ORIGINAL ARTICLE

Childhood obesity and skeletal maturation assessed with Fishman's hand-wrist analysis

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Introduction: The purpose of this study was to determine whether increased body mass index is associated with accelerated skeletal maturation. **Methods:** The skeletal ages of 107 children, aged 9 to 16 years, were determined by using Fishman's hand-wrist analysis. The difference between chronologic age and dental age was analyzed against body mass index, sex, and age. **Results:** The mean differences between chronologic and skeletal ages for normal weight, overweight, and obese subjects were 0.51 years, 0.44 years, and 1.00 years, respectively. Although there was a trend for obese subjects to have accelerated skeletal maturation compared with overweight and normal-weight subjects, the difference was not statistically significant. Skeletal age differences significantly decreased with increasing age. The mean skeletal age differences were 0.90 year for 9- to 13-year-olds and 0.26 year for 13- to 16-year-olds. Mean skeletal age did not differ significantly by sex. **Conclusions:** Overweight or obese children did not have significantly accelerated skeletal maturation after adjusting for age and sex. (Am J Orthod Dentofacial Orthop 2007;132:185-90)

Childhood obesity is an escalating problem nationwide[1-3] that can result in cardiovascular effects such as hypercholesterolemia, dyslipidemia, and hypertension[4-7] or endocrine effects such as hyperinsulinism, insulin resistance, impaired glucose tolerance, and type 2 diabetes (noninsulin dependent diabetes mellitus).[8-10] In girls, obesity can result in the early onset of puberty and considerably accelerated linear growth[11] as well as increased risks of breast cancer[12-14] and polycystic ovary disease[15] into adulthood. In boys, there can be considerable variation in the timing of puberty (accelerated or delayed).[15] Other systemic effects include pulmonary problems such as asthma, obstructive sleep apnea,[15] and Pickwickian syndrome[16-21]; orthopedic problems[22,23]; and gastrointestinal/hepatic problems such as nonalcoholic steatohepatitis.[24] Furthermore, many who are obese also suffer from depression and low self-esteem.[25,26] The Surgeon General expects future obesity-related health-care costs and the morbidity and mortality rates might exceed that associated with cigarette smoking.[26,27]

Body mass index (BMI) is a commonly used measure of adiposity; it is easy to calculate, quick to measure, and noninvasive. It is widely used to screen adults for obesity, but its use in adolescents is controversial, and it is a fairly poor index of fatness in individual children unless age and sex are considered.

Childhood BMI changes significantly with age. At birth, the median is as low as 13 kg per square meter. It increases to 17 at age 1, decreases to 15.5 at age 6, and increases to 21 at age 20. Increases in BMI during both later childhood and adolescence can be attributed primarily to increases in fat-free tissue rather than fat.[28] Therefore, to more accurately define childhood obesity, a cutoff point relative to age is necessary. In the United States, the 85th and 95th percentiles of BMI for age and sex are commonly used and are based on nationally represented survey data. Unfortunately, however, BMI values are increasing in children nationwide, and many children are not properly categorized as overweight because of a relatively heavy American population.

An international survey, recommended by the International Obesity Task Force,[29] has established a standard definition for childhood obesity for global monitoring[30] as well as clinical practice and public health measures.[31] It was developed after a survey of 97,876 males and 94,851 females from birth to 25 years of age for 6 large, nationally representative cross-sectional growth studies from Brazil, Great Britain, Hong Kong, the Netherlands, Singapore, and the

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United States. It provides cutoff points for BMI in childhood that are based on international data and linked to the widely accepted adult cutoff points of BMI values of 25 and 30 kg per square meter for men and women.[30] Neovius et al[29] evaluated the sensitivity and specificity of this BMI-based classification system and found BMI to be highly specific in both sexes.

More accurate methods than BMI are available to assess adiposity, but they are impractical for epidemiologic use. The ideal definition of obesity is based on percentage of body fat, which requires more sophisticated measures, such as x-ray densitometry[32] or other multi-component techniques.[28] Triceps skinfold thickness is a more sensitive assessment than BMI, but it has limitations. It still uses 85th and 95th percentiles operationally to define obesity and superobesity, respectively, and requires the use of population-based, race-specific, or age-specific criteria. Furthermore, its use is not practical in a dental setting.[33]

