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Sex-based prevalence of growth faltering in an urban pediatric population

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Abstract

Objective—To determine the sex-based prevalence of growth faltering in a pediatric primary care setting.

Study design—33,476 children attending four urban pediatric primary care practices affiliated with a tertiary pediatric hospital from July 2002 to June 2005 were studied. Growth faltering was defined as height: <5th percentile or z-score dropping by 1.5 standard deviations (SD) before age 18 months or 1 SD thereafter. Growth faltering and non-faltering groups were compared by sex, race, age, number of clinic visits, insurance and by US census tract, socioeconomic status and parental education. Similar comparisons were made for children with height z-scores below –2.25 SD.

Results—Growth faltering was present in 3,007 (9%) children. Univariate and multivariate logistic regression analyses revealed growth faltering to significantly associate with younger age ($P<0.0001$), white race ($P<0.0001$), fewer clinic visits ($P<0.0001$) and Medicaid insurance ($P<0.005$), but not sex nor within residential census tract, median income or proportion with less than high school education. Height below –2.25 SD associated with male sex ($P<0.01$), Medicaid insurance ($P<0.01$) and more primary care visits ($P<0.0005$).

Conclusion—Sex disparity in subspecialty growth center referrals (2:1 male:female) was not due to male predominance in growth faltering among children in the urban primary care setting.

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Keywords

height; z-score; Medicaid; census; race; ascertainment bias

In a retrospective review of all children referred to a subspecialty growth center in 2001 for the evaluation of poor growth, we found that boys outnumbered girls 182 to 96.¹ The referred boys also had a smaller height deficit, relative to both the general population and their midparental target heights. To further our understanding, we examined the demographics of children with growth faltering among urban pediatric primary care practices in our pediatric healthcare network to determine whether the aforementioned disparities reflect the prevalence of growth problems in the primary care setting.

METHODS

Subject selection and height data

Records of all children aged 0.5 to 20 years attending four urban pediatric primary care practices affiliated with a tertiary pediatric hospital in Philadelphia were retrospectively reviewed. 39,420 children attending these practices had at least one recorded height measurement from July 2002 to June 2005. Approval of the Institutional Review Board of the Children's Hospital of Philadelphia was obtained prior to commencing this study.

Patient heights and lengths were measured in all clinics by nurses or medical assistants using wall-mounted and recumbent stadiometers, as age appropriate. Heights and lengths were converted to age- and sex-specific percentiles and z-scores using the methods defined by the National Center for Health Statistics. The growth percentile data files published by the Centers for Disease Control and Prevention (CDC) in May 2000 were downloaded,^{2,3} and the LMS calculation was implemented in ActiveState Perl (www.activestate.com). To maximize precision, patient age in days was calculated and linear interpolation was used to determine the L, M and S values from the data tables.⁴ This improved the accuracy in z-score calculation for children under 12 months of age.

Because we aimed to understand referral practices, we used growth cut-offs that are often used by primary care pediatricians to signal a problem. Growth faltering was defined as: 1) height <5th percentile ("below the curves" on the growth charts used most often in our network) or 2) fall in height z-score. To reduce the impact of physiologic rechanneling on this study, the z-score drop was required to exceed 1.5 standard deviations (SD) before age 18 months or 1 SD thereafter. To improve validity and reliability, growth data were excluded for all children before age 6 months and for low-birth-weight or premature infants before age 2 years. Outlier points, defined as height z-score beyond -5 or +3 SD, were excluded as biologically implausible values, according to World Health Organization and CDC recommendations.^{5,6}

Two independent checks of data quality were performed. First, growth charts containing multiple episodes of short stature with intervening point(s) of normal stature were manually reviewed by a pediatric endocrinologist (A.G.); based on how the data point fit within the overall growth pattern, the endocrinologist verified whether or not the patient met the

inclusion criteria for growth faltering. Second, due to the complexity of electronic health record data transformation for this project, probabilistic samples of patient data sets were reviewed to ensure there were no errors introduced by the data extraction process. After these exclusions, 33,476 children constituted the patient population for analysis.

