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## Neighborhood Racial and Economic Privilege and Timing of Pubertal Onset in Girls

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### Abstract

**Purpose:** Early puberty is associated with adverse health outcomes over the life course, and Black and Hispanic girls experience puberty earlier than girls of other racial/ethnic backgrounds. Neighborhood racial and economic privilege may contribute to these disparities by conferring differential exposure to mechanisms (e.g., stress, obesity, endocrine disruptors) underlying early puberty. We examined associations between neighborhood privilege, measured by the Index of Concentration at the Extremes (ICE), and age at pubic hair onset (pubarche) and breast development onset (thelarche) in a large multiethnic cohort.

**Methods:** A cohort of 46,299 girls born 2005–2011 at Kaiser Permanente Northern California medical facilities were followed until 2021. Pubertal development was assessed routinely by pediatricians using the Sexual Maturity Rating scale. ICE quintiles for race/ethnicity, income, and income + race/ethnicity were calculated using American Community Survey 2010 5-year estimates and linked to census tract at birth. We fit multilevel Weibull regression models accommodating left, right, and interval censoring for all analyses.

**Results:** ICE measures were monotonically associated with pubertal onset, with the strongest associations observed for ICE–race/ethnicity. Adjusting for maternal education, age at delivery, and parity, girls from the least versus most privileged ICE–race/ethnicity quintiles were at increased risk for earlier pubarche (hazard ratio: 1.30, 95% confidence interval: 1.21, 1.38) and thelarche (hazard ratio: 1.45, 95% confidence interval: 1.36, 1.54). These associations remained significant after adjusting for girls' race/ethnicity and childhood body mass index. Additionally, adjustment for ICE partially attenuated Black–White and Hispanic–White disparities in pubertal onset.

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**Conflicts of interest:** The authors have no conflicts of interest to disclose.

**Discussion:** Neighborhood privilege may contribute to pubertal timing and related disparities.

### Keywords

Puberty; Pubertal timing; Pubarche; Thelarche; Adolescence; Index of Concentration at the Extremes (ICE); Neighborhood; Racial/ethnic disparities; Health equity

Earlier puberty is associated with adverse health outcomes over the life course, including psychopathology in adolescence [1] and cancer and cardiovascular disease in adulthood [2,3]. In the United States, age of onset of puberty has declined dramatically over the last four decades [4], and there is marked variation in pubertal timing across racial/ethnic groups. Studies have consistently shown that non-Hispanic Black (hereafter, Black) and Hispanic girls experience puberty earlier than non-Hispanic White (hereafter, White) girls [5–8]. These differences correspond to widely documented disparities in reproductive health and chronic disease later in life, making understanding the drivers of pubertal timing and racial/ethnic disparities therein critical for advancing health equity.

The timing of puberty is regulated by a complex interplay of genetic and environmentally mediated factors, indicating a need to examine determinants at multiple levels. While genetics play an important role [9], the probabilistic impact of genetics on pubertal timing is highly dependent on girls' physical and social environment [10,11]. Ample research has linked earlier puberty with stressful conditions in childhood, including socioeconomic hardship [12], trauma [13] and father absence [14]. At the same time, rapid childhood growth and increased adiposity are associated with earlier pubertal timing in girls [15]. Researchers have proposed that psychosocial and energetic factors both impact pubertal timing in high-resource settings such as the United States, where place-based social disadvantage contributes both to chronic stress and reduced access to resources that mitigate obesity [11]. However, upstream factors that shape girls' vulnerability to psychosocial and energetic risks have not been widely studied in relation to puberty.

In the United States, policies and practices of racial exclusion, disinvestment, and displacement have afforded structural advantages to White communities while systematically denying communities of color access to resource- and opportunity-rich neighborhoods [16]. Racial/ethnic disparities in neighborhood disadvantage persist across all socioeconomic levels [17], and may contribute to variation in pubertal timing by conferring differential exposure to chronic stress, obesogenic conditions, and other potential mechanisms underlying earlier puberty such as environmental endocrine-disruptors [18]. While some studies have linked neighborhood characteristics with girls' pubertal timing, they have been limited by small sample sizes, limited racial/ethnic diversity, and/or reliance on parent-reported pubertal development or age at menarche as an outcome (which occurs relatively late in the pubertal transition) [19–22]. Moreover, none of these studies explicitly examined how place-based social stratification reflecting structural racism may contribute to racial/ethnic disparities in pubertal timing.

Recently, the *Index of Concentration at the Extremes* (ICE) has emerged as a promising tool for monitoring place-based health inequities. ICE captures the multidimensional nature of structural racism by quantifying spatial concentrations of both racial and economic

privilege/disadvantage in a single metric [23]. Unlike traditional segregation, poverty, and inequality measures, ICE takes into account both the least and most privileged groups, indicates directionality of concentration toward either extreme, and can be meaningfully employed at multiple geographic levels (e.g., census tract, zip code) [24]. While ICE has been associated with adverse birth outcomes, cancer, and other health indicators [24], it has not been studied in relation to puberty.

To address these gaps, we examined associations between neighborhood (i.e., census tract) ICE at birth and timing of pubic hair onset (pubarche) and breast development onset (thelarche) in a large, multiethnic cohort of girls in Northern California. We hypothesized that residence in a neighborhood with less racial and economic privilege would be independently associated with earlier pubertal onset. Additionally, we investigated the contribution of ICE to racial/ethnic variation in pubertal timing and hypothesized that adjustment for ICE would attenuate Black–White and Hispanic–White disparities.

## Methods

### Participants

This study was conducted using a prospective cohort of girls who are members of Kaiser Permanente Northern California (KPNC), an integrated healthcare delivery system that serves over 4.5 million socioeconomically and racially/ethnically diverse members and participates in California’s Medicaid program. KPNC’s catchment area includes San Francisco, the Greater Bay and Sacramento Areas, Silicon Valley, and Central Valley. Its membership is generally representative of the Northern California population [25].

Electronic health records (EHRs) were used to identify girls born singleton and full-term (>36 weeks gestation) at a KPNC facility from 2005 to 2011. We excluded girls with medical conditions that may affect pubertal development (e.g., congenital adrenal hyperplasia). Of the 47,356 records that met initial inclusion criteria, we excluded an additional 1,057 who were missing data on covariates. Puberty data were collected longitudinally at routine pediatric appointments through September 30, 2021. Approximately 3% (N = 1,227) of the 46,299 eligible records were missing pubic hair data and 2% (N = 987) were missing breast development data, resulting in a final sample of 45,072 girls for the pubarche analysis and 45,312 for the thelarche analysis. Study protocols were approved by the KPNC Institutional Review Board.

