Childhood Size and Life Course Weight Characteristics in Association With the Risk of Incident Type 2 Diabetes

EDWINA H. YEUNG, PHD[1]
CUILIN ZHANG, MD, PHD[1]
GERMAINE M. BUCK LOUIS, PHD[1]
WALTER C. WILLETT, MD, DRPH[2,3]
FRANK B. HU, MD, PHD[2,3]

OBJECTIVE — To determine how childhood overweight, in conjunction with other life course weight characteristics, relates to the development of type 2 diabetes in adulthood.

RESEARCH DESIGN AND METHODS — Among 109,172 women in the Nurses' Health Study II, body fatness at ages 5, 10, and 20 years was assessed by recall using 9-level pictorial diagrams (somatotypes) representing extreme thinness (category 1) to obesity (category 9). Recalled weights at age 18 years and adulthood were used to derive BMI. Self-reported cases of type 2 diabetes were confirmed by supplementary questionnaire.

RESULTS — Somatotypes at ages 5 and 10 years were positively associated with diabetes risk ($P_{trend} < 0.0001$). The adjusted relative risk (RR) of women with somatotype ≥6 (vs. 2) at age 5 years was 2.19 (95% CI 1.79–2.67) and at age 10 years was 2.57 (2.20–3.01). Increases in size by somatotype or by weight gain since age 18 were associated with increased risk. Compared with women who were never overweight at any age, women who were overweight as an adult (BMI >25 kg/m^2) but not previously had an adjusted RR of 8.23 (7.41–9.15). The adjusted RR was 15.10 (13.21–17.26) for women who were also overweight at age 10 (somatotype ≥5) and 18 (BMI >25 kg/m^2). Increased childhood size was not associated with risk among women who did not continue to be overweight in adulthood.

CONCLUSIONS — Increased body size starting from childhood is associated with a greater risk of diabetes in adulthood. However, women who become lean in adulthood do not have an increased risk.

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Large proportions of children in the U.S. are currently at risk for or are overweight. Immediate and long-term health problems have arisen because of childhood overweight, including poor lipid profile, earlier onset of type 2 diabetes, and other metabolic syndrome traits (1). Although the rise in prevalence of type 2 diabetes in the pediatric population is cause for concern in itself, the risk as these children continue into adulthood will undoubtedly be a greater public health burden.

Despite strong ties between the development of insulin resistance from increased adiposity via multiple biological mechanisms, few studies have looked at the long-term consequences of childhood overweight and the risk of type 2 diabetes in adulthood and findings have been inconsistent (2–8). One study using birth and medical records data from Finland, found that BMI at ages 7–11 years in women was significantly and positively associated with future risk of diabetes (4). However, the study did not investigate the roles of adolescent and adulthood obesity in this association, and the number of type 2 diabetes cases was small (n = 185) (4). In contrast, a more recent study that accounted for life course weight, found that thinness, rather than overweight, from childhood through young adulthood was associated with increased diabetes risk (8). However, these findings were from an older cohort (born 1925–1950) of French women, with a large percentage being extremely lean in childhood, whose early nutritional status might have been affected by World War II (1939–1945). Thus, the objective of this study was to determine the longitudinal association between childhood overweight in combination with other life course weight characteristics and the risk for type 2 diabetes in a more recent birth cohort of young women.

RESEARCH DESIGN AND METHODS

— The Nurses' Health Study II (NHSII) is an ongoing prospective study of U.S. female nurses aged 25–42 years. Follow-up is conducted using biennial questionnaires from 1989 to 2005. A total of 109,172 participants remained after exclusion for diagnosis of any type of diabetes, cancer, or cardiovascular disease at baseline (2%) or for missing information on childhood body shape (2.5%), BMI at age 18 years (0.7%), or baseline BMI (0.3%). This study was approved by the institutional review board of the Partners Health Care System (Boston, MA).

Assessment of childhood size and weight characteristics

At baseline, body fatness at ages 5, 10, and 20 years of age was assessed by recall of somatotypes or 9-level pictorial diagrams developed by Stunkard et al. (9) representing sizes ranging from extreme thinness (category 1) to obesity (category 9). The use of recalled somatotypes has been validated in both older (10) and younger (11) women by comparison with childhood records. Somatotypes at ages 5, 10, and 20 years correlated moderately with recorded BMI (r = 0.60, 0.65, and 0.66, respectively) (10). The validity did not differ by adult BMI at the time of report

From the [1]Epidemiology Branch, Division of Epidemiology, Statistics, and Prevention Research, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, Bethesda, Maryland; the [2]Departments of Nutrition and Epidemiology, Harvard School of Public Health, Boston, Massachusetts; and the [3]Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts.
Corresponding author: Edwina Yeung, yeungedw@mail.nih.gov.
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(10). Overweight was defined as somatotype ≥5. (12).

