

Serum Kisspeptin in Obese Children and Its Relation to Glucose Metabolism

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Background

Previous studies have demonstrated a possible favorable effect of kisspeptin on glucose metabolism including negative association between serum kisspeptin and body mass index, homeostatic model assessment of insulin resistance (HOMA-IR) and plasma insulin. However, some studies reported conflicting results. Data on serum kisspeptin in obese children are limited.

Objective

Results

Median (IQR) age of enrolled children was 11.8 (10.8, 13.5) years and 57 of them (43%) were males. There were 18 (14%), 83 (62%) and 32 (24%) patients with normal glucose tolerance (NGT), hyperinsulinemia with normal glucose (HI) and abnormal glucose tolerance (AGT), respectively. Serum kisspeptin levels were significantly higher in males as compared with females [68 (37, 83) vs. 48 (25, 73) pg/mL,

To determine serum kisspeptin in obese children and its relation to glucose metabolism

Methods

There were 133 obese children included. All children underwent an oral glucose tolerance test (OGTT) with fasting serum kisspeptin level measurement. Insulin secretion [homeostatic model assessment of β -cell function (HOMA- β) and insulinogenic index (IGI)] and insulin sensitivity [whole body insulin sensitivity index (WBISI) and HOMA-IR] indices were assessed using serum glucose and insulin levels derived during the OGTT. Serum kisspeptin in relation to glucose metabolism was analyzed.

p = 0.043]. Patients with Tanner stages II & III had higher serum kisspeptin levels than those with Tanner stage I [63 (37, 79) vs. 15 (9, 73) pg/mL, p = 0.049]. Serum kisspeptin levels were not different among the 3 groups of glucose metabolism [NGT: 67 (32, 83), HI: 48 (27, 78) and AGT: 69 (35, 80) pg/mL, p = 0.538] (Table 1). There were no correlations between serum kisspeptin levels and indices of insulin secretion and insulin sensitivity (Table 2). However, after adjusting for sex and puberty, serum kisspeptin level was negatively correlated with fasting plasma glucose (FPG) only in the HI group ($\beta = -1.487$, p = 0.006).

Glucose metabolism All children Parameters P (N = 133)**NGT (N=29)** HI (N=118) AGT (N=48) 12.9(11.4, 14.8) 11.3(10.6, 12.8) 13.3(11.1, 14.3) Δqe (vears) 11.8 (10.8, 13.5) 0.003

Table 1. Clinical characteristics of all children according to their glucose metabolism