### Measurements

**Exposures.**—American Community Survey 2010 5-year estimates were used to compute census-tract level ICE scores for racial privilege only (ICE–race/ethnicity), economic privilege only (ICE–income), and racial and economic privilege combined (ICE–income + race/ethnicity) [23]. Participants were assigned ICE scores for the census tract corresponding to their primary residence at or within one year of birth. ICE measures spatial social polarization by quantifying neighborhood concentrations toward extremes of privilege and disadvantage in a single metric. Scores range from –1 to 1, with –1 indicating 100% concentration of the population in the least privileged extreme, and 1 indicating 100%

concentration in the most privileged extreme. ICE measures were calculated using the formula:

$$ICE_i = (A_i - P_i) / T_i$$

where, for each census tract  $i$ ,  $A_i$  corresponds to the number of residents belonging to the most privileged extreme,  $P_i$  corresponds to the number of residents belonging to the least privileged extreme, and  $T_i$  corresponds to the total population for whom privilege was measured. The extremes for ICE–income correspond to the 20th and 80th percentiles of the 2010 national household income distribution (<\$20k and \$100k, respectively). Based on the enduring legacy of pro-White and anti-Black policies and practices in the United States, ICE–race/ethnicity considers White residents as belonging to the most privileged extreme and Black residents as belonging to the least privileged extreme. For ICE–income + race/ethnicity, the extreme groups correspond to the Black population below the 20th percentile of household income (least privileged) and the White population at or above the 80th percentile of household income (most privileged). ICE scores were categorized into quintiles based on California state-wide distributions.

**Outcomes.**—Breast and pubic hair development were assessed by KPNC pediatricians using the 5-stage Sexual Maturity Rating (SMR) scale at routine well-child visits, which occur every 1–2 years. Breast development was determined by a combination of palpation and visual inspection and pubic hair development was determined by visual inspection only. Previous work confirming the validity of KPNC puberty data is described in detail elsewhere [14]. We examined thelarche and pubarche as separate outcomes, with “onset” defined as the age at transition from SMR Stage 1 (prepubertal) to Stage 2+.

Although KPNC encourages pediatricians to conduct SMR exams for all children ages 6+ during each well-child visit, providers may skip the exams due to lack of time, refusal by the patient, or obvious full maturation. We limited analyses to exams conducted at 5 years of age. In our sample, the number of well-child visits with a recorded SMR ranged from 1 to 11 (median: 2; standard deviation: 1.33).

**Covariates.**—Data on maternal factors (education level, parity, and age at delivery) and girls’ race/ethnicity were extracted from girls’ birth certificates. Maternal education level was categorized as high school or less, some college, college graduate, or postgraduate education. Parity was categorized as 0, 1, or 2+ prior livebirths. Age at delivery was treated continuously. Girls’ race/ethnicity was categorized as White, Black, Hispanic, Asian/Pacific Islander, Indigenous, and other/unknown. Because few girls were identified as Indigenous, we collapsed the Indigenous and other/unknown categories in analyses to avoid positivity violations.

Girls’ height and weight were obtained from clinic visits occurring between 5 and 6 years of age. Continuous body mass index (BMI)-for-age was calculated using age- and sex-specific Centers for Disease Control and Prevention standard population distributions for the year 2000.

## Statistical analyses

We performed all analyses using R version 4.1.2 (R Foundation for Statistical Computing). Multilevel Weibull regression models accommodating left, right, and interval censoring were used to estimate time ratios (TRs), hazard ratios (HRs), and their respective 95% confidence intervals (CIs). Associations between each ICE measure and age of pubertal onset were determined using three models: unadjusted (Model 1), adjusted for maternal factors (age at delivery, parity, education; Model 2), and adjusted for maternal factors and girls' race/ethnicity (Model 3). The TR represents the median time to pubertal onset for a given ICE quintile compared to the referent quintile (Q5). We considered girls left censored if they had transitioned to SMR Stage 2+ at the time of their first exam and right censored if they had not transitioned to Stage 2+ by the end of follow-up.

Since childhood overweight/obesity are well-established risk factors for earlier pubertal timing [15], we conducted sensitivity analyses expanding our models with adjustments for continuous childhood BMI-for-age among a subsample of girls for whom we had BMI data (Model 4). We hypothesized that associations between ICE and pubertal onset would be attenuated by the inclusion of BMI.

To assess racial/ethnic disparities, we used Weibull regression models to estimate associations between girls' race/ethnicity and age at thelarche and pubarche (with White race/ethnicity as reference). Adapting methods from Shrimali et al. [26], we examined the contribution of ICE to Black–White and Hispanic–White disparities by comparing the TRs and HRs from models adjusting for each ICE measure with the TRs and HRs from models adjusting for individual-level covariates only. The percentage reduction in disparities due to ICE was calculated using the formula (where “estimate” = HR or TR):

$$(\text{Estimate}_{(-\text{ICE})} - \text{Estimate}_{(+\text{ICE})}) / (\text{Estimate}_{(-\text{ICE})} - 1)$$

## Results

### Participant characteristics

Table 1 describes health and demographic characteristics of girls and their mothers, overall and by the least and most privileged ICE quintiles. The sample was comprised of 46,299 girls from 2,594 census tracts in Northern California, with an average of 18 girls per census tract (range: 1–140). Black and Hispanic girls, girls whose mothers had no college education, and girls with overweight/obesity were disproportionately represented in the least privileged ICE quintiles and underrepresented in the most privileged quintiles; the opposite pattern was observed for White girls. Girls identified as Asian/Pacific Islander were notably underrepresented in the most privileged quintile of ICE–race/ethnicity.

### Primary analyses

**Neighborhood privilege and pubarche.**—Among the 45,072 girls included in the pubarche analysis, 14% (N = 6,522) were left censored and 51% (N = 22,778) were right censored. A general dose–response pattern emerged across the three ICE measures wherein each quintile of declining privilege corresponded to higher hazards of earlier pubarche

relative to the most privileged quintile (Table 2). In models adjusting for maternal age at delivery, parity, and education (Model 2), the HR for the least privileged quintile was 1.30 for ICE–race/ethnicity (95% CI: 1.21, 1.38), 1.11 for ICE–income (95% CI: 1.03, 1.20), and 1.24 for ICE–income + race/ethnicity (95% CI: 1.17, 1.32). The corresponding TRs for girls from the least versus most privileged quintiles of ICE–race/ethnicity, ICE–income, and ICE–income + race/ethnicity translate to approximately 3.4, 1.4, and 2.9 months earlier pubarche, respectively. Effects of ICE–race/ethnicity and ICE–income + race/ethnicity were attenuated but remained significant after including girls’ race/ethnicity as a covariate, while effects of ICE–income became nonsignificant (Model 3).

