HHS Public Access

Author manuscript

Pediatrics. Author manuscript; available in PMC 2015 June 09.

Published in final edited form as:

Pediatrics. 2010 September ; 126(3): e583–e590. doi:10.1542/peds.2009-3079.

Pubertal Assessment Method and Baseline Characteristics in a Mixed Longitudinal Study of Girls

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Abstract

OBJECTIVES—The objective of this study was to describe the assessment methods and maturation status for a multisite cohort of girls at baseline recruitment and at ages 7 and 8 years.

METHODS—The method for pubertal maturation staging was developed collaboratively across 3 sites. Girls at ages 6 to 8 years were recruited at 3 sites: East Harlem, New York; greater Cincinnati metropolitan area; and San Francisco Bay area, California. Baseline characteristics were obtained through interviews with caregivers and anthropometric measurements by trained examiners; breast stage 2 was defined as onset of pubertal maturation. The κ statistic was used to evaluate agreement between master trainers and examiners. Logistic regression models were used to identify factors that are associated with pubertal maturation and linear regression models to examine factors that are associated with height velocity.

RESULTS—The baseline cohort included 1239 girls. The proportion of girls who had attained breast stage 2 varied by age, race/ethnicity, BMI percentile, and site. At 7 years, 10.4% of white, 23.4% of black non-Hispanic, and 14.9% of Hispanic girls had attained breast stage ≥2; at 8 years, 18.3%, 42.9%, and 30.9%, respectively, had attained breast stage ≥2. The prime determinant of height velocity was pubertal status.

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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

The views in this article are those of the authors and do not necessarily represent the views of the National Institute of Environmental Health Sciences, the National Cancer Institute, the National Center for Research Resources, the National Institutes of Health, the Environmental Protection Agency, or the Agency for Toxic Substances and Disease Registry.

CONCLUSIONS—In this multisite study, there was substantial agreement regarding pubertal staging between examiners across sites. The proportion of girls who had breast development at ages 7 and 8 years, particularly among white girls, is greater than that reported from studies of girls who were born 10 to 30 years earlier.

Keywords

puberty; girls; maturation assessment

Determinants of the timing of puberty are not entirely established. Although body fatness is associated with onset of puberty in girls,[1–6] genetics and environment are also thought to play roles in pubertal onset. For better understanding of the role of genetic and environmental influences on breast cancer, acting through the risk factor of early maturation, the Breast Cancer and the Environment Research Centers (BCERC) were established in late 2003 as a consortium in a partnership between the National Institute of Environmental Health Science (NIEHS) and National Cancer Institute (NCI). The focus in the BCERC epidemiologic studies is directed toward prepubertal and early pubertal stages, in recognition of puberty as a potential window of susceptibility for breast cancer. Several epidemiologic studies have observed that women with breast cancer have a younger age of menarche, and those with younger age at menarche have an increased risk for breast cancer.[7–10] A pooled analysis of studies of premenopausal and postmenopausal women revealed that the risk for breast cancer was decreased by 9% and 4%, respectively, for each year that menarche was delayed.[11] There are several possible reasons that later menarche reduced risk for breast cancer, including the relationship between onset of puberty and lifelong exposure to estrogen[12,13] and progesterone,[14] number of proliferating cells in the intralobular terminal ducts,[15] and susceptibility of rapidly developing breast tissue to environmental exposures.[16–18]

The principal end point in the BCERC epidemiology studies is the age when breast development begins. Although there is recent interest in studying causative factors in pubertal onset, few epidemiologic studies have been conducted of general populations. Some of the methodologic limitations include reliability and standardization across multiple sites and obtaining data on risk factors prospectively. Reynolds and Wines[19] proposed a procedure to define pubertal maturation stages that included pubic hair maturation as well as 3 scales for breast maturation. Tanner revised these criteria and published his classic monograph *Growth at Adolescence* regarding pubertal maturation.[20] He proposed using 5 stages of breast development, recognized by many as “Tanner breast stages,” and 5 stages of pubic hair development. A longitudinal study published subsequently by Marshall and Tanner[21] provided descriptive detail of these stages in a group of girls. Independent of Marshall and Tanner’s work was a series by van Wieringen et al,[22] that used high-quality photographs of pubic hair and breast maturation stages to standardize outcome assessment. Other breast/areolar maturation systems have been proposed, such as Garn and Falkner’s[23] areolar maturation system, which was independent of breast size or degree of obesity.

