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Longitudinal Tracking of DXA Bone Measures Over 6 Years in Children and Adolescents: Persistence of Low Bone Mass to Maturity

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Abstract

Objectives—Early assessment of bone mass may be useful for predicting future osteoporosis risk if bone measures “track” during growth. This prospective longitudinal multicenter study examined tracking of bone measures in children and adolescents over 6 years to sexual and skeletal maturity.

Study design—240 healthy males and 293 healthy females, ages 6–17 years, underwent yearly evaluations of height, weight, body mass index, skeletal age, Tanner stage, and dual-energy x-ray absorptiometry (DXA) bone measurements of the whole body, spine, hip, and forearm for 6 years. All subjects were sexually and skeletally mature at final follow-up. Correlation was performed between baseline and 6-year follow-up measures, and change in DXA Z-scores was examined for subjects who had baseline $Z < -1.5$.

Results—DXA Z-scores ($r = 0.66$ – 0.87) had similar tracking to anthropometric measures ($r = 0.64$ – 0.74). Tracking was stronger for bone mineral density (BMD) compared with bone mineral

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content (BMC) and for girls compared with boys. Tracking was weakest during mid to late puberty, but improved when Z-scores were adjusted for height. Almost all subjects with baseline Z < -1.5 had final Z-scores below average, with the majority remaining below -1.0.

Conclusions—Bone status during childhood is a strong predictor of bone status in young adulthood, when peak bone mass is achieved. This suggests that bone mass measurements in children and adolescents may be useful for early identification of individuals at risk for osteoporosis later in life.

Keywords

bone density; osteoporosis; growth

Due to the difficulties in longitudinally studying subjects from childhood to an elderly age, the contention that senile osteoporosis is the result of inadequate bone acquisition during growth lacks supporting data. This notion is supported, however, by knowledge that the prevalence of osteoporosis is substantially lower in men than women, and in subjects of African American than European and Asian descent, due to sex and racial differences in bone mass that are already present in childhood (1–5). Additional support for this concept comes from studies showing a strong resemblance between mother-daughter bone traits and that this resemblance is present even before the daughters have begun puberty (6, 7). If bone loss was indeed the exclusive determinant of late life bone mass, then one would not expect such a strong resemblance in bone traits between girls and mothers.

Peak bone mass is nearly achieved by the end of sexual and skeletal development (8, 9). Early assessment of bone mass is only useful if bone measurements “track” during growth, with children who have low bone mass continuing to have low bone mass as they become young adults. We have previously shown that dual-energy x-ray absorptiometry (DXA) bone measures track longitudinally over 3 years during childhood and adolescence (10). The purpose of this prospective longitudinal multicenter study was to examine tracking of DXA bone measures in subjects from the same large pediatric cohort who were followed for 6 years and had reached sexual and skeletal maturity by the final follow-up. We hypothesized that tracking would be maintained over this longer period.

METHODS

The study sample derived from the Bone Mineral Density in Childhood Study (BMDCS), a multi-center longitudinal study examining bone accretion in a racially diverse cohort of 1,554 healthy boys and girls in the United States (U.S.) (11). Subjects were recruited from July 2002 to November 2003 at five medical centers across the U.S. Consent and assent were obtained from parents/guardians and participants following IRB approved protocols. Detailed information about the study participants and procedures has been published previously (11). Briefly, the BMDCS cohort represents healthy children with height, weight, and body mass index (BMI) between the 3rd and 97th percentile and no previous or current conditions that might affect bone acquisition. The subjects were evaluated annually for up to 6 years.

At each visit, height, weight, and BMI were measured, and Z-scores for sex and age were determined using normative data from the Centers for Disease Control and Prevention 2000 growth charts. A pediatrician or pediatric endocrinologist assessed sexual maturation based on breast stage in girls and testicular volume in boys using the Tanner criteria (12). A pediatric radiologist assessed skeletal maturity based on roentgenograms of the left hand and wrist according to the method of Greulich and Pyle (13). DXA measures were obtained on the same day.