**Neighborhood privilege and thelarche.**—Among the 45,312 girls included in the thelarche analysis, 19% (N = 8,707) were left censored and 42% (N = 18,913) were right censored. In models adjusting for maternal age at delivery, parity, and education (Model 2), the HR for the least privileged quintile was 1.45 for ICE–race/ethnicity (95% CI: 1.36, 1.54), 1.09 for ICE–income (95% CI: 1.02, 1.17), and 1.33 for ICE–income + race/ethnicity (95% CI: 1.25, 1.41). The corresponding TRs translate to approximately 4.8, 1.2, and 3.7 months earlier thelarche, respectively. As with the pubarche models, effects of ICE–race/ethnicity and ICE–income + race/ethnicity on thelarche were attenuated but remained significant after including girls’ race/ethnicity as a covariate, while effects of ICE–income became nonsignificant (Model 3).

**Sensitivity analyses.**—In all, 40,573 and 40,750 girls with childhood BMI-for-age data were retained in sensitivity analyses for pubarche and thelarche, respectively (Model 4). Adjusting for childhood BMI-for-age in addition to maternal education, parity, age at delivery, and girls’ race/ethnicity resulted in estimates very similar to those observed in Model 3 for both pubarche (Table 2) and thelarche (Table 3).

## Secondary analyses

**Contribution of ICE to racial/ethnic disparities in pubertal onset.**—There were substantial racial/ethnic disparities for pubarche (Table 4) and thelarche (Table 5). Adjusting for maternal age at delivery, parity, and education, Black girls had 2.16 times greater risk of experiencing pubarche (HR: 2.16, 95% CI: 2.01, 2.31) and 1.81 times greater risk of experiencing thelarche (HR: 1.81, 95% CI: 1.69, 1.93) at earlier ages relative to White girls. The corresponding TRs translate to approximately 9.7 and 7.5 months earlier pubarche and thelarche, respectively. Adjustment for ICE–race/ethnicity yielded approximately 10% and 18% reductions in the Black–White disparity in pubarche (HR: 2.04, 95% CI: 1.90, 2.19) and thelarche (HR: 1.66, 95% CI: 1.55, 1.79). Adjustment for ICE–income + race/ethnicity yielded more modest reductions of approximately 6% for the disparity in pubarche (HR: 2.09, 95% CI: 1.94, 2.25) and 11% for the disparity in thelarche (HR: 1.72, 95% CI: 1.60, 1.85) between Black and White girls.

Adjusting for maternal age at delivery, parity, and education, Hispanic girls had 1.16 times greater risk of experiencing pubarche (HR: 1.16, 95% CI: 1.11, 1.21) and 1.23 times greater risk of experiencing thelarche (HR: 1.23, 95% CI: 1.18, 1.28) at earlier ages relative to White girls. The corresponding TRs translate to Hispanic–White differences of

approximately 1.9 and 2.7 months earlier pubarche and thelarche, respectively. In models adjusting for ICE–race/ethnicity, Hispanic–White disparities declined by approximately 25% for pubarche (HR: 1.12, 95% CI: 1.07, 1.17) and 29% for thelarche (HR: 1.16, 95% CI: 1.11, 1.22). Adjustment for ICE–income + race/ethnicity resulted in reductions of approximately 13% in the Hispanic–White disparity for pubarche (HR: 1.14, 95% CI: 1.09, 1.19) and 14% for thelarche (HR: 1.20, 95% CI: 1.14, 1.25).

## Discussion

Our study suggests that neighborhood racial and economic privilege, as measured by ICE, may contribute to girls' pubertal timing and racial/ethnic disparities therein. Compared to girls born into neighborhoods of concentrated privilege, girls born into neighborhoods of concentrated disadvantage were significantly more likely to experience pubarche and thelarche at earlier ages, even after controlling for maternal characteristics such as education level, age at delivery, and parity. Associations for ICE–race/ethnicity and ICE–income + race/ethnicity were slightly attenuated but remained significant after including girls' race/ethnicity as a covariate, suggesting that the harmful effects of ICE operate independently of a girl's socially constructed race or ethnicity. However, Black and Hispanic girls were disproportionately represented in the least privileged ICE quintiles and experienced pubarche and thelarche significantly earlier than their White counterparts. In secondary analyses, Black–White and Hispanic–White differences in pubertal onset narrowed after adjustment for ICE–race/ethnicity and ICE–income + race/ethnicity, implicating the role of structural racism in perpetuating these disparities.

Our findings lend support to a growing literature demonstrating the utility of ICE for measuring structural racism at the census tract level, and extend previous research linking ICE to well-documented precursors and sequelae of earlier pubertal timing in women, including preterm birth, cardiovascular disease, and breast, cervical, and ovarian cancer [24]. Our results differ somewhat from the broader ICE literature: approximately two-thirds of epidemiologic studies using ICE have reported larger effects for the combined measure of racial and economic privilege (ICE–income + race/ethnicity) than for the measures of racial (ICE–race/ethnicity) and economic (ICE–income) privilege alone [24]. In our study, ICE–race/ethnicity exhibited stronger effects on pubertal timing than ICE–income + race/ethnicity, while effects of ICE–income were markedly weaker and became nonsignificant in fully adjusted models. We suspect that ICE–income, calculated based on national household income distributions, does not adequately capture extremes of economic privilege and disadvantage in Northern California, given the region's exceptionally high cost of living [27]. The majority (73%) of census tracts in our sample had ICE–income values greater than 0, indicating a propensity for neighborhoods to have larger concentrations of households living above the 80th percentile of the national household income distribution. This likely contributed to our divergent results.

Findings build on prior studies documenting earlier onset of secondary sexual characteristics among Black and Hispanic girls relative to White girls [6–8]. We found evidence to suggest that neighborhood racial and economic privilege contribute in part to these disparities. Although we calculated ICE measures based on neighborhood concentrations of White



versus Black residents, we conceptualized ICE–race/ethnicity and ICE–income + race/ethnicity as proxies for the enduring effects of pro-White and anti-Black policies and practices that segregated and marginalized racially minoritized communities. This legacy has sustained inequitable distributions of resources, opportunities, and risks that drive health across the life course and intergenerationally [28]. Lower ICE scores have been associated with a range of pathogenic conditions—including obesogenic conditions (lack of greenspace) [29] and greater exposure to ambient air pollution [30] and violence [31,32]—that could theoretically accelerate the onset of puberty, for example by limiting physical activity, disrupting normal endocrine production, and/or influencing hormonal stress responses that trigger puberty.

