Ülekaaluliste poiste luutihedus ja vereseerumi parameetrid

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Sissejuhatus [1]

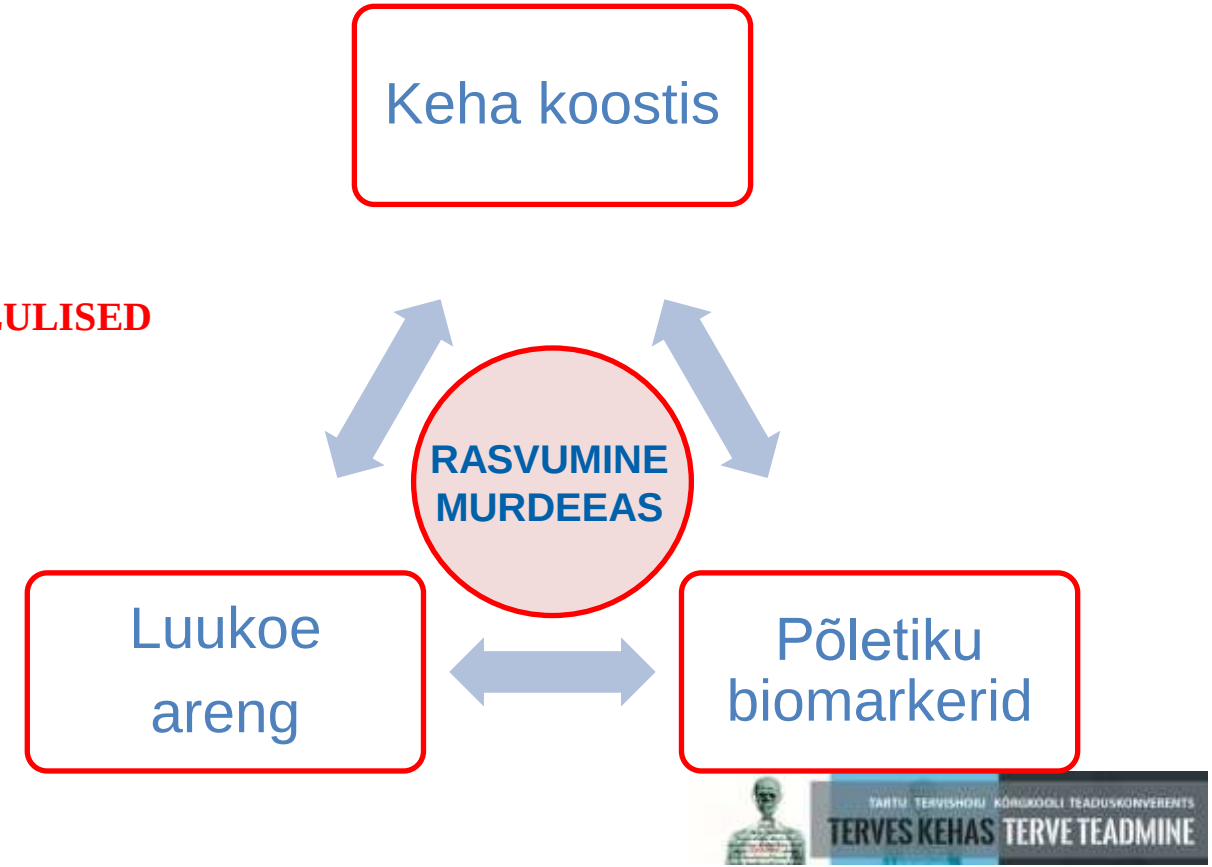
- Ülekaalulisus ja rasvumine mõjutavad kasvamise ja arenguga seotud protsesse murdeeas (De Leonibus et al. 2013)
- Rasvumine lapseeas on seotud mitmete erinevate haigustega hilisemas elus (Denzer et al. 2009; Dimitri et al. 2010; Emanuela et al. 2012; Palermo et al. 2016)
- Lapse- ja noorukiiga on olulised perioodid luukoemassi lisandumisel (Baxter-Jones et al. 2010; Rizzoli et al. 2010)
- Keha mass oluline tegur, mis mõjutab luukoemassi ja luu mineraliseerumist (Bachrach 2001; El Hage 2012; Mosca et al. 2013)

