP, older age at menarche) in the National Health Examination Survey I (1959–1962) has reversed so that in the 2005–2008 National Health and Nutrition Examination Surveys, lower SEP was associated with a younger age at menarche. Recently, Braithwaite et al\textsuperscript{11} investigated the variation in age at menarche by socioeconomic status (SES) (determined by household income and parental education) and race. Black girls in the highest quartile of household income were at an increased risk of early menarche, whereas white girls in the highest quartile were at a significantly lower risk. However, both studies relied on recalled age at menarche, which is not necessarily a sign of initiation but rather a late event in puberty, and their cross-sectional design limited causal inference and deeper exploration of potential confounders.

From a cumulative advantage–disadvantage perspective, small disadvantages at an early stage of a process are likely to grow larger over time.\textsuperscript{12} Household-level SEP is frequently used as an index of childhood disadvantage.\textsuperscript{13} However, other aspects of childhood social context (including features of one’s neighborhood such as availability of and access to services and facilities, neighborhood poverty, low education, and unemployment) may also be important indices.\textsuperscript{14}

We hypothesized that cumulative exposure to socioeconomic disadvantage would predict pubertal timing, with immediate-family SES being more predictive than neighborhood disadvantage. Using national birth cohort data from Growing Up in Australia, several potentially confounding factors related to puberty (eg, parental puberty history, perinatal factors, adiposity, psychosocial stress, physical activity, and sleep duration)\textsuperscript{15} could be taken into account.

**METHODS**

**Study Population**

This study was conducted using B (baby) cohort data from Growing Up in Australia: The Longitudinal Study of Australian Children (LSAC). The sampling frame for the study was the Australian Medicare enrollments database, which is the most comprehensive database of Australia’s population, particularly of young children. Approximately 8921 children (aged 0–1 years) were sampled from this database using a 2-stage, clustered sampling frame to be representative of the proportions of Australian children living in each state and of Australia’s urban–rural mix.\textsuperscript{16} In relation to the key predictors, 61.4% of children at Wave 1 (W1) lived in metropolitan areas, which is very similar to the estimates for this study (69%). At age 10 to 11 years, mean neighborhood SES was 1011 (SD = 59, range: 660–1160), which is slightly higher than the national population mean of 1000 (t test = −7.7; P < .001). The results presented here are broadly representative of Australian children except for those living in very remote areas.

In this paper, data are based on W1 to W6 of the B cohort. Participants (n = 5107, 64% response) were aged 0 to 1 years when recruited in 2004 and 10 to 11 years (n = 3764, 74% retention) at W6 of data collection in 2015.\textsuperscript{17} Data were collected through biennial home visits with direct anthropometric measurements and parental questionnaires. Sample size and response rates for the B cohort are depicted in Fig 1.

**Measures**

**Early Puberty**

We assessed early onset of puberty at W6 (10–11 years old) by adapting items from the Pubertal Development Scale (PDS) for parental reports.\textsuperscript{18} An inference was made that children with parent-reported physical signs of puberty were experiencing an early onset of puberty.\textsuperscript{3} Puberty in girls was indicated by 4 items (growth spurt, pubic hair, skin changes, and breast growth) and in boys by 5 items (growth spurt, pubic hair, skin changes, voice deepening, and facial hair). For each, the parent rated his or her child’s development as “has not started yet (1),” “has...
barely started (2),” “has definitely started (3),” or “seems complete (4).” Items were summed up and averaged to obtain a mean PDS score for each adolescent. Because pubertal development has been shown to vary by age and sex, timing based on pubertal status is usually normed within these subgroups. According to stage-normative pubertal timing, an adolescent was classified as “early” when his or her PDS score was more than 1 SD above the mean pubertal stage by gender. In our analysis, the cutoff PDS score was ≥2.8 for girls and ≥1.8 for boys, respectively. Girls who had their first period (menarche) were also classified as “early” pubertal timing. Previous research found that stage-normative timing predicts substance use, disordered eating, risk-taking, and depression. Stage-normative measures are modestly correlated with physician ratings of Tanner stages and with pubertal hormones. Family and Neighborhood Social Disadvantage At each wave, the LSAC releases composite indicators of family SEP and neighborhood socioeconomic disadvantage. The family SEP variable is an internally standardized summary measure (mean = 0, SD = 1) of parental reports of equalized annual family income, years of education, and current or most recent occupational prestige using the composite variable provided in the LSAC data sets (detailed in Supplemental Table 3). Neighborhood socioeconomic disadvantage is determined by linking the Socio-Economic Indexes for Areas (SEIFA) index of disadvantage with families’ most recent postcode of residence. This census-based index (national mean = 1000, SD = 100) is 1 of 4 indices that have been created by the Australian Bureau of Statistics from social and economic information obtained in the 2011 Census of Population and Housing (detailed in Supplemental Table 3). Information on household SEP and the SEIFA index of disadvantage was available for nearly 90% of respondents in W2, 85% in W3, 80% in W5, and 73% in W6. Potential Confounding Factors Covariates including parental early puberty history, perinatal factors (birth weight, gestational age, delivery mode, and breastfeeding), childhood and current adiposity, physical activity, sleep duration, having a stepfather (for girls only, in line with paternal investment theory), and stressful life events are presented in Supplemental Table 3. Paternal investment theory posits that the developmental pathways presaging adult reproductive behavior are especially sensitive to the father’s family role and parenting behavior. The finding that familial composition (eg, the absence of a biological father or stepfather) is associated with earlier age at menarche suggests that social cues, similarly for those that occur in other animals, can dynamically influence the timing of pubertal maturation. We classified children as underweight, normal weight, overweight, or obese using international definitions for classifying BMI.
Statistical Analysis

