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The pattern of growth and growth retardation of patients with hypophosphataemic vitamin D-resistant rickets: a longitudinal study

R. Steendijk[1] **and R. C. Hauspie**[2]

[1]Department of Paediatrics, Academic Medical Centre, University of Amsterdam, The Netherlands
[2]National Science Foundation (Belgium), Free University of Brussels, Belgium

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Abstract. Growth in height of 16 patients (5 boys and 11 girls) with hypophosphataemic rickets (HR) was studied in a longitudinal survey. The data shortly before and during puberty were analysed on the basis of Preece Baines curves, fitted to the original data; for the analysis at the age of 5 years, the original data were used. It appeared that the overall shape of the individual and average growth pattern could be adequately described by the Preece Baines method. The results further showed that from the age of 5 years onwards, average height was approximately two standard deviations below the normal mean for Dutch children. The patients showed a normal pubertal growth spurt which was, in general, insufficient to restore the growth retardation already established before adolescence. The four children who did show catch-up growth between the age of 5 years and adulthood had minimal rachitic lesions. The greater impact of the disease on growth in early childhood than on adolescent growth could be explained by the fact that HR mainly affects the growth of the legs, the major contributor to body size in early childhood. Finally, it was found that the difference between bone age, as determined by the Tanner Whitehouse (TW_2)-method, and chronological age was not significant and the adult height in all patients except two could be adequately predicted from bone age and height.

Key words: Growth – Hypophosphataemic rickets – Curve fitting

Introduction

Hypophosphataemic vitamin D-resistant rickets (HR) is a disease characterized by rachitic changes in the growth-plates of the long bones, skeletal deformities and disproportionate short stature. Plasma calcium is normal, plasma inorganic phosphate is reduced as a result of impaired tubular reabsorption of this electrolyte, and plasma alkaline phosphatase is usually moderately increased [5]. The disease is hereditary, but also occurs sporadically: the mode of inheritance is X-linked dominant.

Administration of large doses of vitamin D, dihydrotachysterol (DHT) 1,25$(OH)_2$ vitamin D_3 or 1-α(OH) vitamin D_3 is usually followed by a rise of plasma phosphate to less subnormal values, but growth rate remains unaffected [4] except in the least severe cases where it may increase slightly [19]. Addition to this treatment of high doses of phosphate, given orally five times per day, results in intermittent attainment of normal plasma phosphate levels, further improvement of the rachitic signs and usually an increase in growth rate [4, 12, 14]. Recently Balsan and Tieder [1] found in 40 children that treatment with 1-α(OH) vitamin D_3 plus oral phosphate had a more favourable effect on growth than administration of vitamin D_3 and phosphate, or 25(OH) vitamin D_3 and phosphate.

As obtained from cross-sectional data in untreated cases, average stature is 2 standard deviations (SD) below the normal mean with a SD approximately equal to that in normal children [21]. The short stature is mainly, but not exclusively, caused by impairment of growth in the legs [20]. Herweijer and Steendijk [11] found in 13 patients, who had reached adult height, a significant negative correlation between the severity of the radiological signs of rickets during growth and attained adult height, implying a relation between the severity of rickets and growth rate. Possibly as a result of the increased growth rate in puberty, the rachitic signs at the metaphyses may temporarily become more severe and deformities may appear to become more obvious [18]. It is possible that this increase in severity may in turn impair growth rate.