Sissejuhatus [2]

- Kehamassi lisandumine kasvuperioodi jooksul mõjutab luukoemassi moodustumist ja selle kadumist (Mosca et al. 2014)
- Kiire kehamassi lisandumine mõjutab metaboolset profiili ja tõstab põletiku fooni organismis(Calarge et al. 2012)
- Rasvkude toodab põletikuga seotud proteiine (tsütokiine), mis avaldavad kahjustavat mõju luukoe arengulistele protsessidele (Iwaniec & Turner 2016)

Sissejuhatus [3]

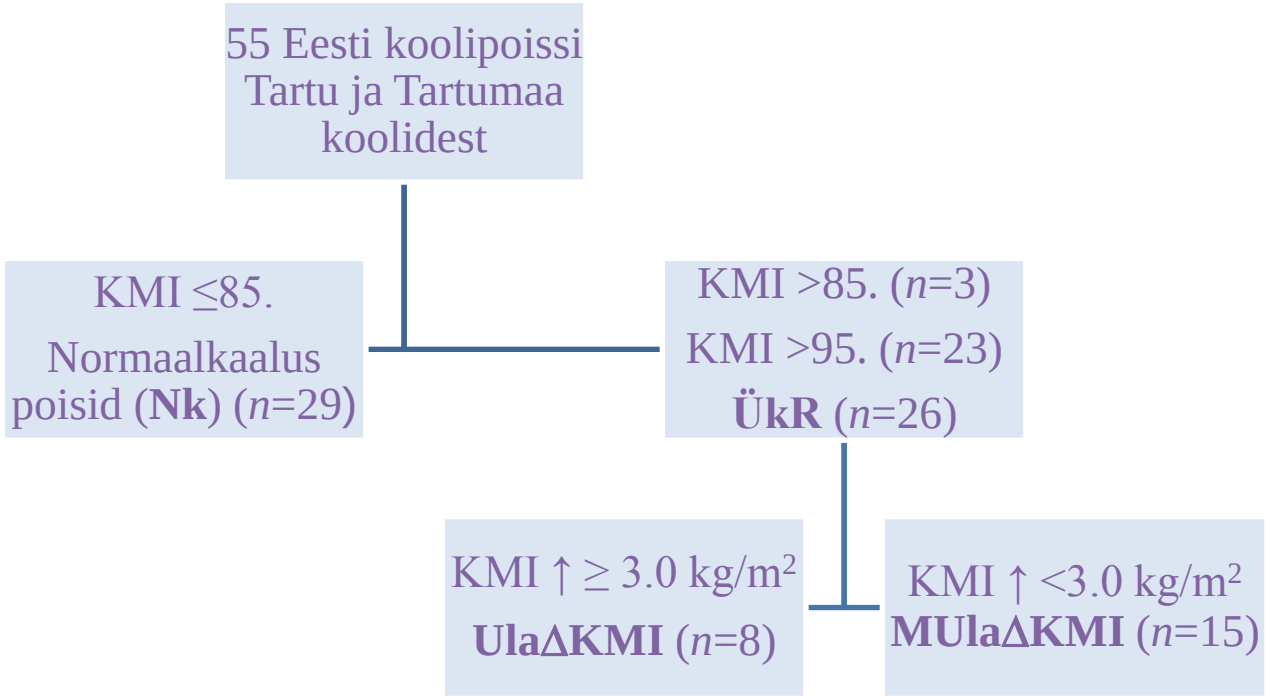
PIIRATUD JA VASTUOLULISED TEADMISED →



Uurimustöö eesmärk

- välja selgitada **muutused** keha koostises, sh luutiheduse näitajates ja vereseerumi põletiku biomarkerites erineva KMI (+ KMI tõusuga) poistel murdeeas;
- uurida **seoseid** nende parameetrite vahel erineva KMI tõusuga ülekaalulistel ja rasvunud (ÜkR) poistel murdeeas.