Since childhood overweight and obesity are well-established risk factors for earlier puberty [15] and were more prevalent among girls from less privileged neighborhoods, we expected childhood BMI to attenuate associations between ICE and pubertal timing. Contrary to our hypothesis, the magnitude of associations was not substantively different in sensitivity analyses controlling for childhood BMI. This finding is consistent with a smaller study by our research team, which found that associations between neighborhood availability of recreational outlets and Black girls’ pubertal timing were not attenuated by prepubertal BMI [19]. While the “change in coefficient” approach remains the most common method for mediation in studies with time-to-event outcomes [33], causal inference approaches, along with a more comprehensive set of adiposity measures, are needed to examine childhood adiposity as a potential mediator more rigorously.

We acknowledge that large racial/ethnic inequities in pubertal timing remained in our study even after accounting for ICE, suggesting a role for factors not captured in our data that may influence girls’ reproductive development. For example, a large body of research supports a relationship between racial discrimination, experienced both directly and vicariously through caregivers, and adverse health outcomes in childhood and adolescence [34]. It has been demonstrated that experiences of racial discrimination are less prevalent among adolescents living in more racially congruent neighborhoods [35]. Protection against racial discrimination, access to culturally appropriate resources, and greater perceived social cohesion, social support, and safety in racially homogenous environments may help mitigate deleterious effects of racial residential segregation and explain why we observed relatively small effects of ICE on Black–White disparities in pubertal timing [36]. Supporting this notion, studies have found that girls who report more experiences of racial discrimination [37,38] and who perceive their neighborhoods as unsafe [21] are at increased risk of earlier puberty. As such, future research should examine how experiences of discrimination and perceptions of the neighborhood social environment modify effects of neighborhood privilege on pubertal timing. Future studies may also examine alternative dimensions of ICE, such as Hispanic versus White or immigrant versus US-born polarization, in relation to pubertal timing.

This study has important limitations and strengths. Reliance on EHRs meant that we lacked detailed information on potentially important confounders such as individual-level socioeconomic status. While we were able to control for maternal education level, socioeconomic status is a multidimensional construct and its indicators (e.g., education,

income, wealth) are not interchangeable [39]. Information on diet, physical activity, and food insecurity is not routinely documented in the KPNC EHR system and are factors that would merit investigation in future studies. Moreover, we only had access to girls' addresses at birth, limiting our ability to examine changes in neighborhood environment across childhood or account for duration of exposure at a given level of ICE. Finally, we analyzed a fully insured patient sample presenting for routine pediatric care, which may restrict generalizability to uninsured and otherwise medically underserved populations. However, the KPNC membership is generally representative of the underlying service area population [25]. Despite some limitations, access to EHRs spanning 16 years of follow-up permitted us to conduct one of the largest prospective studies with direct clinical assessments of puberty in a racially/ethnically diverse sample.

Structural racism, a fundamental cause of racial/ethnic health disparities, is rarely considered in the context of puberty. This study demonstrates that structural racism as measured by ICE is independently associated with timing of girls' puberty onset and racial/ethnic disparities therein. The observed impact of ICE on pubertal timing contributes to a life-course understanding of women's health inequities, with earlier pubertal maturation representing one potential "pathway of embodiment" [40] from prenatal and/or childhood experiences of place-based disadvantage to health deterioration later in life. While individual-level risk factors are important and should be addressed in the immediate term, our findings indicate that efforts to remediate inequities in early pubertal timing and its consequences may not be sufficient without redressing the health-damaging structures of racial exploitation and oppression that created them in the first place. Studies must continue to identify structural barriers to healthy aging in childhood and adolescence and rigorously assess causal mechanisms to better inform upstream interventions.

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## Data availability statement:

The datasets generated and/or analyzed during the current study are not publicly available due to our institutional policy. Individuals who are interested in accessing the data may contact the corresponding author regarding [or to discuss or set up] a data use agreement

## References

- [1]. Deardorff J, Marceau K, Johnson M, et al. Girls' pubertal timing and tempo and mental health: A longitudinal examination in an ethnically diverse sample. *J Adolesc Health* 2021;68:1197–203. [PubMed: 33637403]
- [2]. Fuhrman BJ, Moore SC, Byrne C, et al. Association of the age at menarche with site-specific cancer risks in pooled data from nine cohorts. *Cancer Res* 2021;81:2246–55. [PubMed: 33820799]

- [3]. Ley SH, Li Y, Tobias DK, et al. Duration of reproductive life span, age at menarche, and age at menopause are associated with risk of cardiovascular disease in women. *J Am Heart Assoc* 2017;6:e006713. [PubMed: 29097389]
- [4]. Eckert-Lind C, Busch AS, Petersen JH, et al. Worldwide secular trends in age at pubertal onset assessed by breast development among girls: A systematic review and meta-analysis. *JAMA Pediatr* 2020;174:e195881. [PubMed: 32040143]
- [5]. Shirazi TN, Rosinger AY. Reproductive health disparities in the USA: Self-reported race/ethnicity predicts age of menarche and live birth ratios, but not infertility. *J Racial Ethn Health Disparities* 2021;8:33–46. [PubMed: 32378159]
- [6]. Wu T, Mendola P, Buck GM. Ethnic differences in the presence of secondary sex characteristics and menarche among US girls: The Third National Health and Nutrition Examination survey, 1988–1994. *Pediatrics* 2002;110:752–7. [PubMed: 12359790]
- [7]. Biro FM, Greenspan LC, Galvez MP, et al. Onset of breast development in a longitudinal cohort. *Pediatrics* 2013;132:1019–27. [PubMed: 24190685]
- [8]. Goldberg M, D'Aloisio A, O'Brien K, et al. Early-life exposures and age at thelarche in the sister study cohort. *Breast Cancer Res* 2021;23:111. [PubMed: 34895281]
- [9]. Zhu J, Kusa TO, Chan YM. Genetics of pubertal timing. *Curr Opin Pediatr* 2018;30:532–40. [PubMed: 29771761]
- [10]. Ellis BJ. Timing of pubertal maturation in girls: An integrated life history approach. *Psychol Bull* 2004;130:920–58. [PubMed: 15535743]
- [11]. Glass DJ, Geerkens JT, Martin MA. Psychosocial and energetic factors on human female pubertal timing: A systematized review. *Evol Hum Sci* 2022;4:e28. [PubMed: 37588922]
- [12]. Hiatt RA, Stewart SL, Deardorff J, et al. Childhood socioeconomic status and menarche: A prospective study. *J Adolesc Health* 2021;69:33–40. [PubMed: 34172141]
- [13]. Colich NL, Rosen ML, Williams ES, McLaughlin KA. Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. *Psychol Bull* 2020;146:721–64. [PubMed: 32744840]
- [14]. Aghaee S, Deardorff J, Greenspan LC, et al. Early life household intactness and timing of pubertal onset in girls: A prospective cohort study. *BMC Pediatr* 2020;20:464. [PubMed: 33109126]
- [15]. Zhou X, Hu Y, Yang Z, et al. Overweight/obesity in childhood and the risk of early puberty: A systematic review and meta-analysis. *Front Pediatr* 2022;10:795596. [PubMed: 35722495]
- [16]. Bailey ZD, Feldman JM, Bassett MT. How structural racism works — racist policies as a root cause of U.S. racial health inequities. *N Engl J Med* 2021;384:768–73. [PubMed: 33326717]
- [17]. Williams DR, Collins C. Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Rep* 2001;116:404–16. [PubMed: 12042604]
- [18]. James-Todd TM, Chiu YH, Zota AR. Racial/ethnic disparities in environmental endocrine disrupting chemicals and women's reproductive health outcomes: Epidemiological examples across the life course. *Curr Epidemiol Rep* 2016;3:161–80. [PubMed: 28497013]
- [19]. Deardorff J, Fyfe M, Ekwaru JP, et al. Does neighborhood environment influence girls' pubertal onset? Findings from a cohort study. *BMC Pediatr* 2012;12:27. [PubMed: 22414266]
- [20]. Thorpe D, Klein DN. The effect of neighborhood-level resources on children's physical development: Trajectories of body mass index and pubertal development and the influence of child biological sex. *J Youth Adolesc* 2022;51:967–83. [PubMed: 35028875]
- [21]. Amir D, Jordan MR, Bribiescas RG. A longitudinal assessment of associations between adolescent environment, adversity perception, and economic status on fertility and age of menarche. *PLoS One* 2016;11:e0155883. [PubMed: 27249338]
- [22]. Sun Y, Mensah FK, Azzopardi P, et al. Childhood social disadvantage and pubertal timing: A national birth cohort from Australia. *Pediatrics* 2017;139:e20164099. [PubMed: 28562276]
- [23]. Krieger N, Kim R, Feldman J, Waterman PD. Using the Index of Concentration at the Extremes at multiple geographical levels to monitor health inequities in an era of growing spatial social polarization: Massachusetts, USA (2010–14). *Int J Epidemiol* 2018;47:788–819. [PubMed: 29522187]

