

Metabolic Parameters and Glucose Homeostasis in Children and Adolescents with Childhood-Onset Growth Hormone Deficiency at Time of Initial Evaluation and Retesting

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Background

Growth hormone (GH) is known to have several effects, involving bone, body composition, lipid and glucose homeostasis. However, the complex interplay between these parameters is rather poorly studied in children with childhoodonset-GH deficiency (CO-GHD).

Objectives

To investigate lipids, adipokines (leptin- adiponectin- resistin) and glucose homeostasis and their relationship with bone and body composition in children and adolescents with CO-GHD at time of diagnosis and retesting at final height.

Results contd

The correlations between the metabolic data and other clinical data

Figure 1 Scatterplots showing correlations between total cholesterol levels at retesting and age of starting childhood rhGH

Figure 2 Scatterplots showing correlations between total cholesterol levels at retesting and duration of childhood rhGH

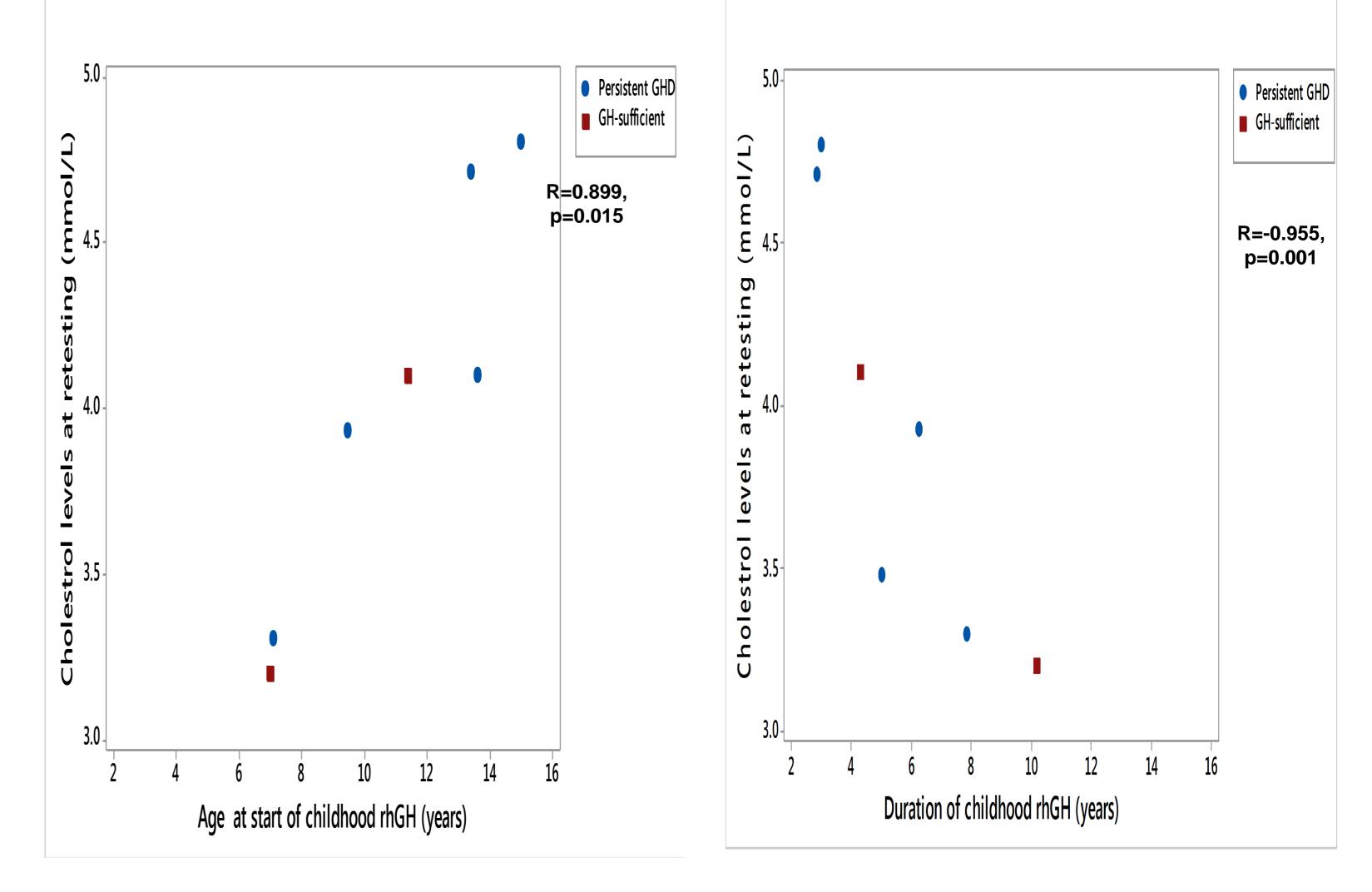
Population and Method

A cross-sectional study of children undergoing GH stimulation tests for short stature and biochemical re-evaluation at final height after withdrawal GH therapy.

Results

 Table 1 Auxological and clinical characteristics of first time

 assessment groups and retesting groups



First time assessment

Retesting

Figure 3 Scatterplots showing correlations between leptin and osteocalcin in first time assessment groups Figure 4 Scatterplots showing correlations between leptin and osteocalcin in retesting groups

	Naïve -GHD (n=15)	Normal (n=10)	Persistent GHD (n=7)	GH-sufficient (n=2)
M/F	13/2	7/3	3/4	0/2