In the BCERC consortium, breast development is assessed at each of 3 sites by trained clinical or research staff on the basis of Tanner’s criteria, with cross-site training and quality

measures, to optimize validity and comparability. In this article, we describe the methods for pubertal assessment and report baseline characteristics of the consortium cohort. The objectives of this article are (1) to describe the methods and training of personnel for determining pubertal stages and (2) to report the maturation status of the cohort of girls at ages 7 and 8 years.

METHODS

Data Collection

This project was conducted as part of the NIEHS/NCI BCERC, 4 centers with transdisciplinary research collaborations across biological and epidemiologic research projects and with community members and advocates fully integrated into all study aspects. The observational longitudinal study of pubertal maturation is conducted at 3 sites: (1) Mount Sinai School of Medicine (MSSM)/Fox Chase Cancer Center/University of Alabama Birmingham group, which recruited through clinics, schools, and neighborhood centers in East Harlem, New York, with girls seen annually; (2) Cincinnati Children's Hospital/ University of Cincinnati (Cincinnati), which recruited through public and parochial schools in the Cincinnati metropolitan area and through the Breast Cancer Registry of Greater Cincinnati, with girls seen semiannually; and (3) the Kaiser Permanente Northern California (KPNC)/University of California San Francisco group, which recruited KPNC Health Plan members in the San Francisco Bay Area, with girls seen annually. Enrollment occurred between the years 2004 and 2006, and the girls were 6 to 8 years of age at recruitment. Parents or guardians of the participants identified the girls as black, white, Asian, or other race and ethnicity as Hispanic or non-Hispanic. Girls were assigned race/ethnicity by using the criteria that black race superseded other race/ethnicity designations and Hispanic superseded all others; all participants were uniquely defined with these criteria.

Before recruitment began, the investigators and study coordinators from each center met to prepare a written protocol for the clinical assessment method and to finalize a training manual and staging forms. Pubertal maturation was assessed by using Marshall and Tanner criteria for breast maturation and pubic hair stages,[21] with photographs that demonstrated the maturation stages, published by van Wieringen et al.[22] The written protocol instructed examiners to inspect and palpate for the presence of breast tissue and to use accessory light sources for pubic hair, if necessary. During training, the investigators and coordinators independently reviewed sets of photographs ("unknown stage") of anonymous girls at various stages of development, and complete agreement was established between the examiners and 3 authors (Drs Biro, Galvez, and Greenspan) by examining initial disparities and establishing unanimity. Clinical staff were certified after completion of training and photographs and then performing dual examinations with an expert on a group of peripubertal girls.

The 3 clinical centers conducted the maturation assessments in different settings, as noted already. Evaluations are performed by physicians and nurse practitioners at 2 sites (MSSM and Cincinnati) and research staff members at KPNC. At each center, training was performed by the local expert investigators (Dr Galvez at MSSM, Dr Biro at Cincinnati, and Dr Greenspan at KPNC). To maintain comparability between the centers, the same

examination form is used, and 1 of the local experts (Dr Galvez) visited each clinical site to perform interrater assessments for quality assurance. Examiners circled the appropriate stage on a standard form that illustrated and described each stage; half-stages were not permitted, and, therefore, examiners graded down unless all criteria were met for the next stage. As noted already, participants were seen annually for follow-up assessments at MSSM and KPNC; at Cincinnati, participants were seen semiannually. For the 7- and 8-year age cohort analyses reported here, data from the first visit at a given age at all sites were used for the analysis of that age cohort. Age was defined as calendar age (age 7 as any age from 7.00 to 7.99).

During the examination visits, trained staff members obtained standardized anthropometric measurements, including height and weight, making at least 2 measurements of each parameter, unless the difference exceeded a preset amount or the amount was outside the 5th or 95th percentile values; in these situations, a third measurement was taken and an average of the closest 2 values was taken. BMI was calculated from the mean values of height and weight, weight divided by height squared. BMI *z* score was determined from the Centers for Disease Control and Prevention (www.cdc.gov/growthcharts/zscore.htm). Height velocity was calculated from the difference of mean heights obtained from consecutive visits, divided by time between visits, and adjusted to a 12- month interval.

