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GHRH plus arginine and arginine administration evokes the same ratio of GH isoforms levels in young patients with Prader-Willi syndrome.

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Human GH is present in pituitary and circulation as several isoforms, the prevalent being 22 kDa- and 20 kDa-GH. Recently, we have demonstrated the preservation of a normal balance in GH isoforms after GH releasing hormone (GHRH) plus arginine (ARG) administration in adult patients with Prader-Willi syndrome (PWS), one of the most common causes of syndromic obesity, often associated with GH deficiency (GHD). Aim of the present study was to measure circulating levels of 22 kDa- and 20 kDa-GH in young PWS patients (n =24; F/M: 10/14; genotype UPD/DEL/met+: 11/11/2; age:  $10.8 \pm 5.3$  years; BMI SDS:  $2.0 \pm 2.0$ ; GHD: 16/24; obesity: 12/24) after combined GHRH+ARG or ARG administration. The results were analysed subdividing the GHRH +ARG and ARG groups on the basis of PWS genotype, GHD status and obesity. Circulating levels of 22 kDa- and 20 kDa- GH were measured by a chemiluminescent or fluorescent method based on specific pairs of monoclonal antibodies. GHRH +ARG or ARG significantly stimulated the secretion of 22 kDa-GH but not that of 20 kDa-GH in all PWS patients. No significant GHRH+ARG- vs. ARG-induced changes in the ratios of 22 kDa- to 20 kDa-GH peaks were observed in all PWS patients, although 22 kDa- or 20 kDa-GH peaks were significantly higher in the GHRH +ARG than ARG group. When subdividing PWS patients in UPD vs. DEL, obese vs. non obese and GHD vs. non GHD subgroups, GH peaks were significantly higher in nonobese than obese patients and in non GHD than GHD patients administered with either GHRH +ARG or ARG test, apart from the comparisons in the DEL/UPD subgroups. Anyway, the ratios of peak levels of 22 kDa- to 20 kDa-GH were similar after GHRH+ ARG vs. ARG in all subgroups investigated. In conclusion, this study shows that administration of two different pharmacological tests, i.e. ARG, capable of reducing hypothalamic somatostatinergic tone, and GHRH (+ARG), that directly acts at pituitary level on the somatotropic cell, evokes the same ratios of GH isoforms in young PWS patients, suggesting that the hypothalamic dysfunction in this genetic disorder does not alter the qualitative and quantitative composition of GH isoforms present in circulation.

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