Longitudinal growth data of patients with HR, including the pubertal growth spurt and the attainment of adult height have occasionally been studied, but not thoroughly analysed [2, 3, 18, 19]. A study in which longitudinal growth data have been analysed, mainly to document in

Offprint requests to: R. Hauspie, Laboratorium Antropogenetica, Vrije Universiteit Brussel, Pleinlaan 2, B-1050 Brussel, Belgium

Abbreviations: CA = chronological age; DHT = dihydrotachysterol; HR = hypophosphataemic, vitamin D-resistant rickets; PAH = predicted adult height; PB1 = Preece Baines model I; SD = standard deviation; sds = standard deviation score(s)

Table 1. Severity of the rachitic signs on X-ray examination, some details on treatment and some observations on the growth of 16 patients with hypophosphataemic rickets

Patient	Sex	Rachitic signs on X-ray during therapy		Oral phosphate from age (years)	$1,25(OH)_2D_3$ or 1α-$(OH)D_3$ from age (years)	Increase of deformities at puberty	Catch-up growth	Preece Baines analysis	
		Slight[a]	Moderate[a]					Age range	First year
1	M	×		–	–	–	–	10.8–20.2	1967
2	M	×		–	–	–	–	8.8–20.3	1953
3	M		×	–	–	×	–	8.2–20.0	1967
4	M	×		–	–	–	×	9.3–20.4	1967
5	M	×		–	–	–	×	8.0–20.9	1965
6	F	×		–	–	×	–	8.6–17.7	1955
7	F		×	9.8	14.7	×	–	9.8–16.9	1975
8	F	×		10.9	–	–	×	9.2–15.7	1977
9	F	×		–	–	–	–	8.5–17.9	1967
10	F		×	5.1	12.5	–	–	7.6–18.0	1976
11	F		×	2.7	10.0	–	–	4.9–15.8	1975
12	F	×		6.9	–	–	×	9.1–15.5	1978
13	F	×		–	–	–	–	8.6–16.6	1963
14	F	×		11.7	16.0	–	–	8.4–17.4	1972
15	F		×	–	–	×	–	9.2–19.0	1956
16	F		×	–	–	×	–	7.5–16.0	1963

[a] *Slight,* the worst lesion in the wrist consisted of cupping of the ulnar metaphysis, without cupping of the radial metaphysis; *moderate,* cupping of both metaphyses in the wrist; widening of the epiphyseal plate, slight or no fraying of the metaphysis

the effect of different therapeutic regimens on growth has recently been published [1].

The aim of the present study was to analyse the growth curves of patients with HR to find out whether growth rate was mainly impaired before or during puberty and whether episodes of conspicuous relative increase or decrease in height, i.e. the attainment of a higher or lower percentile, had occurred. In addition to these growth studies we have followed the development of bone age and predicted adult height (PAH) in these patients. To our knowledge, this has not been done before.

Patients

The sample consisted of 16 patients, 11 girls and 5 boys. Of these patients, 5 girls and 3 boys were familial cases. None of the patients had glycosuria or aminoaciduria. Plasma creatinine was normal in all patients throughout the study. All but two patients (patients 4 and 9) had visible deformities of the legs on first examination. Severe deformities of the legs were present in one boy (patient 2) and in one girl (patient 7). None of the patients had scoliosis or craniostenosis. Rachitic signs on X-ray films of the wrists and knees were present in all patients in varying degrees. After institution of therapy, these improved in all patients and became slight or inconspicuous in all boys and in 6 girls (Table 1); in the other patients, some cupping of the metaphyses of the ulna and, to a lesser extent, the radius persisted. In 5 patients, a clinically apparent increase of the deformities occurred at the beginning of puberty (Table 1). All data were collected during treatment which initially consisted of high doses of vitamin D_3 (0.30–3.0 mg/day). Between 1965 and 1967, this was replaced by DHT (0.4–1.2 mg/day). From 1974 onwards, oral phosphate (4.0–6.5 g PO_4 per day divided in five doses) was added to the regimen in the girls. Since the rachitic signs in the boys were inconspicuous no phosphate was administered. From 1980 onwards, DHT was replaced by $1,25(OH)_2$ vitamin D_3 (0.5–2.0 mcg/day) or 1-α(OH) vitamin D_3 (1.0–4.0 mcg/day) in four of the six patients (all girls) who were still growing at that time.

