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ORIGINAL ARTICLE

Thirty-year persistence of obesity after presentation to a pediatric obesity clinic

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Abstract
Background: Few large, long-term studies are available on the relationship between childhood and adult obesity.
Aim: The present study examined the 30-year association between childhood and adult obesity in a large sample of girls with essential and uncomplicated obesity.
Subjects and methods: 318 girls who had visited our Pediatric Obesity Clinic between January 1972 and December 1974 were re-contacted between January 2002 and December 2005. All had undergone an assessment of weight, height and pubertal status at the baseline visit. Anthropometry was performed again on those who agreed to take part in the follow-up visit. The women's general practitioners were also asked to compile a health questionnaire. Hypertension, hypercholesterolemia, hypertriglyceridemia and diabetes were defined according to current guidelines. Rates are expressed as number of cases per 1000 person-years (PY). Multivariable Poisson regression was used to identify predictors of persistent obesity.
Results: 224 (70%) of the 318 girls took part to the 30-year follow-up study. They had the same baseline anthropometry of those not available at follow-up. Sixteen per cent of them were still obese at the 30-year follow-up, giving a persistence rate of obesity of 5.2×1000 PY. Tanner stages ≥ 1 [rate = ratios (RR) from 4.73 to 7.74 for different stages, $p \leq 0.021$] and Z-score of BMI (RR = 2.72 for one SDS, $p = 0.019$) were independent predictors of obesity persistence. Having a university degree vs. an elementary degree was instead protective (RR = 0.32, $p = 0.009$). The most prevalent complication was hypertriglyceridemia (8.8×1000 PY), followed by hypercholesterolemia (rate = 8.4×1000 PY), hypertension (rate = 5.2×1000 PY) and diabetes mellitus (rate = 1.0×1000 PY).
Conclusion: The study reinforces the notion that obesity should be prevented at an early age and shows that adolescents with severe obesity and low educational degree are at greater risk of becoming obese adults.

Keywords: *Childhood, obesity, incidence, body mass index, risk factors*

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Introduction

Obesity is one of the most important health problems in industrialized countries (Flegal et al. 2007). In the USA, the prevalence of obesity has doubled in 6–12-year-old children and tripled in 13–17-year-old children in just 20 years (Ogden et al. 2002; Slyper 2004). A similar epidemic is undergoing in Italy, where overweight and obesity affect nearly 40% of children in central and southern regions (SIEDP 1992).

There is increasing evidence that childhood obesity is associated with cardiovascular and metabolic disease in adult age (Must et al. 1992; Srinivasan et al. 1996; Vanhala et al. 1998; Freedman et al. 1999). Moreover, the same constellation of cardiovascular risk factors, which plagues adult obesity, is frequently observed in obese children (Barlow and Dietz 1998). Childhood obesity is in fact associated with hyperinsulinemia, type 2 diabetes, hypertension, increased low-density lipoprotein cholesterol and decreased high-density lipoprotein cholesterol (Barlow and Dietz 1998; Freedman et al. 1999; Weiss et al. 2004). There is also some evidence that childhood obesity is a risk factor for adult morbidity independently from its persistence into adulthood (Srinivasan et al. 1996; Baker et al. 2007).

Obese children have an estimated 22–100% risk of remaining overweight in adulthood (Stark et al. 1981; DiPietro et al. 1994). The risk factors for the persistence of childhood obesity into adulthood have been systematically reviewed (Parsons et al. 1999). There are, however, few large long-term studies of the relationship between childhood obesity and adult obesity. We therefore evaluated the long-term relationship between childhood obesity and adult obesity in a large sample of female obese children referred to our Pediatric Obesity Clinic.

Materials and methods

Study design

We performed a follow-up study of the female children who had been consecutively visited at the Outpatient Clinic of the Pediatric Obesity Center of L'Aquila University (Italy) between January 1972 and December 1974. These years were chosen because they were the first 2 years of activity of our Clinic. Only subjects with simple and uncomplicated obesity were studied. Immediately after the first visit, the girls underwent dietary counseling and were referred to their general practitioners. The subjects were contacted again between January 2002 and December 2005. The local Ethical Committee approved the study protocol and all subjects gave written informed consent.