Valim



- Vanus uuringuperioodi alguses 10-11 aastat
- Kolme aasta jooksul 12-kuulise intervalliga
- Jaotus ÜkR gruppides KMI tõusu alusel (+3.0 kg/m²) tuginedes Eesti poiste KMI kõveratele

Meetodid kokkuvõtlikult

- Antropomeetria ja suguline küpsemine
- Vere biokeemilised markerid
  - 12 põletiku biomarkerit, 2 rasvkoe hormooni, testosteroon, 2 metaboolset biomarkerit
- Luutiheduse ja keha koostise parameetrid
  - Kogu keha ja lülisamba lumbaalosa luutihedus ja –mass
    - Sh volumeetriline luutihedus
  - Rasvamass ja selle %, rasvavaba mass

Tulemused [1]

Table 1. The main clinical and body composition characteristics of participants at T0 and T3 {mean with 95% confidence intervals [e.g. X (Y–Z)] or median with 25th and 75th percentile [e.g. X (Y; Z)]}, and changes of these parameters presented as slopes through a 3-year study period {mean with 95% confidence intervals [e.g. X (Y–Z)]} in different study subgroups

Variable	Time Point	Group			
		OWB (n = 26)	NWB (n = 29)	OWB	
				EΔBMI (n = 8)	NEΔBMI (n = 15)
Age (y)	T0	11.1 (10.9–11.4)	10.9 (10.7–11.1)	11.0 (10.3–11.7)	11.1 (10.8–11.5)
	T3	14.1 (13.8–14.4)	13.8 (13.6–14.1)	13.9 (13.2–14.7)	14.1 (13.8–14.5)
Bone age (y)	T0	11.7 (11.2–12.3)*	10.4 (10.0–10.8)	11.1 (9.7–12.5)	12.1 (11.5–12.7)
	T3	14.7 (14.3–15.2)*†	13.7 (13.3–14.0)†	14.1 (13.0–15.1)†	15.0 (14.5–15.6)†
	Slo	1.0 (0.9–1.2)	1.1 (0.9–1.2)	1.1 (0.9–1.3)	0.96 (0.7–1.2)
Tanner (n/stage)	T0	3/21/2/0/0 *	11/16/2/0/0	0/7/1/0/0	2/12/1/0/0
	T3	0/1/4/11/9	0/1/5/16/6	0/1/1/5/1	0/0/2/7/6
Body mass (kg)	T0	63.2 (57.4–68.9)*	35.8 (33.9–37.7)	59.1 (42.1–76.2)	64.4 (57.1–71.8)
	T3	87.4 (78.8–96.1)*	54.0 (50.7–57.4)	90.6 (64.4–116.8)	87.5 (78.3–96.7)
Body height (m)	T0	1.52 (1.49–1.56)*	1.46 (1.44–1.49)	1.53 (1.44–1.63)	1.53 (1.49–1.57)
	T3	1.73 (1.69–1.76)*	1.67 (1.64–1.71)	1.72 (1.62–1.83)	1.74 (1.70–1.78)
BMI (kg/m^2)	T0	27.0 (25.2–28.7)*	16.6 (16.1–17.1)	24.4 (20.8; 27.8)	26.4 (24.2; 29.8)
	T3	29.0 (26.9–31.0)*†	19.2 (18.5–19.9)†	27.9 (25.6; 31.6)	28.1 (26.3; 31.3)
	Slo	0.69 (0.32–1.06)	0.88 (0.72–1.04)	1.75 (0.85–2.66)*	0.51 (0.35–0.68)
Testo (nmol/L)	T0	0.73 (<0.35; 1.69)	0.48 (<0.35; 0.82)	0.36 (0.35; 0.81)	0.76 (0.44; 1.69)
	T3	10.1 (6.2; 13.8)†	12.5 (8.0; 15.7)†	6.0 (4.7; 13.3)†	11.5 (6.3; 13.8)†
	Slo	2.8 (1.8; 4.0)	3.9 (2.3;4.9)	1.8 (1.2; 3.2)	3.3 (2.1; 4.0)

Tulemused [2]