Weight at age 18 and adult height and weight were self-reported. Weight change was the difference between weight at age 18 years and weight at baseline in 1989. The correlation between recalled weight at age 18 and documented weight from college or nursing school records was 0.84, and the correlation between self-reported and technician-measured adult weight was 0.96 (13).

Type 2 diabetes ascertainment

A supplementary questionnaire was mailed to confirm the self-report of diabetes diagnosis and to distinguish different types of diabetes (14). The National Diabetes Data Group diagnostic criteria were used for cases reported through 1997 and required confirmation of at least one of the following: *1*) one or more symptoms (weight loss, hunger, thirst, or polyuria) and elevated glucose (fasting ≥7.8 mmol/l [140 mg/dl] or random plasma or 2-h glucose ≥11.1 mmol/l [200 mg/dl]), *2*) no symptoms but occurrence of elevated plasma glucose as described above on at least two different occasions, or *3*) use of insulin or oral hypoglycemic medication (15). For cases occurring after 1998, the cutoff of fasting plasma glucose was changed to 7.0 mmol/l (126 mg/dl) in accordance with revised criteria (16). We excluded women classified as having only gestational diabetes mellitus or type 1 diabetes. In the Nurses' Health Study, the supplemental questionnaire was highly reliable regarding diabetes diagnosis (14). In a random sample of 84 women classified as being a case subject, medical records were available for 62 of these women and an endocrinologist confirmed the diagnosis in 61 women (98%) (14).

Assessment of covariates

Age was calculated as months from the reported birth date to date of questionnaire return. Race, smoking status, birth weight, prematurity, multiple gestation birth, age of menarche, being breast-fed, alcohol consumption, and family history of diabetes were self-reported at baseline or in 1991. Parity and age at first birth were measured biennially. Physical activity, in MET units, was derived from the average time spent in certain activities (e.g., jogging or running) in 1989, 1991, 1997, and 2001.

Statistical analysis

Differences in participant characteristics by childhood somatotype at age 10 years were compared using χ^2 or linear regression. Person-years were calculated based on date of return to date of diagnosis, death, or 1 July 2005, whichever came first. Multivariate Cox proportional hazard models were used to estimate the relative risk (RR) for the associations of somatotypes at ages 5, 10, and 20 years and BMI at age 18 years with type 2 diabetes risk. Somatotypes were categorized by 1 to ≥6. We used category 2 as the reference because most women had reported this category at both ages 5 and 10 years. We collapsed the uppermost categories because few women reported those categories.

In one multivariate model, estimates were adjusted for age (continuous), race (African American, Hispanic, Asian, or white), parity (0, 1–2, or ≥3) in combination with age at first birth (>24 years), family history of diabetes (maternal, paternal, or both), smoking (current, past, or never), and physical activity (quintiles). In a second model among participants not missing birth weight information (n = 87,349, case subjects = 2,771), estimates were adjusted for characteristics influencing childhood size including age, race, family history of diabetes, birth weight (<5.5, 5.5–6.9, 7.0–8.4, 8.5–9.9, or ≥10 pounds), prematurity or multiple gestation birth status of the woman (yes or no), and age of menarche (<12, 12, 13, 14, or >14 years). To test for significant trends, linear models were fitted using the median values of each category of exposure (e.g., 17.0, 19.0, 21.0, 23.5, 28.0, and 35.0 for BMI at age 18 years).

The differences in somatotype categories (between ages 5 and 10 and between ages 10 and 20) or in weight (between age 18 years and baseline) were used to assess whether change in size was associated with diabetes development. Models were additionally adjusted for starting size (e.g., adjustment for age 5 somatotype in evaluating the difference between ages 5 and 10), because where a woman began in size may reflect weight gain or loss (i.e., heavier women have the possibility to lose more weight).

The cumulative effect of overweight across the life course before reported diabetes was evaluated using the combination of somatotype ≥5 at age 10, BMI ≥25 kg/m^2 at age 18, and BMI ≥25 kg/m^2 at baseline (i.e., to represent adulthood). The reference group comprised women who reported no overweight at any of those time points. Analyses were performed using SAS (version 8.2; SAS Institute, Cary, NC).