Statistical Analyses

For these analyses, we collapsed breast stage 2 and breast stage 3 to contrast girls who were prepubertal (breast stage 1) from those who had breast development (breast stage ≥2), and contrasted pubic hair stage 1 from those with pubic hair stage ≥2. Maturation status was described at age 7 and 8 years, as described already. A given participant could have contributed to analyses at both ages 7 and 8 for age-specific maturation status.

Logistic regression models were used to examine factors that are potentially related to breast stage (1 vs ≥2) as the outcome. The initial models included BMI percentile, race, age, and site, as well as all interaction terms of main effects with site. A final logistic regression model, derived from backward elimination, included only variables that were significant at a probability level of .05. Linear regression was used to estimate the strength of the relationship between height velocity and potential predictors, including age, race, breast stage, site, and all sites by main effect interactions. A backward elimination procedure was used to derive a final model with only significant main effects and site interactions.

To assess interrater agreement for pubertal staging, 1 of the experts (Dr Galvez) visited each clinical site to perform maturation assessment in tandem with clinical staff. The degree of agreement between the master trainers and the research staff members who were conducting the examinations was measured in 127 dual examinations by using the κ statistic, which evaluates observed agreement contrasted to agreement that is attributable chance.

RESULTS

The baseline cohort included 1239 girls; across the 3 sites, 8 girls did not have baseline breast stage information. The racial and ethnic composition of the study varied by clinical

center, as noted in Table 1. At baseline, 33.7% of the girls were identified by the parents/guardians as white non-Hispanic, 31.6% as black non-Hispanic, 29.9% as Hispanic, and 4.6% as Asian. Mean ages of the cohort at recruitment, by site, were 7.34 years (MSSM cohort), 7.13 years (Cincinnati cohort; 7.63 for the second visit), and 7.38 years (KPNC cohort).

Pubertal breast maturation at 7 and 8 years of age, regardless of examination cycle, is reported in Table 2. At 7 years, breast maturation was at stage ≥2 in 10.4% of white non-Hispanic participants, 23.4% of black participants, and 14.9% of Hispanic participants; at 8 years, breast maturation was at stage ≥2 in 18.3%, 42.9%, and 30.9%, respectively. In analyses that used logistic regression with pubertal status at age 7 as the dependent variable, breast stage ≥2 was associated with higher BMI percentile, older age, black race, and being from the MSSM or Cincinnati site. Site interactions were not significant and did not remain in any of these models. Pubic hair maturation results are shown in Table 3. When analyses that used pubic hair status at age 7 as the dependent variable were conducted, significant associations were seen with higher BMI percentile and black race. In a linear regression model, the strongest predictor for height velocity was pubertal status (breast development; $P < .0039$); the only other statistically significant parameter was Asian race (lower height velocity when compared with other groups; $P = .017$).

The proportion of white participants in the BCERC consortium who had attained breast stage ≥2 at 7 years was significantly greater than that reported by Herman-Giddens in the Pediatric Research in Office Settings (PROS)[24]: for white girls, 10.4% vs 5.0% ($z= 3.72$, $P < .001$), and for black non-Hispanic girls, 23.4% vs 15.4% ($z = 1.76$, $P = .09$). For participants at age 8, the proportion of girls in the BCERC who were at breast stage ≥2 among white participants was 17.9% contrasted to 10.5% from PROS ($z = 3.77$, $P < .0002$) and among black BCERC participants was 37.0%, contrasted to 36.6% (differences not significant) in PROS.

When the dual maturation assessments were combined across all 3 sites, 127 dual examinations resulted in 17 inconsistencies between the master trainers and the research staff; the estimated κ statistic was 0.67 (with 87% agreement observed between examiner and master trainers), indicating “substantial” agreement.[25] When restricted to girls at breast stage 1 or 2 ($n = 117$), the κ statistic was 0.68, indicating substantial agreement. The values of the κ statistic were similar when girls above and below the 85th percentile for BMI were compared.

