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ORIGINAL RESEARCH COMMUNICATION

Effect of the melanocortin-3 receptor C17A and G241A variants on weight loss in childhood obesity^{1, 2, 3}

Nicola Santoro, Laura Perrone, Grazia Cirillo, Paolo Raimondo, Alessandra Amato, Carmine Brienza and Emanuele Miraglia del Giudice

¹ From the Department of Pediatrics "F Fede," Seconda Università degli Studi di Napoli, Napoli, Italy

Background: The central melanocortin system is critical for the long-term regulation of energy homeostasis. Melanocortin-3 receptor (*MC3R*) knock-out mice, despite being hypophagic, have increased fat mass and higher feed efficiency than do their wild-type littermates.

Objective: The aim was to evaluate whether, in childhood obesity, *MC3R* variants are associated with changes in fatness reduction as a consequence of a weight-reduction program.

Design: Molecular screening of the *MC3R* coding region in 184 obese children, 77 girls and 107 boys [\bar{x} (\pm SEM) body mass index (BMI; in kg/m²) z score: 3.3 \pm 2.3; age 9.2 \pm 2 y], was performed. BMI was evaluated at baseline and after 6 and 12 mo of the weight loss program.

Results: No new mutations were found. Two previously described polymorphisms, C17A (Thr6Lys) and G241A (Val81IIe), were observed in 20 patients in almost complete linkage disequilibrium. No significant differences in BMI z scores were observed at baseline of the weight-loss program between the genotypes; however, at follow-up, heterozygotes showed a significantly higher BMI z score (P = 0.03). When the patients were divided according to the amount of weight lost, a higher prevalence of heterozygotes was observed among subjects who lowered their BMI z score <1.5 (P = 0.03).

Conclusion: These results suggest a gene-diet interaction between the *MC3R* C17A and G241A variants and a weight loss program for the ability to lose weight in childhood obesity.

Key Words: MC3R gene • polymorphism • childhood obesity • weight loss • BMI z score



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