

Gender Differences of Final Height Contributed by Parents' Height Among Healthy Individuals

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Key Words:corrected midparental; height; final parental height; gender difference; growth evaluation; target height

Background: Estimation of children's final height is of great interest for pediatric endocrinologists in diagnosing and evaluating the treatment of short stature. The current study was performed to characterize the feature of offsprings' final heights by their parents' heights by gender in Taiwan.

Methods: Healthy participants aged 25e35 years who underwent health examinations were recruited for body height measurements with standard processes regulated by a protocol and were enquired about their parental peak adult heights in 2003e2004. Differential contributions from each parent to the tallest/shortest child's height in the family were then assessed with simple linear regressions with scatter plots by gender. Meanwhile, statistical comparisons with the corrected midparental height method and final height for parental height model were performed.

Results: A total of 1113 male and 1036 female participants were enrolled. The fathers' height contributed the most to the tallest son's height in the family (adjusted $R^2=0.20$), and mother's height contributed the most to the shortest daughter's height in the family (adjusted $R^2=0.18$). Specifically, the final height for parental height line worked better for the contribution of midparental height to the tallest son's height in prediction.

Conclusions: For clinical practice, our results provided a reasonable estimation of final heights among local Taiwanese population and are also applicable for the evaluation of growth hormone replacement therapies for patients with short stature of non-growth-hormone defect.

1. Introduction

The final body height for adults is a polygenic trait influenced by numerous determinants, including nutrition; tempo of maturation; and genetic and environmental factors during fetal life, childhood, and adolescence.^{1e5} Estimation of a child's final height is of major significance for pediatric endocrinologists in diagnosing, evaluating the need for therapeutic intervention, and responding to parents' counseling.⁶ Stature of children is noticeably influenced by parental height,^{3,7,8} and meanwhile, the genetic basis of adult height is fairly perceptible in monozygotic twins who grow up apart.³ Genetic potential height or target height is usually determined as a function of parental height.^{9e12} Corrected midparental height (CMH) method computes the target height by adding or subtracting 6.5 cm to the mean of the parental heights for boys or girls, respectively,⁹ which has been commonly used by clinicians in the evaluation of growth-promoting therapies on growth hormone (GH)-deficient¹³ and non-GH-deficient children with short stature.^{14e16} However, midparental height might contribute more during earlier stage in an offspring's life¹¹ and be limited for assessing short children of parents with extreme height.¹⁷ Another approach proposed for estimating target height is known as the final height for parental height (FPH) model.¹⁰ The FPH model is preferred for target-height estimation among Swedish children, as the CMH method leads to an underestimating bias of about 6 cm for children with short parents.⁹ One study compared the validities of CMH and FPH methods for estimating target height in Hong Kong Chinese children.¹² On average, this population was shorter than the Swedes by more than 10 cm. Their secular increase in height over the two generations, however, was 4.2e4.8 cm, which was much greater than that of the Swedes (0.7e1.0 cm). Taiwanese people have experienced a dramatic socioeconomic transformation in the past 5 decades. Thus, the association between parental height and children's final height may also have changed.¹⁸ The aim of this study was to suggest a new approach to characterize the feature of tallest/shortest boys' and girls' final heights, predicted by the height of their parents in a large sample of healthy Taiwanese who were born in the 1970s. Our results may provide further insight into the basis for enhancing accuracy in the estimation of target height by each gender and further evidence of differential contribution from male/female parents for the achieved final height for boys and girls.

2. Methods

2.1. Study setting During the period from July 1, 2003, to June 30, 2004, the investigators recruited

ted 1229 families with healthy offspring aged between 25 years and 35 years for health examinations at a medical center in central Taiwan. Age, sex, height, and examined medical and family histories were recorded at clinical visits. Families with either parents or offspring having hypopituitarism, hypothyroidism, chromosomal abnormality, history of poliomyelitis, antenatal betamethasone exposure, or malnutrition were excluded from our analyses.