- [24]. Larrabee Sonderlund A, Charifson M, Schoenthaler A, et al. Racialized economic segregation and health outcomes: A systematic review of studies that use the Index of Concentration at the Extremes for race, income, and their interaction. *PLoS One* 2022;17:e0262962. [PubMed: 35089963]
- [25]. Gordon N Similarity of adult Kaiser Permanente members to the adult population in Kaiser Permanente's Northern California service area: Comparisons based on the 2017/2018 cycle of the California health interview survey. Oakland, CA: Kaiser Permanente Division of Research; 2020.
- [26]. Shrimali BP, Pearl M, Karasek D, et al. Neighborhood privilege, preterm delivery, and related racial/ethnic disparities: An intergenerational application of the Index of Concentration at the Extremes. *Am J Epidemiol* 2020;189:412–21. [PubMed: 31909419]
- [27]. How has income inequality changed in the Bay area over the last decade? Bay Area Council Economic Institute. 2021. Available at: <http://www.bayareaeconomy.org/report/bay-area-income-inequality>. Accessed September 25, 2022.
- [28]. Bailey ZD, Krieger N, Agénor M, et al. Structural racism and health inequities in the USA: Evidence and interventions. *Lancet* 2017;389:1453–63. [PubMed: 28402827]
- [29]. Casey JA, James P, Cushing L, et al. Race, ethnicity, income concentration and 10-year change in urban greenness in the United States. *Int J Environ Res Public Health* 2017;14:1546. [PubMed: 29232867]
- [30]. Krieger N, Waterman PD, Gryparis A, Coull BA. Black carbon exposure, socioeconomic and racial/ethnic spatial polarization, and the Index of Concentration at the Extremes (ICE). *Health Place* 2015;34:215–28. [PubMed: 26093080]
- [31]. Krieger N, Feldman JM, Waterman PD, et al. Local residential segregation matters: Stronger association of census tract compared to conventional city-level measures with fatal and non-fatal assaults (total and firearm related), using the Index of Concentration at the Extremes (ICE) for racial, economic, and racialized economic segregation, Massachusetts (US), 1995-2010. *J Urban Health* 2017;94:244–58. [PubMed: 28130678]
- [32]. Schleimer JP, Buggs SA, McCort CD, et al. Neighborhood racial and economic segregation and disparities in violence during the COVID-19 pandemic. *Am J Public Health* 2022;112:144–53. [PubMed: 34882429]
- [33]. Lapointe-Shaw L, Bouck Z, Howell NA, et al. Mediation analysis with a time-to-event outcome: A review of use and reporting in healthcare research. *BMC Med Res Methodol* 2018;18:118. [PubMed: 30373524]
- [34]. Cave L, Cooper MN, Zubrick SR, Shepherd CCJ. Racial discrimination and child and adolescent health in longitudinal studies: A systematic review. *Soc Sci Med* 2020;250:112864. [PubMed: 32143088]
- [35]. Pasco MC, White R, Seaton EK. A systematic review of neighborhood ethnic–racial compositions on cultural developmental processes and experiences in adolescence. *Adolesc Res Rev* 2021;6:229–46.
- [36]. Gao X, Kershaw KN, Barber S, et al. Associations between residential segregation and incident hypertension: The multi-ethnic study of atherosclerosis. *J Am Heart Assoc* 2022;11:e023084. [PubMed: 35048712]
- [37]. Argabright ST, Moore TM, Visoki E, et al. Association between racial/ethnic discrimination and pubertal development in early adolescence. *Psychoneuroendocrinology* 2022;140:105727. [PubMed: 35344870]
- [38]. Seaton EK, Carter R. Perceptions of pubertal timing and discrimination among African American and Caribbean Black girls. *Child Dev* 2019;90:480–8. [PubMed: 30737778]
- [39]. Braveman PA, Cubbin C, Egerter S, et al. Socioeconomic status in health research: One size does not fit all. *JAMA* 2005;294:2879–88. [PubMed: 16352796]
- [40]. Krieger N Embodiment: A conceptual glossary for epidemiology. *J Epidemiol Community Health* 2005;59:350–5. [PubMed: 15831681]

### IMPLICATIONS AND CONTRIBUTION

Few studies have examined neighborhood-level determinants of pubertal timing, a predictor of women’s lifelong health. The current study provides preliminary evidence that neighborhood racial and economic privilege influence girls’ pubertal timing and racial/ethnic disparities therein. Findings support the need for greater attention to socio-structural drivers of puberty and place-based interventions.