Table 1. The main clinical and body composition characteristics of participants at T0 and T3 {mean with 95% confidence intervals [e.g. X (Y–Z)] or median with 25th and 75th percentile [e.g. X (Y; Z)]}, and changes of these parameters presented as slopes through a 3-year study period {mean with 95% confidence intervals [e.g. X (Y–Z)]} in different study subgroups

Variable	Time Point	Group			
		OWB (n = 26)	NWB (n = 29)	OWB	
				EΔBMI (n = 8)	NEΔBMI (n = 15)
TBF%	T0	41.3 (36.7; 46.3)*	16.2 (12.3;19.5)	41.0 (35.9; 43.0)	40.9 (36.3; 46.3)
	T3	39.4 (30.6; 42.1)*	16.2 (11.1; 24.2)	40.9 (38.2; 42.0)	34.5 (30.0; 42.3)
	Slo	-1.8 (-2.6; -0.9)*	0.4 (-0.8; 1.6)	0.01 (-2.5; 1.8)*	-1.7 (-3.4;-0.9)
TB FM (kg)	T0	24.6 (18.3; 30.1)*	5.1 (4.0; 7.0)	23.0 (14.8; 29.3)	25.1 (18.4; 30.1)
	T3	30.5 (22.6; 35.0)*†	8.6 (6.5; 11.4)†	33.0 (26.3; 34.9)†	28.3 (22.3; 36.6)†
	Slo	1.9 (1.2–2.7)	1.3 (0.86–1.7)	3.7 (2.0–5.5)*	1.5 (0.8–2.3)
TB FFM (kg)	T0	33.9 (31.5; 38.6)*	27.4 (25.6; 29.2)	33.1 (26.2; 38.2)	34.6 (32.6; 39.1)
	T3	50.8 (40.8; 59.0)*†	40.2 (35.6; 45.2)†	47.3 (37.0; 67.3)†	52.6 (43.6; 59.0)†
	Slo	5.6 (4.5–6.8)	4.9 (4.0–5.9)	6.3 (3.5–9.0)	5.4 (3.9–7.0)
TR FM (kg)	T0	10.8 (7.3; 14.4)*	1.8 (1.3; 2.1)	10.6 (6.6; 14.1)	10.5 (7.3; 14.4)
	T3	13.5 (10.1; 16.5)*†	3.7 (2.9; 4.5)†	14.4 (12.4; 17.0)†	12.8 (89.0; 17.9)†
	Slo	0.9 (0.6–1.3)	0.6 (0.4–0.8)	1.8 (0.9–2.6)*	0.8 (0.4–1.2)

Original Article
PEDIATRIC OBESITY

Obesity

Changes in Inflammatory Markers in Estonian Pubertal Boys with Different BMI Values and Increments: A 3-Year Follow-Up Study

Eva Mengel [1,2], *Vallo Tillmann* [2,3], *Liina Remmel* [1], *Pille Kool* [2,3], *Priit Purge* [1], *Evelin Lätt* [1], *and Jaak Jürimäe* [1]

Objective: Serum inflammatory markers could help to identify those boys with overweight (OWB) who gain weight more extensively during puberty. This study aimed to examine the longitudinal changes in different serum inflammatory markers through puberty in boys with different BMI values and increments.
Methods: Twenty-six OWB and 29 normal-weight boys (NWB) were followed yearly for 3 years to measure changes in BMI and serum concentrations of 12 inflammatory markers.
Results: OWB had higher ($P < 0.033$) baseline interleukin (IL)-6, IL-8, interferon gamma (IFN-γ), tumor necrosis factor alpha (TNF-α), and IL-1α concentrations in comparison with NWB. Over the 3-year period, IFN-γ ($P = 0.0001$) and TNF-α ($P = 0.0042$) decreased more in OWB compared to NWB. Serum IL-8, monocyte chemoattractant protein (MCP)-1, and leptin increased further in those OWB who gained BMI more extensively through puberty compared to OWB who gained weight at slower rates ($P < 0.033$).
Conclusions: Serum IFN-γ and TNF-α levels decreased more during pubertal years in OWB compared to NWB, indicating that pubertal maturation itself may have a favorable impact on the inflammation of obesity. Serum IL-8, MCP-1, and leptin could help to identify OWB who gain BMI more extensively during pubertal years.

