

Predicting Adult Stature Without Using Skeletal Age: The Khamis-Roche Method

Khamis and Roche developed a modification of the Roche-Wainer-Thissen (RWT) stature prediction model in which the skeletal age was not used to calculate the predicted height. The parameters considered for the calculation of predicted adult height were current height and weight and midparental height, ie, the mean of the parents' heights. They obtained these data from a group of white American children (223 males and 210 females) residing in southwest Ohio; they were participants of the Fels Longitudinal Study and were followed with measurements of height and weight every 6 months from the age of 3 years until 18 years. The stature of each parent also was measured. Linear regressions of adult stature (considered for their purpose as the stature attained at age 18 years) were calculated using the 3 variables. The following equation was used: predicted adult stature = $\beta_0 + \beta_1$ stature + β_2 weight + β_3 midparental stature. The tables for males and females list the intercepts (β_0) and the coefficients of the 3 variables (β_1 , β_2 , and β_3 , respectively) for each chronologic age, expressed in 6- and 12-month intervals. The accuracy of the prediction method was measured using the median absolute deviation (MAD), which is the median of the absolute differences, regardless of the signs, between actual and predicted stature at age 18. The smaller the MAD, the better the accuracy. There was only a slight deterioration of the accuracy with this method as compared with RWT, which uses estimations of skeletal age.

Khamis HJ, Roche AF. *Pediatrics* 1994;94:504-507.

Editor's comment: The authors present an ingenious method of predicting final adult stature in children without using skeletal age. This method might be a useful adjuvant in the clinic and allows comparisons of predicted adult height by anthropometric determinations. Large discrepancies between the 2 methods may indicate inaccurate measurements and/or inaccurate bone

age estimation. Two problems may still preclude its use in a pediatric endocrine setting: first, its accuracy seems to be worse in the peripubertal years, especially in males, where it overestimates predicted heights. Second, the predictability is good only in the absence of pathologic conditions that alter the potential for linear growth. **Thus, caution must be exercised if it is used as an adjuvant diagnostic tool in children with short or tall stature.** However, it may be of a great value as a descriptive instrument for prediction of adult stature in normal children, and in epidemiologic studies of population when adult height predictors without bone age estimates may be an important index of health status.

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2nd Editor's comment: After publication of this article, the authors noted an error in Tables 1 and 2 presenting the weight coefficients necessary for calculating adult stature with the above equation. The decimal point was displaced one space to the left. The weight coefficients may be corrected by shifting the decimal points one space to the right of their present locations. This error is to be corrected in a "Letter to the Editor" of *Pediatrics*. Readers may wish to correct the tables in the original article for their own use or watch for publication of the corrected tables in *Pediatrics*.

As the authors point out, the described method for prediction of adult height is based on measurements of healthy white children who are growing normally and, therefore, strictly applicable only to this group. This reviewer seldom predicts stature in normal children, because if the prediction is below that which the parents desire, pressure for intervention—no matter how futile—may be increased.

Allen W. Root, MD

Growth of Short Normal Children in Puberty Treated for 3 Years With Growth Hormone Alone or in Association With Gonadotropin-Releasing Hormone Agonist

Thirty early pubertal short normal subjects received growth hormone (GH) at 0.1 IU/kg/d, 6 d/wk (~0.2 mg/kg/wk) for 3 years. These included 16 males, aged 14.4 ± 0.8 years, and 14 females, aged 12.2 ± 1.2 years. All were at pubertal stage 2 or 3, with slow pubertal growth (4.2 ± 1.2 cm/y) and a mean bone age delay of 2 years. There was no detected GH deficiency or other cause for short stature. Their mean birth length was 48.6 to 49.5 cm at term; the mean of midparental heights was -0.6 to -0.8 standard deviations (SD) below the mean of the general adult population. They were randomized in 2 groups: group A received GH alone; group B received gonadotropin hormone-releasing hormone agonist (GnRHa) plus daily GH injections for 2 years, and for year 3.

The annual growth velocity (GV) increased during the first year in both groups and sexes, the increase being significant ($P < 0.01$) in group A only. The patients of group A maintained an improved GV in the second year, and then returned to pretreatment GV in the third year, while completing their sexual development and bone maturation. Their height, expressed as SD score (SDS) for bone age, improved in the first 2 years but decreased thereafter. Group B patients returned to pretreatment GV in the second year, and demonstrated no significant

improvement when treated with GH alone during the third year of the study. They had no significant progress of height for age at any time. Their bone maturation, slow when they were receiving GnRHa, accelerated when sexual development resumed.

At the end of the 3 years, height, expressed as SDS for age, improved in group A from -2.5 ± 0.6 SD to -1.5 ± 0.4 SD in males ($P < 0.05$) and from -2.8 ± 0.5 SD to -2.1 ± 0.9 SD in females (NS). Expressed as SDS for bone age, mean height slightly improved in males (NS) but not in females. In both groups and sexes, the mean predicted height according to Bayley and Pinneau was only slightly increased at the end of 3 years on GH, with a gain of 2 to 5 cm on the average. There was a wide interindividual variability in these results within each group. Pretreatment characteristics of the patients did not account for individual differences. Annual measurement of plasma insulin-like growth factor 1 showed different degrees of increase, not correlated with any parameter of the patients' growth.

The authors drew the following conclusions: (1) Inhibiting sexual development in short early pubertal subjects has no advantage. This was previously demonstrated with GnRHa alone (see *GGH* 1993;9[4]:13), and now is confirmed for GnRHa plus GH. (2) GH alone, at the dose used, can accelerate for