Latent Class Analysis

We created latent class analysis (LCA) models with Mplus to classify children into longitudinal SEP and the SEIFA index of disadvantage classes. Only children with at least 2 waves of SEP and SEIFA information were included in the LCA models. A 2-class model was estimated first with maximum-likelihood methods and was followed by models with additional classes. We did not specify a priori any defined minimum percentage of individuals in the smallest LCA class. In selecting the model with the optimal number of trajectories, we used several criteria: (1) the Bayesian information criterion (BIC), seeking the most parsimonious model with the smallest absolute value of the BIC; (2) entropy (values closer to 1 indicate better fit); (3) average latent class posterior probabilities; (4) the number and percentage of children in the smallest class; and (5) interpretability of results. Using these criteria, interpretability, and verifying model fit with BIC, we selected 5 family SEP classes and 4 SEIFA indices of disadvantage classes.

All statistical analyses were performed in Stata/IC 14.1 (StataCorp LP; College Station, TX). Comparisons of proportions of early puberty among different household SEP and SEIFA latent classes were assessed by $\chi^2$ test.

Stepwise, multiple logistic regression models were run separately for boys and girls for the association between early puberty and family- and neighborhood-disadvantage variables. These were tested for trends in logistic regression models.

We used multiple imputation to address the potential bias of loss of precision that could result from complete-case analysis. The missing information for all confounding factors is presented in Supplemental Table 4. Fifty imputed data sets were generated based on a set of variables that were selected because of their strong hypothesized association with early pubertal timing by using the ice command in Stata. This method assumes data are missing at random, which is a reasonable assumption given the ability of many variables to predict the absence of data. Rubin’s rules were used when combining the imputed data sets for analysis.

Distributions of all variables were compared between observed and imputed data (Supplemental Table 5); results indicated no obvious problems with the imputation process.

Given the large number of covariates targeted for exploration in analyses, a series of multiple models was conducted that included different sets of covariates in each model. The baseline model (model 1) controlled self-reported early pubertal development of mothers and fathers in W5. Models 2 to 4 added the following covariates to the baseline model: model 2, perinatal factors (birth weight, gestational age, delivery mode, breastfeeding); model 3, early childhood (W3, 4–5 years old), peripuberty (W5, 8–9 years old), current adiposity (W6, 10–11 years old), ΔBMI-SDS between ages 4 and 5 and 10 and 11 years old, and physical activity and sleep duration in W5 and W6; and model 4, psychosocial stress composed of 2 dimensions—ever having stepfathers for girls and high-stress life events in W5 and W6.

RESULTS

Almost all children were born in Australia (95.8%) and spoke English at home (87.5%), and the majority had parents who were born in Australia. In Wave 6 (10–11 years old), 19.2% of boys (363 of 1886) and 21.1% of girls (374 of 1770) were classified in the early puberty group.

