# Growth patterns in non-syndromic childhood overweight: comparing children with early of late onset weight gain Alina German<sup>1,3</sup>, Yulia Vaisbourd<sup>1</sup>, Kerstin Albertsson-Wikland<sup>2</sup>, Lars Gelander<sup>2</sup>, Anton Holmgren<sup>2</sup>, Aimon Niklasson<sup>2</sup>, Ze'ev Hochberg<sup>3</sup> <sup>1</sup>Pediatric Endocrinology, Clalit HMO and Bnei Zion Medical Center, Haifa, Israel <sup>2</sup> Sahlgrenska Academy at the University of Gothenburg, Sweden, <sup>3</sup>Technion – Israel Institute of Technology, Haifa, Israel

BACKGROUND	RESULTS

- A rapid weight gain during infancy increases adult lean body mass, whereas weight gain during adiposity rebound at age 4-7 years results in increased adult fat mass and an increased risk of the metabolic syndrome and T2D.
- To understand the impact of age of obesity onset on growth,  $\bullet$ we classified non-syndromic childhood overweight into an early

Study participants divided according to their sex and obesity onset age.



onset (EO, age 0-3) and a late onset (LO, age 3-7) group and characterized the growth patterns of the two.

### **METHODS**

Study population:

A retrospective study based on the "Grow up Gothenburg" 1974 cohort of children born in Sweden. (Holmgren A et al BMC Pediatrics 2017) International cut off points for BMI for overweight by sex at age 2-18 years defined to pass through body mass index of 25 at age 18 and through BMI 17.56 for girls and 17.89 for girls at age 3 years. (Tim J Cole, BMJ, 2000)





### BMI by age for the study and the control groups



Growth periods according to the life history theory were defined as:

1-infancy, 4-8 month

- 2- childhood 1.5-3.5Y
- 3- Juvenility 7-9y girls
- 4- adult height >16Y girls, >17Y boys

# **CONCLUSION** and **DISCUSSION**

During the 1.8 Myr of a hunter-gatherers (HGs) lifestyle, physical activity was intense and metabolic fuels were high in animal protein and low in CHO, the genome of HGs was adapted for low insulin sensitivity. Owing to severe seasonal food shortages, the 'thrifty' HG genome was preserved until the 'escape from hunger' in the past 200 years.

The agrarian epoch 12 000 years ago characterized by a transition to a sedentary lifestyle, and the consumption of a high-CHO and low protein diet, which required the genome to adapt to low activity levels and high insulin sensitivity.

#### Adiposity rebound age in EO and LO obesity according to sex.



Adiposity rebound: Early onset – 6±1.7Y P value = 0.017

Adiposity rebound:	Early onset – 5±1.55Y
	Late onset – 4.7±2.1Y
	P value = 0.73

Late onset – 5.3±2.2Y

Table 1. Height in EO and LO girls during life history growth periods						
Height (cm)	ΕΟ	LO	Control	P Value		
Infantile height	65.9±1.9	65.6±2.67	65.7±2.2	0.73		
Childhood height	89.6±5.5	89.2±5.3	89.7±4.2	0.32		
Juvenile height	131.9±5.7	128.6±7.1	129.1±5.6	0.015		
Adult height	168.2±6.0	165.4±7.2	167.8±5.9	0.046		
Mother height	169.3±5.0	163.6±5.9	166.5±5.6	0.005		
<b>ΟΙΙΝΛΝΛΟΥ</b>						

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Obesity in individuals with a HG genome (more males) starts after the age of 3 years and is likely to develop into metabolic syndrome and type 2 diabetes mellitus. Late onset obesity is best treated with a HG routine: increase physical activity and low CHO and high protein diet.

Obesity in individuals (more females) with an adapted farmer genome has its onset within the first 6 months of life, and is unlikely to develop into metabolic syndrome and type 2 diabetes mellitus. In this instance, obesity is best treated with a low-calorie diet. Early onset is associated with taller stature.

The EO group had relatively more girls, they were more affected by obesity than the LO group and had later adiposity rebound. 2. From juvenility onwards and at final height, girls of the EO group, and also their mothers, were taller than girls of the LO group. 3. The timing of onset of overweight can be used to distinguish between obese children who are likely (LO) or unlikely (EO) to embark on a trajectory, which leads to insulin resistance, the metabolic syndrome, and T2D.