Similar analyses were performed for the subgroup of children with height z-scores below -2.25 SD, the FDA-approved threshold for growth hormone (rhGH) treatment of idiopathic short stature (ISS).⁷

Covariates

Patient data, including sex, race, ethnicity, insurance provider and primary care clinic attendance, were extracted from the electronic health records. Age was calculated from dates of birth and dates of the first visit within the observation window that either met inclusion criteria for growth faltering or if none qualified, then the first visit under observation. In our health care system, race and ethnicity were determined by parental self-report, which were then assigned to categories based on the recommendations of the National Institutes of Health and US Census Bureau⁸ and reviewed with the parents for accuracy. Insurance providers at the first qualifying visit were recorded for 20,527 of the patients and were categorized into private, Medicaid or self-pay for analyses.

To obtain surrogate markers of family socioeconomic status, each patient residing in Philadelphia County was assigned to a residential census tract using ArcView 9.1 (ESRI, Inc., Redlands, CA). All non-matching addresses were manually reviewed for spelling or formatting and corrected; a total of 28,656 matches were made. Socioeconomic indicators for each census tract were based on 2000 US Census data.⁹ Each patient was assigned the median income, percent employment over age 16 years, and percent of females and males of their respective census tracts with an education level less than high school completion.

For each census tract, the ratio of patients with growth faltering to the total number of patients seen in the primary care network was calculated. To enhance data reliability, analyses were limited to census tracts containing at least 10 patients. The ratios were transformed into quartiles and mapped. The locations of all pediatric and family practices providing primary care to children in Philadelphia County (KIDS Registry, Philadelphia Department of Public Health, Division of Disease Control Immunization Program), as well as the four pediatric practices that served our study population, were geocoded to evaluate whether proximity to a health care provider was associated with growth faltering.

Statistical analyses

Descriptive statistics were calculated using JMP Software (SAS Institute, Inc.; Cary, NC) and presented as mean $\pm$ standard deviation (SD). Differences between means of continuous variables, like height z-score, age and number of encounters, were compared by two-sided t-test, and between frequencies of categorical variables (i.e. sex, race, ethnicity and payor) by Pearson Chi Square. The presence of growth faltering was modeled against potential contributing variables; logistic regression analyses with Effect Wald tests were performed to assess univariate associations, and GenMod procedure (SAS Institute, Inc.) was used for multivariate analyses. Model results are presented as odds ratio with 95% confidence

intervals (CI) and Chi Square P value. Median income was transformed into strata of \$10,000 to allow comparison of clinically significant differences in the models. Sex differences among children with and without growth faltering were compared by the Mixed Procedure (SAS Institute, Inc) for continuous variables, with results presented as Least Squares Means $\pm$ SD and P values including Bonferroni's adjustment for multiple testing. Categorical variables were compared by logistic regression analyses. If the Breslow-Day Test for Homogeneity of the Odds Ratios showed no effect modification, Cochran-Mantel-Haenszel Test scores were provided for the P value. For variables with more than two levels, Mantel-Haenszel Chi Square P values were provided for cases and controls separately.

RESULTS

Growth faltering occurred in 3,007 (9%) children. Mean height z-score was -1.5 ± 1.0 SD vs 0.3 ± 0.9 SD in patients without faltering ($P < 0.0001$), and their characteristics are compared in Table I. Males comprised 53% of the growth faltering children and 51% of the non-faltering group ($P < 0.01$). Mean height z-scores of the boys were slightly lower than that of the girls in both the faltering and non-faltering groups (Table II). Characteristics of the two groups by sex are further compared in Table II.

Figure 1 shows the age distribution of children in the study population (panel A). The proportion of children with growth faltering showed a bimodal distribution, peaking in those under age 3 years and again in the second decade (panel B). The frequency of growth faltering in the early years lowered the median age of the growth-faltering group to 2 years, compared with the non-faltering median of 6 years ($P < 0.0001$). Males were especially prone to growth faltering in the younger years, such that the mean age of growth-faltering boys was lower than growth-faltering girls (Table II).

The study population was predominantly African American (87%). The remainder was comprised of white (7%), self-reported other (5%), Asian (1%) and American Indian/Eskimo/Alaska native (0.1%) children. The racial distribution of children with growth faltering differed significantly from those without (Table I), but not by sex (Table II). One percent of patients were self-identified as Hispanic or Latino, and their proportion did not differ significantly by growth pattern (Table I) or sex (Table II).