Obesity (2017) **25**, 600-607. doi:10.1002/oby.21756

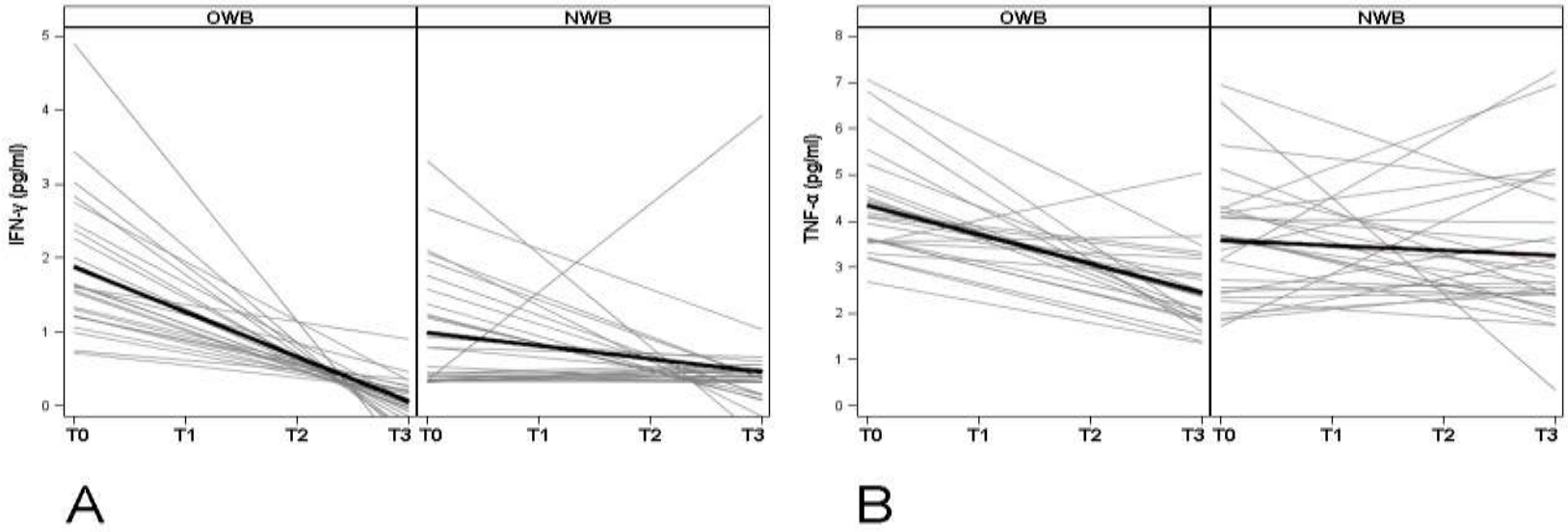


Figure 1. Longitudinal changes in different cytokines in OWB and NWB. Panel A is slope lines for IFN-γ; panel B is slope lines for TNF-α. For panels A-B, gray lines represent longitudinal changes of an individual value, and the bold lines are the best fitting lines representing the group mean.