2.2. Data collection

Data about offspring's body heights were collected by a questionnaire after the standard measurements of their parents' heights with a calibrated Harpenden stadiometer (the height bar on a standard hospital scale) by a well-trained staff in health examinations. Parents were requested to report their adult peak height, number of offspring in each gender, and height of their tallest/shortest sons and daughters. Thus, each family provided just one set of height information to avoid the violation of independent observation assumption in statistics. Self-reported peak adult height for parents was obtained, because most of these individuals were in their 50s and 60s. They had experienced aging-related decreases in height.

Midparental height was defined as the average of father's and mother's reported heights. This study was approved by the hospital ethics committee, and informed consent was obtained from all participants. 192.3. Statistical analyses Two analytical schemes of attempts were taken for this study. We tried to distinguish the differential contributions from each parent to the tallest/shortest child's height in the family by gender. If there was only one son or daughter in a family, the only son or daughter was taken as the shortest and also the tallest one in the family for analyses. Simple linear regressions were used for the estimation of target heights of the offspring by parental heights. The explainable variation of independent variables, parental heights, with the dependent variables, children's final heights, was expressed by adjusted R^2 , the R^2 adjusted for the number of parameters involved in the model. Scatter plots with linear regression lines were used to depict the distribution of children's final height over parental heights. B values (slopes), intercepts, and their 95%

confidence intervals were estimated for the effect of parental height on children's final height. Meanwhile, for the purpose of detecting the differences between models of parental heights predicting tallest/shortest children in the family, statistical tests for comparing intercepts and slopes between regressions were performed. Then, the comparisons of these scenarios of tallest/shortest son/daughter predicted by midparental height with the CMH method²⁰ and the FPH model¹⁰ were performed. CMH method predicts the target height by adding 6.5 cm to the midparental height in boys or subtracting 6.5 cm from the midparental height in girls.²⁰ The FPH model for estimating target height (Y) as a simple linear function of midparental height (X) in centimeters (boys: $YZ = 45.99 + 0.78X$; girls: $YZ = 37.85 + 0.75X$) was based on a Swedish series.¹⁰ SPSS 13.0 for Windows (SPSS Inc., Chicago, IL, USA) and Stata/SE version 8.0 (Stata Corp LP, College Station TX, USA) were used to perform the statistical analyses, and the significance level (alpha value) was set as 0.05. 184P.-H. Su et al

3. Results

3.1. General features of the study participants A total of 1113 male participants, 1036 female participants, and their parents were enrolled for the analyses. The mean and standard deviation for tallest/shortest sons, daughters, their fathers, and mothers were 173.19(5.20)cm/168.30(5.77)cm, 161.65(4.87)cm/156.99(4.70)cm, 168.34(5.59)cm, and 157.04(4.85)cm, separately. Among these families, the average numbers (standard deviation) of sons and daughters were 2.42(1.25) and 2.55(1.39), respectively, with a range of 1 to 9 sons or daughters.

3.2. Predicting offspring's final height by parent's height The statistics for predicting tallest/shortest child in the family by parental heights with linear regressions are presented in Table 1. All the p values for testing intercepts and slopes differing from zero were less than 0.01. Father's height contributed the most to the tallest son's height in the family (adjusted R^2 0.20) and the least to the shortest daughter's height in the family (adjusted R^2 0.11). Nonetheless, mother's height contributed the most to the shortest daughter's height in the family (adjusted R^2 0.18) and the least to the shortest son's height in the family (adjusted R^2 0.13).

