Early Adiposity Rebound and the Risk of Adult Obesity

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ABSTRACT. *Objective.* **At 5 to 6 years of age, body fatness normally declines to a minimum, a point called adiposity rebound (AR), before increasing again into adulthood. We determined whether a younger age at AR was associated with an increased risk of adult obesity and whether this risk was independent of fatness at AR and parent obesity.**

Design. **A retrospective cohort study using lifelong height and weight measurements recorded in outpatient medical records.**

Setting. **Group Health Cooperative of Puget Sound (GHC), a health maintenance organization based in Seattle, Washington.**

Participants. **All 390 GHC members (and their parents) born at GHC between January 1, 1965, and January 1, 1971, who had at least one recorded adult height and weight measurement plus two visits with recorded height and weight measurements in each of three age intervals: 1.5 to 4, 4 to 8, and 8 to 16 years.**

Main Outcome Measures. **We calculated the mean body mass index (BMI) of each subject during young adulthood (age 21 to 29 years) and the BMI of the parents when each subject was 1.5 years of age. Adult obesity was defined as a BMI ≥27.8 for males and ≥27.3 for females. Curves were fit to each subject's BMI values between ages 1.5 and 16 years, and the age and BMI at AR were calculated from these curves. Subjects were divided into tertiles of age at AR (early, middle, and late), BMI at AR, and parent BMI (heavy, medium, and lean).**

Results. **The mean age at AR was 5.5 years, and 15% of the cohort was obese in young adulthood. Adult obesity rates were higher in those with early versus late AR (25% vs 5%), those who were heavy versus lean at AR (24% vs 4%), those with heavy versus lean mothers (25% vs 5%), and those with heavy versus lean fathers (21% vs 5%). After adjusting for parent BMI and BMI at AR, the odds ratio for adult obesity associated with early versus late AR was 6.0 (95% CI, 1.3–26.6).**

Conclusion. **An early AR is associated with an increased risk of adult obesity independent of parent obesity and the BMI at AR. Future research should examine the biological and behavioral determinants of AR.** *Pediatrics* **1998;101(3).**

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This paper was presented in part at the 37th Annual Meeting of the Ambulatory Pediatric Association, Washington, DC, May 3, 1997.

Received for publication Jul 2, 1997; accepted Oct 30, 1997.

Reprint requests to (R.C.W.) Children's Hospital Medical Center, Division of General and Community Pediatrics, CH-1S, 3333 Burnet Ave, Cincinnati, OH 45229-3039.

URL: http://www.pediatrics.org/cgi/content/full/101/3/e5; ***obesity, child, body mass index, parents.***

ABBREVIATIONS. BMI, body mass index; AR, adiposity rebound; GHC, Group Health Cooperative of Puget Sound.

Changes in body composition occur with normal growth. Age-related change in body fatness can be shown by radiographs[1] or by measurement of skin-fold thickness.[2,3] However, for longitudinal studies, height and weight measurements are easier to obtain than direct measures of fatness. The body mass index (BMI = weight [kilograms] divided by stature [meters]^2) is the best and most widely used surrogate measure of fatness among those indices derived from height and weight measurements.[4,5] Children have a rapid increase in BMI during the first year of life. After 9 to 12 months of age, BMI declines and reaches a minimum, on average, at 5 to 6 years of age before beginning a gradual increase through adolescence and most of adulthood. The point of maximal leanness or minimal BMI has been called the adiposity rebound (AR).[6]

The time of AR may be a critical period in childhood for the development of obesity.[7] An early AR (younger age at the point of AR) is associated with higher BMI in adolescence[6,8,9] and in early adulthood,[10] but no study has examined the association between the age at AR and adult obesity. Therefore, we determined whether an early AR is associated with an increased risk of adult obesity and whether this association is independent of three other risk factors for adult obesity—the BMI at AR, maternal BMI, and paternal BMI.[11]

METHODS

In this retrospective cohort study, we abstracted height and weight measurements from the outpatient medical records of a cohort of young adults and their parents, all of whom had been long-term enrollees at Group Health Cooperative of Puget Sound (GHC), a staff-model health maintenance organization based in Seattle, Washington. GHC was established in 1947 and, at the time of this study, was the sixth-largest nonprofit health maintenance organization in the United States, enrolling ~10% of the population in the Seattle metropolitan area.

Subject Selection

Using GHC's computerized enrollment and outpatient visit databases, we identified all 1333 GHC members who were born at GHC between January 1, 1965, and January 1, 1971, and who had at least one outpatient visit at GHC after 21 years of age. The medical records of 8 subjects could not be located or were missing volumes, 18 subjects were excluded because of premature birth (gestational age <36 weeks), and 44 were excluded because of a

chronic condition that significantly affected weight or stature (eg, cancer). Of the remaining 1263 subjects, 854 had at least one adult weight measurement (age ≥21 years) and at least one adult height measurement (age ≥18 years for males and age ≥16 years for females). Our final study sample included the 390 subjects who also had at least two visits with recorded height and weight measurements in each of the following age intervals: 1.5 to 4 years, 4 to 8 years, and 8 to 16 years. The 390 subjects were born to 364 pairs of parents, all of whom were also GHC members. One pair of parents had three offspring in the cohort and 24 pairs of parents had two offspring in the cohort. Thus, 51 subjects (13.0%) had at least one sibling in the cohort.