Skeletal age is a more reliable and precise method for assessing physical maturity than chronologic age,[34-36] and a person's maturational status can considerably influence orthodontic diagnosis, treatment planning, and treatment outcome. Other areas of the skeleton have been used to assess skeletal maturation such as the foot, the ankle, the hip, and the elbow.[37] However, orthodontists most frequently use radiographs of the hand, because the hand-wrist more accurately assesses biological maturation variations relative to chronologic age than the knee.[38-40] Fishman[41] developed the system using the 4 stages of ossification—initial ossification, width, capping, and fusion—at 6 anatomic sites to judge the remaining growth potential of the maxilla and the mandible. The updated system considers not only bone staging (skeletal maturation stage) but also skeletal maturation level (advanced, normal, or delayed) and accounts for sex by sex-matched charts that reflect earlier female maturation.[42,43] Studies reported that the analysis is accurate and significantly related to skeletal maturation in both sexes.[44]

Current literature reported that obesity is correlated with the early onset of puberty due to hormonal fluctuation.[45,46] The amount of remaining facial growth potential is vitally important in orthodontic growth modification and orthognathic surgical treatment planning. It is most often determined by the assessment of hand-wrist radiographic ossification.[44] The widely used system of hand-wrist skeletal maturation indicators (SMI) uses 4 stages of bone maturation (initial ossification, width, capping, and fusion) at 6 anatomic sites.[42] This analysis is the most widely used when judging remaining growth potential of the jaws before orthodontic treatment,[47,48] although the cervical vertebrae maturation analysis has been increasingly used to determine skeletal maturation.[49] Using the lateral profiles of the second, third, and fourth cervical vertebrae, practitioners can evaluate the potential for future adolescent growth. Additionally, some studies used permanent tooth maturation rather than skeletal maturity with mixed results.[50-52]

To our knowledge, no previous study has evaluated the relationship between childhood obesity and early skeletal growth maturation. Therefore, our purpose was to determine whether increased BMI is associated with accelerated skeletal age as determined by Fishman's hand-wrist analysis.

Table I. International index for BMI for overweight and obesity by sex and age[30]

	Overweight		*Obese*	
Age (y)	*Male BMI*	*Female BMI*	*Male BMI*	*Female BMI*
7	17.9	17.75	20.63	20.51
8	18.44	18.35	21.60	21.57
9	19.10	19.07	22.77	22.81
10	19.84	19.86	24.00	24.11
11	20.55	20.74	25.10	25.42
12	21.22	21.68	26.02	26.67
13	21.91	22.58	26.84	27.76
14	22.62	23.34	27.63	28.57
15	23.29	23.94	28.30	29.11
16	23.90	24.37	28.88	29.43
17	24.46	24.70	29.41	29.69
18	25.00	25.00	30.00	30.00

MATERIAL AND METHODS

This study comprised a chart review of new patients between 9 and 16 years of age who were seen between January 1 and December 31, 2004, in the Department of Orthodontics and Pediatric Dentistry at the University of Louisville School of Dentistry, Louisville, Ky. Appropriate institutional review board approval was received by the University of Louisville.

BMI was calculated to determine patients who were overweight or obese.[53-55] Body adiposity status was determined by the international classification system for childhood obesity recommended by the International Obesity Task Force, which formed a standard regardless of ethnicity.[30] Published age- and sex-matched tables (cutoff points listed in Table I) defined subjects as "overweight" or "obese" to comprise the study groups. "Normal-weight" subjects who were age- and sex-matched to the study groups comprised the control group. Charts were excluded from review if the patients had several congenitally missing teeth, were underweight (low BMI), or had a history of chronic infectious disease, nutritional disturbance, or endocrine disorder.

Table II. Estimated skeletal maturation age values for adolescent SMI[43]

SMI	Female Mean	Female SD	Male Mean	Male SD
1	9.94	0.96	11.01	1.22
2	10.58	0.88	11.68	1.06
3	10.88	0.99	12.12	1.00
4	11.22	1.11	12.33	1.09
5	11.64	0.90	12.98	1.12
6	12.06	0.96	13.75	1.06
7	12.34	0.90	14.38	1.08
8	13.10	0.87	15.11	1.03
9	13.90	0.99	15.50	1.07
10	14.77	0.96	16.40	1.00
11	16.07	1.25	17.37	1.26

Patients' skeletal ages were determined by the primary investigator (M.A.) by evaluating available hand-wrist radiographs using the Fishman method.[41] Each subject's SMI was calculated and then converted into skeletal age with the published sex-matched table (Table II). Skeletal age differences were calculated by subtracting the chronologic age from the calculated skeletal age range (skeletal age ± standard deviation listed in Table II). Positive differences above the skeletal age range reflected accelerated skeletal maturation, and negative differences below the skeletal age range reflected delayed maturation.