This paper examines the 533 subjects who completed the full 6 years of follow-up and were sexually and skeletally mature at final follow-up. Skeletal maturity was defined as closure of the long bone physes, which occurs at a skeletal age of 17 years in boys and 16 years in girls. Of the 1,554 subjects in the original BMDCS cohort, 977 completed the year 6 visit with 533 of these being mature at final follow-up. The mean $\pm$ standard deviation (SD) chronological age of the 533 subjects at baseline was 13.2 ± 1.8 years with a comparable skeletal age (13.5 ± 2.2 years) (Table I). At final follow-up, average chronological and skeletal age were 19.2 ± 1.8 and 17.7 ± 0.9 years, respectively.

Whole body, anterior-posterior (AP) lumbar spine, non-dominant forearm, and left proximal femur DXA scans were performed using Hologic, Inc. (Bedford, MA) bone densitometers (QDR4500A, QDR4500W, and Delphi A models) and analyzed using pediatric software (Hologic version 12.3). Scanner calibration was described previously (11). The precision errors were coefficients of variation $<1.4\%$ for BMC and $<1\%$ for BMD of the spine and whole body less head, and $<1.7\%$ for BMD of all other sites (14). Standard and height-adjusted DXA Z-scores were calculated using the appropriate black or non-black normative data for sex and age as reported previously by the BMDCS (11, 15, 16).

Statistical Analyses

Statistical analyses were conducted using Stata (version 12.0; StataCorp LP, College Station, TX). Pearson correlations were performed between baseline anthropometric and DXA measures and the same measures 6 years later. Correlations were calculated by sex and by Tanner stage at baseline. Pearson correlations were also used to compare the change in BMC and BMD Z-scores over 6 years to the change in Z-scores for anthropometric measures.

To assess further changes over time, we categorized children by their DXA Z-scores at baseline (<-1.5 , -1.5 to 1.5 , or >1.5) and calculated the mean Z-score by baseline category for each study year. Cutoffs of -1.5 and $+1.5$ were used rather than -2.0 and $+2.0$ because the subjects were required to be between the 3rd and 97th percentiles for height, weight, and BMI, resulting in only a small number with Z-scores above 2.0 or below -2.0 . Differences in mean Z-scores at the final 6-year follow-up were compared among subjects in the different baseline groups using analysis of variance with Bonferroni post-hoc tests.

RESULTS

Baseline Z-scores for anthropometric and DXA bone measures were close to zero with standard deviations close to one (Table I). The tracking of DXA Z-scores over 6 years ($r =$

0.66 to 0.87) was similar in magnitude, or slightly greater, than the tracking of anthropometric measures (Table II). In general, tracking was stronger for BMD than for BMC and for height-adjusted DXA Z-scores compared with unadjusted Z-scores. Tracking also tended to be better for girls compared with boys.

When examined as a function of sexual maturity at baseline, tracking of unadjusted Z-scores was strong before puberty (Tanner 1; boys: $r = 0.73$ to 0.89 , $N = 24$, girls: $r = 0.83$ to 0.93 , $N = 26$) and after sexual maturity had been achieved (Tanner 5; boys: $r = 0.74$ to 0.85 , $N = 90$, girls: $r = 0.75$ to 0.89 , $N = 90$). However, tracking was weaker during puberty (Tanner 2–4; boys: $r = -0.03$ to 0.78 , $N = 126$, girls: $r = 0.56$ to 0.88 , $N = 177$), particularly in Tanner stage 3 (boys: $r = -0.03$ to 0.65 , $N = 31$; girls: $r = 0.60$ to 0.75 , $N = 58$) (Figure 1, A; available at www.jpeds.com). Tracking generally improved with height adjustment, especially in Tanner stage 3 (Figure 1, B).

There was a significant relationship between the change in BMC Z-scores and the change in Z-scores for height ($r = 0.50$ to 0.68) and, to a lesser extent, weight ($r = 0.38$ to 0.48). A similar, though weaker, relationship was observed for BMD Z-scores ($r = 0.31$ to 0.53 for height; $r = 0.27$ to 0.48 for weight). Almost all subjects with poor tracking of BMC Z-scores (change exceeding ± 1.5) experienced a change in height in the same direction with a median magnitude of change of 0.3 to 1.7 . Similar but weaker trends were observed for BMD Z-scores.

