The Enigmatic Pursuit of Puberty in Girls

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KEY WORDS: puberty, thelarche, menarche, endocrine disruptors

ABBREVIATIONS: BCERC—Breast Cancer and the Environment Research Centers; NHANES—National Health and Nutrition Examination Survey; PROS—Pediatric Research in Office Settings

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www.pediatrics.org/cgi/doi/10.1542/peds.2013-3058
doi:10.1542/peds.2013-3058
Accepted for publication Sep 24, 2013
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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275)
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FINANCIAL DISCLOSURE: The author has indicated she has no financial relationships relevant to this article to disclose.

FUNDING: No external funding

POTENTIAL CONFLICT OF INTEREST: The author has indicated she has no potential conflicts of interest to disclose.

COMPANION PAPER: A companion to this article can be found on page 1019, and online at www.pediatrics.org/cgi/doi/10.1542/peds.2012-3773.

Nuances of puberty, the actual trigger, influencing factors, secular and tempo changes, continue to plague us. Uncertainty, as in quantum physics, may continue. Niels Bohr concluded decades ago that what you see depends on how you look. In the robust Breast Cancer and the Environment Research Centers (BCERC) study in this issue of Pediatrics by Biro et al, onset of Tanner 2 breast development is the main end point.1 This article adds to studies providing the unsettling findings that the age of onset of breast development, in synch with, though not entirely explained by, the “obesity epidemic,” has continued to drop. It will inspire continued investigations of causes and implications for psychosocial public health. Remedies will follow.

The 1997 cross-sectional Pediatric Research in Office Settings (PROS) study on 17 000 US girls, followed by nationally representative data from the National Health and Nutrition Examination Survey III (NHANES), reported earlier onset of secondary sexual characteristics in girls over the previous several decades.2 The considerable controversy and angst generated by these findings led to lay and scientific essays and studies here and abroad. Now there is considerable agreement that puberty is occurring earlier.3 Adding further complexity are recent data finding male puberty is also occurring earlier.4

This BCERC study seeks to learn more about the role of genetic and environmental influences on breast cancer, acting through the risk factor of early maturation. Its methodology has benefited from lessons learned from recent pubertal studies. Although the 1200 girls are not nationally representative, their diversity provides data requiring our attention. Onset of breast stage 2 occurred at 8.8 years of age for African American girls, 9.3 for Hispanic girls, and 9.7 for white non-Hispanic and Asian girls. White girls experienced breast development 4 months earlier than PROS subjects 15 years prior, a change not entirely explained by current obesity prevalence. Although overall age of breast development onset for BCERC black girls was not different from PROS girls, their proportion of African American first-graders at Tanner 2 tripled in the 15 years between the studies. In the BCERC population, 18% of first-grade black girls are Tanner 2, rising to 38% in the third grade; for white girls it is 4% rising to 21%. BMI did not explain the entire change.1

Whether this early breast development represents true centrally mediated puberty has been questioned. In 2005, NHANES data 10 years apart found slight menarcheal age declines in white girls from 12.57 to 12.52 and in black girls from 12.09 to 12.06.5 NHANES data from the 2003–2008 cohort found further declines to 12.1 years of age for white girls and 11.5 for black girls, incidentally with an inverse relationship between levels of environmental phenols and age of menarche.6 These data support the suggestion by Biro et al that the age span for the entire spectrum of puberty has declined, although age of thelarche and menarche appear less linked than before.
A considerable literature supports the obesity epidemic as an important factor in the decline. This article found BMI accounted for 14.2% of the variance of all covariates in their model. Although obesity is implicated as a “prime driver,” factors involved in these secular changes are far more complex. Extensive interacting variables are known to be associated with earlier development in addition to weight and genetics: certain intrauterine conditions and exposures, preschool high-meat diets, dairy products, low fiber intake, isoflavones, high-stress families, absent fathers, certain endocrine disruptors, the microbiome as it influences weight, epigenetics, light exposure, hormone-laced hair products, insulin resistance, activity level, geographical location, and others. Animal husbandry has known for decades that growth and/or the age of puberty can be manipulated by altering nutrition, light exposure, exogenous hormones, and activity. Each individual girl is exposed to multiple factors in today’s environment, many not present decades ago, that may potentially influence her pubertal onset. Given that the exact trigger for pubertal initiation is still unknown, we hardly can fully understand the interactions of factors known to affect puberty, even with current sophisticated statistical modeling. We may have to live with uncertainty for a long time. Furthermore, because early puberty and menarche are associated with many detrimental health and psychosocial issues, we must not accept this premature development as the “new normal.” Data may appropriately alter clinical decisions; for example, when invasive procedures are required for evaluating a particular early developer, but they should not alter forward movement on creating a healthier environment for our children.

Companies now market “smaller than regular (menstrual) pads” with sparkling designs for elementary school girls and a genre of deodorants for children aged 8 to 10 with “younger, sensitive skin.” With each new study in the past 2 decades, we hope the age of “early puberty” has bottomed out. When each “new study” has been published, however, we find the trend toward early puberty has continued. Fortunately, we have moved beyond controversy about the data and are responding to the wake-up call.

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*Pediatrics* 2013;132;1125
DOI: 10.1542/peds.2013-3058 originally published online November 4, 2013;

The online version of this article, along with updated information and services, is located on the World Wide Web at:

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