RESTING ENERGY EXPENDITURE AND BODY COMPOSITION IN CHILDREN AND ADOLESCENTS WITH SEVERE OBESITY DUE TO (SUSPECTED) MEDICAL CAUSES:

COMPARISON BETWEEN DIFFERENT SUBTYPES OF OBESITY

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BACKGROUND

Paediatric obesity is a multifactorial disease characterized by an imbalance between energy intake and expenditure.

In rare cases, paediatric obesity is caused by underlying medical causes, *i.e.*, genetic, hypothalamic, and medication-induced obesities. These disorders arise from disruptions in the central regulation of satiety and energy expenditure.

AIM

To investigate resting energy expenditure (REE) in relation to body composition in children and adolescents with non-syndromic and syndromic genetic, hypothalamic, medication-induced or idiopathic severe obesity.

METHODS

Prospective observational study of children and adolescents referred to our academic paediatric obesity centre due to suspicion of an underlying medical cause.[1]

Diagnostic workup: extensive medical history taking and physical examination, growth charts analysis, biochemical and hormonal assessment, and genetic testing.

REE measurement: indirect calorimetry after an overnight fast using a metabolic cart (Quark RMR) under strictly controlled environmental conditions.

Body composition (fat-free-mass, FFM): air displacement plethysmography (BOD POD).

REE% = Ratio measured REE (mREE) / predicted REE (using Schofield formula).
Lowered mREE = REE% ≤ 90%.
Elevated mREE = REE% ≥ 110%.

RESULTS

N = 285 patients included, of which:
- 28 (10%) non-syndromic genetic obesity
- 27 (9%) syndromic genetic obesity
- 6 (2%) hypothalamic obesity
- 4 (1%) medication-induced obesity
- 220 (77%) idiopathic obesity

Mean age 10.7 ± 4.4 years, 171 (60%) were female.
Mean BMI standard deviation score (SDS) was 3.7 ± 1.1, corresponding to adult BMI ≥ 40 kg/m$^{2.}$

Across all patients, large intra-individual variability in mREE was found, with a lowered mREE in 21% of patients (Table, Figure).

Table. REE characteristics of the study population

	mREE kcal/day	**mREE** % of predicted	**Lowered/Elevated mREE**	**mREE/FFM** kcal/day/kg
All patients (n=285)	1706 (499)	100.1 (13.6)	60 (21%) / 67 (24%)	46.8 (10.8)
Non-syndromic genetic (n=28)	1860 (655)	104.5 (14.6)	4 (14%) / 10 (36%)	45.9 (10.2)
Syndromic genetic (n=27)	1475 (358)	98.5 (9.0)	5 (19%) / 1 (4%)	50.8 (14.8)
Hypothalamic (n=6)	1523 (285)	91.6 (17.6)	2 (33%) / 1 (17%)	42.0 (7.1)
Medication-induced (n=4)	1517 (229)	91.1 (20.3)	1 (25%) / 1 (25%)	-
Idiopathic (n=220)	1645 (523)	99.9 (13.7)	48 (22%) / 54 (25%)	46.5 (10.4)

Data presented as mean (SD) or count (%)

Patients with hypothalamic and medication-induced obesities showed lower REE% than patients with idiopathic obesity (Table, Figure), although these differences were not statistically significant (p-values >0.05), possibly due to small sample size.

mREE was strongly associated with FFM (r = 0.87, p<0.001).

mREE adjusted for FFM was not associated with BMI SDS (r = -0.00, p=0.98) and did not differ between patients with underlying medical causes and patients with idiopathic obesity after adjustment for sex (p-values >0.05, Figure).

DISCUSSION

In this cohort of children and adolescents with severe obesity, **21%** of patients had a lowered REE → highlighting the importance of measuring REE in the diagnostic workup of children and adolescents with severe obesity

REE adjusted for FFM was similar in patients with non-syndromic and syndromic genetic obesity vs patients with idiopathic severe obesity.

Measuring REE in children and adolescents with severe obesity can aid patient-tailored treatment:
- Personalized dietary and exercise plans.
- Pharmacologic treatment affecting central energy expenditure regulation.

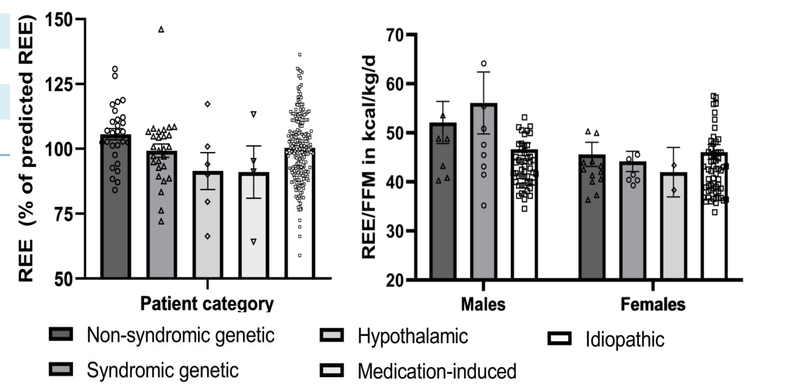

Figure. REE% (left) and mREE adjusted for FFM (right) of the study population.

REFERENCES [1]Kleinendorst, Abawi *et al.*, *PLOS ONE 2020* 15(5): e0232990
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Fat, metabolism and obesity
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