Figure 2 presents the household SEP for each class: extremely favorable, moderately favorable, average, moderately unfavorable, and extremely unfavorable. It also presents the 4 classes of trajectories for neighborhood disadvantage: consistently favorable, average, moderately unfavorable, and deteriorating. For detailed indices of model fit for LCA, see the Supplemental Information. Household SEP correlated moderately with neighborhood SES ($r = 0.38; P < .001$). Among the 209 children from families with extremely unfavorable household SEP, 44 (21.1%) were also in the most disadvantaged neighborhood SES class (deteriorating group).

About 31.5% and 8.3% of children were in the moderately unfavorable and extremely unfavorable trajectories for household SEP, respectively, compared with 12.7% in the extremely favorable SEP group (Table 1).

Proportions of 4 neighborhood disadvantage classes were 19.9%, 37.2%, 35.2%, and 7.6% in the consistently favorable, average, moderately unfavorable, and deteriorating groups, respectively (Table 1).

Figure 2 illustrated that SEP trajectories were virtually flat with little evidence of change over time for any trajectory. However, the lowest SEIFA trajectory had a clear downward shift in neighborhood disadvantage for 7.9% of children, which equates to a fall from a mean SEIFA of 947 at baseline to a mean SEIFA of 884 at Wave 6. No trajectories overlapped, which enabled them to be considered ordinal variables.

Overall, a strong gradient was found between the household SEP and SEIFA with the proportion of early puberty, especially in boys (Fig 3). Early puberty was more common among boys and girls from the most...
unfavorable (lowest) household SEP and was less common in the most favorable family, with significant sex differences ($\chi^2 = 39.97$ for boys, $\chi^2 = 28.95$ for girls; both $P < .001$). Early puberty was highest only in boys from deteriorating neighborhoods and lowest in most favorable neighborhoods ($\chi^2 = 15.14; P = .002$).

Table 2 shows that boys from extremely unfavorable SEP families had more than 4.2 times the risk of developing early (odds ratio [OR] = 4.22; 95% confidence interval [CI]: 2.27–7.86). For girls, extremely unfavorable family SEP also increases the risk of early puberty.

### TABLE 1: Proportion of Different Latent Classes of Family and Neighborhood Disadvantage in Boys and Girls From W1 (0–1 years old) to W6 (10–11 years old), Growing Up in Australia

<table>
<thead>
<tr>
<th>Household SEP, n (%)</th>
<th>SEIFA Index of Disadvantage, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Extremely Favorable</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Total</td>
<td>4785</td>
</tr>
<tr>
<td>Boys</td>
<td>2446</td>
</tr>
<tr>
<td>Girls</td>
<td>2339</td>
</tr>
</tbody>
</table>

In the LCA, from W1 (0–1 year old) to W6 (10–11 years old), children with family and neighborhood disadvantage information in at least 2 waves are included.

![FIGURE 2](image-url) Latent class categories of z score of household SEP (top) and z score of SEIFA index of disadvantage (bottom) from birth to adolescence.
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(OR = 1.96; 95% CI: 1.08–3.56) after adjusting for parental early puberty history, perinatal factors, childhood, peripuberty, current adiposity, change in adiposity between ages 4 and 5 and 10 and 11 years old (ΔBMI-SDS), physical activity, sleep duration, and psychosocial stress.

In multivariable logistic regression models (Table 2), extremely unfavorable family SEP increases the risk of early puberty by nearly 2.8 times (OR = 2.82; 95% CI: 1.84–4.32). Children from moderately unfavorable family SEP also have increased risk of early puberty (OR = 1.58; 95% CI: 1.16–2.16) compared with those with consistently favorable backgrounds. No relation was found between early puberty with family SEP and neighborhood disadvantage among either boys or girls.

For boys, other predictors of early puberty included older age (OR = 1.46; 95% CI: 1.13–1.88), paternal background of early puberty (OR = 1.83; 95% CI: 1.16–2.88), and current adiposity (OR = 1.72; 95% CI: 1.08–2.75). For girls, other predictors included maternal (OR = 2.08; 95% CI: 1.56–2.78) and paternal early puberty (OR = 1.80; 95% CI: 1.13–2.85); childhood obesity (OR = 1.45; 95% CI: 1.01–2.07); peripubertal obesity (OR = 2.57; 95% CI: 1.38–4.80); higher ΔBMI-SDS between ages 4 and 5 years old to 10 and 11 years old (OR = 1.34; 95% CI: 1.16–1.55); and current high-stress life events (OR = 1.88; 95% CI: 1.14–3.09). For girls, high birthweight ≥4000 g (OR = 0.48; 95% CI: 0.29–0.77) was protective against early puberty.

