THE GROWTH HORMONE TREATMENT RESULTS IN THE INCREASE OF IRISIN CONCENTRATION IN PLASMA

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Introduction

Brown adipose tissue metabolism is of remarkable pathophysiological interest, because it could be a target for future therapies for obesity and metabolic syndrome.
Irisin (Ir), recently identified novel adipomyokine is essential in a white-to-brown fatty tissue transdifferentiation, and mediates some of the positive influences on metabolic disorders through increase of energy expenditure.
The exact regulation of Ir secretion and action is unknown but significant positive associations of circulating Ir with growth hormones and IGF-1 were found.
We studied Ir response in a group of Turner Syndrom (TS) patients treated with supraphysiological doses of growth hormone (rGH).

Study group and rGH treatment

The study group consisted of 36 patients with Turner syndrome (TS) diagnosed in one pediatric tertiary center. Their median age was 7,06 years (range 3.2–16.07 years). The patients were diagnosed by karyotyping. Conventional G-banding and fluorescence *in situ* hybridization on peripheral blood cultures confirmed numerical or structural abnormalities of X chromosome or mosaicism. X chromosome monosomy was established in 19 patients (52,8%).
Because of short stature the patients were treated with rGH given subcutaneously once daily at bedtime, in a dose 0,05 mg/kg/day. The dose of rGH was adjusted to body weight every 3 months.
The mean treatment period was 1.5 yr (range 0.4-4.0yr). No other medication including estrogen replacement therapy was conducted during the study. Patients with coexisting endocrine diseases or other conditions interfering with glucose metabolism were excluded.

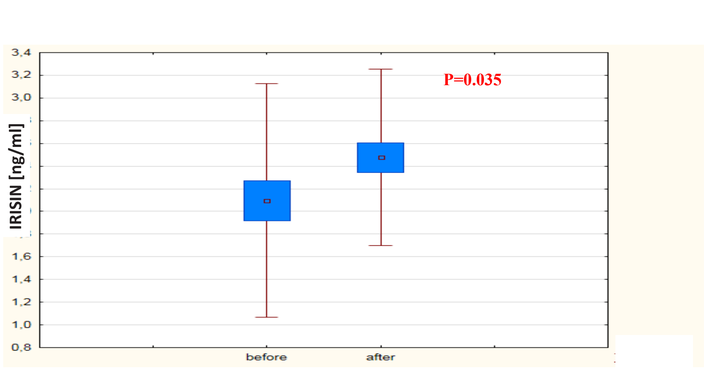
Measurements

Prior to and following the rGH treatment (rGHt) anthropometrical data were recorded as well as biochemical parameters were measured: Ir, OGTT, insulin, IGF-1, and IGFBP-3.

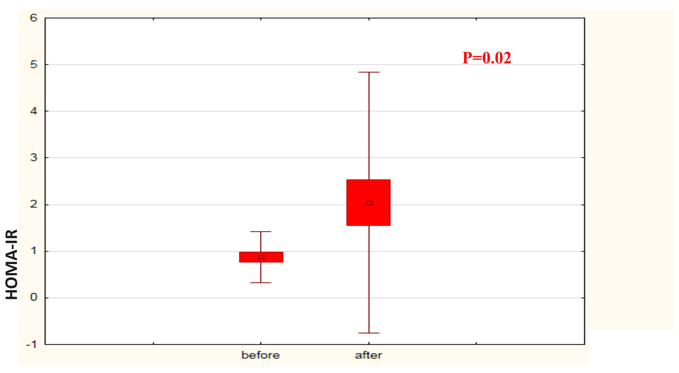
The description of TS group and the effect of rGH therapy

	before rGHt	after rGHt
Height [cm]	112.14 ± 16.3	126.14 ± 15.29
Height SDS	-2.45 ± 0.91	-1.8 ± 0.83
BMI SDS	0.18 ± 1.27	0.34 ± 1.1
Fasting plasma glucose [mg/dl]	87.77 ± 9.13	86.69 ±15.26
Fasting plasma insulin [uIU/ml]	3.67 ± 2.3	8.38 ±10.79
IGF-1 [ng/ml]	119.40 ± 62.47	439.08 ± 209.91
IGF-1 SDS	-1.70 ± 0.48	0.41 ± 1.29
IGFBP3 [ug/ml]	3.94 ± 0.83	5.73 ± 1.2

