Relation between obesity from childhood to adulthood and the metabolic syndrome: population based study

Mauno Vanhala, Pasi Vanhala, Esko Kumpusalo, Pirjo Halonen, Jorma Takala

Most researchers agree that obesity is an important modulator of the metabolic syndrome,[1 2] which is a clustering of cardiovascular risk factors associated with insulin resistance—such as hypertension, hypertriglyceridaemia, a low concentration of high density lipoprotein cholesterol, abnormal glucose metabolism, and hyperinsulinaemia.[3] Little is known, however, about the association between relative weight change from childhood to adulthood and the development of metabolic syndrome in adulthood.

Material, methods, and results

We recently published data of a population study for the metabolic syndrome, performed during 1993-4 in Pieksämäki, Finland. All subjects (n = 1008) born in the years 1947, 1952, and 1957 were examined according to a protocol described elsewhere.[4] Data on both weight and height at age 7 years (the start of primary school) were also collected.

Altogether, 712/1008 (70.6%) subjects participated in the study. Weights and heights at age 7 were traced for 439/712 (61.7%) participants. Obesity was defined both in childhood and in adulthood as a sex specific highest third of the body mass index (weight(kg)/(height (m)^2)). The metabolic syndrome was defined as a cluster of *(a)* hypertension (a systolic blood pressure ⩾140 mm Hg, a diastolic blood pressure ⩾90 mm Hg, or treatment with antihypertensive drugs); *(b)* dyslipidaemia (hypertriglyceridaemia (⩾1.70 mmol/l) or a high density lipoprotein cholesterol concentration of <1.00 mmol/l (<1.20 mmol/l in women), or both dyslipidaemia and hypertriglyceridaemia); and *(c)* insulin resistance (abnormal glucose metabolism according to the World Health Organisation's criteria or hyperinsulinaemia (⩾78 pmol/l), or both).[2–4]

Of the 439 subjects, 75 had been obese and 219 not obese in both childhood and adulthood; 71 had not been obese as children but were obese as adults; and 74 had been obese as children but were not obese as adults. The metabolic syndrome was present in 18/219 (8%) men and in 12/220 (5%) women. Of the 30 subjects having this syndrome, 28 were obese as adults; 21 of them had also been obese as children (table). In exact logistic regression analysis (LogXact), the risk of metabolic syndrome was 2.9 (95% confidence interval 1.1 to 7.6) for the subjects who had been obese as children and 26.7 (6.4 to 237) for the subjects who were obese as adults, compared with their non-obese controls. None of the 74 subjects who had been obese as children but who were not obese as adults had the metabolic syndrome. The increased risk of the metabolic syndrome was still present when the population was split into thirds for weight but not when it was split into thirds for height.

Comment

Our results show that half of the obese children had become obese adults with an especially high risk of the metabolic syndrome and that childhood obesity overall increases the risk for the metabolic syndrome in adulthood. The risk of the syndrome was lower among the obese adults who had not been obese as children than among the obese adults who had also been obese as children. Independent of childhood obesity, the risk for developing the syndrome was lowest among the non-obese subjects overall. This finding suggests that obesity in adulthood that became established in childhood may be a more harmful than obesity that has appeared in adulthood. The possible mechanism is that continuous obesity from childhood to adulthood serves as a "generator" for prolonged insulin resistance, which results in the clustering of hypertension and metabolic abnormalities in the same individual.[5] Our results also show that if an obese child reduces his or her relative weight to become a non-obese adult, this may protect against the metabolic syndrome. Thus, the identification of obese children could lead to early intervention to prevent adult obesity, the metabolic syndrome, and cardiovascular risk.

Contributors: MV had the original idea for the present study, coordinated the formulation of the primary study hypothesis, discussed core ideas, designed the protocol, and participated in the data collection, analysis, and writing of the paper. He will also act as the guarantor for the paper. PV collected the data on weights and heights at age 7, discussed the study hypothesis and core ideas, and participated in the writing of the paper. PH participated in the statistical analysis and in the discussion of the core ideas. EK and JT discussed the study hypothesis and core ideas, participated in the planning of the design and practical implementation of the study, and edited and contributed to the paper.

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Pieksämäki District Health Centre, PO Box 65, 76101 Pieksämäki, Finland
Mauno Vanhala, *general practitioner*
Pasi Vanhala, *general practitioner*
Kuopio University, Department of Public Health and General Practice, PO Box 1627, FIN-70211 Kuopio, Finland
Esko Kumpusalo, *lecturer*
Pirjo Halonen, *statistician*
Jorma Takala, *professor*
Correspondence to: Dr M Vanhala, Community Health Centre of the City of Imatra, Virastokatu 2, 55100 Imatra, Finland
Mauno.Vanhala@pp.inet.fi

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Prevalence and risk (odds ratio) of metabolic syndrome for 146 obese adults (73 men and 73 women), according to childhood obesity status, compared with 293 non-obese adults (146 men and 147 women). Values are numbers (percentages) of subjects unless stated otherwise

		Obese adults†	
	Non-obese adults*	Not obese in childhood‡ (men, n=32; women, n=39)	Obese in childhood§ (men, n=41; women, n=34)
Men	2 (1)	4 (13)	12 (29)
Women	0	3 (8)	9 (26)
Total	2 (1)	7 (10)	21 (28)
Odds ratio (95% CI)	1	16 (2.9 to 159)	56 (13 to 504)

*Body mass index <27.7 in men, <26.6 in women. †Body mass index ⩾27.7 in men, ⩾26.6 in women. ‡Body mass index <15.8 in boys, <15.6 in girls. §Body mass index ⩾15.8 in boys, ⩾15.6 in girls.