Anthropometry

Weight and height were measured according to standard guidelines (Lohman et al. 1988). Body mass index (BMI) was calculated as weight (kg)/stature (m)^2. The Z-score of BMI was calculated using US reference values and is given as standard deviation score (SDS) (Kuczmarski et al. 2000). In children, overweight was defined as $BMI \geq 85th$ (1.036 SDS) and <95th (1.645 SDS) and obesity as a value of $BMI \geq 95th$ (1.645 SDS) percentile for gender and age. Adult obesity was diagnosed as a $BMI \geq 30.0 \, kg/m^2$ and overweight as a $BMI \geq 25.0$ and $<30.0 \, kg/m^2$ (NIH 1998). Pubertal status was evaluated in five stages using Tanner's criteria (Tanner 1990).

Assessment of cardiovascular risk factors

A structured questionnaire was sent to the general practitioners of the women to obtain a history of past and present disease. The variables of the questionnaire employed for the present analysis were: (1) hypertension (yes/no), i.e. systolic blood pressure >140 mm Hg and/or diastolic blood pressure >90 mm Hg (Chobanian et al. 2003); (2) hypercholesterolemia (yes/no), i.e. cholesterol >240 mg dL^{-1} (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults 2001); (3) hypertriglyceridemia (yes/no), i.e. fasting triglycerides >150 mg dL^{-1} (NIH 1998); and (4) diabetes mellitus (yes/no), i.e. fasting glucose >126 mg dL^{-1} or use of antidiabetic drugs (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults 2001).

Statistical analysis

Continuous variables are given as medians and inter-quartile ranges (IQR) because of skewed distributions. IQR was calculated as the difference between the 75th and the 25th percentile. Between-group comparisons of continuous variables were performed with the Wilcoxon–Mann–Whitney test and those of categorical variables with the Fisher's exact test. Persistent obesity (1 = yes; 0 = no) was defined as obesity present at both baseline and follow-up visits. Hypertension (1 = yes; 0 = no), hypertriglyceridemia (1 = yes; 0 = no), hypercholesterolemia (1 = yes; 0 = no) and type 2 diabetes mellitus (1 = yes; 0 = no) at the 30-year follow-up were defined as described above. Rates are expressed as number of cases per 1000 person-years (PY). Multivariable Poisson regression with robust 95% confidence intervals (95% CI) was used to evaluate the contribution of baseline age (continuous, modeled as 1-year increase), pubertal status (categorical, modeled using all Tanner stages ≥1 vs. stage 1), educational level [categorical, modeled using all levels ≥1 (2 = high school, 3 = university school) vs. level 1 (elementary school)] and *Z*-BMI (continuous, modeled as SDS) to persistent obesity, incident hypertension, incident hypertriglyceridemia and incident hypercholesterolemia (Lumley et al. 2006). Exact multivariable Poisson regression was used to model the relationship between incident type 2 diabetes mellitus and the predictors of interest because of the low number of incident outcomes (Hirji et al. 1987). Statistical significance was set to a *p*-value <0.05 for all tests. All statistical tests are two-tailed. Statistical analysis was performed using STATA 10.0 (STATA Corporation, College Station, TX, USA) and LogXact 8.0 (Cytel, Cambridge, MA, USA).

Results

Of the 318 Caucasian girls aged 3–18 years who had been evaluated at our Clinic between January 1972 and December 1974, 224 (70%) agreed to participate in the 30-year follow-up study. Two hundred and sixty-eight (84%) of the girls were obese and 50 (16%) were overweight at the baseline visit.

Table I shows that the women available and not available at the 30-year follow-up had similar anthropometric measurements at the baseline visit.

Table II gives the measurements of the 224 women available at the 30-year follow-up. The median (IQR) follow-up time of the cohort was 31 (2) years (range: 29–33 years), corresponding to 6911 PY.

Table I. Baseline measurements of children available and not available at the 30-year follow-up. Values are medians and interquartile ranges for continuous variables and number of subjects for categorical variables. Abbreviations: SDS, standard deviation score; Z, Z-score; BMI, body mass index.