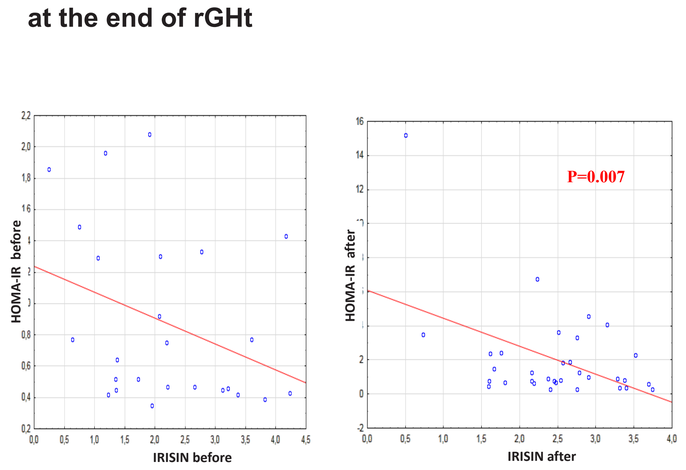
Results



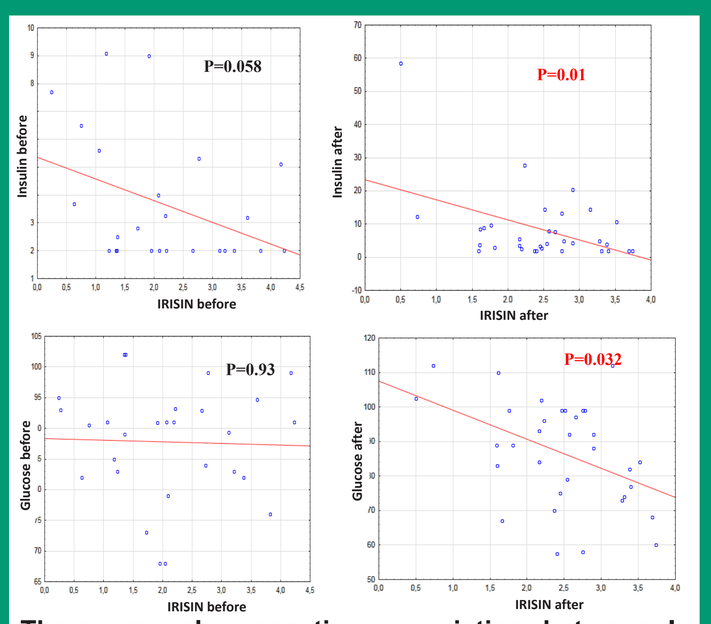
Irisin concentration increased significantly after rGHt



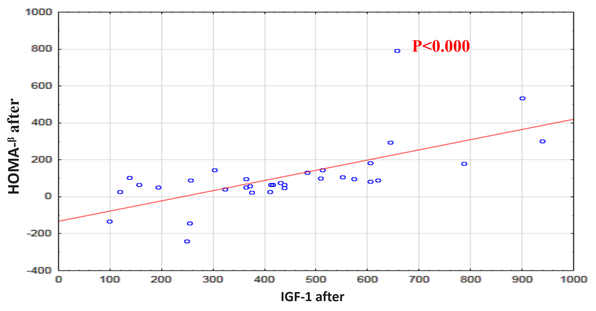
We observed the increase of HOMA-IR value at the end of rGHt



There was a negative association between Ir concentration and HOMA-IR after rGHt



There was also negative association between Ir concentration and fasting insulin and glucose levels after rGHt



After rGHt a pronounced HOMAβ value increase related to IGF-1 level was observed but there was no association between Irisin concentration and HOMA-β before or after rGHt.

Conclusions

1) **rGH therapy has been shown to increase the concentration of circulating Irisin**
2) **rGH treatment seems to restore physiological function of Irisin**
3) **This study has shown that in the Turner Syndrome group the rGH medication improved β-cell function**
4) **No relation between Ir and IGF-1 levels or IGF-1/ IGFBP3 ratio was recorded, therefore the Irisin rise seemed not to be IGF-1 mediated.**
5) **The complex functional interrelationships between GH, IGF-1 and insulin resistance/sensitivity must be a subject of further investigations. Our data have shown that Irisin may be a new relevant player in this branch of energy metabolism.**

To the best of our knowlege this is the first study on Irisin in Turner Syndrome patients.

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