DISCUSSION

Variations in the timing of pubertal maturation may be sensitive “sensors” of the effects of environmental exposure in human populations. An expert panel that discussed secular trends in pubertal maturation noted that recent data suggested a trend toward earlier breast development onset.[26] They recommended longitudinal studies with regular observations; maturation assessment by using Tanner staging with breast palpation by trained examiners; geographic variation; broad socioeconomic representation; defined race/ethnicity; and body fat and weight covariates, such as BMI.[26] These components have been incorporated into

the epidemiologic projects of the BCERC. Inspection and breast palpation are the only practical methods to assess secondary sex characteristics in an epidemiologic research setting; therefore, the accuracy of pubertal stage assignment will be influenced by interrater reliability. In this longitudinal investigation, a standardized protocol for pubertal staging and recertification was developed to minimize measurement error between sites and examiners. The κ statistic of 0.67 indicates that there is "substantial" agreement between the master examiners and the research staff.[25] This value is consistent with other published studies that investigated agreement between examiners: 0.50 (n = 25 comparisons),[27] 0.78 (n = 20 comparisons), [28] and 0.86 (n = 56 comparisons). [24] Although our study consisted of a greater number of dual comparisons (n = 127) than the other studies, we had a limited number of inconsistent dual observations (17 of 127) to study the impact of factors such as BMI and race on lack of agreement between raters. We did examine those examinations included in this article, as well as an additional 40 dual examinations, and found no difference in the κ statistic between participants whose BMI was above and below at the 85th percentile.

There were important differences among the 3 clinical sites by racial and ethnic characteristics of the participants. Indeed, there were significant differences at baseline by site in the proportion of participants who had entered puberty. These differences resolved when examined by single-year chronological age groups, rather than all baseline participants, in the adjusted models. In addition, the analyses of height velocity, a biological change that is associated with pubertal maturation, provided a physiologic validation of maturation assessment, in addition to the methodologic validation provided by the κ statistic. Site differences could result from differential exposure to endocrine disruptors or from dietary differences (eg, differences in fiber intake, because dietary fiber has been noted to affect timing of pubertal maturation).[29,30] These issues will be examined in future analyses.

The proportion of white participants in the BCERC consortium who had breast development at ages 7 and 8 years was greater than that reported from studies of girls who were born 10 to 30 years earlier, such as the PROS study.[24] Earlier onset of breast development was noted in a recent report from the Copenhagen Puberty Study.[31] When they examined differences in age at onset of puberty contrasting 2 groups who were born 15 years apart, they noted that the differences in age of onset remained significant after adjusting for BMI.[31] Several studies and reviews have addressed the impact of timing of pubertal maturation. Earlier maturation in girls is associated with lower self-esteem and less favorable body image,[32,33] as well as greater rates of eating problems, [34] depression,[35] and suicide attempts. [36,37] They were more likely to be influenced by deviant peers,[38] with earlier onset of sexual intercourse[39] and norm-breaking behaviors,[40,41] although this may be mediated through greater involvement with and influence by deviant peers.[39] Health risks included greater risk for breast cancer[7,42] and endometrial cancer.[43,44] Although girls who matured earlier had a greater BMI in adulthood, most of the apparent effect of early maturation on obesity is attributable to the association of childhood obesity on both earlier menarcheal age and adult obesity.[4] Earlier maturation is also associated with hyperinsulinemia and elevated blood pressure.[45] A recent review commented that some of the adverse outcomes that are

associated with altered timing of puberty may have been attributable to exposure to potential endocrine-disrupting chemicals that affect pubertal timing as well as physiologic or metabolic processes.[46]