	Not available at follow-up ($n = 94$)	Available at follow-up ($n = 224$)	p-value★
Age (years)	10.1 (5.0)	10.0 (4.0)	0.319
Weight (kg)	52.4 (26.7)	49.8 (23.1)	0.241
Z-weight (SDS)	2.1 (0.9)	2.0 (0.9)	0.792
Height (m)	1.45 (0.26)	1.40 (0.23)	0.267
Z-height (SDS)	0.6 (1.3)	0.6 (1.3)	0.979
BMI (kg/m^2)	26.3 (4.4)	25.5 (3.9)	0.337
Z-BMI (SDS)	2.1 (0.6)	2.1 (0.5)	0.835
Tanner stage (1/2/3/4/5)	33/21/17/9/14	97/36/33/32/26	0.310

★Wilcoxon–Mann–Whitney test for continuous variables and Fisher's exact test for categorical variables.

Table II. Measurements of the 224 women available at the 30-year follow-up. Abbreviations: IQR = interquartile range; other abbreviations as in Table I.

	Median	IQR	Minimum	Maximum
Age (years)	40.1	3.1	34.1	50.0
Weight (kg)	63.8	17.9	41.9	112.0
Height (m)	1.60	0.07	1.40	1.74
BMI (kg/m^2)	25.1	7.3	15.4	42.5

Table III. Persistence of obesity and incidence of hypertension, hypertriglyceridemia, hypercholesterolemia and type 2 diabetes at the 30-year follow-up.

	n	Rate (95% CI)★
Obesity	36	5.2 (3.8–7.2)
Hypertension	36	5.2 (3.8–7.2)
Hypertriglyceridemia	61	8.8 (6.9–11.3)
Hypercholesterolemia	58	8.4 (6.5–10.9)
Type 2 diabetes	7	1.0 (0.5–2.1)

★Rate is number of cases per 1000 person-years.

Table III gives the persistence rate of obesity and the incidence rate of cardio-metabolic disease at the 30-year follow-up. The persistence rate of obesity was of $5.2 \times 1000\,\text{PY}$. In detail, 9% of the women were underweight ($n = 20$), 40% normal weight ($n = 91$), 34% overweight ($n = 77$), and 16% obese ($n = 36$). The most prevalent complication was hypertriglyceridemia (rate $= 8.8 \times 1000\,\text{PY}$), followed by hypercholesterolemia (rate $= 8.4 \times 1000\,\text{PY}$), hypertension (rate $= 5.2 \times 1000\,\text{PY}$) and type 2 diabetes mellitus (rate $= 1.0 \times 1000\,\text{PY}$).

Table IV gives the multivariable analysis of the predictors of persistent obesity and incident hypertension, hypercholesterolemia, hypertriglyceridemia and type 2 diabetes mellitus.

The rate of persistent obesity was 4.73–7.74 times higher in pubertal than in prepubertal children. An increase of one SDS of Z-BMI increased the rate of persistent obesity

Table IV. Multivariable Poisson regression of persistent obesity and incident hypertension, hypercholesterolemia, hypertriglyceridemia and type 2 diabetes mellitus at the 30-year follow-up.

	Obesity RR [robust 95% CI] (p-value)	Hypertension RR [robust 95% CI] (p-value)	Hypercholesterolemia RR [robust 95% CI] (p-value)	Hypertriglyceridemia RR [robust 95% CI] (p-value)	Type 2 diabetes mellitus RR [exact 95% CI] (exact p-value)
Age (years)	1.04 [0.86–1.26] (0.657)	1.25 [1.02–1.54] (0.031)	1.14 [0.98–1.33] (0.097)	1.16 [1.00–1.35] (0.056)	2.43 [1.20–5.82] (0.009)
Tanner stage 2*	5.53 [1.77–17.31] (0.003)	2.07 [0.66–6.48] (0.212)	2.18 [1.04–4.55] (0.038)	1.09 [0.55–2.19] (0.799)	1.41 [0.69–27.18] (1.000)
Tanner stage 3*	4.73 [1.37–16.28] (0.014)	2.76 [0.81–9.39] (0.103)	1.38 [0.57–3.34] (0.479)	0.53 [0.22–1.28] (0.158)	0.26 [0.00–9.59] (0.784)
Tanner stage 4*	7.74 [1.92–31.18] (0.004)	1.87 [0.46–7.54] (0.379)	1.73 [0.65–4.58] (0.273)	1.23 [0.53–2.89] (0.630)	0.12 [0.00–2.64] (0.1760)
Tanner stage 5*	6.88 [1.33–35.58] (0.021)	0.67 [0.08–5.34] (0.704)	1.67 [0.49–5.65] (0.411)	0.50 [0.15–1.72] (0.273)	0.00 [0.00–8.34] (0.454)
Z-BMI (SDS)†	2.72 [1.18–6.28] (0.019)	1.85 [0.80–4.29] (0.154)	1.74 [0.90–3.36] (0.102)	1.92 [1.01–3.66] (0.049)	22.36 [1.08–528.79] (0.044)
Superior school‡	0.53 [0.27–1.04] (0.063)	0.96 [0.52–1.78] (0.895)	0.84 [0.50–1.40] (0.504)	1.24 [0.73–2.12] (0.427)	0.44 [0.06–2.83] (0.516)
University school‡	0.32 [0.14–0.75] (0.009)	0.56 [0.25–1.22] (0.142)	0.80 [0.46–1.41] (0.439)	0.90 [0.50–1.65] (0.744)	0.13 [0.00–1.02] (0.052)