Most children were insured by Medicaid (70% over all). Children with growth faltering were more likely to have Medicaid than children without a growth problem (Table I). Among the children with growth faltering, the proportion of boys with Medicaid exceeded that of girls (Table II). There was no sex difference in the non-faltering group (Table II).

The average number of pediatric primary care encounters was 0.5 fewer for children with growth faltering than those without ($P < 0.0001$) (Tables I and II). Boys were more likely than girls to see a male pediatrician, regardless of growth pattern (Table II). Patients and primary care practices were geocoded to evaluate whether distance to a healthcare office may contribute to the fewer visits by the growth faltering children. As seen in Figure 2 (available at www.jpeds.com), geography did not create high risk areas of growth faltering;

there were equally high prevalences of growth faltering in areas near and far from a pediatric provider.

Modeling growth faltering against the variables explored (Table I) revealed significant associations with age ($P<0.0001$), race ($P<0.0001$), number of pediatrician visits ($P<0.0001$) and insurance category ($P<0.005$), but not sex ($P=0.36$) or ethnicity ($P=0.43$). Markers of socioeconomic status, including median income, employment over the age of 16 years, and education level, did not correlate with growth faltering (Tables I and Table II). This was a predominantly low-income urban population, whose median household income was \$24,600 with 25th and 75th quartiles of \$20,600 and \$28,800, respectively. The children lived in census tracts with a median 29% of inhabitants below the federal poverty line (25th and 75th quartiles, 21% and 35%).

Additional analyses showed that 534 children (1.6%) had height z-scores below -2.25 SD, the FDA-approved threshold for the ISS indication for rhGH therapy.⁷ Modeling this <-2.25 SD group against the same variables (Table III) revealed significant associations with male sex ($P<0.01$), Medicaid insurance ($P<0.01$) and greater number of pediatric primary care encounters ($P<0.0005$). This group had 57% males, 77% Medicaid coverage and an average 0.4 primary care visits more than all other children.

DISCUSSION

We found that growth faltering among children in urban pediatric primary care practices was not related to sex, but was related to age, race, medical insurance and clinic visits. This is in striking contrast to the male predominance of endocrine subspecialist growth evaluations and pediatric rhGH therapy.

The average number of pediatric primary care encounters was 0.5 fewer for children with growth faltering than those without, and was the most powerful factor in modeling growth faltering. This was statistically and clinically significant, given that our observation spanned three years and the recommended frequency for well-child care is annual beyond age 3 years.¹⁰ Further, this population had good access to healthcare, attending primary care practices that are part of a major children's hospital network.

Race was the second most powerful predictor of growth faltering. This is consistent with past US reports. African American children, with and without correction for socioeconomic status, were found to be taller than white children from infancy through adolescence,^{11,12} have more advanced skeletal development and dentition,¹¹ and enter puberty at younger ages,^{13,14} but not demonstrate a greater variability in height than white children.¹⁵

The third most informative factor was insurance. Medicaid is often a surrogate marker for lower socioeconomic status and access to healthcare. Smaller stature was linked to low per capita income, and growth stunting in children living below the poverty line was found across races and ethnicities.^{11,12,16–18} Height was further related to father's employment status, and sex differences were detected in the relationships among socioeconomic measures and child height in a study of an Eastern Kentucky high-poverty community.¹⁸ The lack of association with socioeconomic factors in our study may have been due to the

socioeconomic homogeneity of our population, the reduced number of children with addresses matching the census, and the use of census tract-level factors. Although socioeconomic data at the individual patient-family level were desirable, such information was not available.

Age was the last variable to associate significantly with growth faltering. Prevalence of growth faltering peaked during the first 3 years of life, with a second rise during the second decade. The early peak corresponds to the age range when failure to thrive is fairly common.¹⁹ Physiologic rechanneling, the crossing of major percentiles when the factors that determined intrauterine growth get replaced by postnatal growth determinants, especially genetic height potential,²⁰ may also have contributed to the early peak. The impact of rechanneling was minimized by omitting data before age 6 months, excluding growth data for low-birth-weight or premature infants before age 2 years, and using a more stringent criterion for drop in z-score before the age of 18 months. Pubertal delay or absence is the principal mediator of growth faltering in adolescence, the time of the second peak, and constitutional delay of growth and puberty (CDGP) is the most common cause.²¹

