Muscle strength and power, maximum oxygen consumption, and body composition in middle-aged short-stature adults with childhood-onset growth hormone deficiency.

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Archives of Medical Research 39: 78-83, 2008.

Background: growth hormone (GH) replacement in adult GH-deficient (GHD) patients is reported to have a long-term beneficial effect on muscle mass and function, these effects being greater in young males and in adult-onset compared with those with childhood-onset GHD. To date, more discordant data are reported on the degree of muscle impairment in untreated GHD patients, due to the large heterogeneity of this syndrome.

Methods: muscle maximum total isotonic strength (ST), lower limb maximum power output (W), maximum aerobic capacity (VO_2max) and body composition (by tetrapolar bio-impedentiometry) were evaluated in seven short-stature adults with childhood-onset GHD and in seven age-matched normal-stature controls with comparable lifestyle and daily physical activity.

Results: significant differences were found in body composition between control subjects and GHD patients, who presented higher adiposity (mean BMI ± SD: GHD, 27.8 ± 5.8 kg/m^2; controls, 22.1 ± 0.8 kg/m^2; p = 0.047), larger fat mass (GHD, 21.8 ± 10.7 kg; controls, 8.8 ± 3.5 kg; p = 0.008), and lower fat-free mass (GHD, 65.8 ± 11.4 %; controls, 87.0 ± 6.5 %; p = 0.002). In absolute terms, GHD patients attained significantly lower values in ST (GHD, 2479 ± 493 N; controls, 4578 ± 1476 N; p = 0.008), W (GHD, 1092 ± 452 W; controls, 1910 ± 781 W; p = 0.035) and VO_2max (GHD, 1.68 ± 0.40 l/min; controls, 2.67 ± 0.84 l/min; p = 0.035) than those attained by controls. The differences were still evident when the results were normalized by unit body mass, whereas they disappeared when the parameters were expressed per unit fat-free mass, suggesting for these patients the presence of an intrinsic muscle function in the same range as that of control subjects.

Conclusions: middle-aged and short-stature adults with childhood-onset GHD, who received discontinuous pit-GH substitution therapy only during childhood and have uncorrected long-lasting GHD, still retain a normal intrinsic muscle capability in attaining isotonic strength, generating anaerobic power as well as accomplishing oxidative processes. Nonetheless, it is not known which age-dependent evolution in motor dysfunction could be expected in this subgroup of GHD patients, when ageing processes add up to hormonal deficiencies.