We examined the persistence of values at the tails of the distribution by categorizing children according to their baseline Z-score as LOW (< -1.5), HIGH (> 1.5), or INTERMEDIATE (-1.5 to 1.5). Although regression towards the mean was observed over time, final Z-scores remained significantly different among subjects in the different baseline groups (Figure 2). Notably, among boys and girls who had initial Z-scores below -1.5 , almost all remained below the mean of normal ($Z = 0$) and the majority stayed below -1.0 at final follow-up (Figure 3). This was true for both BMC and BMD, regardless of measurement site and whether Z-scores were adjusted for height (Figure 4; available at www.jpeds.com). Likewise, final values for subjects who had initial Z-scores > 1.5 tended to remain above the mean (Figure 3). Only 3.8% of the children crossed from INTERMEDIATE to LOW; 12 of 256 girls and 6 of 209 boys. We were not able to identify any characteristics distinguishing the children who crossed Z-score categories.

DISCUSSION

This study indicates that DXA measures of bone strength track through childhood until the time of skeletal and sexual maturity. This was true in both the axial and appendicular skeleton, for both males and females. On average, DXA measures of BMC and BMD during childhood accounted for approximately 50–70% of the variation observed 6 years later when subjects were mature. Almost all boys and girls with low bone mass (Z-score < -1.5) became young men and women with values for BMC and BMD below the mean (Z-score < 0). These results corroborate previous findings indicating that the susceptibility for osteoporosis is present early in life.

Ample data suggest that the amount of bone accrued by skeletal maturity is the main contributor to peak bone mass, which, in turn, is a major determinant of osteoporosis and fractures in the elderly (17). We have also recently shown in the same cohort that bone mass during childhood is a predictor of pediatric fractures (18). Although the time of life when DXA values reach their peak has been of considerable controversy (19, 20), most estimates indicate that bone mass does not significantly increase after the third decade (5). Because the degree of tracking during young adulthood should be comparable or greater than during growth, the findings of our study underscore the potential of these measures to predict bone mineral status in older adulthood. Support for this notion is that the degree of tracking for DXA measures of bone mineral accrual in our large, nationally representative cohort of healthy children was comparable with that of height and weight.

We were not able to identify any characteristics that predicted which subjects would cross Z-score categories, such as changing from low to intermediate or intermediate to low. Changes in bone status may have been related to growth, nutrition, changes in activity, or many other factors. It is important to recognize that although there is strong overall tracking of DXA Z-scores, it is still possible for the bone status of a given individual to worsen or improve.

The large sample of well characterized healthy children representative of the current U.S. population, the relatively long follow-up period until skeletal maturity, and the highly standardized assessments of bone density measured by DXA are strengths of this study. The findings of this 6-year longitudinal study corroborate previous studies with shorter follow-ups suggesting that children retain their bone phenotypes throughout growth (9, 21). Most studies to date, however, have been constrained by small sample sizes (22–24) and/or BMD determinations using techniques not widely used in clinical practice (23, 25). Our results are also consistent with smaller studies of pre- and early pubertal children that were followed for 7 to 8 years (26–29). However, the current study is limited to the natural history of bone mineral accrual in healthy children. Studies are needed for children who have chronic medical conditions that may alter bone mineral accrual and cause fluctuations in tracking.

Bone mineral status during childhood is a strong predictor of bone mineral status in the axial and appendicular skeletons in young adulthood. These results suggest that bone mass measurements may be useful for early identification of children at risk for osteoporosis later in life. They also provide a distinct example of how phenotypic traits, like genetic information, could assume a major role in personalized medicine and the customization of healthcare. Because current treatment for osteoporosis in the elderly does not significantly restore loss of bone, efforts should be directed toward developing preventive measures that increase bone accrual before the completion of skeletal maturity. Studies are needed to establish whether DXA bone measures associated with weaker bones in childhood can be altered as a result of simple nutritional, mechanical, or pharmacological intervention.

