

intake and expenditure. In rare cases, it is caused by underlying medical disorders arising from disruptions in the leptin-melanocortin pathway which regulates satiety and energy expenditure.

Aim: To investigate and compare resting energy expenditure (REE) and body composition characteristics of children and adolescents with severe obesity with and without underlying medical causes.

Methods: This prospective observational study included pediatric patients who underwent an extensive diagnostic workup in our academic center in which endocrine, non-syndromic and syndromic genetic, hypothalamic, and medication-induced causes of obesity were evaluated. Patients in whom no underlying medical cause was identified were classified as multifactorial obesity. REE was assessed by indirect calorimetry; body composition by air displacement plethysmography. The ratio measured REE (mREE) vs predicted REE (Schofield equations) was expressed as REE%, with decreased mREE defined as REE% \leq 90% and elevated mREE as \geq 110%. Additionally, the ratio mREE vs fat-free-mass (FFM) was calculated.

Results: We included 292 patients, of which 218 (75%) patients had multifactorial obesity and 74 (25%) had an underlying medical cause: non-syndromic and syndromic genetic (n= 29 and 28, respectively), hypothalamic (n= 10), and medication-induced (n= 7) obesity. Mean age was 10.8 ± 4.3 years, 59% were female, mean BMI SDS was 3.8 ± 1.1 , indicating severe obesity. Mean REE% was higher in children with non-syndromic genetic obesity ($107.4\% \pm 12.7$) and lower in children with hypothalamic obesity ($87.6\% \pm 14.2$) compared to multifactorial obesity ($100.5\% \pm 12.6$, both $p < 0.01$). Measured REE was decreased in 60 (21%) patients (corresponding to an average overprediction of daily caloric needs of 341 kcal/day) and elevated in 69 (24%) patients. Only in hypothalamic obesity, a larger proportion of patients showed a decreased REE compared to multifactorial obesity (6/10 vs 41/218, $p < 0.01$). FFM was higher in children with non-syndromic obesity compared to multifactorial obesity (+7.5kg, $p < 0.001$), but lower in syndromic obesity (-5.2kg, $p = 0.03$), hypothalamic obesity (-12.6kg, $p < 0.001$), and similar in medication-induced obesity (+1.5kg FFM, $p = 0.80$). Mean mREE/FFM was 46.5 ± 10.6 kcal/day/kg FFM and did not differ between patients with underlying medical causes compared to multifactorial obesity (all $p > 0.05$).

Conclusion: In this cohort of children with severe obesity due to various etiologies, large inter-individual differences in mREE were found. Almost half of patients had decreased or elevated mREE. When relating mREE to FFM, no differences were found between children with underlying medical causes versus multifactorial obesity. Thus, our study underlines the importance of measuring REE and relating mREE to FFM in children with early-onset severe obesity with or without underlying medical causes. This knowledge is important for patient-tailored treatment, e.g. personalized dietary or physical activity interventions and consideration of pharmacotherapy affecting central energy expenditure regulation in children with decreased mREE.

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Resting Energy Expenditure and Body Composition in Children and Adolescents with Genetic, Hypothalamic, Medication-Induced or Multifactorial Severe Obesity

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Background: Pediatric obesity is a multifactorial disease characterized by a prolonged imbalance between energy