RESULTS — There were 3,307 incident cases of type 2 diabetes over 16 years of follow-up, for an incidence rate of 197 cases per 100,000 person-years. Table 1 shows the characteristics for all women and by reported childhood somatotype at age 10 years categorized from 1 to ≥6. Women with somatotype 1 at age 10 years were more likely to be of minority race, to have birth weight <5.5 pounds, to have been born prematurely, and to have had a later age at menarche; whereas women with somatotype ≥6 were more likely to have birth weight >10 pounds, to actively smoke, to be nulliparous, and to have had a positive parental history of diabetes. Similar associations were found at age 5 years. Somatotypes at ages 5, 10, and 20 years were associated with each other. Of the women reporting somatotype ≥6 at age 10 years, 41% reported somatotype ≥6 at age 5 years, and 28% at age 20 years. Weight and BMI were also positively associated with somatotype; a 1–2 kg/m^2 increase in adolescent or adult BMI was observed per increase in somatotype category.

Somatotypes at ages 5, 10, and 20 years and BMI at age 18 years (Table 2) were all significantly and positively associated with diabetes risk (all P_{trend} < 0.0001). Compared with having a somatotype of 2, the RR of diabetes associated with a somatotype ≥6 at age 5 was 2.19 (95% CI 1.79–2.67), at age 10 years was 2.57 (2.20–3.01), and at age 20 years was 5.67 (4.92–6.54). The RR among women who reported a BMI at age 18 >30 kg/m^2 was 8.72 (7.58–10.02) compared with women with a BMI of 18–19 kg/m^2. Among a subgroup not missing birth weight information (n = 87,175), adjustment for childhood factors including birth weight did not substantially change these estimates.

Type 2 diabetes risk increased among women who reported increases in size whether by somatotype between ages 5 and 10 and between ages 10 and 20 or by weight gain since age 18 (Table 3). Compared with women reporting no change in somatotype, one or more unit increases in somatotype at these ages were associated with approximately twice the risk, whereas decreases in somatotype at these ages were associated with reduced risk.

Table 1—*Baseline characteristics of NHSII participants by somatotype at age 10 years*

		Childhood somatotype at age 10 years					
	Total	1	2	3	4	5	≥6
Women (%)	100	18.9	30.7	22.5	15.8	9.0	3.1
Age (years)	34.3 ± 5	34.7 ± 5	34.0 ± 5	34.1 ± 5	34.4 ± 5	34.7 ± 5	34.8 ± 5
White (%)	95.2	92.0	95.0	96.4	96.7	96.7	95.6
Birth weight <5.5 pounds (%)*	7.9	10.2	7.8	7.1	6.9	7.4	7.2
Birth weight >10 pounds (%)*	1.3	0.9	0.9	1.3	1.5	1.9	3.1
Premature (%)*	6.5	7.1	6.4	6.2	6.3	6.5	6.4
Multiple gestation (%)*	1.4	1.5	1.5	1.3	1.2	0.9	0.7
Breast-fed (%)*	34.0	33.0	34.2	35.5	33.4	33.1	31.9
Menarche at age 12 years (%)*	30.2	27.1	29.8	32.5	31.4	30.7	29.6
Menarche at age ≥15 years (%)*	7.6	12.1	8.7	5.7	4.7	4.6	4.9
Nulliparous (%)	30.4	29.6	29.3	29.3	31.5	34.4	37.9
Age at first birth >24 years (%)	41.0	40.5	42.1	42.3	40.6	37.3	34.7
Parental history of diabetes (%)	15.2	15.0	13.6	14.4	16.6	18.7	22.1
Active smoker (%)	13.4	13.4	11.6	12.4	14.4	17.8	20.8
Nondrinker (%)	35.0	33.4	34.5	36.0	35.9	35.3	36.2
Physical activity (METs/week)	24.9 ± 37	26.3 ± 40	25.0 ± 37	24.3 ± 36	24.1 ± 34	24.4 ± 35	26.0 ± 41
Somatotype ≥6 at 5 years (%)	1.6	0.1	0.1	0.1	0.4	2.3	41.0
Somatotype ≥6 at 20 years (%)	3.3	0.3	0.5	1.5	4.4	13.9	27.7
BMI at age 18 (kg/m^2)	21.3 ± 3	19.3 ± 2	20.2 ± 2	21.6 ± 3	22.8 ± 3	23.9 ± 4	25.6 ± 6
Adult weight (kg)	65 ± 14	59.9 ± 10	61.8 ± 11	66.3 ± 14	70 ± 17	73.2 ± 18	76.5 ± 19
Adult height (m)	1.65 ± 0.07	1.65 ± 0.07	1.65 ± 0.07	1.65 ± 0.07	1.65 ± 0.07	1.65 ± 0.07	1.66 ± 0.07
Adult BMI (kg/m^2)	24.1 ± 5	22.0 ± 3	22.8 ± 4	24 ± 5	26.1 ± 6	26.9 ± 6	27.9 ± 7
Weight change since age 18 (kg)	7.61 ± 11	7.31 ± 8	6.94 ± 9	7.92 ± 11	8.74 ± 13	8.26 ± 14	6.30 ± 16