Thus, 12 of the patients never received vitamin D metabolites and none of the other 4 patients (all girls) received these metabolites before puberty. Five of the girls never received oral phosphate, whereas in the other girls the addition of oral phosphate had been started between the ages of 2.7 and 11.7 years (Table 1), i.e. before the beginning of puberty.

Methods

Data on growth in height were collected at regular intervals of 3–4 months in the period July 1949 – February 1988. All measurements were taken or supervised by one of us (R.S.). In the period before 1967, height was measured with a simple stadiometer with a precision of 5 mm. In the period after 1967, height was measured with a precision of 1 mm, using a Harpenden type stadiometer (Holtain Ltd., Crymych, Wales). At the time of the last measurement, six of the patients were still growing slowly at a declining rate; the others had reached their adult height.

To obtain comparable data, individual growth curves were estimated by fitting the Preece Baines model 1 (PB1) to each individual's height for age data [13]. The quality of the fit was verified by examining the residual variances, while checking for systematic bias in the residuals was done by means of the runs test [17]. The PB1 fits were used to derive the following biological parameters; AH, adult height; T_1, H_1, V_1, age, height and velocity at take-off, i.e. the point of minimal pre-pubertal growth velocity. T_2, H_2, V_2, age, height and velocity at peak velocity in puberty; AG, adolescent growth (= AH-H1). In this study puberty was supposed to begin at T_1. Instantaneous growth velocity was obtained by taking the mathematical first derivative of the curve fitted to the height-for-age data.

In addition to the PB1-derived data, empirical growth velocities were calculated according to Tanner and Davies [25]. These actually correspond to whole-year velocities converted from the increments taken over the intervals of not less than 0.8 and not

more than 1.2 years, and plotted at the centre of the respective intervals.

Although the PB1 model generally fits growth data for height in the age range of 2–18 years without systematic bias [8, 9], it tends to underestimate age at take-off. This bias can be minimized by excluding data from the early childhood and mid-childhood period. In this study, the cut-off point for excluding early growth data has been determined empirically for each subject by comparing the growth velocity curve of the PB1 model fitted to subsets of data, varying in the lower bounds of the age range, with the graph of the subject's empirical yearly increments in height. The subset of data yielding the PB1 estimate of age at take-off nearest to the graphical estimate of age at take-off was retained for further analysis. The age ranges defined in this way varied from 6.4 to 12.9 years (average = 9.5 years ± 1.8 SD). The average range of the corresponding chronological ages of the patients was 8.5–18 years. The average number of measurements per subject was 28 ± 7.

Mean-constant curves (distance and velocity) [7, 23], representing the growth pattern of the "typical average" child in the sample, were constructed and have been used to break down the adult height differences (D) between boys and girls into three additive components [10, 26]: DA, difference in adolescent growth; DP, difference in pre-pubertal growth, i.e. the difference in height between boys and girls at the girls' take-off; DT, the amount of growth achieved by the boys, due to their later onset of the adolescent growth spurt, i.e. the boys' gain in height between age at take-off of the girls and their own age at take-off.

These three components add up to the sex difference in adult height, thus D = DA + DP + DT

PB1 estimates of height at take-off and adult height were available in all patients. In the absence of appropriate longitudinal growth standards for the Dutch population, we used the cross-sectional Dutch nation-wide growth survey [15] as a reference to express height at take-off and adult height in terms of standard deviation scores (sds). We could thus estimate the relative increase or decrease of height at the beginning and at the end of puberty with respect to the family of cross-sectional standard curves. To obtain some insight in growth before puberty we decided to use height at approximately 5 years of age (5.0–5.3 years), which was available in nine children. Since the cut-off point for excluding early growth data for the PB1 model was over 5 years, except in one patient, the actual growth data, expressed as sds, were used at this age.

Between 5 and 18 years of age, bone age was determined in all patients on several occasions (between 2 and 8 times, average 4.5 times per patient), using the Tanner-Whitehouse (TW2-RUS) method for bone age [28]. The total number of bone age assessments was 72. Advance or delay in skeletal maturation was expressed as the difference between bone age (RUS) and chronological age (CA). In all patients adult height was predicted at least twice on the basis of RUS, CA and height, according to the method of Tanner et al. [27]. Estimations of bone age and predictions of adult height were carried out by one of us (R.S.).