3.3. Figures for fitted regression lines and comparison with existing height estimation methods Figures 1A-E illustrate the height scenarios for the distribution of each observation and fitted linear regression lines. For better visual impression, vertical and horizontal scales for all the figures were unified (from 150 cm to 200 cm). In terms of the comparison of intercepts and slopes among models of parental heights predicting tallest/shortest children in the family, all the intercepts were significantly different from each other (all $p < 0.01$), but only the slope differences between fathers' height predicting tallest and shortest sons (i.e., 0.42 vs. 0.37) revealed borderline statistical significance ($p = 0.07$, details not shown). Figures 2A-E present the predicting scenarios of midparental height to tallest/shortest sons and daughters with CMH and FPH regression lines as benchmark. When we used the Taiwanese midparental height to predict the tallest son's height (adjusted R^2 0.32), the regression line and FPH line almost overlapped from 160 cm to 180 cm of midparental height (Figure 2A). In Figure 2, the prediction line of the current study is parallel to the FPH line constantly all the way. In addition, CMH prediction was close to the present study participants at lesser midparental heights, but it shifted toward the FPH line at greater midparental heights. Figure 2C illustrates the prediction of the contribution of midparental height to the tallest daughters' heights in the study families, comparing the CMH and FPH lines. They differ the most at low midparental height, and then they reach a common prediction point around the midparental height of 178 cm. Finally, the pattern shown in Figure 2D was very similar to that in Figure 2B. The distance between the present study line and FPH did not alter much from the midparental height of 150 to 180 cm. Its CMH line was close to the present study line at lesser midparental height, but shifted toward FPH line at greater midparental height. In summary, the FPH line performs

med a better prediction of the contribution of midparental height to the heights of the tallest sons in the families of the present study, and the achievements varied in other scenarios. To ascertain the extent, specific patterns in the observations among the Taiwanese population for final height prediction with respect to the tallest/shortest sons/daughters were characterized by our study results.

4. Discussion With a huge number of healthy participants, we found that father's height contributed the most to the tallest sons' height in the family and mother's height contributed the most to the shortest daughter's height in the family.

Furthermore, in the present study, we compared the linear regression lines using Taiwanese midparental height to predict tallest/shortest sons' and daughters' heights with CMH and FPH regression lines. Specifically, the FPH line performed a better prediction on contribution of midparental height to the tallest son's height in the family but not in the other scenarios. We found that the generational Table 1 Predicting the tallest/shortest sons' and daughters' final heights by parental heights with simple linear regressions*

Variables	Sons' heights in cm (Y; n=113)	Daughters' heights in cm (Y; n=106)					
Tallest son	Shortest son	Tallest daughter	Shortest daughter	Father (X)	Adjusted R ²	Z	0.20
Adjusted R ²	0.13	Adjusted R ²	0.13	Adjusted R ²	0.11	Intercept (cm) (95% CI)	102.40 (94.12e110.68)
Slope (95% CI)	0.42 (0.37e0.47)	0.37 (0.32e0.43)	0.31 (0.26e0.36)	0.28 (0.23e0.33)	Mother (X)	Adjusted R ²	0.14
Adjusted R ²	0.14	Adjusted R ²	0.13	Adjusted R ²	0.15	Adjusted R ²	0.18
Intercept (cm) (95% CI)	109.26 (100.09e118.43)	99.82 (89.58e110.06)	101.14 (92.24e110.04)	92.33 (83.90e110.77)	Slope (95% CI)	0.41 (0.35e0.47)	0.44 (0.37e0.50)
Slope (95% CI)	0.41 (0.35e0.47)	0.44 (0.37e0.50)	0.39 (0.33e0.44)	0.41 (0.36e0.47)			

*All p values less than 0.01 for testing intercepts and slopes differing from zero.

CI Z confidence interval.

Final height predicted by parents' height 185

increases in height of Taiwanese parents and their offspring were between those of Hong Kong Chinese and Swedish people, all born in similar years of the 1970s. The CMH method underestimated the final height, whereas the Swedish FPH model may overestimate it.¹⁸ The contribution from a parent to the tallest/shortest child in the family varied. Nonetheless, the performance of FPH model favored the prediction of midparental height to the tallest son in a family among this Taiwanese population.

The major advantage of the present study was the relatively large number of participants in Taiwan. Although factors about diet intake, physical activity, and puberty age, which also cooperatively determine final height, were not available, the approach of innovative attempts by predicting the final height of the tallest/shortest boys/girls provided insight into this issue in Taiwanese population.