*Categorical and modeled *vs.* Tanner stage 1.
†Continuous.
‡Categorical and modeled *vs.* Elementary school.

by 2.72 times. Lastly, the rate of persistent obesity was 68% lower in subjects with a university degree as compared to those with an elementary degree. The rate of incident hypertension increased for increasing age at baseline but the 95% CI were wide (1.02 to 1.54); the fact that hypercholesterolemia was more incident in Tanner 2 stage is likely a matter of chance since no trend was seen for pubertal stages from 2 to 5; the rate of incident hypertriglyceridemia was associated with baseline Z-BMI but the 95% CI were very wide (1.01–3.66); lastly, the incidence of type 2 diabetes mellitus was associated with age (RR = 2.43, exact-95% CI 1.20–5.82) and baseline Z-BMI, but the precision of this latter estimate is very low (exact-95% CI 1.08–528.79) because of the very low number of incident outcomes ($n = 7$).

Discussion

In the present study, one in every two obese girls was still obese (16%) or overweight (34%) at 30 years of follow-up. These prevalence rates are higher than those reported by the Italian Institute of Statistics for women of this age from this part of Italy (obesity = 5% and overweight = 19%) (ISTAT 2005). However, if one considers obesity alone, our data are more optimistic than others available in the literature (Abraham et al. 1971; Must et al. 1992; DiPietro et al. 1994; Srinivasan et al. 1996; Power et al. 1997; Must and Strauss 1999; Togashi et al. 2002; Field et al. 2005; Freedman et al. 2005a, b; Viner and Cole 2006; Thompson et al. 2007). For instance, from 26% to 41% of obese pre-school children and from 42% to 63% of obese school children have been reported to become obese adults (Must and Strauss 1999). On the other hand, our data are in agreement with those obtained from other studies (Charney et al. 1976; Stark et al. 1981; Garn and LaVelle 1985; Rolland-Cachera et al. 1987; Mossberg 1989; Deshmukh-Taskar et al. 2006; Juonala et al. 2006) (Table V).

In the present study, the risk of persistent obesity increased for increasing values of baseline Z-BMI. This finding confirms the results obtained by other studies that showed a significant association between BMI in childhood and adulthood (Sandhu et al. 2006). In our subjects, the relative risk of adult obesity was 2.72 (95% CI 1.18–6.28) for every SDS of baseline Z-BMI. Thus, we confirm that the degree of childhood obesity is a risk factor for adult obesity, even if the incidence of adult obesity was lower in our study than in most previous studies (Must and Strauss 1999). It is possible that the dietary counseling offered at the baseline visit may have contributed to the lower incidence of adult obesity as compared to other studies available in the literature.

It is known that the risk of becoming a severely obese adult increases exponentially over the entire range of childhood BMI (Sørensen and Sonne-Holm 1988). The risk of adult obesity is also greater in children who are obese at older ages (Serdula et al. 1993; Guo and Chumlea 1999). In our cohort, however, pubertal status but not age at baseline did predict persistence of obesity at the 30-year follow-up. The rate ratio for persistent obesity was in fact 4.73–7.74 higher in pubertal than in pre-pubertal children while age was not associated with the outcome. On the other hand, an earlier timing of puberty has been shown to be independently associated with an increased BMI in adulthood (Sandhu et al. 2006).

The present study also shows that, other factors being equal, a high social status – evaluated using the surrogate measurement of a university degree – protects from becoming an obese adult (RR = 0.32). This finding is in agreement with the evidence that a

Table V. Persistence of childhood obesity into adulthood: A review of published studies. Abbreviations: M, male; F, female; Wt, weight; RWt, relative weight; TSF, triceps skinfold; other abbreviations as in Table I.