The absence of a sex effect on growth faltering was unexpected, especially during the second decade. Many textbook discussions of CDGP relate it to male sex^{22–25} though at least one attributed it to ascertainment bias from increased social pressures on males.²⁶ Although fathers frequently report a similar pattern, analysis of 53 CDGP pedigrees showed the delay can be evident on the maternal side as well.²⁷ The contrast between the absence of male predominance in the prevalence of growth faltering among the adolescents in our primary care practices and the increase in sex disparities after the age of 9 years among children referred to our subspecialty growth center¹ supports the notion of an ascertainment bias. A sex-based referral bias for CDGP was recently documented in a national study in Finland, whose racial composition and healthcare system differ from ours.²⁸

Male predominance (64%) was seen among children with growth failure identified through a 1994 Utah elementary school-based study.²⁹ The growth failure group, comprising 0.7% of children screened, was defined as height <3rd percentile and growth rate <5 cm between the two annual measurements. This study used a more restrictive definition of growth failure, an older growth chart for reference, and a demographically different population (89% white, 75% urban, and living at an average altitude of 1311 m [4300 feet] above sea level). Further, the fixed growth velocity criterion did not account for the usually earlier pubertal onset among girls than boys, and the slight growth deceleration that frequently predates the pubertal increase, especially in late developers. In contrast, our study used fall in height z-score as the measure of abnormally slow growth, which corrects for sex differences in pubertal timing.

Generalizability of our results may be limited by the socioeconomic and racial composition of our population. By 2006, 72,387 children were enrolled in four US post-marketing surveillance studies of rhGH. Of the 69,834 children with racial data, African Americans comprised <5%, and 80% were white. Combining all pediatric indications including Turner Syndrome, which occurs in girls only, 65% of the US rhGH recipients were male. (Personal communication, Lippe B.M., Genentech, Inc.; Wajnrajch M.P., Pfizer, Inc.; Quigley C. and

Scott R., Eli Lilly and Company; and Germak J.A., Novo Nordisk, Inc.). Such sex and race disparities have been evident since the first US post-marketing surveillance report (1985–1987), wherein 71% were male, 88% white and 6% African American.³⁰ Follow up studies with non-urban pediatric primary care practices in our network are planned to examine the prevalence of growth faltering in this setting.

When our population was divided by a height z-score of -2.25 SD, the threshold for the ISS indication for rhGH therapy,⁷ we too found a male predominance (57%). This severely short group was more likely to be insured by Medicaid and had more pediatric primary care encounters, likely reflecting increased pediatrician concern and monitoring. The higher prevalence of severe short stature among males may explain the presence of a male predominance among rhGH recipients, but not its magnitude. Of all patients in a global rhGH post-marketing surveillance study, including girls with Turner Syndrome, US recipients were 64% male, close to Asia (mostly Japan, at 65%) but exceeding Europe/Australia/New Zealand (55% male).³¹ Nor can the sex-based prevalence of severe short stature explain the disparities in short stature evaluations in our subspecialty growth center; the referred girls had greater height deficits than the referred boys (median height z-score of -2.4 vs -1.9 SD, respectively).¹

Ascertainment bias can lead to missed or delayed diagnosis of underlying pathology. Among the children referred to our subspecialty growth center for evaluation of poor growth, girls were 2.7 times more likely than boys to have an identifiable underlying organic disease, extending beyond the indications for rhGH therapy.¹ Because growth faltering can be the first or only presenting sign of various diseases,¹⁹ pediatric care providers must strive to overcome ascertainment biases in order to prevent under diagnosis of health problems among children whose growth faltering may elicit less social pressure, such as females and low-income children.

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Acknowledgments available at www.jpeds.com.

ABBREVIATIONS

SD	standard deviation
CI	confidence interval

PCP	primary care pediatrician
rhGH	recombinant human growth hormone
ISS	idiopathic short stature
CDGP	constitutional delay of growth and puberty

