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Editorial

Why We Need More Biocultural Studies of Pubertal Timing

Early puberty is arguably one of the first signs of weathering—the premature aging believed to be a consequence of racial/ethnic or socioeconomic group disadvantages [1]. The disparities in pubertal development track to disparities in chronic diseases later in life, such as breast cancer. To date, the most established predictors of age at puberty are body mass index (BMI), race, and stress [2]. Yet, despite the biocultural nature of puberty, researchers seldom study the biological and social drivers of pubertal timing concurrently. The false dichotomies between biological and social drivers for pubertal timing—and between biological and social exposures during puberty for adult diseases—hinder our research from moving forward. Rather more biocultural approaches are needed [3]. The relationship between socioeconomic status (SES) and pubertal timing illustrates where such an approach would help.

In this issue of the Journal of Adolescent Health, Hiatt et al [4]. found that lower SES is associated with earlier pubertal development, extending the results of previous studies both methodologically and conceptually. First, they examine the association of SES with menarche taking into account onset of breast development, which typically occurs 2–4 years before menarche. They also considered SES simultaneously with BMI and race/ethnicity. Furthermore, they contextualize their findings within a life course approach aimed at understanding how upstream social factors, such as SES, affect breast cancer risk. They also take a historic perspective by comparing the direction of the association in their contemporary cohort with a cohort born in the 1960s.

Owing to their robust methodology, Hiatt et al [4]. were able to provide much more nuanced findings than previous studies. They report results from an urban, multiethnic longitudinal cohort of girls aged six at baseline and followed up for 11 years starting in 2004. Researchers collected data annually from girls and their mothers using questionnaires to derive an SES index based on household income, mother's education, and home ownership. To assess pubertal development, the researchers used Tanner staging [5] with palpation for the presence of breast tissue, which is the gold standard. This is a clear methodological strength in that it bypasses the critiques of previous landmark studies that declared high prevalence of precocious puberty but did not use palpation [6]. Since those landmark studies, there is growing evidence that mothers' and girls' reports of the pubertal development, as well as hormones, correlate highly with Tanner staging. Therefore, while palpation remains a clinical gold standard, other methods can be used in large-scale studies when direct clinical assessment of breast onset is not feasible [7,8].

By measuring breast development and menarche in the same girls, Hiatt et al. determined that the relationship with SES was stronger for breast development than menarche. When stratifying by race/ethnicity, SES emerged as a particularly strong predictor for age at menarche in Hispanic girls. In contrast, SES was a not significant predictor of puberty for black girls, perhaps owing to the unbalanced distribution of SES, as the authors also note. Racial differences in pubertal timing remain after taking account of SES, BMI, and race concurrently. Thus, other social factors must be at play explaining the earlier puberty observed in black and Hispanic girls when compared with non-Hispanic white girls. These findings warrant a more refined investigation into the causes of this disparity, such as environmental, material and stressful factors that SES does not fully capture. Furthermore, an intersectional approach investigating race/ethnicity and SES in relation to puberty is also warranted.

The results of this study pose new questions when it comes to interpreting how SES and puberty are related to breast cancer. Historically within the United States, high childhood SES increases breast cancer risk [9]. In contrast, this article suggests that high childhood SES *decreases* risk through delaying puberty, thus pointing to a disconnection between SES, puberty and breast cancer risk. To reconcile this contradiction, the authors compare their results with those from a 1960s cohort and observe that low SES was associated with delayed menarche [10]. They speculate that in recent decades the relationship of SES with menarche appears to be reversed, which means a possible reversal of the relationship of childhood SES to breast cancer. Longitudinal follow-up of pubertal cohorts for breast cancer or intermediate outcomes could help answer this question. An alternative explanation is that prior studies only explored the association of SES with menarche and not breast development. Over time, the relationship between breast development and menarche is changing, in that the period of time between them is lengthening owing to the declining age of breast development, while the age of menarche remains stable

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[11]. Given that the effect of SES is stronger for breast development, future studies interested in the relation of pubertal timing to adult diseases should consider the onset of breast development rather than menarche. Another possibility is the construct of SES, in terms of what it represents in relation to pubertal timing and breast cancer, has changed over time. The authors suggest a standardized SES index may help explain inconsistencies between studies, but this would only narrow the construct. Rather, a biocultural assessment of SES would widen the lens and help distinguish the material from psychosocial facets of SES.

In summary, puberty is a fascinating stage of the life course with drastic changes in biological, physiological, and social development. Its timing is linked with breast cancer risk later in life. The study by Hiatt et al. contributes evidence that both social (SES) and biological (BMI) factors influence the timing of puberty and these associations differ by race/ethnicity. Clearly, social context matters for pubertal development. To build on this, future studies should to delve into what it means to have low SES in terms of pubertal timing. Integrating refined social measures and biomarkers into biocultural studies of puberty will advance our understanding of the drivers of puberty, which will also inevitably shed light onto the racial/ethnic disparities of pubertal timing and the etiology of chronic diseases with similar racial/ethnic disparities. With these advancements we will begin to understand why puberty is a "critical opportunity for health intervention" [12].

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