Means ± SD presented unless indicated. n = 109,172. *Reported in 1991 questionnaire (rather than at baseline in 1989 for all other covariates).

These associations were strengthened after adjustment for earlier somatotype. Weight gain since age 18, which is the difference between baseline weight and weight at age 18, was also significantly associated with risk of diabetes. Compared with women who had little change in weight (i.e., ±4.9 pounds), even a weight gain of 5–8 pounds doubled risks, whereas weight gain of >25 pounds increased risk by >20 times. Weight loss was associated with increased risk until after adjustment for BMI at age 18 to account for greater weight loss being associated with larger adolescent body size. In analyses stratified by BMI at age 18 years, weight loss of ≥10 pounds was significantly associated with a reduced risk of diabetes among women who were overweight (RR 0.45 [95% CI 0.22–0.91]) or obese (0.45 [0.28–0.72]) in adolescence but not among those who were lean (1.72 [0.76–3.90]).

The prevalences of overweight by somatotype ≥5 at ages 5, 10, and 20 years were 7, 12, and 11%, respectively. Most of the cases (83%) in the cohort occurred among women who were overweight (BMI ≥25 kg/m^2) at baseline with an incidence rate of 562 per 100,000 person-years. Among these women, the prevalences of overweight by somatotype ≥5 were 12, 21, and 23%, respectively.

To evaluate the cumulative effect of being overweight across the life course, women were jointly categorized as being overweight using somatotype at age 10, BMI at age 18 years, and BMI at baseline (with mean age of 34 years) (Fig. 1). Women who were overweight only as an adult (BMI >25 kg/m^2) had an adjusted RR of 8.23 (95% CI 7.41–9.15) compared with women who were never overweight at any age. The adjusted RR increased to 15.10 (13.21–17.26) for women who were also overweight at age 10 (somatotype ≥5) and age 18 (BMI >25 kg/m^2). However, women who were overweight at age 10 but came off the trajectory and became lean in adulthood (i.e., not being overweight in adulthood) did not have a significantly increased risk of diabetes (1.02, 0.74–1.40).

CONCLUSIONS — Among 109,172 women followed for >16 years, somatotypes at ages 5 and 10 years were positively associated with the risk of incident type 2 diabetes after adjustment for major risk factors. However, women who were overweight at age 10 but were lean in adulthood did not have an increased risk of diabetes associated with childhood overweight, underscoring the importance of continued efforts to control adiposity among overweight children.

Limited studies have investigated the long-term consequences of childhood overweight and the risk of type 2 diabetes in adulthood, with inferences hindered by small numbers of case subjects and/or lack of differentiation of diabetes type (2–8). In addition, some studies included case subjects with young age at onset (<30 years old) (3,5,7). One study using data based on records (185 cases) showed that the cumulative incidence of diabetes was doubled (from 4.2 to 8.4%) among women in the largest BMI group at age 11 years (>17.4 kg/m^2) compared with those in the smallest BMI group (≤15.3 kg/m^2) (4). We found a similar increase in risk using recalled somatotypes. In contrast, a study of older French women using recalled body shape, found an association between childhood thinness and diabetes risk (8). However, this birth cohort vastly differed from the NHSII as demonstrated by >60% of their case subjects having reported extreme leanness (somatotype 1) at age 8 years, possibly due to their nutritional status having been affected by World War II (8). Thus, our

Table 2—*Childhood and adolescent size and RR (95% CI) of type 2 diabetes in adulthood in NHSII*