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ABBREVIATIONS

BMC	bone mineral content
BMD	bone mineral density (areal)
BMDCS	Bone Mineral Density in Childhood Study
BMI	body mass index
DXA	dual-energy x-ray absorptiometry

References

1. Gilsanz V, Perez FJ, Campbell PP, Dorey FJ, Lee DC, Wren TA. Quantitative CT reference values for vertebral trabecular bone density in children and young adults. *Radiology*. 2009; 250:222–7. [PubMed: 19001149]
2. Gilsanz V, Kovanlikaya A, Costin G, Roe TF, Sayre J, Kaufman F. Differential effect of gender on the size of the bones in the axial and appendicular skeletons. *J Clin Endocrinol Metab*. 1997; 82:1603–7. [PubMed: 9141557]
3. Gilsanz V, Roe TF, Mora S, Costin G, Goodman WG. Changes in vertebral bone density in black girls and white girls during childhood and puberty. *N Engl J Med*. 1991; 325:1597–600. [PubMed: 1944449]
4. Burrows M, Baxter-Jones A, Mirwald R, Macdonald H, McKay H. Bone mineral accrual across growth in a mixed-ethnic group of children: are Asian children disadvantaged from an early age? *Calcif Tissue Int*. 2009; 84:366–78. [PubMed: 19308631]
5. Clarke BL, Khosla S. Female reproductive system and bone. *Arch Biochem Biophys*. 2010; 503:118–28. [PubMed: 20637179]
6. Fernandez-Ojeda R, Moruno RM, Miranda MJ, Giner M, Montoya MJ, Vazquez MA, et al. Study of bone mass in young daughters of women with fracture of the distal end of the radius. *J Clin Densitom*. 2013; 16:87–91. [PubMed: 22980490]
7. Seeman E, Hopper JL, Bach LA, Cooper ME, Parkinson E, McKay J, et al. Reduced bone mass in daughters of women with osteoporosis. *N Engl J Med*. 1989; 320:554–8. [PubMed: 2915666]
8. Gilsanz V, Gibbens DT, Roe TF, Carlson M, Senac MO, Boechat MI, et al. Vertebral bone density in children: effect of puberty. *Radiology*. 1988; 166:847–50. [PubMed: 3340782]
9. Bonjour JP, Theintz G, Buchs B, Slosman B, Rizzoli R. Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. *J Clin Endocrinol Metab*. 1991; 73:555–63. [PubMed: 1874933]
10. Kalkwarf HJ, Gilsanz V, Lappe JM, Oberfield S, Shepherd JA, Hangartner TN, et al. Tracking of bone mass and density during childhood and adolescence. *The Journal of clinical endocrinology and metabolism*. 2010; 95:1690–8. [PubMed: 20194709]
11. Kalkwarf HJ, Zemel BS, Gilsanz V, Lappe JM, Horlick M, Oberfield S, et al. The bone mineral density in childhood study: bone mineral content and density according to age, sex, and race. *The Journal of clinical endocrinology and metabolism*. 2007; 92:2087–99. [PubMed: 17311856]
12. Tanner JM. Growth and maturation during adolescence. *Nutr Rev*. 1981; 39:43–55. FAU - Tanner, J M. [PubMed: 7010232]
13. Greulich, WW.; Pyle, SI. *Radiographic Atlas of Skeletal Development of the Hand and Wrist*. 2. California: Stanford University Press; 1959.
14. Shepherd JA, Wang L, Fan B, Gilsanz V, Kalkwarf HJ, Lappe J, et al. Optimal monitoring time interval between DXA measures in children. *Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research*. 2011; 26:2745–52.
15. Zemel BS, Kalkwarf HJ, Gilsanz V, Lappe JM, Oberfield S, Shepherd JA, et al. Revised reference curves for bone mineral content and areal bone mineral density according to age and sex for black and non-black children: results of the bone mineral density in childhood study. *The Journal of clinical endocrinology and metabolism*. 2011; 96:3160–9. [PubMed: 21917867]