REFERENCES

1. Grimberg A, Kutikov JK, Cucchiara A. Sex differences in patients referred for evaluation of poor growth. *J Pediatr*. 2005; 146:212–216. [PubMed: 15689911]
2. National Center for Health Statistics. CDC Growth Charts: United States. Percentile Data Files with LMS Values. Available from: <http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.html>
3. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, et al. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat* 11. 2002; 246:1–190. [PubMed: 12043359]
4. Cole TJ. The LMS method for constructing normalized growth standards. *Eur J Clin Nutr*. 1990; 44:45–60. [PubMed: 2354692]
5. World Health Organization. WHO technical report series: 854. Geneva, Switzerland: World Health Organization; 1995. Physical Status: The use and interpretation of anthropometry.
6. Centers for Disease Control and Prevention. Cut-offs to define outliers in the 2000 CDC Growth Charts. Available from: <http://www.cdc.gov/nccdphp/dnpg/growthcharts/00binaries/BIV-cutoffs.pdf>
7. U.S. Food and Drug Administration. [July 25, 2003] FDA Talk Paper: FDA Approves Humatrope for Short Stature. Available at: <http://www.fda.gov/bbs/topics/ANSWERS/2003/ANS01242.html>
8. US Department of Health and Human Services. NIH Policy and Guidelines on The Inclusion of Women and Minorities as Subjects in Clinical Research – Amended October 2001. Available from: http://grants.nih.gov/grants/funding/women_min/guidelines_amended_10_2001.htm
9. US Census Bureau. United States Census 2000. Available from: <http://www.census.gov/main/www/cen2000.html>
10. American Academy of Pediatrics Policy Statement - Committee on Practice and Ambulatory Medicine. Recommendations for Preventive Pediatric Health Care. *Pediatrics*. 2000; 105:645.
11. Garn SM, Clark DC. for the Ad Hoc Committee to Review the Ten-State Nutrition Survey. Nutrition, growth, development, and maturation: Findings from the Ten-State Nutrition Survey of 1968–1970. *Pediatrics*. 1975; 56:306–319. [PubMed: 169509]
12. Kerr GR, Lee ES, Lorimor RJ, Mueller WH, Lam MM. Height distributions of US children: Associations with race, poverty status and parental size. *Growth*. 1982; 46:135–149. [PubMed: 7173700]
13. Sun SS, Schubert CM, Chumlea WC, Roche AF, Kulin HE, Lee PA, Himes JH, Ryan AS. National estimates of the timing of sexual maturation and racial differences among US children. *Pediatrics*. 2002; 110:911–919. [PubMed: 12415029]
14. Anderson SE, Dallal GE, Must A. Relative weight and race influence average age at menarche: results from two nationally representative surveys of US girls studied 25 years apart. *Pediatrics*. 2003; 111:844–850. [PubMed: 12671122]
15. Godoy R, Goodman E, Levins R, Leonard WR. Anthropometric variability in the USA: 1971–2002. *Ann Hum Biol*. 2005; 32:469–486. [PubMed: 16147396]
16. Yip R, Scanlon K, Trowbridge F. Trends and patterns in height and weight status of low-income US children. *Crit Rev Food Sci Nutr*. 1993; 33:409–421. [PubMed: 8357504]
17. Wiecha JL, Casey VA. High prevalence of overweight and short stature among Head Start children in Massachusetts. *Public Health Rep*. 1994; 109:767–773. [PubMed: 7800786]
18. Crooks DL. Child growth and nutritional status in a high-poverty community in Eastern Kentucky. *Am J Phys Anthropol*. 1999; 109:129–142. [PubMed: 10342469]