**DISCUSSION**

To our knowledge, this is the first prospective study of the cumulative effects of early social disadvantage from birth on pubertal development. In this national birth cohort study, cumulative exposure to extremely unfavorable household SEP in boys independently predicted a fourfold increase in the rate of early puberty. In girls, the increase was nearly twofold.

Determinants of pubertal timing recently have received greater attention because of an ongoing, secular trend toward earlier pubertal onset.²⁻⁶ Possible confounding influences include growth in the pre- and postnatal environment (eg, weight for gestational age and childhood obesity) and aspects of the early social environment (eg, presence of a stepfather and stressful family life events).³²⁻³⁴ We therefore adjusted for birth weight, gestational age, childhood, peripubertal and current adiposity as well as adiposity changes over time in the regression models. Although current adiposity was strongly correlated with early pubertal onset, the effects of household social disadvantage remained significant. It is possible there could be other confounding factors beyond this study, such as environmental endocrine disruptors.³⁵ However, given the breadth of exposure to such agents,
it is difficult to see that the effects of cumulative household disadvantage on early puberty could be explained by these unmeasured covariates.

It is possible that greater early social disadvantage might partially explain earlier breast development and menarche in African-American girls compared with other ethnic groups.\textsuperscript{11,36} Considerable research demonstrates important associations between early social disadvantage and cascading negative outcomes later.\textsuperscript{37,38} Our findings raise the possibility that pubertal timing may play a mediational role in the links between early social disadvantage and health disparities later in life. A greater understanding of this mediational role may also inform the design of public health interventions to improve health and well-being over the life course.

The mechanisms by which long-term household socioeconomic disadvantage might contribute to early puberty risk are not yet clear.

A recent study found early chronic adversity may prompt earlier, adultlike (negative) neuroendocrine coupling between hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes, which may contribute to early pubertal development.\textsuperscript{39} The effects of early life stress on dual-system (HPA-HPG axes) coupling is one possible mechanism through which early life may affect pubertal development.\textsuperscript{40} In other species, the HPG axis can be affected by the hypothalamic-pituitary-gonadal (HPA-HPG axes) coupling is another possible mechanism. It is possible that greater early social disadvantage might partially explain earlier breast development and menarche in African-American girls compared with other ethnic groups.\textsuperscript{11,36}

### Table 2: Stepwise, Multiple Logistic Regression Estimates Showing ORs for Early Puberty at 10–11 Years in Boys and Girls, Separately and Jointly by Trajectories of Household SEP and the SEIFA Index of Disadvantage