Statistical analysis

Three-way ANOVA was used to analyze the skeletal age difference (dependent variable) against the independent variables, which were the BMI categories (obese, overweight, and normal weight), sex, and chronologic age (9-12 and 13-15 years). The Pearson correlation test was used to determine the correlation between skeletal age and chronologic age by sex. To determine intraexaminer reliability, 10 hand-wrist radiographs were reassessed after 2 weeks, and skeletal ages were compared by using Cronbach's alpha. To determine hand-wrist analysis accuracy by the primary investigator, 25 hand-wrist radiographs were blindly assessed by 2 experts (A.M.S. and W.S.) in Fishman's hand-wrist analysis and compared using Cronbach's alpha.

RESULTS

One hundred seven children met all inclusion criteria and were included in the study—44 boys and 63 girls (Table III). Ages ranged from 9.0 to 15.5 years, BMI values from 10.6 to 39.3, and skeletal ages from 9.94 to 16.4 years. No ethnic data were available.

Table III. Mean differences between skeletal and chronologic ages

Sex	BMI	Chronologic age	Mean skeletal age difference	n	SD
Male	Normal	9-12.9	0.77	11	0.95
		†13-15.5	0.06	19	1.41
		Total	0.32	30	1.29
	Overweight	9-12.9	0.92	3	1.45
		†13-15.5	0.54	1	0.00
		Total	0.82	4	1.20
	*Obese	9-12.9	2.38	2	0.70
		†13-15.5	0.62	8	1.27
		Total	0.97	10	1.36
	Total	9-12.9	1.00	16	1.10
		†13-15.5	0.24	28	1.35
		Total	0.52	44	1.30
Female	Normal	9-12.9	0.89	25	1.08
		†13-15.5	0.20	15	0.91
		Total	0.63	40	1.06
	Overweight	9-12.9	0.49	9	0.51
		†13-15.5	–0.09	4	1.19
		Total	0.31	13	0.78
	*Obese	9-12.9	1.27	5	1.38
		†13-15.5	0.79	5	0.82
		Total	1.03	10	1.10
	Total	9-12.9	0.85	39	1.02
		†13-15.5	0.27	24	0.94
		Total	0.63	63	1.02
Total	Normal	9-12.9	0.85	36	1.03
		†13-15.5	0.12	34	1.20
		Total	0.50	70	1.17
	Overweight	9-12.9	0.60	12	0.78
		†13-15.5	0.03	5	1.07
		Total	0.43	17	0.88
	*Obese	9-12.9	1.60	7	1.28
		†13-15.5	0.69	13	1.08
		Total	1.00	20	1.21
	Total	9-12.9	0.89	55	1.03
		†13-15.5	0.25	52	1.17
		Total	0.58	107	1.14

*$P < .1$: A trend was seen between normal and obese subjects and between overweight and obese subjects for those with higher BMI values to have accelerated skeletal ages.
†$P < .05$: Subjects in the older chronologic age group had skeletal ages significantly closer to their chronologic age than did those in the younger age group.

However, most patients seen at the University of Louisville are white or black. Overall, skeletal age correlated fairly well with chronologic age (0.753) (Table IV). Differences between skeletal age and chronologic age varied from –2.07 to +4.10 years. When assessed by sex, skeletal age was significantly closer to chronologic age in the girls (0.790) than in the boys (0.694) ($P < .001$).

Although not significant statistically, skeletal age differences tended to increase with increases in BMI from normal to overweight ($P = .074$) and from

Table IV. Correlation between skeletal and chronologic ages by sex

Sex	*Age (y)*	*Mean*	*SD*	*n*	*Correlation*
Male	Chronologic	13.06	1.56	44	0.69
	Skeletal	13.63	1.75	44	
Female	Chronologic	12.53	1.53	63	0.79
	Skeletal	13.17	1.60	63	
Total	Chronologic	12.75	1.56	107	0.75
	Skeletal	13.36	1.67		

All 3 measures of correlation were statistically significant at P = .000.

overweight to obese (P = .118). The mean skeletal age differences were 0.59 for all subjects, 0.51 for normal weight subjects, 0.44 for overweight subjects, and 1.00 for obese subjects. Skeletal age differences significantly decreased with increasing age (P = .018). The mean skeletal age differences were 0.90 year for 9- to 12.9-year-olds and 0.26 year for 13- to 15.5-year-olds. Mean skeletal age difference did not differ significantly by sex (P = .364), but girls tended to have greater skeletal age differences (0.63) than boys (0.52). There was no statistical interaction between age, sex, and BMI when analyzing skeletal age differences.