Finally, age at menarche was recorded in the girls.

Results

PB1 fit to the growth of HR patients

The PB1 model described the growth pattern of these HR patients fairly accurately, the pooled residual variance being 0.190 cm^2 (d.f. 125) for boys and 0.142 cm^2 (d.f. 246) for girls. The overall pooled residual variance (boys + girls) was 0.158 cm^2 (d.f. 371), which was significantly below the value of 0.41 cm^2 for girls reported by Hauspie et al. [8] ($P < 0.001$).

Out of the 16 subjects in our sample, two were significant ($P < 0.05$) for the runs test [17]. In one of them (patient 4) the PB1 model could not satisfactorily cope with the quite sharp adolescent growth spurt (maximal increment 10 cm/year) and the preceding rise in growth velocity. The other subject (patient 16) showed some irregularities in her growth pattern, which resulted in a slightly biased fit. Despite this systematic bias, these two subjects still showed a reasonably acceptable overall fit with relatively low residual variances (respectively 0.206 and 0.164 cm^2) and without major bias in the estimation of take-off.

The median of the difference between the estimate of final height and the last measurement was −0.04 cm

Table 2. Means and standard deviations (SD) of the PB1 derived biological variables for HR patients

Parameter	Boys ($n = 5$)		Girls ($n = 11$)	
	Mean	SD	Mean	SD
AH: adult height	170.4	8.6	156.1	7.6
T1: age at take-off	11.9	1.4	10.3	0.8
H1: height at take-off	137.7	6.4	130.8	8.2
V1: velocity at take-off	4.4	0.7	4.4	0.7
T2: age at peak velocity	15.2	1.3	12.9	0.6
H2: height at peak velocity	156.3	7.3	144.4	7.6
V2: peak height velocity	7.3	1.2	6.3	0.8
AG: adolescent growth	32.7	5.7	25.3	5.4

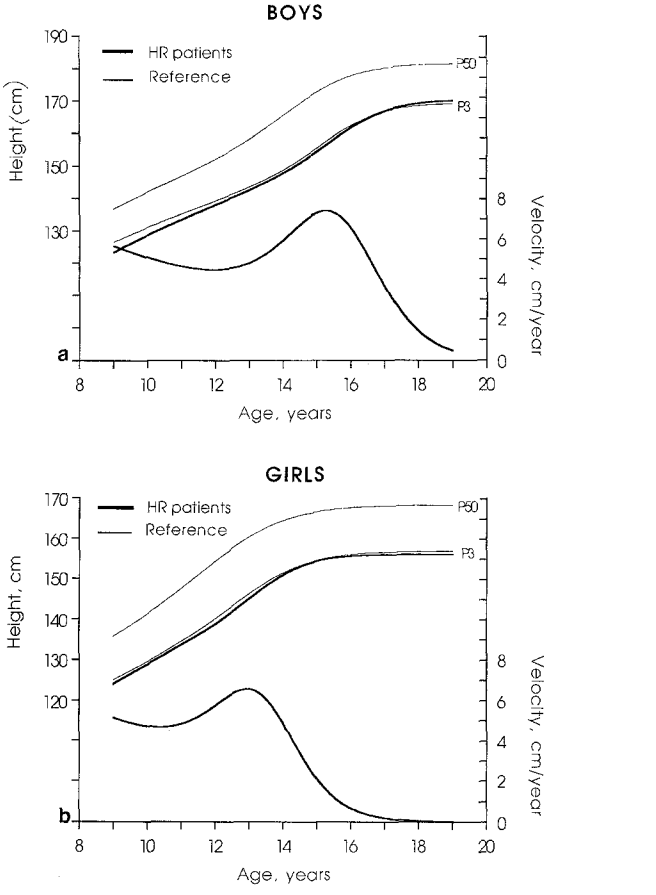

Fig. 1. Mean-constant distance and velocity curves for height of Preece Baines model I fitted to 5 HR boys (**a**) and 11 HR girls (**b**), compared to the P50 and P3 height-for-age percentiles of Dutch nation-wide cross-sectional standards [15]