	PY	Cases of diabetes	Age-adjusted	Model 2*	Model 3†
Age 5 somatotype					
1	417,996	756	1.13 (1.02–1.25)	1.05 (0.95–1.16)	1.01 (0.90–1.13)
2	533,990	807	1.00	1.00	1.00
3	396,742	774	1.28 (1.15–1.41)	1.25 (1.14–1.38)	1.20 (1.08–1.34)
4	212,080	568	1.72 (1.54–1.91)	1.65 (1.48–1.84)	1.63 (1.45–1.84)
5	88,885	289	2.04 (1.78–2.33)	1.85 (1.62–2.12)	1.80 (1.55–2.09)
≥6	26,619	113	2.61 (2.14–3.18)	2.19 (1.79–2.67)	2.00 (1.59–2.51)
P_{trend}			<0.0001	<0.0001	<0.0001
Age 10 somatotype					
1	317,663	441	1.04 (0.92–1.17)	0.98 (0.87–1.11)	0.96 (0.83–1.10)
2	516,959	649	1.00	1.00	1.00
3	377,995	716	1.50 (1.35–1.67)	1.49 (1.34–1.66)	1.49 (1.32–1.67)
4	262,795	765	2.27 (2.05–2.52)	2.17 (1.95–2.41)	2.15 (1.92–2.42)
5	149,808	525	2.68 (2.39–3.01)	2.45 (2.18–2.75)	2.52 (2.21–2.86)
≥6	51,091	211	3.09 (2.64–3.61)	2.57 (2.20–3.01)	2.41 (2.02–2.88)
P_{trend}			<0.0001	<0.0001	<0.0001
Age 20 somatotype					
1	75,871	83	0.97 (0.77–1.23)	0.91 (0.72–1.15)	0.96 (0.73–1.25)
2	443,762	443	1.00	1.00	1.00
3	633,117	928	1.54 (1.37–1.72)	1.52 (1.36–1.70)	1.55 (1.36–1.76)
4	346,718	924	2.83 (2.53–3.17)	2.71 (2.42–3.04)	2.78 (2.44–3.16)
5	122,949	564	4.85 (4.28–5.49)	4.42 (3.90–5.01)	4.79 (4.16–5.51)
≥6	53,897	365	7.24 (6.30–8.32)	5.67 (4.92–6.54)	6.17 (5.26–7.23)
P_{trend}			<0.0001	<0.0001	<0.0001
BMI at age 18					
<18 kg/m^2	158,206	164	1.13 (0.94–1.35)	1.06 (0.88–1.26)	1.06 (0.86–1.30)
18–<19 kg/m^2	507,641	458	1.00	1.00	1.00
20–<22 kg/m^2	530,083	727	1.52 (1.35–1.71)	1.49 (1.32–1.67)	1.51 (1.32–1.72)
22–<25 kg/m^2	312,876	848	3.02 (2.69–3.38)	2.79 (2.49–3.13)	2.86 (2.51–3.25)
25–<30 kg/m^2	127,826	721	6.32 (5.62–7.11)	5.32 (4.72–5.99)	6.10 (5.35–6.97)
≥30 kg/m^2	39,682	389	11.55 (10.08–13.23)	8.72 (7.58–10.02)	9.26 (7.92–10.82)
P_{trend}			<0.0001	<0.0001	<0.0001

Data are RR (95% CI) unless otherwise indicated. n = 109,172. *Model 2 adjusted for age, race, smoking status, parental history of diabetes, parity, age at first birth, and adult physical activity. †Model 3 adjusted for age, race, parental history of diabetes, birth weight, multiple birth, prematurity, age of menarche (n = 87,175, case subjects = 2,681 in subgroup not missing birth weight information). PY, person-years.

findings among a more recent birth cohort may be more relevant.

In addition to absolute size, longitudinal changes through different ages are also relevant to the risk of diabetes (17,18). Under normal development, infants lose weight after 6 months of age and continue doing so until ~5 years of age when their adiposity rebounds (18). Although we were unable to assess age at adiposity rebound, earlier rebound has been associated with increased risk of type 2 diabetes (17), and there is some suggestion that age at rebound matters more than the absolute size of the child at any point in time because it corresponds to weight gain. Our findings of increased risk associated with increases in somatotype and weight gain support the importance of change in size in addition to absolute size. These findings offer support for continued weight reduction efforts across the life span.