<table>
<thead>
<tr>
<th>Variables</th>
<th>Model 1\textsuperscript{a}</th>
<th>Model 2\textsuperscript{b}</th>
<th>Model 3\textsuperscript{c}</th>
<th>Model 4\textsuperscript{d}</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Boys (n = 1886)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Household SEP (reference = extremely favorable)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Favorable</td>
<td>1.52 (0.97–2.37) \textsuperscript{*}</td>
<td>1.33 (0.98–2.40) \textsuperscript{*}</td>
<td>1.48 (0.93–2.36) \textsuperscript{*}</td>
<td>1.46 (0.91–2.33) \textsuperscript{*}</td>
</tr>
<tr>
<td>Average</td>
<td>1.64 (1.06–2.53) \textsuperscript{**}</td>
<td>1.66 (1.07–2.57) \textsuperscript{**}</td>
<td>1.57 (0.98–2.48) \textsuperscript{**}</td>
<td>1.56 (0.99–2.47) \textsuperscript{**}</td>
</tr>
<tr>
<td>Unfavorable</td>
<td>2.38 (1.49–3.89) \textsuperscript{***}</td>
<td>2.42 (1.58–3.70) \textsuperscript{***}</td>
<td>1.98 (1.26–3.11) \textsuperscript{***}</td>
<td>1.94 (1.23–3.08) \textsuperscript{***}</td>
</tr>
<tr>
<td>Extremely unfavorable</td>
<td>4.49 (2.59–7.78) \textsuperscript{***}</td>
<td>4.69 (2.68–8.20) \textsuperscript{***}</td>
<td>4.40 (2.57–7.17) \textsuperscript{***}</td>
<td>4.22 (2.27–7.86) \textsuperscript{***}</td>
</tr>
<tr>
<td><strong>SEIFA index of disadvantage (reference = consistently favorable)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>1.60 (1.15–2.22) \textsuperscript{**}</td>
<td>1.60 (1.15–2.23) \textsuperscript{**}</td>
<td>1.36 (0.98–1.93) \textsuperscript{**}</td>
<td>1.34 (0.95–1.90) \textsuperscript{**}</td>
</tr>
<tr>
<td>Moderately unfavorable</td>
<td>1.65 (1.17–2.31) \textsuperscript{**}</td>
<td>1.65 (1.17–2.31) \textsuperscript{**}</td>
<td>1.54 (0.94–1.91) \textsuperscript{**}</td>
<td>1.31 (0.92–1.87) \textsuperscript{**}</td>
</tr>
<tr>
<td>Deteriorating</td>
<td>1.52 (0.91–2.56)</td>
<td>1.53 (0.91–2.58)</td>
<td>1.07 (0.58–1.97)</td>
<td>1.05 (0.79–1.44)</td>
</tr>
<tr>
<td><strong>Girls (n = 1770)</strong></td>
<td></td>
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<tr>
<td>Household SEP (reference = extremely favorable)</td>
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</tr>
<tr>
<td>Favorable</td>
<td>1.16 (0.77–1.75)</td>
<td>1.14 (0.76–1.73)</td>
<td>1.14 (0.73–1.78)</td>
<td>1.15 (0.74–1.80)</td>
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<tr>
<td>Average</td>
<td>1.22 (0.82–1.82)</td>
<td>1.20 (0.81–1.80)</td>
<td>1.15 (0.74–1.77)</td>
<td>1.14 (0.74–1.77)</td>
</tr>
<tr>
<td>Unfavorable</td>
<td>1.41 (0.95–2.09)</td>
<td>1.43 (0.98–2.12)</td>
<td>1.29 (0.84–2.00)</td>
<td>1.24 (0.80–1.92)</td>
</tr>
<tr>
<td>Extremely unfavorable</td>
<td>2.38 (1.40–4.04) \textsuperscript{***}</td>
<td>2.42 (1.41–4.15) \textsuperscript{***}</td>
<td>2.00 (1.11–3.82) \textsuperscript{***}</td>
<td>1.96 (1.08–3.58) \textsuperscript{***}</td>
</tr>
<tr>
<td><strong>SEIFA index of disadvantage (reference = consistently favorable)</strong></td>
<td></td>
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</tr>
<tr>
<td>Average</td>
<td>1.12 (0.82–1.54)</td>
<td>1.12 (0.82–1.54)</td>
<td>1.13 (0.80–1.59)</td>
<td>1.13 (0.80–1.60)</td>
</tr>
<tr>
<td>Moderate unfavorable</td>
<td>1.12 (0.81–1.56)</td>
<td>1.11 (0.80–1.55)</td>
<td>1.04 (0.73–1.49)</td>
<td>1.01 (0.70–1.44)</td>
</tr>
<tr>
<td>Deteriorating</td>
<td>1.28 (0.76–2.10)</td>
<td>1.29 (0.76–2.20)</td>
<td>0.98 (0.54–1.78)</td>
<td>0.95 (0.51–1.89)</td>
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<tr>
<td><strong>Total (n = 3658)</strong></td>
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<tr>
<td>Household SEP (reference = extremely favorable)</td>
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<td></td>
</tr>
<tr>
<td>Favorable</td>
<td>1.32 (0.98–1.79)</td>
<td>1.33 (0.98–1.79)</td>
<td>1.30 (0.95–1.80)</td>
<td>1.30 (0.95–1.79)</td>
</tr>
<tr>
<td>Average</td>
<td>1.41 (1.05–1.89) \textsuperscript{**}</td>
<td>1.42 (1.06–1.90) \textsuperscript{**}</td>
<td>1.33 (0.95–1.85)</td>
<td>1.35 (0.99–1.84)</td>
</tr>
<tr>
<td>Unfavorable</td>
<td>1.84 (1.33–2.50) \textsuperscript{***}</td>
<td>1.88 (1.41–2.50) \textsuperscript{***}</td>
<td>1.84 (1.20–2.33) \textsuperscript{***}</td>
<td>1.58 (1.16–2.16) \textsuperscript{***}</td>
</tr>
<tr>
<td>Extremely unfavorable</td>
<td>3.27 (2.23–4.78) \textsuperscript{***}</td>
<td>3.42 (2.25–5.02) \textsuperscript{***}</td>
<td>3.93 (1.92–4.47) \textsuperscript{***}</td>
<td>2.82 (1.84–4.32) \textsuperscript{***}</td>
</tr>
<tr>
<td><strong>SEIFA index of disadvantage (reference = consistently favorable)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>1.34 (1.06–1.68) \textsuperscript{**}</td>
<td>1.35 (1.07–1.69) \textsuperscript{**}</td>
<td>1.24 (0.98–1.59)</td>
<td>1.23 (0.96–1.57)</td>
</tr>
<tr>
<td>Moderately unfavorable</td>
<td>1.35 (1.07–1.71) \textsuperscript{**}</td>
<td>1.36 (1.08–1.73) \textsuperscript{**}</td>
<td>1.19 (0.93–1.53)</td>
<td>1.16 (0.90–1.49)</td>
</tr>
<tr>
<td>Deteriorating</td>
<td>1.38 (0.95–1.99)</td>
<td>1.40 (0.97–2.03)</td>
<td>1.04 (0.68–1.58)</td>
<td>1.00 (0.65–1.53)</td>
</tr>
</tbody>
</table>