Table 3. Data for age height (observed measurement and sds) at approximately 5 years of age, at take-off (to) and at adulthood (ad). The differences in sds for height between adulthood and take-off (ad-to), and between take-off and 5 years of age (to-5 years)

No	Sex	Approximately 5 years			Take-off			Adulthood		Differences	
		Age	Height	sds	Age	Height	sds	Height	sds	ad-to	to-5 years
1	M	5.2	102.5	−2.43	10.8	131.0	−2.43	167.4	−3.18	0.25	0.00
2	M	5.3	106.0	−1.72	12.4	141.3	−1.74	171.3	−1.60	0.14	−0.02
3	M	5.0	98.1	−3.11	12.7	131.6	−3.26	157.4	−3.67	−0.41	−0.15
4	M	5.1	106.5	−1.42	10.0	138.5	−0.61	178.7	−0.49	0.12	0.81
5	M	5.0	104.0	−1.83	13.6	146.1	−1.95	177.3	−0.70	1.25	−0.12
6	F				9.6	122.6	−2.77	151.0	−2.79	−0.02	
7	F				10.7	126.5	−2.78	150.8	−2.82	−0.05	
8	F				10.6	139.3	−0.82	168.2	−0.02	0.80	
9	F				10.6	139.2	−0.83	164.4	−0.63	0.20	
10	F	5.0	104.3	−1.64	9.8	128.2	−1.98	158.1	−1.64	0.34	−0.34
11	F	5.0	104.2	−1.67	8.4	121.4	−1.96	157.5	−1.74	0.22	−0.29
12	F	5.0	107.9	−0.87	10.6	144.5	−0.04	166.4	−0.31	−0.27	0.83
13	F				11.2	130.5	−2.55	152.2	−2.60	−0.05	
14	F				10.7	138.8	−0.97	155.2	−2.13	−1.16	
15	F				10.6	126.8	−2.69	149.2	−3.08	−0.39	
16	F	5.0	95.0	−3.67	10.3	121.6	−3.36	144.3	−3.87	−0.51	0.31
Means boys				−2.10			−2.00		−1.73	0.27	0.10
SD				0.67			0.97		1.28	0.60	0.40
Means girls				−1.96			−1.89		−1.97	−0.08	0.13
SD				1.20			1.06		1.23	0.51	0.55
Means all				−2.04			−1.92		−1.89	0.03	0.11
SD				0.88			1.00		1.21	0.55	0.44

(min. −0.18, max. +2.44 cm). Only one girl (patient 11) had a rather large increase in height between the last two measurements (2.69 cm/year). PB1 estimated her final height 2.44 cm above the height at the last measurement occasion, but the plot of the height data and the fitted curve indicated that this extrapolation was acceptable.

Table 2 shows the mean values and standard deviations of a number of PB1 derived biological variables for HR patients. Decomposition of sex differences in adult height into prepubertal and pubertal components revealed that HR boys were taller than HR girls by 14.3 cm. Such as in normal children, growth rate before the onset of puberty was approximately the same in boys and girls. HR boys entered puberty 1.6 years later than HR girls, had a higher peak height velocity and added 7.2 cm more to their stature during adolescence, whilst the difference between boys and girls resulting from the later onset of puberty in boys was also 7.2 cm. These figures are derived from the mean-constant curves, reflecting the typical average child in the sample, and therefore slightly differ from the figures which can be derived from the sample means shown in Table 2. The latter are subject to phase-difference effects. Finally, pre-pubertal differences were negligible.