16. Zemel BS, Leonard MB, Kelly A, Lappe JM, Gilsanz V, Oberfield S, et al. Height adjustment in assessing dual energy x-ray absorptiometry measurements of bone mass and density in children. *The Journal of clinical endocrinology and metabolism*. 2010; 95:1265–73. [PubMed: 20103654]
17. Bonjour JP, Theintz G, Law F, Slosman D, Rizzoli R. Peak bone mass. *Osteoporos Int*. 1994; 4 (Suppl 1):7–13. [PubMed: 8081064]
18. Wren TA, Shepherd JA, Kalkwarf HJ, Zemel BS, Lappe JM, Oberfield S, et al. Racial disparity in fracture risk between white and nonwhite children in the United States. *The Journal of pediatrics*. 2012; 161:1035–40. [PubMed: 22974572]
19. Bachrach LK, Hastie T, Wang MC, Narasimhan B, Marcus R. Bone mineral acquisition in healthy Asian, Hispanic, black, and Caucasian youth: a longitudinal study. *The Journal of clinical endocrinology and metabolism*. 1999; 84:4702–12. [PubMed: 10599739]
20. Wren TA, Kim PS, Janicka A, Sanchez M, Gilsanz V. Timing of peak bone mass: discrepancies between CT and DXA. *The Journal of clinical endocrinology and metabolism*. 2007; 92:938–41. [PubMed: 17164299]
21. Theintz G, Buchs B, Rizzoli R, Slosman D, Clavien H, Sizonenko PC, et al. Longitudinal monitoring of bone mass accumulation in healthy adolescents: evidence for a marked reduction after 16 years of age at the levels of lumbar spine and femoral neck in female subjects. *J Clin Endocrinol Metab*. 1992; 75:1060–5. [PubMed: 1400871]
22. Loro ML, Sayre J, Roe TF, Goran MI, Kaufman FR, Gilsanz V. Early identification of children predisposed to low peak bone mass and osteoporosis later in life. *The Journal of clinical endocrinology and metabolism*. 2000; 85:3908–18. [PubMed: 11061556]
23. Ruff C. Growth tracking of femoral and humeral strength from infancy through late adolescence. *Acta Paediatr*. 2005; 94:1030–7. [PubMed: 16188845]
24. Van Coeverden SC, De Ridder CM, Roos JC, Van't Hof MA, Netelenbos JC, Delemarre-Van de Waal HA. Pubertal maturation characteristics and the rate of bone mass development longitudinally toward menarche. *J Bone Miner Res*. 2001; 16:774–81. [PubMed: 11316006]
25. Magarey AM, Boulton TJ, Chatterton BE, Schultz C, Nordin BE, Cockington RA. Bone growth from 11 to 17 years: relationship to growth, gender and changes with pubertal status including timing of menarche. *Acta Paediatr*. 1999; 88:139–46. [PubMed: 10102144]
26. Cheng S, Volgyi E, Tylavsky FA, Lyytikainen A, Tormakangas T, Xu L, et al. Trait-specific tracking and determinants of body composition: a 7-year follow-up study of pubertal growth in girls. *BMC Med*. 2009; 7:5. [PubMed: 19171028]
27. Ferrari SL, Chevalley T, Bonjour JP, Rizzoli R. Childhood fractures are associated with decreased bone mass gain during puberty: an early marker of persistent bone fragility? *J Bone Miner Res*. 2006; 21:501–7. [PubMed: 16598368]
28. Foley S, Quinn S, Jones G. Tracking of bone mass from childhood to adolescence and factors that predict deviation from tracking. *Bone*. 2009; 44:752–7. [PubMed: 19103314]
29. Budek AZ, Mark T, Michaelsen KF, Molgaard C. Tracking of size-adjusted bone mineral content and bone area in boys and girls from 10 to 17 years of age. *Osteoporos Int*. 2010; 21:179–82. [PubMed: 19387763]

Appendix

Collaborators in the pediatric endocrine divisions of each Bone Mineral Density in Childhood Study Group clinical centers include: Children's Hospital of Philadelphia: Andrea Kelly, David Langdon, Thomas Moshang, Steve Willi, Lorraine Katz, Charles Stanley, and Craig Alter; Children's Hospital Los Angeles: Lynda Fisher, Mitchell Geffner, Debra Jeandron, Steven Mittelman, Pisit Pitukcheewanont, and Francine Kaufman; Cincinnati Children's Hospital Medical Center: Susan Rose, Frank Biro, Peggy Stenger, Debbie Elder, and James Heubi; Columbia University Medical Center-St. Luke's Hospital: Mary Horlick, Natasha Leibel, and Abeer Hassoun; and Creighton University: Jean-Claude Desmangles.

Data Safety and Monitoring Board members: Clifford Rosen, Ralph D'Agostino, Ingrid Holm, James Reynolds, and Reginald Tsang.