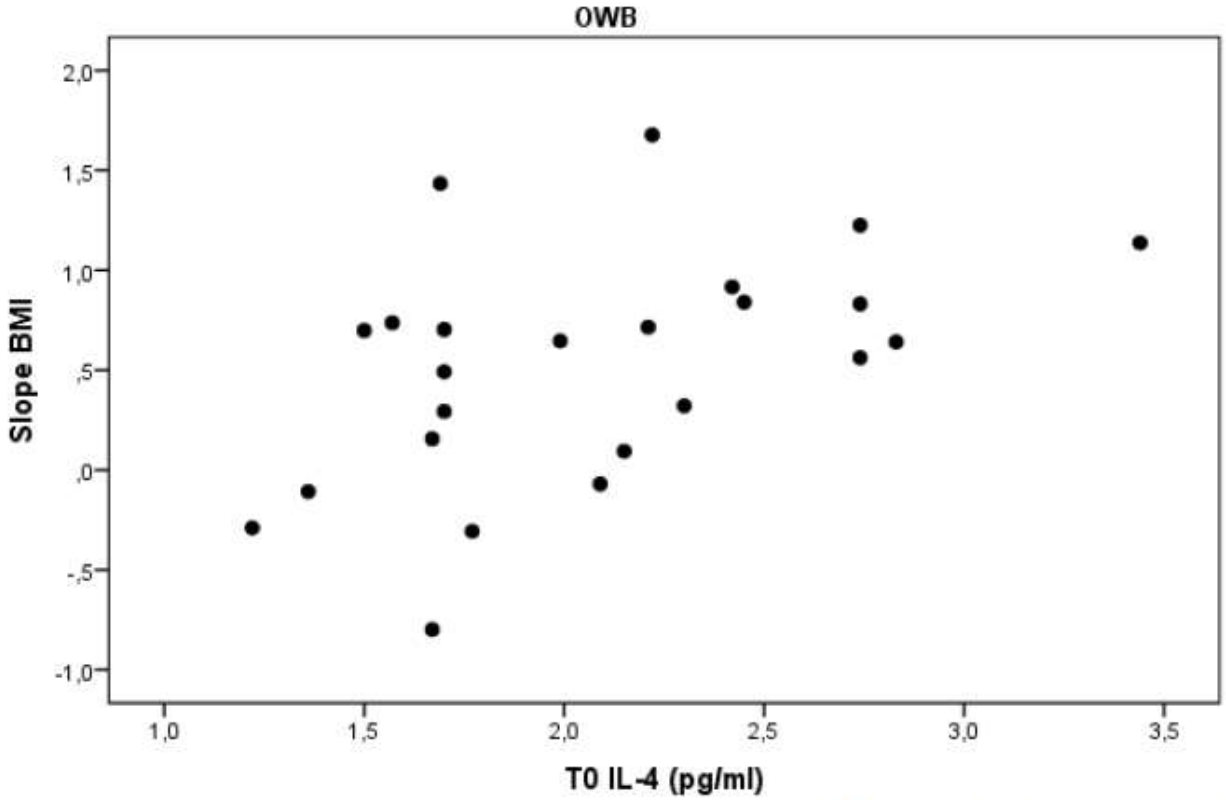


Figure 3. Spearman correlation between baseline interleukin-4 and BMI slope in the OWB group ($r = 0.48$, $P = 0.02$).

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ORIGINAL RESEARCH

Extensive BMI Gain in Puberty is Associated with Lower Increments in Bone Mineral Density in Estonian Boys with Overweight and Obesity: A 3-Year Longitudinal Study

Eva Mengel[1,3] · Vallo Tillmann[2,3] · Liina Remmel[1] · Pille Kool[2] · Priit Purge[1] · Evelin Lätt[1] · Jaak Jürimäe[1]

Luutiheduse näitajate muutused erineva KMI-ga poistel

Var	Time point	OWB (n = 26)	NWB (n = 29)
TB BMD (g/cm^2)	T0	1.01 (0.99–1.03)*	0.94 (0.93–0.96)
	T3	1.05 (1.03–1.08)*†	1.03 (1.01–1.05)†
	Slo	0.05 (0.04–0.06)*	0.03 (0.02–0.04)
TB BMAD (g/cm^3)	T0	0.09 (0.08–0.09)*	0.09 (0.09–0.10)
	T3	0.08 (0.08–0.09)	0.09 (0.08–0.09)†
	Slo	-0.0004 ((-0.001)– 0.001)*	-0.003 ((-0.003)–(-0.002))
TB BMC (g)	T0	1844 (1568; 1938)*	1392 (1310; 1504)
	T3	2745 (2409; 3188)*†	1985 (1861; 2363)†
	Slo	308 (264–351)*	235 (203–266)
TB BMC/height	T0	12.1 (10.7; 12.9)*	9.7 (9.1; 10.0)
	T3	15.6 (14.4; 17.8)*†	12.0 (11.7; 13.9)†
	Slo	1.29 (1.08–1.50)*	0.98 (0.84–1.13)
LS BMD (g/cm^2)	T0	0.84 (0.8; 0.9)*	0.77 (0.73; 0.81)
	T3	1.05 (0.94; 1.19)*†	0.89 (0.85; 1.01)†
	Slo	0.08 (0.06–0.09)*	0.05 (0.04–0.06)
LS BMAD (g/cm^3)	T0	0.15 (0.15; 0.16)	0.14 (0.14; 0.15)
	T3	0.16 (0.16; 0.17)*†	0.15 (0.14; 0.16)†
	Slo	0.005 (0.003–0.006)	0.003 (0.002–0.004)
LS BMC (g)	T0	26.9 (22.7; 30.0)*	21.0 (19.8; 23.7)
	T3	46.1 (34.5; 53.5)*†	35.3 (29.6; 37.3)†
	Slo	6.3 (5.2–7.5)*	4.2 (3.4–5.0)