Figure 1 shows the mean-constant curves of HR patients. The distance curves are compared to the P50 and P3 percentiles of the Dutch cross-sectional growth standards. It is apparent that the average growth pattern of HR patients, as well as the sex differences in growth pattern, do not reveal obvious abnormalities in the shape of the overall growth curve of these patients.

Percentile changes during the growth period

Average adult height of these children was approximately 2 SD below the average height of the population, with a SD slightly larger than 1 (Table 3). The average for height at take-off and adult height, expressed as sds, were not very different. The average gain in boys was 0.27 sds while girls lost only 0.08 sds. In this period a relative loss of height occurred in one boy and seven girls; a relative gain in four boys and four girls. If we assume – arbitrarily – a difference of 0.5 sds in height to be meaningful, only one boy (patient 5) and one girl (patient 8) showed an important gain in height of 1.25 and 0.8 sds respectively. Two girls (patients 14 and 16) showed a relative decrease in height, 1.16 and 0.51 sds respectively.

Before puberty, the average height of nine patients changed only slightly between the ages of approximately 5 years and take-off (0.11 sds). One boy (patient 4) and one girl (patient 12) experienced a gain in height of respectively 0.81 and 0.83 sds in this period. Adult height in the six girls, who were treated with oral phosphate in addition to vitamin D or DHT was 159.3 ± 6.7 cm; in the 5 girls who did not receive oral phosphate, this value was 152.2 ± 7.4 cm. The difference is not significant ($P > 0.05$). The adolescent growth of these two groups was 24.04 ± 2.79 cm (−P) and 26.37 ± 6.99 cm (+P). The difference is also not significant ($P > 0.05$).

None of the five patients (Table 1), who showed a clinically demonstrable increase in leg deformities at the beginning of puberty, gained height during this period and one of them (patient 16) lost 0.51 sds.

Age at menarche

Average age at menarche of the girls was 14.25 ± 0.77 years, which was significantly later than average age at menarche in the Dutch female population ($0.01 < P < 0.05$), which was 13.28 ± 1.24 years [15]. The age at menarche occurred 1.37 ± 0.65 years after age at peak height velocity.

Skeletal age and prediction of adult height

The averge value for RUS - CA in the patients was 0.29 ± 0.84 years. This value was not significantly different from zero. In general, adult height was slightly, but not significantly, overpredicted. The difference between predicted adult height (PAH) and actual adult height was 0.54 ± 3.99 cm. Individually, the average value for PAH was overestimated in nine patients, underestimated in six patients, and correct in one patient. In 2 patients, PAH was considerably outside the normal range of prediction, by 8.12 and -9.22 cm. Although the mean value of RUS-CA and PAH-AH differed somewhat, none of these means differed significantly from zero.

Discussion

The PB1 model

This study has shown that the growth pattern of HR can be adequately described by the PB1 curve since the resulting residual mean squares were fairly low. Despite the marked reduction in absolute size and the considerable variation in the shape of the growth pattern, the PB1 model could cope well with the growth data of these patients. The PB1 model did slightly worse when short-term variations in growth velocity were present in the data. It can indeed be expected that the PB1 curve will be generally less suitable to describe growth patterns in pathological conditions with marked periods of growth retardation and/or catch-up growth.

Growth of the patients

Since longitudinal data on growth in normal Dutch children are not available, we could not compare the application of the Preece Baines method in our patients with normal controls. We decided not to use other longitudinal standards, such as the Belgian or Swiss ones for example, since adult height of those references differed considerably from that of the Dutch children and there might also be essential differences in features of the growth pattern, such as the adolescent growth spurt.