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**Table 1**  
 Characteristics of participating KPNC female patients born 2005–2011 and their mothers (N = 46,299 mother-daughter pairs), overall and by Index of Concentration at the Extremes quintiles 1 (least privileged) and 5 (most privileged)

	Overall (N = 46,299)		ICE-race/ethnicity <sup>a</sup>		ICE-income <sup>a</sup>		ICE-income + race/ethnicity <sup>a</sup>	
	Least privileged	Most privileged	Least privileged	Most privileged	Least privileged	Most privileged	Least privileged	Most privileged
<b>Maternal characteristics</b>								
Age at delivery (mean [SD])	30.1 (5.7)	31.2 (5.5)	28.6 (6.0)	31.2 (5.5)	28.2 (6.1)	31.6 (5.2)	28.1 (6.1)	32.0 (5.2)
Parity (n [%])								
0	20,209 (43.6)	2,419 (45.3)	3,223 (41.7)	2,419 (45.3)	1,899 (42.9)	5,620 (43.1)	2,852 (41.7)	3,576 (44.2)
1	16,185 (35.0)	1,964 (36.8)	2,469 (31.9)	1,964 (36.8)	1,378 (31.1)	4,919 (37.8)	2,164 (31.6)	3,064 (37.9)
2+	9,905 (21.4)	960 (18.0)	2,042 (26.4)	960 (18.0)	1,153 (26.0)	2,487 (19.1)	1,824 (26.7)	1,442 (17.8)
<b>Education (n [%])</b>								
High school or less	13,483 (29.1)	1,154 (21.6)	3,393 (43.9)	1,154 (21.6)	2,141 (48.3)	2,022 (15.5)	3,279 (47.9)	1,238 (15.3)
Some college	13,881 (30.0)	1,407 (26.3)	2,558 (33.1)	1,407 (26.3)	1,428 (32.2)	3,360 (25.8)	2,276 (33.3)	1,925 (23.8)
College graduate	12,123 (26.2)	1,680 (31.4)	1,256 (16.2)	1,680 (31.4)	590 (13.3)	4,564 (35.0)	942 (13.8)	2,876 (35.6)
Postgraduate	6,812 (14.7)	1,102 (20.6)	527 (6.8)	1,102 (20.6)	271 (6.1)	3,080 (23.6)	343 (5.0)	2,043 (25.3)
<b>Daughters' characteristics</b>								
<b>Race/ethnicity (n [%])</b>								
White	16,359 (35.3)	3,675 (68.8)	995 (12.9)	3,675 (68.8)	932 (21.0)	5,256 (40.4)	1,046 (15.3)	4,537 (56.1)
Black	3,418 (7.4)	74 (1.4)	1,411 (18.2)	74 (1.4)	783 (17.7)	445 (3.4)	1,394 (20.4)	188 (2.3)
Hispanic	12,257 (26.5)	733 (13.7)	2,876 (37.2)	733 (13.7)	1,660 (37.5)	2,132 (16.4)	2,561 (37.4)	1,211 (15.0)
Asian/Pacific Islander	10,151 (21.9)	506 (9.5)	1,781 (23.0)	506 (9.5)	705 (15.9)	3,817 (29.3)	1,313 (19.2)	1,332 (16.5)
Indigenous	129 (0.3)	16 (0.3)	19 (0.2)	16 (0.3)	10 (0.2)	36 (0.3)	12 (0.2)	21 (0.3)
Other/unknown	3985 (8.6)	339 (6.3)	652 (8.4)	339 (6.3)	340 (7.7)	1340 (10.3)	514 (7.5)	793 (9.8)
<b>Childhood BMI-for-age (n [%])</b>								
Underweight	1,494 (3.2)	156 (2.9)	207 (2.7)	156 (2.9)	117 (2.6)	540 (4.1)	168 (2.5)	260 (3.2)
Normal weight	28,947 (62.5)	3,588 (67.2)	4,394 (56.8)	3,588 (67.2)	2,517 (56.8)	8,806 (67.6)	3,860 (56.4)	5,606 (69.4)
Overweight	6,185 (13.4)	635 (11.9)	1,151 (14.9)	635 (11.9)	615 (13.9)	1,500 (11.5)	1,013 (14.8)	975 (12.1)
Obese	5,003 (10.8)	379 (7.1)	1,190 (15.4)	379 (7.1)	679 (15.3)	1,014 (7.8)	1,072 (15.7)	558 (6.9)
Missing	4,670 (10.1)	585 (10.9)	792 (10.2)	585 (10.9)	502 (11.3)	1166 (9.0)	727 (10.6)	683 (8.5)

BMI = body mass index; ICE = Index of Concentration at the Extremes; KPNC = Kaiser Permanente Northern California.

<sup>a</sup>California statewide ICE quintiles measured at the census-tract level.

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**Table 2**

Associations between Index of Concentration at the Extremes and timing of pubarche in participating KPNC female patients born 2005–2011

ICE measured <sup>d</sup>	Model 1 (unadjusted) (N = 45,072)		Model 2 <sup>a</sup> (N = 45,072)		Model 3 <sup>b</sup> (N = 45,072)		Model 4 <sup>c</sup> (sensitivity analysis; N = 40,573)	
	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)
ICE-race/ethnicity								
Quintile 5, most privileged	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -
Quintile 4	0.99 (0.98, 1.00)	1.09 (1.02, 1.15)	0.99 (0.99, 1.00)	1.08 (1.02, 1.15)	0.99 (0.99, 1.00)	1.07 (1.01, 1.14)	0.99 (0.99, 1.00)	1.08 (1.01, 1.15)
Quintile 3	0.99 (0.98, 0.99)	1.12 (1.06, 1.19)	0.99 (0.99, 1.00)	1.11 (1.04, 1.17)	0.99 (0.98, 1.00)	1.10 (1.04, 1.17)	0.99 (0.98, 1.00)	1.11 (1.04, 1.18)
Quintile 2	0.98 (0.98, 0.99)	1.18 (1.11, 1.25)	0.98 (0.98, 0.99)	1.15 (1.08, 1.22)	0.99 (0.98, 0.99)	1.15 (1.08, 1.22)	0.98 (0.98, 0.99)	1.15 (1.08, 1.23)
Quintile 1, least privileged	0.97 (0.96, 0.97)	1.38 (1.29, 1.47)	0.97 (0.97, 0.98)	1.30 (1.21, 1.38)	0.98 (0.97, 0.99)	1.20 (1.12, 1.29)	0.98 (0.97, 0.99)	1.18 (1.10, 1.27)
ICE-income								
Quintile 5, most privileged	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -
Quintile 4	0.99 (0.99, 1.00)	1.06 (1.02, 1.11)	1.00 (1.00, 1.00)	1.04 (0.99, 1.09)	1.00 (0.99, 1.00)	1.02 (0.98, 1.07)	1.00 (0.99, 1.01)	1.00 (0.95, 1.05)
Quintile 3	0.99 (0.98, 0.99)	1.12 (1.07, 1.18)	0.99 (0.99, 1.00)	1.07 (1.01, 1.13)	1.00 (0.99, 1.00)	1.03 (0.97, 1.08)	1.00 (0.99, 1.01)	1.00 (0.95, 1.06)
Quintile 2	0.98 (0.97, 0.99)	1.20 (1.14, 1.26)	0.99 (0.99, 0.99)	1.12 (1.06, 1.18)	0.99 (0.99, 1.00)	1.05 (1.00, 1.11)	1.00 (0.99, 1.01)	1.00 (0.95, 1.07)
Quintile 1, least privileged	0.98 (0.97, 0.99)	1.21 (1.12, 1.30)	0.99 (0.99, 1.00)	1.11 (1.03, 1.20)	1.00 (0.99, 1.01)	1.01 (0.94, 1.09)	1.00 (0.99, 1.01)	0.98 (0.90, 1.06)
ICE-income + race/ethnicity								
Quintile 5, most privileged	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -	1.00 -
Quintile 4	0.99 (0.99, 1.00)	1.07 (1.02, 1.12)	1.00 (1.00, 1.00)	1.04 (0.99, 1.09)	1.00 (0.99, 1.00)	1.05 (0.99, 1.10)	1.00 (0.99, 1.00)	1.04 (0.99, 1.10)
Quintile 3	0.99 (0.98, 0.99)	1.12 (1.06, 1.18)	0.99 (0.99, 1.00)	1.08 (1.02, 1.14)	0.99 (0.99, 1.00)	1.08 (1.02, 1.15)	0.99 (0.99, 1.00)	1.07 (1.01, 1.13)
Quintile 2	0.98 (0.98, 0.99)	1.18 (1.11, 1.25)	0.99 (0.99, 0.99)	1.11 (1.05, 1.18)	0.99 (0.98, 1.00)	1.09 (1.02, 1.16)	0.99 (0.99, 1.00)	1.06 (0.99, 1.13)