Moreover, in analyses of the cumulative effect of overweight through childhood, adolescence, and adulthood, the risk for type 2 diabetes was greatest among women reporting overweight by all three measures, even though the difference was marginal when compared with adolescent overweight. However, women who became lean in adulthood did not have an increased risk of diabetes associated with childhood overweight. Similar observations have been made from other studies of youth overweight and type 2 diabetes or related traits, with findings becoming nonsignificant after accounting for adult BMI (6,19,20). Tracking of metabolic risk factors (e.g., HDL and triglycerides) has also been observed after 21 years of follow-up from childhood (21), supporting the fact that cumulative overweight is linked with prolonged exposure to metabolic irregularities, putting children on the path to β-cell dysfunction earlier. It should be noted that although there is tracking of overweight to obesity from childhood to adulthood, the trajectory is not fixed from youth (18,22). It has been shown that ~10–30% of overweight children do not go on to be overweight as adults with conflicting evidence as to whether there is greater tracking in girls than in boys (22). Conversely, the majority of adults who are overweight were not in childhood (22) as confirmed here with only 12–20% of the overweight women reporting childhood overweight.

Our study had some limitations. Although experts have recommended BMI for the measurement of childhood size

Table 3—*Changes in size and RR (95% CI) of type 2 diabetes in NHSII*

	PY	Cases of diabetes	Age-adjusted	Model 2*	Model 3†
Age 5–10 years					
Decrease	19,572	29	0.93 (0.65–1.35)	0.95 (0.65–1.37)	0.63 (0.43–0.91)
No change	1,235,621	1,943	1.00	1.00	1.00
+1	310,086	950	2.00 (1.85–2.16)	1.86 (1.72–2.01)	1.89 (1.75–2.05)
≥2	111,033	385	2.16 (1.93–2.41)	1.95 (1.75–2.18)	2.19 (1.95–2.45)
Age 10–20 years					
Decrease	109,956	196	1.04 (0.89–1.21)	1.00 (0.86–1.17)	0.53 (0.45–0.62)
No change	758,071	1,294	1.00	1.00	1.00
+1	621,736	1,274	1.27 (1.18–1.37)	1.22 (1.12–1.31)	1.86 (1.71–2.02)
≥2	186,550	543	1.89 (1.70–2.08)	1.72 (1.55–1.90)	2.87 (2.58–3.20)
Weight change since age 18					
Loss ≥10 pounds	31,322	61	5.44 (4.11–7.20)	4.48 (3.38–5.94)	1.26 (0.93–1.71)
Loss 5–<10 pounds	58,170	41	2.02 (1.45–2.82)	1.84 (1.32–2.56)	1.17 (0.84–1.63)
No change (±5 pounds)	714,230	249	1.00 (referent)	1.00 (referent)	1.00 (referent)
Gain 5–<8 pounds	226,283	169	2.05 (1.69–2.49)	1.99 (1.64–2.42)	2.10 (1.73–2.55)
Gain 8–<11 pounds	182,828	219	3.24 (2.70–3.88)	3.05 (2.54–3.66)	3.10 (2.58–3.72)
Gain 11–<15 pounds	177,159	437	6.54 (5.60–7.65)	5.99 (5.12–7.00)	5.78 (4.94–6.77)
Gain 15–<20 pounds	114,008	477	10.92 (9.36–12.74)	9.67 (8.28–11.30)	8.74 (7.48–10.20)
Gain 20–<25 pounds	76,539	464	15.72 (13.46–18.36)	13.42 (11.48–15.70)	11.11 (9.49–13.00)
Gain ≥25 pounds	95,774	1,190	32.28 (28.11–37.06)	27.13 (23.57–31.23)	20.41 (17.70–23.52)

Data are RR (95% CI) unless otherwise indicated. n = 109,172. *Model 2 adjusted for age, race, smoking status, parental history of diabetes, parity, age at first birth, and adult physical activity. †Model 2 variables with additional adjustment for earlier size, i.e., somatotype at age 5 and 10 years and BMI at age 18 years. PY, person-years.