Var	Time point	OWB	
		EΔBMI (n = 7)	NEΔBMI (n = 15)
TB BMD (g/cm^2)	T0	0.99 (0.93–1.05)	1.02 (0.99–1.04)
	T3	1.09 (1.02–1.15)*†	1.18 (1.13–1.22)†
	Slo	0.03 (0.02–0.04)*	0.05 (0.04–0.06)
TB BMAD (g/cm^3)	T0	0.09 (0.08–0.09)	0.09 (0.08–0.09)
	T3	0.09 (0.08–0.09)	0.08 (0.08–0.09)
	Slo	-0.0012 ((-0.003–0.0003))	-0.001 (-0.002)–0.001)
TB BMC (g)	T0	1703 (1307–2098)	1895 (1687–2102)
	T3	2390 (1973–2808)*†	2899 (2632–3167)†
	Slo	235 (173–297)*	340 (283–398)
TB BMC/height	T0	11.1 (9.2–13.0)	12.3 (11.3–13.3)
	T3	13.9 (12.3–15.5)*†	16.6 (15.4–17.8)†
	Slo	0.965 (0.69–1.23)*	1.46 (1.17–1.74)
LS BMD (g/cm^2)	T0	0.80 (0.71–0.89)	0.85 (0.8–0.9)
	T3	0.94 (0.84 – 1.04)*†	1.09 (1.02–1.17)†
	Slo	0.05 (0.03–0.07)*	0.08 (0.07–0.10)
LS BMAD (g/cm^3)	T0	0.16 (0.13; 0.16)	0.15 (0.14; 0.16)
	T3	0.16 (0.15; 0.16)	0.17 (0.15; 0.17)†
	Slo	0.002 ((-0.0003)–0.004)*	0.006 (0.004–0.007)
LS BMC (g)	T0	24.0 (18.6–29.5)	28.2 (24.7–31.8)
	T3	36.5 (25.9–47.0)*†	48.4 (42.5–54.4)†
	Slo	4.2 (2.0–6.5)*	6.9 (5.5–8.3)

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ORIGINAL ARTICLE

The associations between the changes in serum inflammatory markers and bone mineral accrual in boys with overweight and obesity during pubertal maturation: a 3-year longitudinal study in Estonian boys