In clinical settings, the major application of target-height assessment on endocrine conditions would be in Figure 1(A) The contribution of father's height to the tallest son in family (YZ102.40t0.42X; adjusted R² 0.20; p value for slope < 0.01; nZ1113). (B) The contribution of father's height to the shortest son in family (YZ105.26t0.37X; adjusted R² 0.13; p value for slope < 0.01; nZ1113). (C) The contribution of mother's height to the tallest son in family (YZ109.26t0.41X; adjusted R² 0.14; p value for slope < 0.01; nZ1113). (D) The contribution of mother's height to the shortest son in family (YZ99.82t0.44X; adjusted R² 0.13; p value for slope < 0.01; nZ1113). (E) The contribution of father's height to the tallest daughter in family (YZ109.58t0.31X; adjusted R² 0.13; p value for slope < 0.01; nZ1036). (F) The contribution of father's height to the shortest daughter in family (YZ109.72t0.28X; adjusted R² 0.11; p value for slope < 0.01; nZ1036). (G) The contribution of mother's height to the tallest daughter in family (YZ101.14t0.39X; adjusted R² 0.15; p value for slope < 0.01; nZ1036). (H) The contribution of mother's height to the shortest daughter in family (YZ92.33t0.41X; adjusted R² 0.18; p value for slope < 0.01; nZ1036).

determining short stature and precocious puberty. For the issue of short stature of non-GH-defect patients, our study results provide a more reasonable range of final heights among local population, rather than using the biased universal CMH estimations. Meanwhile, for the patients with GH defect, our research outcome is also essential for the evaluation of GH replacement therapy and fine-tuning on prescription dosage. As for precocious puberty, because of early epiphyseal plate development resulting in its premature closure and short adult height, gonadotropin-releasing hormone analog is used for delaying the progress and then letting patients approach ideal final height. Thus, further understanding about the normal range of final height of Taiwanese population facilitates the assessment of gonadotropin-releasing hormone treatments in clinics. Meanwhile, limitations of the present research also exist. First, this is a retrospective study, not a longitudinal one. Second, parental height was self-reported in questionnaires, which might result in information bias. Third, heights among siblings within a family vary. If there was only one boy/girl in a family, he/she served as the tallest/shortest boy/girl in the prediction model at the same time. The more the kids in a family, the better is the possibility of finding participants with extreme heights. Consequently, bigger families might provide more exceptionally tall or short participants in the final height prediction model as suggested by the present research. Lastly, variables of potential confounders about nutrition, menarche age of female offspring, and exercise were not available in the large-scale study for analyses, which might decrease the precision of prediction of offspring's final height. As our suggestions for further studies in future, differential effects of environmental stress on adult height by gender are of major interest. 4 Follow-up studies of populations of different race might be beneficial, in terms of active data collection and final height measurements during the study period. Furthermore, some more data on

Figure 1 (continued).

Figure 2 (A) The contribution of midparental height to the tallest son in family ($YZ49.13 \pm 0.76 X$; adjusted $R^2 = 0.32$; p value for slope < 0.01 ; $n = 1113$), comparing the FPH and CMH regression lines. (B) The contribution of midparental height to the shortest son in family ($YZ48.32 \pm 0.74 X$; adjusted $R^2 = 0.24$; p value for slope < 0.01 ; $n = 1113$), comparing the FPH and CMH regression lines. (C) The contribution of midparental height to the tallest daughter in family ($YZ60.43 \pm 0.62 X$; adjusted $R^2 = 0.25$; p value for slope < 0.01 ; $n = 1036$), comparing the FPH and CMH regression lines. (D) The contribution of midparental height to the shortest daughter in family ($YZ57.69 \pm 0.61 X$; adjusted $R^2 = 0.26$; p value for slope < 0.01 ; $n = 1036$), comparing the FPH and CMH regression lines. CMHZ corrected midparental height; FPHZ final height for parental height. environmental factors would be helpful to study issues regarding gene-environment interactions. Twin studies or adaptation studies could provide advanced insight for such study.

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