19. Grimberg, A.; Lifshitz, F. Worrisome growth. In: Lifshitz, F., editor. *Pediatric Endocrinology*. 5th Ed.. Vol. Volume 2. New York, NY: Informa Healthcare USA, Inc.; 2007. p. 1-50.
20. Roche, A.; Sun, S. *Human growth: Assessment and interpretation*. Cambridge: Cambridge University Press; 2003. p. 106
21. Sedlmeyer IL, Palmert MR. Delayed puberty: analysis of a large case series from an academic center. *J Clin Endocrinol Metab*. 2002; 87:1613–1620. [PubMed: 11932291]
22. Blizzard, RM.; Johanson, A. Disorders of growth. In: Kappy, MS.; Blizzard, RM.; Migeon, CJ., editors. *Wilkins – The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence*. 4th ed.. Springfield, IL: Charles C. Thomas Publisher; 1994. p. 383-455.
23. Lifshitz, F.; Cervantes, CD. Short stature. In: Lifshitz, F., editor. *Pediatric Endocrinology*. 3rd ed.. New York, NY: Marcel Dekker, Inc.; 1996. p. 1-18.revised and expanded.
24. Plotnick, LP.; Long, DN. Puberty and gonadal disorders. In: McMillan, JA.; Feigin, RD.; DeAngelis, C.; Jones, MD., editors. *Oski's Pediatrics: Principles and Practice*. 4th ed.. Philadelphia, Pa: Lippincott Williams and Wilkins; 2006. p. 2079-2084.
25. Plotnick, LP.; Miller, RS. Growth, growth hormone, and pituitary disorders. In: McMillan, JA.; Feigin, RD.; DeAngelis, C.; Jones, MD., editors. *Oski's Pediatrics: Principles and Practice*. 4th ed.. Philadelphia, Pa: Lippincott Williams and Wilkins; 2006. p. 2084-2092.
26. Rosen, D.; Kelch, RP. Precocious and delayed puberty. In: Becker, KL.; Bilezikian, JP.; Bremner, WJ.; Hung, W.; Kahn, CR.; Loriaux, DL.; Nysten, ES.; Rebar, RW.; Robertson, GL.; Wartofsky, L., editors. *Principles and Practice of Endocrinology and Metabolism*. 2nd ed.. Philadelphia, Pa: J.B. Lippincott Company; 1995. p. 830-842.
27. Sedlmeyer IL, Hirschhorn JN, Palmert MR. Pedigree analysis of constitutional delay of growth and maturation: determination of familial aggregation and inheritance patterns. *J Clin Endocrinol Metab*. 2002; 87:5581–5586. [PubMed: 12466356]
28. Wehkalampi K, Widén E, Laine T, et al. Patterns of inheritance of constitutional delay of growth and puberty in families of adolescent girls and boys referred to specialist pediatric care. *J Clin Endocrinol Metab*. 2008; 93:723–728. [PubMed: 18160460]
29. Lindsay R, Feldkamp M, Harris D, Robertson J, Rallison M. Utah Growth Study: growth standards and the prevalence of growth hormone deficiency. *J Pediatr*. 1994; 125:29–35. [PubMed: 8021781]
30. August GP, Lippe BM, Blethen SL, Rosenfeld RG, Seelig SA, Johanson AJ, et al. Growth hormone treatment in the United States: Demographic and diagnostic features of 2331 children. *J Pediatr*. 1990; 116:899–903. [PubMed: 2348293]
31. Grimberg A, Stewart E, Wajnrajch MP. Gender of pediatric recombinant human growth hormone recipients in the United States and globally. *J Clin Endocrinol Metab*. 2008; 93:2050–2056. [PubMed: 18334582]

Figure 1. Age distribution, by sex, of the children in the pediatric primary care practices
 (A) Total number of children seen by each year of age. (B) Percentage of children seen at each year of age that have growth faltering.

Figure 2. Prevalence of growth faltering, as percentage of patients, by census tract in Philadelphia County

Excludes all census tracts with counts fewer than 10 patients in our pediatrics primary care network. All practices providing pediatric primary care, as well as the four practices caring for our patient population, are shown.

Table 1
Characteristics of the growth faltering and non-faltering children in the four urban pediatric primary care practices.

	Growth faltering group (n=3007)	Non-faltering children (n=30469)	P value, univariate analysis	Odds ratio	95% CI	P value, multivariate analysis
Height z-score (SD)	-1.5 ± 1.0	0.3 ± 0.9	<0.0001	_*	_*	_*
Female (%)	47	49	<0.01	0.96	[0.87,1.05]	0.36
Age (yrs)	4.9 ± 5.3	6.5 ± 5.1	<0.0001	0.91	[0.90,0.92]	<0.0001
Race (%)			<0.0001	0.66	[0.55,0.80]	<0.0001
African American	82	87				
White	9	7				
Ethnicity (% Hispanic)	2	1	0.30	0.85	[0.56,1.28]	0.43
Medicaid (%)	74	70	<0.0001	1.20	[1.06,1.35]	<0.005
Number of PCP** encounters	1.8 ± 1.5	2.3 ± 1.4	<0.0001	0.61	[0.57,0.64]	<0.0001
PCP** sex (Male %)	22	24	<0.05	_*	_*	_*
Median income (\$1000)***	25.2 ± 7.4	25.3 ± 7.3	0.80	0.95	[0.87,1.03]	0.20
Employment over 16 yrs (%)****	53 ± 7	53 ± 7	0.24	1.05	[0.99,1.11]	0.10
Education less than HS (%)****						
Female	25 ± 10	25 ± 10	0.10	1.00	[0.99,1.01]	0.98
Male	30 ± 12	30 ± 12	0.05	1.00	[1.00,1.01]	0.27

* Not included in the model of growth faltering; height z-score because the two groups were defined according to height, and PCP sex because it was a clinically implausible contributor to growth outcome.