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Abstract

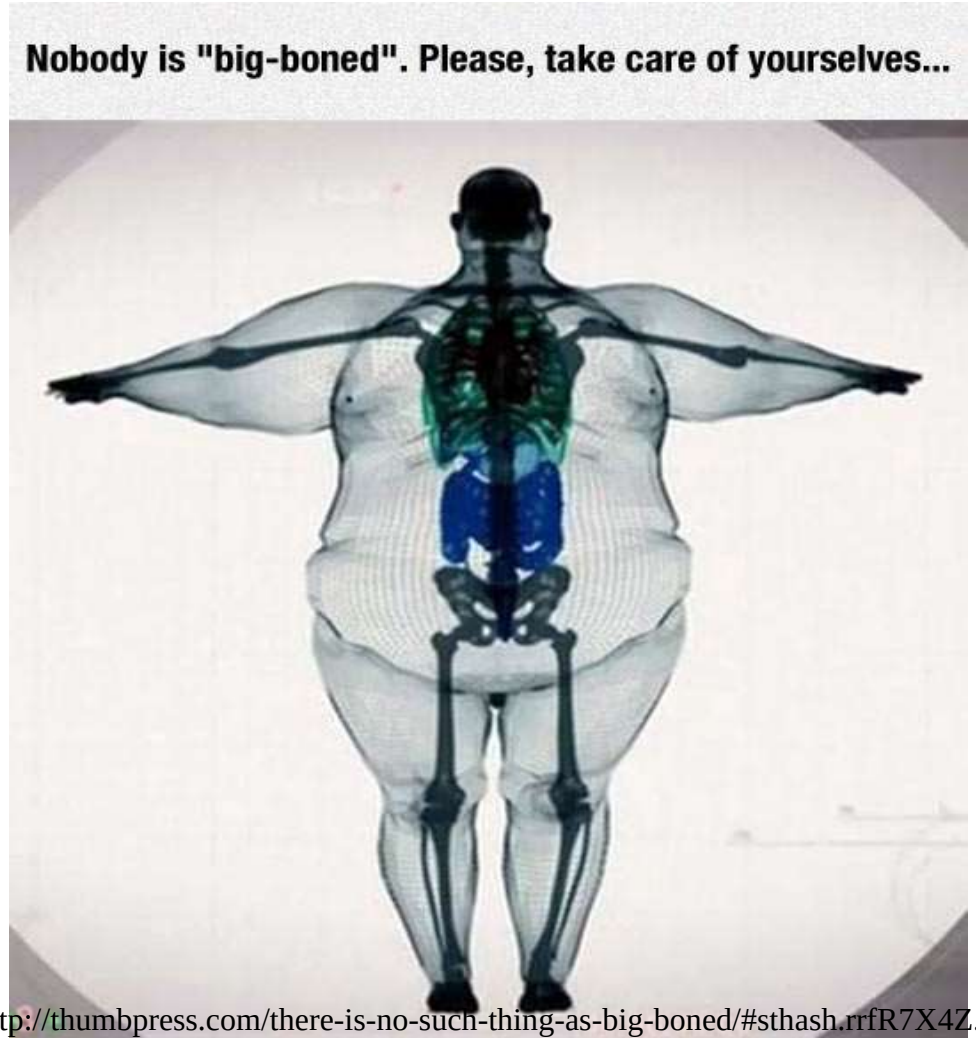
Summary Adipose tissue produces different inflammatory cytokines which compromise bone mineral accrual during puberty. Vascular endothelial growth factor (VEGF), epidermal growth factor (EGF), interleukin (IL)-8, and interferon-gamma (IFN-γ) are significantly related to bone mineral accrual during pubertal maturation in boys with different BMI values.

Introduction This longitudinal study aims to identify the inflammatory markers that most strongly associate with pubertal bone mineral density (BMD) increment in boys with overweight and obesity (OWB).

Methods Twenty-six OWB and 29 normal-weight boys were followed yearly for 3 years to measure changes in 12 serum inflammatory markers, BMD (by DXA), and apparent volumetric BMD. The OWB group was further divided into two subgroups according to their BMI gain during the 3-year period. Data through time points presented as slopes were used to calculate correlation coefficients to explore the possible relationships between variables of interest. In the whole study group, linear mixed effects (LME) models were also used.

Results Increment in serum VEGF concentration was inversely associated with an increase in total body (TB) BMD ($r = -0.82$, $P = 0.02$) and TB bone mineral content (BMC)/height ($r = -0.82$, $P = 0.02$) in those OWB whose BMI gain was higher during pubertal years. In the whole study group, the LME model confirmed the inverse association between VEGF and TB BMC/height ($P < 0.05$). EGF was inversely associated with LS BMD and LS BMAD ($P < 0.05$), whereas there was a positive association between IL-8 and TB BMAD and between IFN-γ and LS BMD ($P < 0.05$).

Conclusions Lower increment in BMD in OWB with higher BMI gain is associated with increasing serum VEGF concentration during pubertal maturation. VEGF, EGF, IL-8, and IFN-γ are significantly associated with BMD during pubertal maturation in boys with different BMI values.



http://thumbpress.com/there-is-no-such-thing-as-big-boned/#sthash.rrfR7X4Z.dpbs

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