Correlation between Circulating Betatrophin Level and Insulin Sensitivity in Centrally-Obese Male Subjects

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Objective

To investigate relationship between serum betatrophin level and insulin sensitivity in centrally-obese male subjects compared to age-matched non-obese subjects.

Materials and Methods

This cross-sectional comparative study was undertaken in 34 centrally-obese [Age: 48.9 ± 6.2 yrs, Body Mass Index (BMI): 26.8 ± 2.4 kg/m2, waist circumference (WC): 98.2 ± 6.3 cm] and 34 non-obese subjects [Age: 49.3 ± 5.3 yrs, BMI: 19.7 ± 2.8 kg/m2, WC: 76.0 ± 7.8 cm]. Insulin sensitivity and β -cell functions were assessed by homeostasis model assessment method (HOMA). Fasting plasma glucose was measured by glucose oxidase method. Serum insulin and betatrophin levels were determined by ELISA method. Results

Significant increase of HOMA-IR in centrally-obese subjects compared with non-obese subjects [median and interquartile range; median (IQR): 4.5 (3.4-6.7) vs. 1.94 (1.3-3.2) p <0.001] indicated that insulin sensitivity was decreased in centrally-obese subjects. Median (IQR) of fasting serum insulin level [19.3 (15.3-28.5) vs. 9.8 (6.5-13.5) μ IU/ml, p<0.001] and HOMA β -cell function [274.5 (192.9-467.0) vs. 167.6 (92.7-239.8), p<0.001] were significantly higher in centrally-obese subjects than that of non-obese subjects, indicating compensatory increased β -cell functions in centrally-obese subjects. Serum betatrophin level in centrally-obese group was significantly lower than that of non-obese group [2.4 (0.8-4.2) vs. 0.8 (0.4-2.4) ng/ml, p<0.05]. There was no significant correlation between serum betatrophin level and HOMA-IR (Spearman's rho=-0.136, p=0.269) as well as HOMA β -cell function (Spearman's rho=0.036, p=0.771) in both groups. Conclusion

Betatrophin could not improve beta-cell function and might not involve in compensatory mechanism of insulin resistance in central obesity.