** PCP, primary care physician

*** Census tract variables

**** For continuous variables, results presented as mean ± SD.

Table 2

Characteristics of the boys and girls in the four urban pediatric primary care practices.

	Growth faltering group (n=3,007)				Non-faltering children (n= 30,469)			
	Boys	Girls	P value	Boys	Girls	P value		
n	1607	1400	<0.01	15,448	15,021	-		
Height z-score (SD)	-1.7 ± 1.0	-1.4 ± 1.1	<0.0001	0.2 ± 0.9	0.3 ± 0.9	<0.0001		
Age (yrs)	4.5 ± 5.0	5.4 ± 5.5	<0.0001	6.4 ± 5.0	6.5 ± 5.2	<0.05		
Race (%)			0.38			0.65		
African American	83	82		87	88			
White	9	10		7	7			
Ethnicity (% Hispanic)	1	2	0.45	1	1	0.45		
Medicaid (%)	77	72	<0.01	70	69	0.47		
Number of PCP* encounters	1.9 ± 1.7	1.7 ± 1.3	<0.001	2.3 ± 1.4	2.3 ± 1.4	0.14		
PCP* gender (Male %)	24	19	<0.0001	27	21	<0.0001		
Median income (\$1,000)**	25.1 ± 7.3	25.4 ± 7.5	0.58	25.3 ± 7.3	25.2 ± 7.2	0.99		
Employment over 16 yrs (%)**	53 ± 7	53 ± 7	0.99	53 ± 7	53 ± 7	0.16		
Education less than HS (%)**								
Female	25 ± 10	25 ± 11	0.60	25 ± 10	25 ± 10	0.99		
Male	31 ± 12	30 ± 13	0.73	30 ± 12	30 ± 12	0.99		

* PCP, primary care pediatrician

** Census tract variables

*** For continuous variables, results presented as Least Squares Means ± SD; P values include Bonferroni's adjustment for multiple testing.

Table 3

Characteristics of children with height z-scores below -2.25 SD (ISS indication) in the four urban pediatric primary care practices.

	ISS group (n=534)	Non-ISS children (n=32 942)	P value, univariate analysis	Odds ratio	95% CI	P value, multivariate analysis
Height z-score (SD)	-2.8 ± 0.6	0.2 ± 1.0	<0.0001	—	—	*
Female (%)	43	49	<0.01	0.70	[0.54,0.90]	<0.01
Age (yrs)	5.7 ± 5.5	6.3 ± 5.1	<0.01	1.00	[0.97,1.02]	0.88
Race (%)			<0.0001	0.95	[0.54,1.70]	0.87
African American	80	87				
White	10	7				
Ethnicity (% Hispanic)	3	1	<0.01	1.44	[0.66,3.15]	0.36
Medicaid (%)	77	70	<0.05	1.58	[1.13,2.23]	<0.01
Number of PCP** encounters	2.6 ± 2.4	2.2 ± 1.4	<0.0001	1.20	[1.09,1.31]	<0.0005
PCP** sex (Male %)	22	24	0.30	—	—	*
Median income (\$1000)***	25.5 ± 7.9	25.2 ± 7.3	0.43	1.01	[0.82,1.25]	0.91
Employment over 16 yrs (%)***	53 ± 8	53 ± 7	0.64	1.12	[0.96,1.30]	0.15
Education less than HS (%)***						
Female	25 ± 11	25 ± 10	0.24	1.01	[0.99,1.02]	0.55
Male	30 ± 12	30 ± 12	0.37	1.00	[0.99,1.02]	0.71

* Not included in the model of ISS; height z-score because the two groups were defined according to height, and PCP sex because it was a clinically implausible contributor to growth outcome.

** PCP, primary care pediatrician

*** Census tract variables

**** For continuous variables, results presented as mean $\pm$ SD.