(18,23), we used recalled somatotype. Misclassification of childhood size is inevitable. However, two previous studies have shown that the accuracy of recall between childhood somatotypes and recorded childhood BMI did not differ by adult BMI (10,11). The misclassification of childhood size is thus more likely to be nondifferential, which cannot explain the

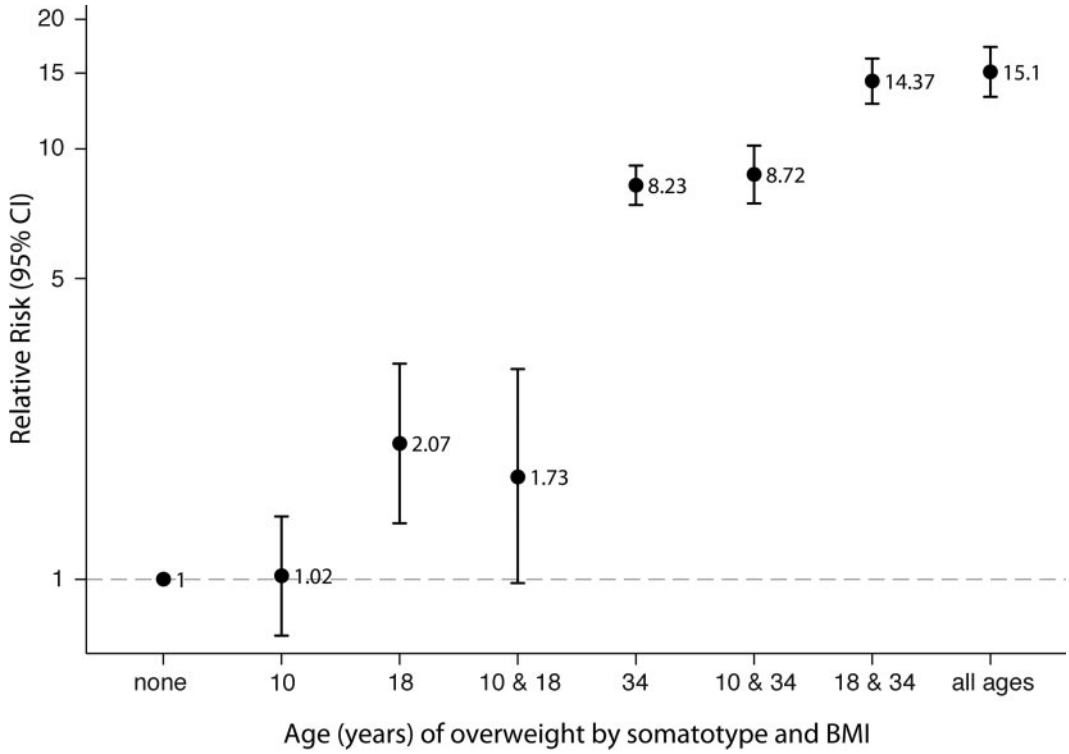

Figure 1—*Adjusted RRs (95% CI) of type 2 diabetes by somatotype ≥5 at age 10, BMI ≥25 kg/m^2 at age 18 years, and BMI ≥25 kg/m^2 at baseline (average age of 34 years), after adjustment for age, race, smoking status, parental history of diabetes, parity, age at first birth, and physical activity. Number of cases of type 2 diabetes for each category: 503 for none; 50 for 10 years; 21 for 18 years; 10 for 10 and 18 years; 1,469 for 34 years; 260 for 10 and 34 years; 664 for 18 and 34 years; and 441 for all ages.*

observed positive association between childhood size and type 2 diabetes risk. In addition, our findings were in agreement with those of other studies that used childhood measures of weight and height (4,20). The generalizability of our findings may be limited to white women who comprised >95% of our study population. More research is needed in minority populations who have higher rates of overweight and diabetes (1,24). Last, we cannot rule out the possibility of residual confounding by unmeasured confounders because of the observational nature of the study.

As for strengths, the NHSII included a large number of women (>3,000 case subjects) and adjusted for many risk factors. Follow-up for previous studies required recontacting participants in adulthood with low to moderate success rates (20–60%) (5,7,19,20), whereas we evaluated somatotypes at baseline and >90% of the women remained for follow-up.

In summary, our findings demonstrate that the importance of childhood overweight stems largely from adult overweight. Women who do not continue to be overweight in adulthood do not have increased risks. It remains important then to promote lifestyle changes from youth so that the adverse trajectory could be avoided. Multiple interventions to address childhood overweight have been suggested (23), but these remain to be fully tested.