	Overweight children, all (M/F)	Diagnosis of overweight	Age at baseline (years)	Follow-up (years)	Adult overweight (%)	Adult obesity (%)
Abraham et al. (1971)	21 (NA/NA)	RWt ≥ 120%	9–13	29–46	63	NA
Charney et al. (1976)	NA (NA/NA)	Wt > 90th percentile	0.1–0.5	20–30	36	NA
Stark et al. (1981)	203 (70/133)	RWt > 120%	7	19	M 43 F 41	NA
Garn and LaVelle (1985)	46 (NA/NA)	TSF ≥ 85th percentile	0.5–5	20–25	26	NA
Rolland-Cachera et al. (1987)	46 (NA/NA)	BMI > 75th percentile	1	17–24	41	NA
Mossberg (1989)	504 (NA/NA)	RWt > 1 SDS	NA	40	NA	47*
Must et al. (1992)	85 (32/53)	BMI > 75th percentile	13–18	53–66	52	NA
DiPietro et al. (1994)	504 (233/271)	BMI – cut-off NA	2 months–16 years	40	100	M 13 F 16†
Srinivasan et al. (1996)	191 (NA/NA)	BMI ≥ 75th percentile	13–17	12–14	58	NA
Power et al. (1997)	3968 (2323/1645)	BMI ≥ 95th percentile	7	26	M 80 F 70	M 38 F 44
Togashi et al. (2002)	276 (176/100)	RWt ≥ 120%	6–15	12	55	NA
Freedman et al. (2005a)	White M 124	BMI ≥ 95th percentile	5–14	17	White M 89	White M 71
	Black M 50	BMI ≥ 95th percentile			Black M 89	Black M 82
	White F 126	BMI ≥ 95th percentile			White F 90	White F 65
	Black F 95	BMI ≥ 95th percentile			Black F 95	Black F 84
Freedman et al. (2005b)	123 (51/72)	BMI ≥ 95th percentile	9–11	17	NA	M 76 F 78
Field et al. (2005)	103 (44/59)	BMI > 85th percentile	8–15	8–12	75	NA
Viner and Cole (2006)	467 (NA/NA)	BMI > 95th percentile	16	14	NA	61‡
Deshmukh-Taskar et al. (2006)	208 (NA/NA)	BMI > 85th percentile	9–11	8–26	22	NA
Juonala et al. (2006)	211 (NA/NA)	BMI ≥ 80th percentile	3–9	21	NA	27
	220 (NA/NA)	BMI ≥ 80th percentile	12–18	21	NA	43
Thompson et al. (2007)	122 (0/122)	BMI ≥ 95th percentile	9	12–14	71	NA

*RWt > 1 SDS.
†BMI ≥ 29 kg/m².
‡Self-reported BMI ≥ 28.5 kg/m².

decrease of BMI between adolescence and adulthood is more common among women of higher social class (Viner and Cole 2006).

The morbidity and mortality for cardiovascular disease is higher in men and women who were overweight during childhood and adolescence (Must et al. 1992; Srinivasan et al. 1996; Must and Strauss 1999; Baker et al. 2007). In The Harvard Growth Study, the men who had a body weight >75th percentile for age as children had two times the risk of cardiovascular death of those with body weight within the 25th and 50th percentile (Must et al. 1992). In the Bogalusa Heart Study, the prevalence of hypertension and dyslipidemia was much higher in overweight than in normal-weight adolescents and this was especially evident for men (Srinivasan et al. 1996). In a recent study (Baker et al. 2007), a high BMI during childhood was associated with a higher risk of coronary heart disease in adulthood. In the present study, however, we did not detect any association between the occurrence of cardio-metabolic disease and the degree of childhood obesity, even if the 30-year incidence of cardio-metabolic disease was not negligible. The absence of association between childhood BMI and cardiovascular complications in adulthood has nonetheless been reported by other studies (Gunnell et al. 1998; Juonala et al. 2006; Lawlor et al. 2006).

