The influence of GH treatment on the Oral Disposition Index (ODI) in Turner Syndrome girls and in GH deficient children : eight years of follow-up

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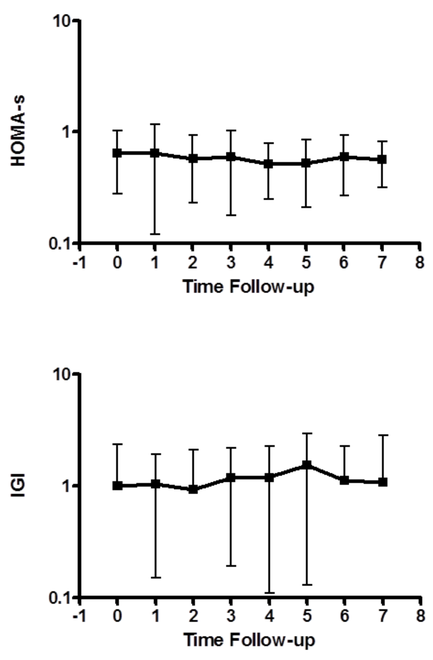
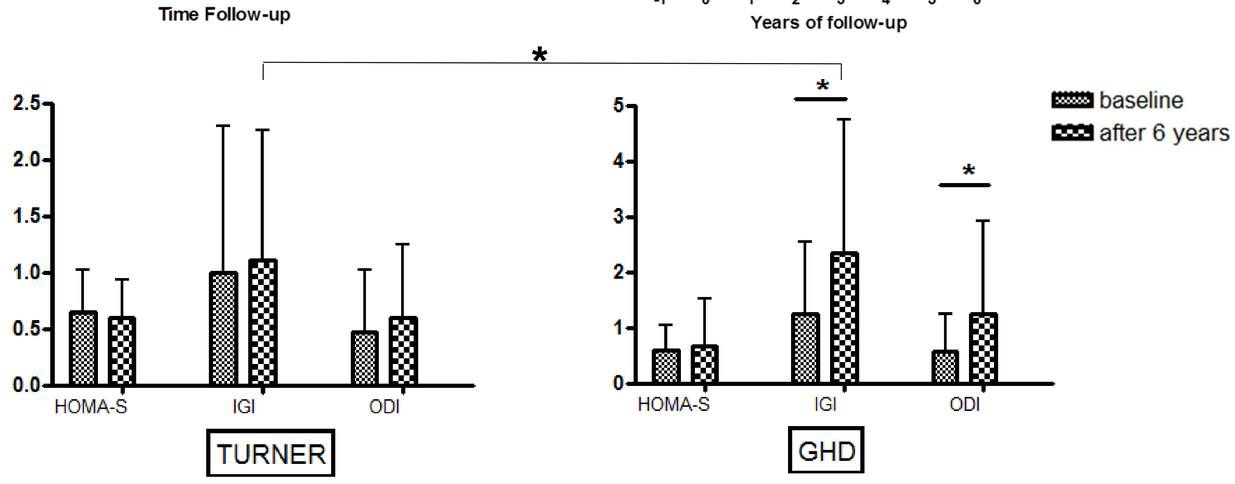
Background: GH has been shown to influence glucose homeostasis through a negative effect on insulin sensitivity followed by a compensatory increase of insulin secretion. However it has been recently reported, in animals and in humans, that GH might stimulate insulin secretion also through a direct effect on the growth and on the function of the pancreatic beta cell.

Objective: to study longitudinally the insulin sensitivity (HOMA-S), the insulin secretion (IGI) and the capacity of the beta cell to adapt to the insulin sensitivity (ODI) in a group of girls affected by Turner's syndrome (TS) and in a group of growth hormone deficient children (GHD).

Methods: we studied **92 TS** (9.7 ± 2.95 years) and **99 GHD** (62 m, 37 f) (8.9 ± 3.5 years for a median period of 7.32 years (range 2.04-13) in TS and 7.7 years (range 3.4-14.7) in GHD. Every year the children underwent an OGTT which was employed to calculate the HOMA-S 1/((insulin*glucose)/22.5), the insulinogenic index, IGI (ΔI30/ΔG30) and the ODI (disposition index=HOMA-S*IGI).





***p<0.01**

Results: in TS no significant changes over the years were observed in term of HOMA-S, IGI or ODI. On the contrary, in GHD children, despite HOMA-S remaining unchanged, an increase of IGI (1.25 ±1.28 vs 2.35 ± 2.38) and ODI (0.57 ± 0.68 vs 1.23 ± 1.68) was observed, which became significant after 6 years of treatment. There was no difference before GH treatment between GHD and TS regarding HOMA-S, IGI and ODI but IGI became significantly higher in GHD after 6 years.

Conclusion: our results suggest a positive influence of GH treatment on the beta cell secretory capacity in children with GH deficiency, while no effect was observed in those (TS) with normal GH secretion. A different sensitivity to GH might explain the differences.