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References

1. Lee WW. An overview of pediatric obesity. Pediatr Diabetes 2007;8(Suppl. 9): 76–87
2. Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. HSMHA Health Rep 1971;86:273–284
3. Al Mamun A, Cramb SM, O'Callaghan MJ, Williams GM, Najman JM. Childhood overweight status predicts diabetes at age 21 years: a follow-up study. Obesity (Silver Spring) 2009;17:1255–1261
4. Forsén T, Eriksson J, Tuomilehto J, Reunanen A, Osmond C, Barker D. The fetal and childhood growth of persons who develop type 2 diabetes. Ann Intern Med 2000;133:176–182
5. Franks PW, Hanson RL, Knowler WC, Moffett C, Enos G, Infante AM, Krakoff J, Looker HC. Childhood predictors of young-onset type 2 diabetes. Diabetes 2007;56:2964–2972
6. Lawlor DA, Davey Smith G, Clark H, Leon DA. The associations of birthweight, gestational age and childhood BMI with type 2 diabetes: findings from the Aberdeen Children of the 1950s cohort. Diabetologia 2006;49:2614–2617
7. Bhargava SK, Sachdev HS, Fall CH, Osmond C, Lakshmy R, Barker DJ, Biswas SK, Ramji S, Prabhakaran D, Reddy KS. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. N Engl J Med 2004;350:865–875
8. de Lauzon-Guillain B, Balkau B, Charles MA, Romieu I, Boutron-Ruault MC, Clavel-Chapelon F. Birth weight, body silhouette over the life course and incident diabetes in 91,453 middle-aged women from the French Etude Epidemiologique de Femmes de la Mutuelle Generale de l'Education Nationale (E3N) Cohort. Diabetes Care 2010;33:298–303
9. Stunkard AJ, Sorensen T, Schulsinger F. Use of the Danish adoption register for the study of obesity and thinness. In *The Genetics of Neurological and Psychiatric Disorders.* Kety SS, Rowland LP, Sidman SW, Mathysee SW, Eds. New York, Ravens Press, 1983, p. 115–120
10. Must A, Willett WC, Dietz WH. Remote recall of childhood height, weight, and body build by elderly subjects. Am J Epidemiol 1993;138:56–64
11. Field AE, Franko DL, Striegel-Moore RH, Schreiber GB, Crawford PB, Daniels SR. Race differences in accuracy of self-reported childhood body size among white and black women. Obes Res 2004;12: 1136–1144
12. Schernhammer ES, Tworoger SS, Eliassen AH, Missmer SA, Holly JM, Pollak MN, Hankinson SE. Body shape throughout life and correlations with IGFs and GH. Endocr Relat Cancer 2007;14:721–732
13. Solomon CG, Willett WC, Carey VJ, Rich-Edwards J, Hunter DJ, Colditz GA, Stampfer MJ, Speizer FE, Spiegelman D, Manson JE. A prospective study of pregravid determinants of gestational diabetes mellitus. JAMA 1997;278:1078–1083
14. Manson JE, Rimm EB, Stampfer MJ, Colditz GA, Willett WC, Krolewski AS, Rosner B, Hennekens CH, Speizer FE. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. Lancet 1991;338:774–778
15. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. National Diabetes Data Group. Diabetes 1979;28:1039–1057
16. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 1997;20:1183–1197
17. Eriksson JG, Forsén T, Tuomilehto J, Osmond C, Barker DJ. Early adiposity rebound in childhood and risk of type 2 diabetes in adult life. Diabetologia 2003; 46:190–194
18. Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. Obes Rev 2004;5(Suppl. 1):4–104
19. Clausen JO, Ibsen H, Ibsen KK, Borch-Johnsen K. Association of body mass index, blood pressure and serum levels of triglycerides and high-density lipoprotein cholesterol in childhood with the insulin sensitivity index in young adulthood: a 13-year follow-up. J Cardiovasc Risk 1996;3:427–433
20. Wright CM, Parker L, Lamont D, Craft AW. Implications of childhood obesity for adult health: findings from thousand families cohort study. BMJ 2001;323: 1280–1284
21. Mattsson N, Rönnemaa T, Juonala M, Viikari JS, Raitakari OT. Childhood predictors of the metabolic syndrome in adulthood. The Cardiovascular Risk in Young Finns Study. Ann Med 2008;40: 542–552
22. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. Obes Rev 2008;9:474–488
23. Barlow SE. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics 2007;120(Suppl. 4):S164–S192
24. Klein DJ, Aronson Friedman L, Harlan WR, Barton BA, Schreiber GB, Cohen RM, Harlan LC, Morrison JA. Obesity and the development of insulin resistance and impaired fasting glucose in black and white adolescent girls: a longitudinal study. Diabetes Care 2004;27:378–383