11.8 (10.8, 13.5)	12.9 (11.4, 14.8)	11.3 (10.6, 12.8)	13.3 (11.1, 14.3)	0.003	Data are presented as
57/76 (43/57)	7/11 (39/61)	38/45 (46/54)	12/20 (38/62)	0.692	
					median (IQR).
9 (7)	1 (6)	7 (8)	1 (3)		Pvalue, comparing
66 (50)	8 (44)	43 (52)	15 (47)	0.795	among the 3 groups of
58 (43)	9 (50)	33 (40)	16 (50)		
4.7 (3.7, 6.0)	4.5 (3.6, 5.9)	4.9 (3.8, 6.0)	4.2 (3.3, 6.3)	0.830	glucose metabolism
2.4 (2.0, 2.8)	2.3 (1.8, 2.7)	2.4 (2.0, 2.9)	2.3 (2.1, 3.2)	0.366	(Kruskal-Wallis test and
137 (121, 161)	123 (101, 149)	138 (123, 162)	136 (118, 162)	0.109	Chi-square test)
55 (28, 79)	67 (32, 83)	48 (27, 78)	69 (35, 80)	0.538	•
5.7 (5.4, 5.9)	5.4 (5.2, 5.7)	5.6 (5.4, 5.9)	5.7 (5.6, 5.9)	0.010	AUC, area under the
81 (77, 87)	80 (77, 87)	81 (77, 86)	83 (77, 89)	0.365	curve
123 (106, 138)	106 (102, 115)	118 (104, 129)	147 (143, 157)	<0.001	
254 (230, 285)	225 (214, 244)	250 (228, 269)	300 (278, 316)	<0.001	
224 (153, 327)	115 (97, 136)	238 (188, 331)	266 (165, 422)	<0.001	
301 (189, 484)	131 (85, 214)	337 (240, 520)	304 (187, 501)	<0.001	
1.8 (1.2, 2.7)	1.6 (0.9, 6.0)	2.0 (1.4, 3.4)	1.3 (0.8, 2.4)	<0.001	
2.3 (1.8, 3.4)	5.6 (4.4, 7.2)	2.3 (1.8, 3.1)	2.0 (1.2, 2.4)	<0.001	
3.3 (2.1, 4.6)	1.4 (0.9, 2.3)	3.6 (2.5, 4.8)	3.6 (2.6, 6.5)	<0.001	
	57/76 (43/57) 9 (7) 66 (50) 58 (43) 4.7 (3.7, 6.0) 2.4 (2.0, 2.8) 137 (121, 161) 55 (28, 79) 5.7 (5.4, 5.9) 81 (77, 87) 123 (106, 138) 254 (230, 285) 224 (153, 327) 301 (189, 484) 1.8 (1.2, 2.7) 2.3 (1.8, 3.4)	57/76 (43/57) $7/11 (39/61)$ 9 (7)1 (6)66 (50)8 (44)58 (43)9 (50)4.7 (3.7, 6.0)4.5 (3.6, 5.9)2.4 (2.0, 2.8)2.3 (1.8, 2.7)137 (121, 161)123 (101, 149)55 (28, 79)67 (32, 83)5.7 (5.4, 5.9)5.4 (5.2, 5.7)81 (77, 87)80 (77, 87)123 (106, 138)106 (102, 115)254 (230, 285)225 (214, 244)224 (153, 327)115 (97, 136)301 (189, 484)131 (85, 214)1.8 (1.2, 2.7)1.6 (0.9, 6.0)2.3 (1.8, 3.4)5.6 (4.4, 7.2)	57/76 (43/57) $7/11$ (39/61) $38/45$ (46/54)9 (7)1 (6)7 (8)66 (50)8 (44)43 (52)58 (43)9 (50)33 (40)4.7 (3.7, 6.0)4.5 (3.6, 5.9)4.9 (3.8, 6.0)2.4 (2.0, 2.8)2.3 (1.8, 2.7)2.4 (2.0, 2.9)137 (121, 161)123 (101, 149)138 (123, 162)55 (28, 79)67 (32, 83)48 (27, 78)5.7 (5.4, 5.9)5.4 (5.2, 5.7)5.6 (5.4, 5.9)81 (77, 87)80 (77, 87)81 (77, 86)123 (106, 138)106 (102, 115)118 (104, 129)254 (230, 285)225 (214, 244)250 (228, 269)224 (153, 327)115 (97, 136)238 (188, 331)301 (189, 484)131 (85, 214)337 (240, 520)1.8 (1.2, 2.7)1.6 (0.9, 6.0)2.0 (1.4, 3.4)2.3 (1.8, 3.4)5.6 (4.4, 7.2)2.3 (1.8, 3.1)	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$

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Fat, metabolism and obesity

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Table 2. Correlations between serum kisspeptin and parameters derived from the OGTT in all children (N = 133)

	Serum kisspeptin					
Parameters	Unadjusted		Adjusted for sex and puberty			
	β	Ρ	β	Ρ		
Fasting plasma glucose	-0.143	0.538	-0.189	0.412		
2-h plasma glucose	0.010	0.905	0.006	0.938		
Glucose area under the curve	0.021	0.654	0.022	0.637		
Insulin area under the curve	-0.007	0.733	-0.015	0.472		
ΗΟΜΑ-β	-0.001	0.933	-0.001	0.878		
IGI	-0.447	0.801	-1.435	0.417		
WBISI	1.330	0.354	1.560	0.268		
HOMA-IR	-0.808	0.476	-1.168	0.296		

Conclusions

Serum kisspeptin levels in obese children were not different among different glucose metabolism categories. Negative correlation between serum kisspeptin and FPG was only found in the HI group.