Although maturation data presented in this article were cross-sectional, longitudinal analyses of our cohort will allow the determination of the mean age at onset of breast development, which cannot be determined at this time because many have not yet attained breast development. The longitudinal design and accompanying potential for greater statistical power will allow the BCERC researchers to investigate individual changes as well as temporal relationships for earlier exposures. Future analyses within the BCERC consortium, by using the wealth of anthropometric, lifestyle, psychological, family history, genetic, and chemical exposure data that are being collected, may help to define more precisely the factors that are associated with onset of puberty. Similar to previous studies, we found that age, race/ethnicity, and BMI were associated with age at onset of puberty.[5,47] The variability in timing, as noted by Parent et al,[48] involves genetic factors, ethnicity, nutritional conditions, and secular trends. Longitudinal analyses from the Bogalusa Heart Study noted a decrease in age of menarche in black and white girls, with the greater decrease in age in black girls suggesting a combination of environmental and race factors.[49] Several studies have examined genetic influences on timing of pubertal events. Treloar and Martin[50] noted that the correlation in age at menarche was greater in monozygotic than in dizygotic twins; genetic variance was nonadditive, typical of a fitness trait yielding a genetic advantage.[51]

There are several potential weaknesses of this study. Although families were recruited at 3 distinctly different areas of the United States, with broad racial/ethnic and socioeconomic diversity, this is not a nationally representative sample. In addition, recruitment levels were relatively low, which could yield recruitment biases. As noted previously, there were baseline differences in maturation by site, suggesting different dietary patterns, chemical exposures, race/ethnic differences, or interactions between these areas. Subsequent analyses that use additional longitudinal observations and additional exposure and dietary information may help to define better these differences.

CONCLUSIONS

Initial observations indicate consistency of assessment of pubertal maturation across the 3 BCERC sites. The ability to capture with reasonable accuracy the timing and tempo of pubertal breast maturation in this prospective study, therefore, should allow us to pool data for detecting associations between specific factors, including diet and environmental chemicals, with variations in patterns of pubertal maturation. The findings of a higher prevalence of onset of breast development among girls at ages 7 and 8 years, especially in white participants, compared with those observed more than a decade earlier by Herman-Giddens et al[24] but similar to a contemporary group of European girls[31] highlights the importance of identifying such factors.

Acknowledgments

This study was supported by grants ES/CA12770, 012771, 012800, and 012801 from the NIEHS and the NCI. MSSM acknowledges support from NIEHS (ES009584 and ES012645), US Environmental Protection Agency

(R827039 and RD831711), Agency for Toxic Substances and Disease Registry (ATU 300014), NCI (CA93447), and National Center for Research Resources (NCRR; MO1-RR-00071). Cincinnati acknowledges grant support from P30-ES06096.

This study was conducted within the BCERC, a network of centers that include the Fox Chase Cancer Center, Michigan State University, the University of Cincinnati, and the University of California San Francisco Comprehensive Cancer Center.

We thank the study investigators and staff at the 3 medical centers involved in this research including Julie Britton, Eunpa Chae, Ana Mejia, Arkeyris Richiez, Jessica Montana, Rochelle Osborne, Lisa Boguski, Joel Forman, and Barbara Brenner (MSSM); Lorah Dorn, Bin Huang, Cendi Dahl, Chrissy Baker, Shannon Myatt, Kanti Ford, Anuradha Josyula, Erin Baker, Rob Herrick, Heather Foote, Bob Bornschein, Katie Brown, Kim Dietrich, Lusine Yaghjyan, Ann Hernick, Kathy Ball, and the Study Helpers (Cincinnati); and Robert Hiatt, Gayle Windham, Barbara Sternfeld, Kathy Koblick, Christine Ashley, Christine Bonnell, Anoushe Mirabedi, Isaac Joshua Ergas, Patricia Atkinson, Angela Beeck, Marissa Brownell, Coralie Chan, Deneshia Davis, Evelyn Landaverde, Gloria Roman, Gina Saelee, Shanita Burleson, Donneva Johnson, and Michaelynn Trotter (KPNC).

ABBREVIATIONS

BCERC	Breast Cancer and the Environment Research Centers
NIEHS	National Institute of Environmental Health Science
NCI	National Cancer Institute
MSSM	Mount Sinai School of Medicine
KPNC	Kaiser Permanente Northern California
PROS	Pediatric Research in Office Settings

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WHAT'S KNOWN ON THIS SUBJECT

Age of onset of puberty in girls has fallen in the past 2 decades. It is unclear whether this trend is continuing or the age of onset of puberty in girls has stabilized.