When we evaluated intraobserver variability, there was a high degree of consistency. Cronbach's alpha was 0.98 for measuring skeletal ages on the repeated 10 hand-wrist radiographs. The primary author (M.A.) interpreted the radiographs and was calibrated by 2 experts in hand-wrist analysis for a high level of accuracy. Cronbach's alpha values were 0.99 between experts, 0.99 between the experts' average and the primary investigator, 0.99 between 1 expert and the primary investigator, and 0.99 between the second expert and the primary investigator for 25 hand-wrist calibration radiographs.

DISCUSSION

Dental practitioners should provide regular obesity screenings simply by recording the height and weight of all patients and calculating BMI status. Screening for early treatment of overweight children and adolescents might increase awareness and limit the associated long-term health consequences.[56,57] As health-care providers, we should furnish dietary education not only to promote oral health, but also to maintain healthy body adiposity levels. These simple measures could increase awareness and limit the long-term health consequences associated with childhood obesity.[58]

In this study, the subjects were selected from a patient base treated in a dental school in an urban area. Therefore, the sample might be of lower socioeconomic status, which has been reported as a risk factor for childhood obesity.[59] It did, however, give us a wide range of subjects of differing ages, BMI values, and dental development stages to address the effect of obesity on dental and chronologic ages.

Skeletal and chronologic ages correlated moderately well, and our results agree with published reports.[60] However, it is controversial whether early puberty and obesity are related. Several studies reported a positive relationship,[45,46] but others suggested that increases in relative weight are a consequence of age at puberty rather than a determinant.

Flores-Mir et al[52] evaluated children in Peru with stunted growth to determine whether skeletal maturation and dental development were also delayed. They used Fishman's analysis[41] to assess skeletal maturation and the method of Demirjian et al[61] to asses dental development. Similar to our study, they found no statistically significant difference for the skeletal maturation or the dental development stages according to nutritional status (determined by BMI status).

Secular changes in BMI and mean age of puberty could be independent phenomena.[61] We found that skeletal ages for the control group (normal weight) were accelerated approximately 0.51 years ahead of chronologic age. Acceleration in skeletal age did not significantly increase with increases in BMI. However, there was a trend for skeletal age acceleration when comparing normal-weight and overweight subjects with obese subjects. A larger sample size organized into more age categories is necessary and might demonstrate a statistically significant relationship rather than a trend.

Furthermore, the age groups were small when broken down by sex, with only 2 groups formed for each sex. As expected, skeletal age differences significantly decreased with increases in age, since growth was nearly completed and minimized marginal error. Intraobserver reliability was excellent, but the radiographic interpretation accuracy might have been lower in younger subjects. There was also a trend for female skeletal maturation to be more accelerated than male, even after accounting for sex through sex-matched charts in the Fishman analysis. Therefore, the secular trend of earlier maturation might affect girls more than boys.

Use of Fishman's maturation system was advantageous, because it analyzes the influence of skeletal level (advanced, average, or delayed maturation) as well as skeletal indicators on remaining craniofacial growth. Because the use of individual ossification events (skeletal indicators) is of limited use in predicting the pubertal growth spurt, an analysis that includes maturation levels and ossification events is recommended.[47]

If obesity is associated with accelerated dental maturation[62] and skeletal maturation, orthodontic treatment timing and treatment options might vary for these patients. For example, overweight patients might require earlier orthodontic consultation because serial extraction timing might need to be altered, as well as space maintenance, growth modification, and orthognathic surgery. Furthermore, we suggest that the early eruption of permanent teeth when the children might not be capable of proper oral hygiene could result in increased caries incidence.

In this study, we found that the skeletal age differences between obese and overweight subjects decreased as adolescence progressed. This was expected, particularly during late adolescence, because advanced maturers tend to "burn out" relative to skeletal growth rate, and average maturers and especially delayed maturers continue to grow for extended periods of time. This applies to all advanced maturers, whether they are obese or not.

Treatment options such as growth modification are most successful when used immediately before peak height velocity growth. If obesity and skeletal maturation were significantly associated, orthodontists would need to consider the patient's BMI in addition to sex when deciding among various treatment options. Further studies with larger sample sizes are warranted to investigate the trend found here. Additionally, studies should assess whether ethnicity effects skeletal maturation, and Fishman's intermediate adolescent stages of maturation should be included also.[41] The current literature does not define a relationship between cranial base growth velocity and skeletal maturity. Hand-wrist radiographic assessment of skeletal maturity for facial growth prediction should be reassessed to include bone staging in addition to the ossification events.[47]

CONCLUSIONS

1. Skeletal age determined by a hand-wrist radiograph and chronologic age correlate moderately well.
2. Skeletal age was not significantly accelerated in children with increased BMI values, but a trend was evident.
3. The difference between skeletal age and chronologic age significantly decreased in older subjects.

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