ICE measure <sup>d</sup>	Model 1 (unadjusted) (N = 45,072)		Model 2 <sup>a</sup> (N = 45,072)		Model 3 <sup>b</sup> (N = 45,072)		Model 4 <sup>c</sup> (sensitivity analysis; N = 40,573)	
	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)
Quintile 1, least privileged	0.97 (0.96, 0.97)	1.35 (1.27, 1.43)	0.98 (0.98, 0.98)	1.24 (1.17, 1.32)	0.99 (0.98, 0.99)	1.14 (1.07, 1.21)	0.99 (0.98, 1.00)	1.10 (1.03, 1.17)

CI = confidence interval; HR = hazard ratio; ICE = Index of Concentration at the Extremes; KPNC = Kaiser Permanente Northern California; TR = time ratio.

<sup>a</sup> Adjusted for maternal age at delivery, maternal education, and maternal parity.

<sup>b</sup> Adjusted for maternal age at delivery, maternal education, maternal parity, and race/ethnicity.

<sup>c</sup> Adjusted for maternal age at delivery, maternal education, maternal parity, race/ethnicity, and continuous BMI-for-age.

<sup>d</sup> California statewide ICE quintiles measured at the census-tract level.

**Table 3**

Associations between Index of Concentration at the Extremes and timing of the larche in participating KPNC female patients born 2005–2011

CE measure <sup>d</sup>	Model 1 (unadjusted) (N = 45,312)			Model 2 <sup>a</sup> (N = 45,312)			Model 3 <sup>b</sup> (N = 45,312)			Model 4 <sup>c</sup> (sensitivity analysis; N = 40,750)		
	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)
ICE–race/ethnicity												
Quintile 5, most privileged	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Quintile 4	0.98 (0.98, 0.99)	1.15 (1.09, 1.22)	0.98 (0.98, 0.99)	1.15 (1.08, 1.22)	0.99 (0.98, 0.99)	1.12 (1.06, 1.19)	0.99 (0.98, 0.99)	1.12 (1.06, 1.19)	0.99 (0.98, 0.99)	1.13 (1.06, 1.20)	0.99 (0.98, 0.99)	1.13 (1.06, 1.20)
Quintile 3	0.97 (0.96, 0.98)	1.30 (1.23, 1.38)	0.97 (0.97, 0.98)	1.29 (1.22, 1.36)	0.98 (0.97, 0.98)	1.23 (1.16, 1.30)	0.98 (0.97, 0.98)	1.23 (1.16, 1.30)	0.98 (0.97, 0.98)	1.23 (1.15, 1.31)	0.98 (0.97, 0.98)	1.23 (1.15, 1.31)
Quintile 2	0.96 (0.96, 0.97)	1.41 (1.33, 1.49)	0.96 (0.96, 0.97)	1.38 (1.31, 1.47)	0.97 (0.97, 0.98)	1.29 (1.21, 1.37)	0.97 (0.97, 0.98)	1.29 (1.21, 1.37)	0.97 (0.97, 0.98)	1.30 (1.21, 1.39)	0.97 (0.97, 0.98)	1.30 (1.21, 1.39)
Quintile 1, least privileged	0.96 (0.95, 0.96)	1.51 (1.41, 1.60)	0.96 (0.96, 0.97)	1.45 (1.36, 1.54)	0.97 (0.96, 0.98)	1.29 (1.21, 1.38)	0.97 (0.96, 0.98)	1.29 (1.21, 1.38)	0.97 (0.97, 0.98)	1.27 (1.18, 1.36)	0.97 (0.97, 0.98)	1.27 (1.18, 1.36)
ICE–income												
Quintile 5, most privileged	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Quintile 4	0.99 (0.99, 1.00)	1.06 (1.01, 1.11)	1.00 (1.00, 1.00)	1.04 (0.99, 1.09)	1.00 (0.99, 1.00)	1.03 (0.98, 1.08)	1.00 (0.99, 1.00)	1.03 (0.98, 1.08)	1.00 (1.00, 1.01)	0.99 (0.95, 1.04)	1.00 (1.00, 1.01)	0.99 (0.95, 1.04)
Quintile 3	0.99 (0.99, 1.00)	1.06 (1.00, 1.12)	1.00 (1.00, 1.00)	1.01 (0.96, 1.07)	1.00 (0.99, 1.01)	1.00 (0.95, 1.05)	1.00 (0.99, 1.01)	1.00 (0.95, 1.05)	1.00 (1.00, 1.01)	0.97 (0.91, 1.03)	1.00 (1.00, 1.01)	0.97 (0.91, 1.03)
Quintile 2	0.99 (0.98, 0.99)	1.14 (1.08, 1.20)	0.99 (0.99, 1.00)	1.07 (1.01, 1.13)	1.00 (1.00, 1.00)	1.03 (0.98, 1.09)	1.00 (0.99, 1.00)	1.03 (0.98, 1.09)	1.00 (1.00, 1.01)	0.99 (0.93, 1.04)	1.00 (1.00, 1.01)	0.99 (0.93, 1.04)
Quintile 1, least privileged	0.98 (0.98, 0.99)	1.17 (1.09, 1.25)	0.99 (0.99, 1.00)	1.09 (1.02, 1.17)	1.00 (0.99, 1.00)	1.02 (0.95, 1.10)	1.00 (0.99, 1.01)	1.02 (0.95, 1.10)	1.00 (0.99, 1.01)	1.00 (0.93, 1.08)	1.00 (0.99, 1.01)	1.00 (0.93, 1.08)
ICE–income + race/ethnicity												
Quintile 5, most privileged	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Quintile 4	0.99 (0.98, 0.99)	1.11 (1.05, 1.16)	0.99 (0.99, 1.00)	1.08 (1.03, 1.14)	0.99 (0.99, 1.00)	1.07 (1.02, 1.12)	0.99 (0.99, 1.00)	1.07 (1.02, 1.12)	0.99 (0.99, 1.00)	1.06 (1.00, 1.11)	0.99 (0.99, 1.00)	1.06 (1.00, 1.11)
Quintile 3	0.98 (0.97, 0.98)	1.22 (1.15, 1.29)	0.98 (0.98, 0.99)	1.18 (1.12, 1.25)	0.99 (0.98, 0.99)	1.14 (1.08, 1.21)	0.99 (0.98, 0.99)	1.14 (1.08, 1.21)	0.99 (0.98, 0.99)	1.13 (1.07, 1.20)	0.99 (0.98, 0.99)	1.13 (1.07, 1.20)
Quintile 2	0.97 (0.97, 0.98)	1.25 (1.18, 1.33)	0.98 (0.98, 0.99)	1.21 (1.13, 1.28)	0.99 (0.98, 0.99)	1.14 (1.07, 1.21)	0.99 (0.98, 0.99)	1.14 (1.07, 1.21)	0.99 (0.98, 1.00)	1.10 (1.03, 1.17)	0.99 (0.98, 1.00)	1.10 (1.03, 1.17)