Outcome Measures

For subjects and both of their parents, all height and weight measurements recorded before January 1, 1994, were abstracted from the outpatient medical record unless these measurements were from an emergency department visit or during a pregnancy. For each subject, we calculated the average BMI between 21 and 29 years of age. Adult obesity was defined as an average BMI ≥27.8 in men and ≥27.3 in women.[12] We standardized the mean adult BMI for sex by converting BMI to a z score. The z score was calculated as (BMI − mean)/SD, where the mean and SD of BMI were from a same-sex reference population of 20- to 29-year-olds in the Second National Health and Nutrition Examination Survey.[13]

Predictor Measures

We used the method of Siervogel and colleagues to determine the age and BMI at AR.[9] This method involved fitting a four-parameter polynomial function to the logarithmic-transformed BMI data on each subject between 1.5 and 16 years of age. The function took the form of $ln\ \text{BMI} = \beta_0 + \beta_1 x + \beta_2 x^2 + \beta_3 x^3 + \epsilon$, where x was the age in years and ϵ was the error term. The age and BMI at AR were derived from the parameter estimates of this cubic polynomial (Fig 1). BMI at AR then was converted to a z score using the reference mean and SD of BMI for that child's age and sex. The reference means and SD units were from the combined data of the First and Second National Health and Nutrition Examination Surveys.[2] Means and SD units of BMI for specific ages (eg, 5.2 years) were found by linear interpolation between the discrete ages (eg, 5 years and 6 years) given in the reference data.

If the parent medical record contained an adult height, the recorded parent weights were used to calculate BMI values. Parent BMI was estimated on the date the child turned 18 months of age. This age was picked to obtain a point before the child's normal age at AR and after the mother had returned to her baseline postpartum weight. To estimate the parent BMI, we used linear interpolation between parent BMI values before and after the date the child turned 18 months of age.

Statistical Analysis

For each subject we computed a weighted average of the BMI values in adulthood (21 to 29 years of age). The weighting was based on the time between available values. Values closely spaced in time received less weight than widely spaced values, so that the average would not be unduly influenced by the values clustered close together in time. The formula used for the average was:

$$\sum_{i=1}^{K+1} \frac{1}{2}[\text{BMIz}(t_i) + \text{BMIz}(t_{i-1})](t_i - t_{i-1})/(t_{K+1} - t_0),$$

where $t_1, \ldots, t_K$ denoted the ages at which BMI values were available and where $t_0 = 21.0$ and $t_{K+1} = 29.0$ years of age. The limits t_0 and t_{K+1} were used in the formula so that the average would reflect an estimate of the average over the entire interval (t_0, t_{K+1}), rather than just the interval over which data were available.

For the predictor variables, the study population was divided into tertiles of age at AR (early, middle, and late), BMI z score at AR, maternal BMI, and paternal BMI (lean, medium, and heavy). Data were also analyzed according to parent obesity status, with parent obesity defined as a BMI ≥27.8 in fathers and ≥27.3 in mothers.[12]

We first summarized the outcome measures (adult BMI and adult obesity) for male and female subjects by tertile of each risk factor: age at AR, BMI z score at AR, maternal obesity, and paternal obesity. Logistic regression models for adult obesity were constructed to examine the combined effects of the four risk factors for adult obesity. The sex of the subject was included in the models because of differing rates of adult obesity observed for males and females in this cohort. Regression parameters for these models were estimated using the generalized estimating equation method of Liang and Zeger.[14] This method incorporated adjustments for correlations between data from siblings and was implemented with the *xtgee* command of STATA (version 5.0), the statistical software used to perform our data analyses.[15]

RESULTS

Ninety-five percent of the subjects were non-Hispanic whites, and 60% were female. More women than men met inclusion criteria for the study. We suspect that this gender disparity occurred because in young adulthood, women are more likely than men to use health care[16] and more often met study criteria by having adult weight and height measurements in their record. At birth of the subject, the mean maternal and paternal ages were 28 and 32 years, respectively. Socioeconomic data for the fam-

