

Irisin levels in genetic and essential obesity: clues for a potential dual role.

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Irisin is conventionally regarded as a myokine involved in the browning of white adipose tissue, energy expenditure and glucose tolerance. Its potential link to fat accumulation and metabolic dysfunction is debated. We sought to explore the relationship between circulating irisin and components of body composition in two different phenotypes of severe obesity. For this purpose, 30 obese adults with Prader-Will syndrome (PWS) (age 35.7 ± 1.5 y, BMI 45.5 ± 1.5 kg/m²) and 30 adult controls with common obesity (age 34.9 ± 1.7 y, BMI 46.8 ± 1.4 kg/m²) underwent analysis of irisin levels, metabolic profile, body composition and resting energy expenditure (REE). Normal irisin levels were obtained from a group of 20 lean donors (age 32.4 ± 1.5 y, BMI 23.8 ± 0.8 kg/m²). Expected differences in body composition and metabolic profile existed between study groups. PWS exhibited lower muscle mass ($p < 0.001$), FFM ($p < 0.001$), REE ($p < 0.001$), as well as insulin ($p < 0.05$), HOMA-IR ($p < 0.05$) and triglycerides levels ($p < 0.05$) than controls with common obesity. In PWS, irisin levels were significantly lower and overall less dispersed than in controls with common obesity ($p < 0.05$), while being similar to values recorded in lean subjects. To explore the relation between irisin and body composition in obesity, univariate correlation analysis in the obese populations as a whole showed positive associations between irisin and muscle mass ($p = 0.03$) as well as REE ($p = 0.01$), which disappeared when controlled for the PWS status. Noticeably, a positive association became evident between irisin and %FM after controlling for the PWS status ($p = 0.02$). Also positive were associations between irisin and insulin ($p = 0.02$), HOMA-IR ($p = 0.02$) and triglycerides ($p = 0.04$). In stepwise multivariable regression analysis, irisin levels were independently predicted by the PWS status ($p = 0.001$), %FM ($p = 0.004$) and triglycerides ($p = 0.008$). Current results suggest that obese adults with PWS harbor lower irisin levels than individuals with common obesity. The divergent models of obesity herein studied suggest a potential link between circulating irisin and muscle mass and metabolic dysfunction relating to adiposity.

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