Our study confirms previous findings that the average height of the patients is approximately two SD's below the average height of the population [21]. As also found recently by Balsan and Tieder [1], most, if not all, of the growth retardation in HR patients took place before and not during puberty. In fact, judging by the height of nine of our patients at the age of 5 years, the retardation occurred even before that age. Harrison et al. [6], Stickler [22] and Schimert and Fanconi [16] observed that conspicuous growth retardation occurred during the first few years of life, whereas length of the children at birth had been virtually normal. Combining their studies and ours we can tentatively conclude that growth retardation in HR occurs very early in life. This may be related to the fact that HR mainly affects growth of the legs [20]. In normal children, pre-pubertal growth velocity in the legs is faster than in the trunk, especially during the first few years of life. In this period of rapid growth, factors which disturb growth, such as rickets, apparently have a greater effect on the legs than on the trunk, causing a disproportion between sitting height and leg length. During adolescence, when the trunk grows slightly faster than the legs [23], there was in general no further growth retardation. The appearance or increase of the rachitic deformities however which occurred in five patients in the beginning of puberty, most probably was related to the increase in the velocity of leg growth. It is of interest that these five patients lost an average of 0.28 sds of height during puberty, one of them losing as much as 0.51 sds and none of them gaining height.

In four children a gain in height, which could be called catch-up growth, was noted at some time between the age of 5 years and the end of growth. There was no obvious relation to a change in therapy, except perhaps the additon of oral phosphate to the regimen in the girls. It is probably more important however that these four patients had minimal signs of rickets, clinically as well as on X-ray films which may therefore be a condition for catch-up growth to occur.

The girl (patient 14) who lost 1.16 sds of height during puberty had a very short period of adolescent growth, which we cannot explain. Her adolescent growth was only 16.4 cm.

Age at menarche

We have no explanation for the observation that age at menarche was significantly later than in normal girls. One might be inclined to think of secular trend phenomena since the reference population was measured in 1980 and a number of the patients entered the study 25 or more years earlier. But in 1965, menarche in Dutch girls occurred at an average age of 13.42 ± 1.21 years [29] and this is also significantly earlier ($P < 0.05$) than the age in our girls.

Skeletal age and prediction of adult height

The normal values for bone age and the equally normal values for PAH in the majority of the patients are in agreement with the presence of normal control of the processes of growth in this disease. We do not know why two of the predictions fell consistently outside the normal range.

The main conclusion of this study is that growth retardation in patients with HR appears to occur in the first few years of life, probably mainly as a result of the impact of the disease on the growth of the legs.