Age(yrs)	10.9 (5.6, 15.2)	12.1 (5.8, 16.5)	16.6 (14.9,18.6)	16.8(16.3, 20.4)
Anthropometry				
	-2.5 (-3.4- 1.3)			
Height -SDS	-1.8 (-3.6- 1.9)	-2.2 (-4.60.1)	-1.2 (-1.9- 1.2)	-1.6 (-3.0, 0.5)
Weight-SDS	0.0 (-1.8- 3.0)	-1.3 (-4.7 - 0.7)	0.6 (-1.8- 1.4)	0.0 (-3.2, 1.1)
BMI-SDS		0.0 (-2.4-1.6)	0.9 (-1.1- 2.0)	1.0 (-1.4, 1.1)
Biochemical data				
GH-peak(µg/l)	2.6 (0.7-4.7)	8.0 (6.7-22.3)	2.0 (0.1-3.8)	8.3 (6.4- 10.2)
IGF1 levels(ng/ml)	65.0 (14.0- 433.0)	85.5(28.0-295.0)	141.0 (18.0-294.0)	241.5 (117-327.0)
IGF1 levels SDS	-3.2 (<-5.0- 0.3)	-2.0 (-4.50.9)	-3.2 (<-5.01.3)	-2.0 (-3.50.9)
Retesting data				
Age of diagnosis (yr)			9.5 (2.6-10.3)	9.2 (7.0-11.4)
Age at starting rhGH(yr)			10.3 (7.1-13.6)	9.2 (7.0-11.4)
Duration of rhGH (yr)			4.7 (2.9-7.8)	7.3 (4.3-10.2)
Age at stopping rhGH(yr)			15.9 (14.4-17.9)	16.4 (15.7-17.0)
Duration of stopping rhGH (yr)			0.6 (0.2-1.0)	0.7 (0.4-1.0)

naive GHD
 naive GHD
 naive GHD
 naive GHD
 normal GH
 normal GH
 R=0.517, p=0.01
 R=0.917, p=0.01
 R=0.917, p=0.001
 R=0.917, p=0.001
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Metabolic data

At time of diagnosis and retesting, lipid profiles, adipokines and glucose homeostasis in both groups were not significantly different between those who were GH deficient and those who had normal GH levels.

Conclusion

Metabolic profiles and glucose homeostasis are not significantly different between those with childhood onset GHD and those with normal GH levels at time of diagnosis and retesting at final height. Although the numbers are small, our data showed that the timing and duration of childhood rhGH treatment may influence adiposity parameters and bone formation biomarkers seen in adolescents with CO-GHD.

Disclosure Statements: The authors have nothing to disclose