WHAT THIS STUDY ADDS

The authors describe the method and comparability of maturation assessment across 3 geographically distinct centers. It seems that age at onset of puberty is continuing to fall in white but not black girls. Black girls continue to mature at younger ages than white girls.

TABLE 1

Site and Race/Ethnicity in the BCERC Cohort

Race/Ethnicity	MSSM	Cincinnati	KPNC	Overall
White non-Hispanic		231	187	418
Black non-Hispanic	168	127	96	391
Latina Hispanic	248	15	107	370
Asian		5	52	57
Other		1	2	3
Total	416	379	444	1239

TABLE 2

Breast Maturation Status at Ages 7 and 8, by Site and Race/Ethnicity, in the BCERC Cohort

Parameter	MSSM			Cincinnati			KPNC			Overall
	B1	B2+, *n* (%)	Total	B1	B2+, *n* (%)	Total	B1	B2+, *n* (%)	Total	B2+, *n*/*N* (%)
7-y-olds										
White				184	29(13.6)	213	179	13(6.8)	192	42/405(10.4)
Black	83	11(11.7)	94	75	34(31.3)	109	75	26(25.7)	101	71/304(23.4)
Hispanic	117	25(17.6)	142	10	1(9.1)	11	79	10(11.2)	89	36/242(14.9)
Asian				4	0(0.0)	4	40	1(2.4)	41	1/45(2.2)
Other				1	0(0.0)	1	8	0(0.0)	8	0/9(0.0)
Total	200	36(15.3)	236	274	64(18.9)	338	381	50(11.6)	431	150/1005(14.9)
8-y-olds										
White				156	57(26.7)	213	152	12(7.3)	164	69/377(18.3)
Black	77	31(28.7)	108	54	58(51.8)	112	55	24(30.4)	79	82/191(42.9)
Hispanic	97	60(38.2)	157	8	4(33.3)	12	78	18(18.8)	96	82/265(30.9)
Asian				4	0(0.0)	4	34	6(15.0)	40	6/44(13.6)
Other				1	0(0.0)	1	7	0(0.0)	7	0/8(0.0)
Total	174	91(34.3)	265	223	119(34.8)	342	326	60(15.5)	386	270/993(27.2)

B1 indicates breast stage 1 (no evidence of breast maturation); B2+, breast stage 2 or 3 for 7-year-olds and 2, 3, or 4 for 8-year-olds.

TABLE 3

Pubic Hair Maturation Status at Ages 7 and 8, by Site and Race/Ethnicity, in the BCERC Cohort

Parameter	MSSM			Cincinnati			KPNC			Overall
	PH1	PH2+, *n* (%)	Total	PH1	PH2+, *n* (%)	Total	PH1	PH2+, *n* (%)	Total	PH2+, *n*/*N* (%)
7-y-olds										
White				201	12(5.6)	213	136	11(7.5)	147	23/360(6.4)
Black	77	17(18.1)	94	88	21(19.3)	109	59	18(23.4)	77	56/280(20.0)
Hispanic	131	11(7.8)	142	10	1(9.1)	11	82	4(4.7)	86	16/239(6.7)
Asian				4	0(0.0)	4	38	1(2.6)	39	1/43(2.3)
Other				1	0(0.0)	1	8	0(0.0)	8	0/9(0.0)
Total	208	28(13.5)	236	304	34(10.1)	3398	323	34(9.5)	357	96/931(10.3)
8-y-olds										
White				192	21(9.9)	213	111	13(10.5)	124	34/337(10.1)
Black	69	38(35.5)	107	81	31(27.7)	112	45	21(31.8)	66	90/285(31.6)
Hispanic	119	38(24.2)	157	9	3(25.0)	12	65	4(5.8)	69	45/238(18.9)
Asian				4	0(0)	3	35	4(10.3)	39	4/43(9.3)
Other				0	1(100)	1	6	0(0.0)	6	1/7(14.3)
Total	188	76(28.8)	264	286	56(16.4)	342	262	42(13.8)	304	174/910(19.1)

PH1 indicates pubic stage 1 (no evidence of pubic hair maturation); PH2+, pubic stage 2, 3, or 4.