References

1. Balsan S, Tieder M (1990) Linear growth in patients with hypophosphataemic vitamin D-resistant rickets: Influence of treatment regimen and parental height. J Pediatr 116:365–371
2. Büller HE, Tigchelaar-Gutter W, Steendijk R (1982) Improved bone structure in hypophosphataemic rickets after treatment with dihydrotachysterol and phosphate. In: Norman AW, Schaeffer K, Herrath D von, Grigoleit HG (eds) Vitamin D, chemical, biochemical and clinical endocrinology of calcium metabolism. de Gruyter, Berlin, pp 1041–1040
3. Evans GA, Arulanantham K, Gage JR (1980) Primary hypophosphataemic rickets: effect of oral phosphate and vitamin D on growth and surgical treatment. J Bone Joint Surg 62:1130–1138
4. Glorieux FH, Scriver CR, Reade TM, Goldman H, Rosenborough A (1972) Use of phosphate salts and vitamin D to prevent dwarfism and rickets in X-linked hypophosphataemia. N Engl J Med 287:481–487
5. Harrison HE, Harrison HC (1979) Disorders of calcium and phosphate metabolism in childhood and adolescence. WB Saunders, Philadelphia, pp 193–246
6. Harrison HE, Harrison HC, Lifshitz F, Johnson AD (1966) Growth disturbance in hereditary hypophosphatemia. Am J Dis Child 112:290–297
7. Hauspie RC (1989) Mathematical models for the study of individual growth patterns. Rev Epidemiol Sante Publique 37: 461–476
8. Hauspie RC, Das SR, Preece MA, Tanner JM (1980) A longitudinal study of growth in height of boys and girls of West Bengal (India) aged six months to 20 years. Ann Hum Biol 7:429–441
9. Hauspie RC, Wachholder A, Baron G, Cantraine F, Susanne C, Graffar M (1980) A comparative study of the fit of four different functions to longitudinal data of growth in height of Belgian girls. Ann Hum Biol 7:347–358
10. Hauspie R, Das SR, Preece MA, Tanner JM, Susanne C (1985) Decomposition of sexual dimorphism in adult size of height, sitting height, shoulder width and hip width in a British and West Bengal sample. In: Ghesquière J, Martin RD, Newcombe F (eds) Human sexual dimorphism. Taylor and Francis, London, pp 207–215
11. Herweijer TJ, Steendijk R (1985) The relation beween attained adult height and the metaphyseal lesions in hypophosphataemic vitamin D-resistant rickets. Acta Paediatr Scand 74:196–200
12. McEnery PT, Silverman FN, West CD (1972) Acceleration of growth with combined vitamin D-phosphate therapy of hypophosphataemic vitamin D-resistant rickets. J Pediatr 80:763–774
13. Preece MA, Baines MJ (1978) A new family of mathematical models describing the human growth curve. Ann Hum Biol 5:1–24
14. Rasmussen H, Pechet M, Anast C, Mazur A, Gertner J, Broadus AE (1981) Longterm treatment of familial hypophosphataemic rickets with oral phosphate and 1α-hydroxyvitamin D_3. J Pediatr 99:16–25
15. Roede MJ, Wieringen JC van (1985) Growth diagrams 1980. Netherlands third nation-wide survey. Tijdschr Soc Gez Zorg 63:1–34
16. Schimert G, Fanconi A (1983) Early history of familial hypophosphataemic vitamin D-resistant rickets. Helv Paediatr Acta 38:383–398
17. Siegel S (1956) Nonparametric statistics for the behavioural sciences. McGraw-Hill, London
18. Steendijk R (1971) Metabolic bone disease in children. Clin Orthop 77:247–275
19. Steendijk R (1976) Aspects of growth and bone structure in hypophosphataemic rickets. In: Bickel H, Stern J (eds) Proceedings of the twelfth symposium of the Society for the study of inborn errors of metabolism. MTP Press, Lancester, pp 93–114
20. Steendijk R, Herweijer TJ (1984) Height, sitting height and leg length in patients with hypophosphataemic rickets. Acta Paediatr Scand 73:181–184
21. Steendijk R, Latham SC (1971) Hypophosphataemic vitamin D-resistant rickets: an observation on height and serum inorganic phosphate in untreated cases. Helv Paediatr Acta 26: 179–184
22. Stickler GB (1969) Familial hypophosphatemic vitamin D-resistant rickets: the neonatal period and infancy. Acta Paediatr Scand 58:213–219
23. Tanner JM (1986) Use and abuse of growth standards. In: Falkner F, Tanner JM (eds) Human growth, Vol. 3, Plenum Press, New York, pp 95–109
24. Tanner JM (1990) Fetus into man: physical growth from conception to maturity, revised and enlarged edition, Harvard University Press, Cambridge, Massachusetts, p 66
25. Tanner JM, Davies PW (1985) Clinical longitudinal standards for height and height velocity for North American children. J Pediatr 107:317–329
26. Tanner JM, Whitehouse RH, Marubini E, Resele LF (1976) The adolescent growth spurt of boys and girls of the Harpenden Growth Study. Ann Hum Biol 3:109–126
27. Tanner JM, Landt KW, Cameron N, Carter BS, Patel J (1983) Prediction of adult height from height and bone age in childhood. Arch Dis Child 58:767–776
28. Tanner JM, Whitehouse RH, Cameron N, Marshall WA, Healy MJR, Goldstein H (1983) Assessment of skeletal maturity and prediction of adult height (TW2 method), 2nd edn. Academic Press, London
29. Wieringen JC van, Wafelbakker F, Verbrugge HP, Haas JH de (1968) Growth diagrams 1965 (in Dutch). Wolters-Noordhoff, Groningen