\textsuperscript{a} Model 1: additionally controlled for age, maternal, and paternal early puberty reported in W5.
\textsuperscript{b} Model 2: additionally controlled for perinatal risk factors including cesarean delivery, birth weight, gestational age, and breastfeeding in W1.
\textsuperscript{c} Model 3: additionally controlled for childhood (W3, 4–5 years old), peripubertal (8–9 years old), and current (W6, 10–11 years old) adiposity, ΔBMI-SDS between W3 and W6, physical activity, and sleep duration in W5 and W6.
\textsuperscript{d} Model 4: additionally controlled for ever having stepfathers for girls in W5 and W6 and stressful life events in W5 and W6.

\textsuperscript{*} \textit{P} < .05.

\textsuperscript{**} \textit{P} < .01.

\textsuperscript{***} \textit{P} < .001.
Fernald41 illustrated how rapidly the social environment can influence the epigenome at every level of the HPG axis, from the expression and secretion of gonadotropin-releasing hormone in the brain to the development of sperm in the gonads, by using the social African cichlid fish as a model. However, less is known about how the social environment may effect change in the systems regulating pubertal timing. The role of both epigenetic changes and small RNA (eg, microRNAs) regulation in potentially mediating socially induced changes along the reproductive axis is an exciting area of future work.42,43

The primary strength of our study is the longitudinal measurement of socioeconomic disadvantage repeatedly collected from adolescents in a nationally representative birth cohort, which demonstrates that persistent and long-term exposure to socioeconomic disadvantage had more deleterious influences on early pubertal timing among both boys and girls. The most significant limitation was the parental report of pubertal development. This study’s use of parental reports needs to be considered in light of the ethics barriers to gaining self-reports in younger children as well as their doubtful validity, particularly in young boys.44 Parents are better at assessing whether their children were in puberty than at describing its very first physical signs.44

Considering that the majority of our participants were in the early stages of puberty, parental reports of puberty might be reliable in our large epidemiologic study in which the distinction of prepuberty versus puberty is important. There is little or no literature on the possibility that parental SES may affect reporting of their children’s pubertal status. It is theoretically possible that parental SES might be associated with a different understanding of pubertal development or a different pattern of observation of their children’s growth. There is no clear a priori basis for believing this may affect the study findings, but the possibility cannot be totally excluded. Another limitation may be using parental rather than child report of life events. Although parental events such as divorce and financial difficulties affect children, it is possible that we have missed other events that are of particular relevance to a child (eg, change of school).

CONCLUSIONS
Cumulative exposure to extremely unfavorable household SEP from birth was associated with 4 times and 2 times the risk of early puberty in boys and girls, respectively, compared with those from a favorable background. The findings raise the possibility that the effects of social disadvantage early in life on later health may be mediated by the effects on the timing of puberty.

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ABBREVIATIONS
BIC: Bayesian information criterion
CI: confidence interval
HPA: hypothalamic-pituitary-adrenal
HPG: hypothalamic-pituitary-gonadal
LCA: latent class analysis
LSAC: Longitudinal Study of Australian Children
OR: odds ratio
SEIFA: Socio-Economic Indexes for Areas
SEP: socioeconomic position
SES: socioeconomic status
W: wave

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