Our study has several limitations. First, 30% of our subjects were not available at the 30-year follow-up. However, this drop-out rate is within acceptable limits, less relevant than in most studies, and baseline anthropometry and pubertal status were similar in subjects available and not available at follow-up. Second, our subjects were young fertile women, a condition involving cardiovascular protection from estrogens, and this may have influenced the incidence of cardio-metabolic disease. Third, the study design did not allow us to take into account other known cardiovascular risk factors. Fourth, our data may be subject to a secular trend effect because the prevalence and incidence of childhood obesity have changed substantially during the last decades. Fifth, all subjects were submitted to dietary counseling and followed up by their general practitioners, so that the persistence of obesity may have been influenced by this treatment.

In conclusion, our study confirms that female childhood obesity tends to persist into adulthood (Stark et al. 1981; Rolland-Cachera et al. 1987; DiPietro et al. 1994; Power et al. 1997; Must and Strauss 1999; Togashi et al. 2002; Freedman et al. 2005b; Deshmukh-Taskar et al. 2006; Thompson et al. 2007). The persistence rate of obesity was lower than previously reported (Guo and Chumlea 1999; Ferraro et al. 2003; Deshmukh-Taskar et al. 2006) and was independently predicted by BMI, pubertal status and educational degree. Our data reinforce therefore the notion that obesity should be prevented at an early age and suggests that adolescents with severe obesity and low educational level are at greater risk for becoming obese adults.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

Abraham S, Collins G, Nordsieck M. 1971. Relationship of childhood weight status to morbidity in adults. HSMHA Health Rep 86:273–284.

Baker JL, Olsen LW, Sørensen TI. 2007. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med 357:2329–2337.

Barlow SE, Dietz WH. 1998. Obesity evaluation and treatment: Expert Committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. Pediatrics 102:E29.

Charney E, Goodman HC, McBride M, Lyon B, Pratt R. 1976. Childhood antecedents of adult obesity. Do chubby infants become obese adults? N Engl J Med 295:6–9.

Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jones DW, Materson BJ, Oparil S, Wright JT, et al. 2003. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 report. JAMA 289:2560–2572.

Deshmukh-Taskar P, Nicklas TA, Morales M, Yang SJ, Zakeri I, Berenson GS. 2006. Tracking of overweight status from childhood to young adulthood: The Bogalusa Heart Study. Eur J Clin Nutr 60:48–57.

DiPietro L, Mossberg HO, Stunkard AJ. 1994. A 40-year history of overweight children in Stockholm: Life-time overweight, morbidity, and mortality. Int J Obes Relat Metab Disord 18:585–590.

Expert Panel on Detection, Evaluation, & Treatment of High Blood Cholesterol in Adults. 2001. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 285:2486–2497.

Ferraro KF, Thorpe RJ, Wilkinson JA. 2003. The life course of severe obesity: Does childhood overweight matter? J Gerontol B Psychol Sci Soc Sci 58:S110–S119.

Field AE, Cook NR, Gillman MW. 2005. Weight status in childhood as a predictor of becoming overweight or hypertensive in early adulthood. Obes Res 13:163–169.

Flegal KM, Graubard BI, Williamson DF, Gail MH. 2007. Cause-specific excess deaths associated with underweight, overweight, and obesity. JAMA 298:2028–2037.

Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. 1999. The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa Heart Study. Pediatrics 103:1175–1182.

Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. 2005a. Racial differences in the tracking of childhood BMI to adulthood. Obes Res 13:928–935.

Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. 2005b. The relation of childhood BMI to adult adiposity: The Bogalusa Heart Study. Pediatrics 115:22–27.

Garn SM, LaVelle M. 1985. Two-decade follow-up of fatness in early childhood. Am J Dis Child 139:181–185.

Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey Smith G. 1998. Childhood obesity and adult cardiovascular mortality: A 57-y follow-up study based on the Boyd Orr cohort. Am J Clin Nutr 67:1111–1118.

Guo SS, Chumlea WC. 1999. Tracking of body mass index in children in relation to overweight in adulthood. Am J Clin Nutr 70:145S–148S.

Hirji KF, Mehta CR, Patel NR. 1987. Computing distributions for exact logistic regression. J Am Stat Assoc 82:1110–1117.

ISTAT. 2005. Stili di vita e condizioni di salute. Indagine multiscopo sulle famiglie. Aspetti della vita quotidiana. Anno 2003. Rome: Istituto Nazionale di Statistica.

Juonala M, Raitakari M, SA Viikari J, Raitakari OT. 2006. Obesity in youth is not an independent predictor of carotid IMT in adulthood. The Cardiovascular Risk in Young Finns Study. Atherosclerosis 185:388–393.

Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, Mei Z, Curtin LR, Roche AF, Johnson CL. 2000. CDC growth charts: United States. Adv Data 8:1–27.

Lawlor DA, Lean M, Sattar N. 2006. ABC of obesity: Obesity and vascular disease. BMJ 333:1060–1063.

Lohman TG, Roche AF, Martorell R. 1988. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics Books.

Lumley T, Kronmal R, Ma S. 2006. Relative risk regression in medical research: Models, contrasts, estimators and algorithms. UW Biostatistical Working Paper Series: http://www.bepress/uwbiostat/paper293

Mossberg HO. 1989. 40-year follow-up of overweight children. Lancet 2:491–493.

Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. 1992. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 327:1350–1355.

Must A, Strauss RS. 1999. Risks and consequences of childhood and adolescent obesity. Int J Obes Relat Metab Disord 23:(Suppl 2)S2–S11.

NIH. 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults – The Evidence Report. National Institutes of Health. Obes Res 6:(Suppl 2)51S–209S.

Ogden CL, Flegal KM, Carroll MD, Johnson CL. 2002. Prevalence and trends in overweight among US children and adolescents, 1999–2000. JAMA 288:1728–1732.

Parsons TJ, Power C, Logan S, Summerbell CD. 1999. Childhood predictors of adult obesity: A systematic review. Int J Obes Relat Metab Disord 23:(Suppl 8)S1–S107.

Power C, Lake JK, Cole TJ. 1997. Body mass index and height from childhood to adulthood in the 1958 British born cohort. Am J Clin Nutr 66:1094–1101.

Rolland-Cachera MF, Deheeger M, Guilloud-Bataille M, Avons P, Patois E, Sempé M. 1987. Tracking the development of adiposity from one month of age to adulthood. Ann Hum Biol 14:219–229.

Sandhu J, Ben-Shlomo Y, Cole TJ, Holly J, Davey Smith G. 2006. The impact of childhood body mass index on timing of puberty, adult stature and obesity: A follow-up study based on adolescent anthropometry recorded at Christ's Hospital (1936–1964). Int J Obes (Lond) 30:14–22.

Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. 1993. Do obese children become obese adults? A review of the literature. Prev Med 22:167–177.

SIEDP. 1992. L'obesità essenziale in età pediatrica: Attuali orientamenti per la terapia. Rivista Italiana di Pediatria 17:487–494.

Slyper AH. 2004. The pediatric obesity epidemic: Causes and controversies. J Clin Endocrinol Metab 89:2540–2547.

Srinivasan SR, Bao W, Wattigney WA, Berenson GS. 1996. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: The Bogalusa Heart Study. Metabolism 45:235–240.

Stark O, Atkins E, Wolff OH, Douglas JW. 1981. Longitudinal study of obesity in the National Survey of Health and Development. Br Med J (Clin Res Ed) 283:13–17.

Sørensen TI, Sonne-Holm S. 1988. Risk in childhood of development of severe adult obesity: Retrospective, population-based case-cohort study. Am J Epidemiol 127:104–113.

Tanner JM. 1990. Foetus into man: Physical growth from conception to maturity. Cambridge, MA: Harvard University Press.

Thompson DR, Obarzanek E, Franko DL, Barton BA, Morrison J, Biro FM, Daniels SR, Striegel-Moore RH. 2007. Childhood overweight and cardiovascular disease risk factors: The National Heart, Lung, and Blood Institute Growth and Health Study. J Pediatr 150:18–25.

Togashi K, Masuda H, Rankinen T, Tanaka S, Bouchard C, Kamiya H. 2002. A 12-year follow-up study of treated obese children in Japan. Int J Obes Relat Metab Disord 26:770–777.

Vanhala M, Vanhala P, Kumpusalo E, Halonen P, Takala J. 1998. Relation between obesity from childhood to adulthood and the metabolic syndrome: Population based study. BMJ 317:319–320.

Viner RM, Cole TJ. 2006. Who changes body mass between adolescence and adulthood? Factors predicting change in BMI between 16 year and 30 years in the 1970 British Birth Cohort. Int J Obes (Lond) 30:1368–1374.

Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, Allen K, Lopes M, Savoande M, Morrison J, et al. 2004. Obesity and the metabolic syndrome in children and adolescents. N Engl J Med 350:2362–2374.