CE measure <sup>d</sup>	Model 1 (unadjusted) (N = 45,312)		Model 2 <sup>a</sup> (N = 45,312)		Model 3 <sup>b</sup> (N = 45,312)		Model 4 <sup>c</sup> (sensitivity analysis; N = 40,750)	
	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)	TR (95% CI)	HR (95% CI)
Quintile 1, least privileged	0.96 (0.96, 0.97)	1.40 (1.33, 1.48)	0.97 (0.97, 0.98)	1.33 (1.25, 1.41)	0.98 (0.97, 0.99)	1.19 (1.13, 1.27)	0.98 (0.98, 0.99)	1.16 (1.09, 1.23)

CI = confidence interval; HR = hazard ratio; ICE = Index of Concentration at the Extremes; KPNC = Kaiser Permanente Northern California; TR = time ratio.

<sup>a</sup> Adjusted for maternal age at delivery, maternal education, and maternal parity.

<sup>b</sup> Adjusted for maternal age at delivery, maternal education, maternal parity, and race/ethnicity.

<sup>c</sup> Adjusted for maternal age at delivery, maternal education, maternal parity, race/ethnicity, and continuous BMI-for-age.

<sup>d</sup> California statewide ICE quintiles measured at the census-tract level.

**Table 4**

Racial/ethnic disparities in timing of pubarche in participating KPNC female patients born 2005–2011, unadjusted and adjusted for Index of Concentrations at the Extremes (N = 31,110)

Model:	Black–White disparity			Hispanic–White disparity		
	TR (95% CI)	% change <sup>a</sup>	HR (95% CI)	TR (95% CI)	% change <sup>a</sup>	HR (95% CI)
1. Unadjusted for ICE <sup>b</sup>	0.92 (0.91, 0.93)	N/A	2.16 (2.01, 2.31)	0.98 (0.98, 0.99)	N/A	1.16 (1.11, 1.21)
2. Adjusted for ICE–race/ethnicity	0.93 (0.92, 0.93)	7.08%	2.04 (1.90, 2.19)	0.99 (0.98, 0.99)	23.99%	1.12 (1.07, 1.17)
3. Adjusted for ICE–income	0.92 (0.91, 0.93)	0.38%	2.15 (2.00, 2.31)	0.98 (0.98, 0.99)	1.47%	1.16 (1.10, 1.21)
4. Adjusted for ICE–income + race/ethnicity	0.92 (0.92, 0.93)	3.98%	2.09 (1.94, 2.25)	0.99 (0.98, 0.99)	11.70%	1.14 (1.09, 1.19)

CI = confidence interval; HR = hazard ratio; ICE = Index of Concentration at the Extremes; KPNC = Kaiser Permanente Northern California; N/A = not applicable; TR = time ratio.

<sup>a</sup>Percent change from Model 1.

<sup>b</sup>Adjusted for maternal age at delivery, maternal education, and maternal parity.

**Table 5**

Racial/ethnic disparities in timing of the larche in participating KPNC female patients born 2005–2011, unadjusted and adjusted for Index of Concentrations at the Extremes (N = 31,378)

Model:	NH Black–NH White disparity				Hispanic–NH White disparity			
	TR (95% CI)	% change <sup>a</sup>	HR (95% CI)	% change <sup>a</sup>	TR (95% CI)	% change <sup>a</sup>	HR (95% CI)	% change <sup>a</sup>
1. Unadjusted for ICE <sup>b</sup>	0.94 (0.93, 0.94)	N/A	1.81 (1.69, 1.93)	N/A	0.98 (0.97, 0.98)	N/A	1.23 (1.18, 1.28)	N/A
2. Adjusted for ICE–race/ethnicity	0.95 (0.94, 0.95)	13.62%	1.66 (1.55, 1.79)	17.55%	0.98 (0.98, 0.99)	26.83%	1.16 (1.11, 1.22)	28.92%
3. Adjusted for ICE–income	0.94 (0.93, 0.94)	1.51%	1.79 (1.67, 1.91)	2.02%	0.98 (0.97, 0.98)	1.89%	1.22 (1.17, 1.28)	2.08%
4. Adjusted for ICE–income + race/ethnicity	0.94 (0.93, 0.95)	8.05%	1.72 (1.60, 1.85)	10.61%	0.98 (0.98, 0.99)	12.69%	1.20 (1.14, 1.25)	13.90%

CI = confidence interval; HR = hazard ratio; ICE = Index of Concentration at the Extremes; KPNC = Kaiser Permanente Northern California; N/A = not applicable; NH = non-Hispanic; TR = time ratio.

<sup>a</sup>Percent change from Model 1.

<sup>b</sup>All models adjusted for maternal age at delivery, maternal education, and maternal parity.