Figure 1.

Correlation between baseline and final (6-year) Z-scores for whole body less head BMC as a function of baseline Tanner stage. Similar trends were observed for other DXA bone measures.

Figure 2.

Mean $\pm$ SD whole body less head BMC Z-scores over time for subjects subdivided by baseline Z-score (<-1.5, -1.5 to 1.5, >1.5). Final Z-scores differed significantly among the different baseline groups ($p < 0.001$).

Figure 3.

Change between baseline and final (6-year) Z-scores for whole body less head BMC subdivided by baseline Z-score: > 1.5 (top row), -1.5 to 1.5 (middle row), < -1.5 (bottom row). Similar trends were observed for other DXA bone measures.

Figure 4.
Final height-adjusted Z-scores for subjects starting with adjusted $Z < -1.5$ at baseline.

Table 1

Baseline characteristics of the study subjects

	Male (N=240)	Female (N=293)	All Subjects (N=533)
<i>Developmental Characteristics</i>			
Chronological Age (yr)	13.7 (1.8)	12.8 (1.7)	13.2 (1.8)
Skeletal age (yr)	13.8 (2.3)	13.3 (2.0)	13.5 (2.2)
Sexual maturation			
Tanner 1	24 (10%)	26 (9%)	50 (9%)
Tanner 2	47 (20%)	45 (15%)	92 (17%)
Tanner 3	31 (13%)	58 (20%)	89 (17%)
Tanner 4	48 (20%)	74 (25%)	122 (23%)
Tanner 5	90 (38%)	90 (31%)	180 (34%)
<i>Anthropometric Characteristics</i>			
Height Z-score	0.2 (0.8)	0.2 (0.8)	0.2 (0.8)
Weight Z-score	0.3 (0.8)	0.5 (0.8)	0.4 (0.8)
BMI Z-score	0.3 (0.9)	0.5 (0.8)	0.4 (0.8)
<i>DXA Z-score</i>			
Whole body less head BMC	0.0 (1.0)	0.2 (0.9)	0.1 (1.0)
Whole body less head BMD	0.1 (0.9)	0.2 (0.9)	0.1 (0.9)
AP spine BMC	-0.1 (1.0)	0.1 (1.0)	0.0 (1.0)
AP spine BMD	-0.1 (1.0)	0.0 (1.0)	0.0 (1.0)
Hip total BMC	0.0 (1.0)	0.1 (1.0)	0.1 (1.0)
Hip total BMD	0.0 (1.0)	0.1 (1.0)	0.1 (1.0)
Hip neck BMC	0.0 (0.9)	0.1 (0.9)	0.1 (0.9)
Hip neck BMD	0.0 (1.0)	0.1 (1.0)	0.0 (1.0)
1/3 forearm BMC	-0.1 (0.9)	0.1 (0.9)	0.0 (0.9)
1/3 forearm BMD	-0.1 (1.0)	0.1 (1.1)	0.0 (1.1)

Continuous variables are shown as mean (standard deviation).

Categorical variables are N (%).

Table 2

Correlation of Z-scores between baseline and 6 years later

	BMC	BMD	Height adjusted BMC	Height adjusted BMD
Boys				
Height	.71	---	---	---
Weight	.64	---	---	---
BMI	.68	---	---	---
DXA whole body	---	.66	.71	.76
DXA spine	---	.69	.78	.78
DXA hip total	---	.70	.78	.80
DXA hip neck	---	.68	.75	.77
DXA forearm	---	.73	.80	.78
Girls				
Height	.72	---	---	---
Weight	.69	---	---	---
BMI	.74	---	---	---
DXA whole body	---	.68	.70	.81
DXA spine	---	.73	.84	.87
DXA hip total	---	.73	.75	.85
DXA hip neck	---	.70	.75	.82
DXA forearm	---	.79	.85	.83
All Subjects				
Height	.71	---	---	---
Weight	.66	---	---	---
BMI	.71	---	---	---
DXA whole body	---	.67	.71	.78
DXA spine	---	.71	.81	.83
DXA hip total	---	.71	.76	.83
DXA hip neck	---	.69	.75	.80
DXA forearm	---	.75	.83	.81