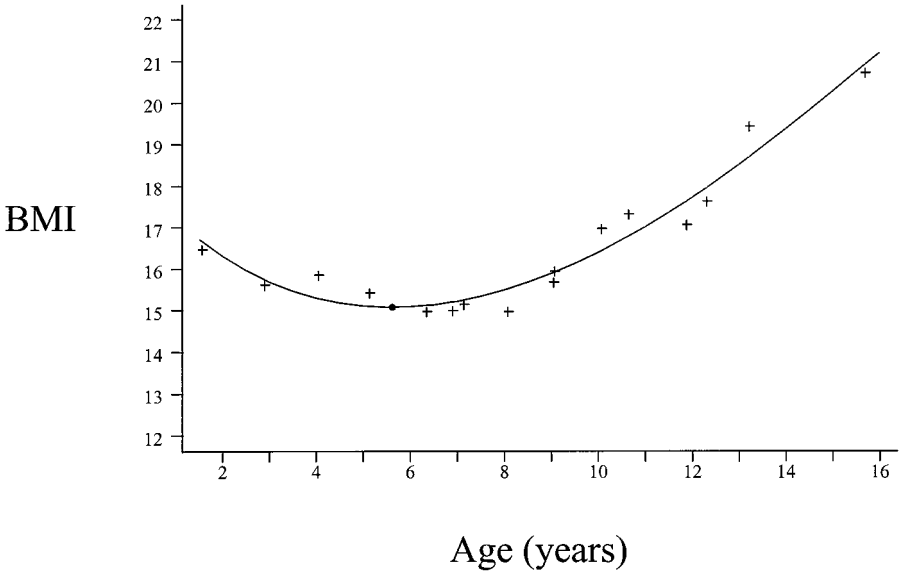

Fig 1. An example of the curve produced by fitting a cubic polynomial in age to the natural log of the available observed BMI values (+) for one subject. The vertical axis has been transformed back to BMI units by exponentiation: $\text{BMI} = \exp(\beta_0 + \beta_1 x + \beta_2 x^2 + \beta_3 x^3)$. The minimum point on the fitted curve (●) is the point of AR.

ilies were not available in the medical record, but previous surveys indicate that the GHC population is primarily middle and upper-middle class and well educated.

Males had a higher rate of adult obesity than did females, but did not differ significantly from females in mean adult BMI *z* score (Table 1). The mean adult BMI *z* score was not significantly different between the 390 study subjects and the 464 subjects who had an adult BMI measurement but who had too few childhood measurements to be included in the study cohort. Girls tended to reach AR earlier than did boys. As expected, the mean BMI *z* score at AR was negative, because the BMI at AR was, by definition, an estimate of each subject's lowest BMI point in childhood. The 11 subjects whose calculated age at AR was <1.5 years were assumed to have reached AR at 1.5 years, and their BMI *z* score at AR was not calculated. BMI data were available on the mother for 87% of subjects, on the father for 73% of subjects, and on both parents for 62% of subjects. Fathers had higher obesity rates than did mothers.

In both sexes, earlier AR, higher BMI at AR, and higher maternal and paternal BMI all were associated with higher BMI and obesity rates in young adulthood (Table 2). These differences in adult obesity rates across tertiles of each risk factor were statistically significant in both sexes for both BM *z* score and age at AR, in females for maternal BMI, and in males for paternal BMI ($P < .05$ using logistic regression to adjust for sibling correlations). In females, rates of adult obesity also increased with increasing paternal BMI and, in males, with increasing maternal BMI, but these trends did not reach statistical significance. The four risk factors for adult obesity were interrelated. Both parent BMI and the BMI *z* score at AR were associated with a younger age at AR (associations examined with nonparametric tests; data not shown).

For both sexes, regardless of the tertile of BMI *z* score at AR, the chance of adult obesity was higher in those who reached AR earlier (Table 3). The risk of adult obesity was highest in those who had early AR and who also were heavy at AR, and was lowest in those who had late AR and who also were lean at AR. The age at AR and the BMI *z* score at AR were independent risk factors for adult obesity when considered in a multivariate logistic model (Table 4). There were no significant interactions between the factors included in this model (data not shown).

Among children with at least one obese parent, the chance of adult obesity was highest for those who with early AR and lowest for those with late AR (Table 5). All four risk factors for adult obesity were examined in a logistic model to determine the statistical significance of each risk factor while adjusting for the others (Table 6). This analysis was limited to those who had data available on all risk factors ($n = 234$). The rate of adult obesity was lower in subjects included in this model compared with those not included (11% vs 21%, $P = .007$). However, there were no differences between these two groups in sex, age at AR, or BMI *z* score at AR. After controlling for all other risk factors in the model, there was a strong indication that early AR was associated with an increased risk of adult obesity ($P = .06$). In this model, the odds ratio for both paternal and maternal obesity also were statistically significant. The odds ratios for the other risk factors were in the anticipated direction and may not have reached statistical significance in the final model because of the smaller sample size and the lower prevalence of obesity in the sample.

DISCUSSION

This is the fourth report demonstrating an association between early AR and later BMI.[8–10] For the first time, however, we have shown that adult obesity occurs more frequently in children who have early AR. Furthermore, the increased risk of adult obesity associated with early AR is independent of both the BMI at the AR and parent obesity.

This study differs from other studies of AR because we used retrospective rather than prospective data and because our subjects were older at follow-up. Our height and weight measurements were obtained from clinical records, and these measurements lack the precision obtainable in a prospective study. However, any measurement error is likely to have been random. Because our subjects differed from each other in the number and timing of their BMI

TABLE 1. Outcome Variables and Predictors by Gender

	Female		Male		Total	
	Mean (SD)	*N*	Mean (SD)	*N*	Mean (SD)	*N*
Outcome variables						
Adult BMI *z* score	0.11 (1.03)	235	0.17 (1.05)	155	0.13 (1.04)	390
Adult BMI	23.6 (4.8)	235	25.0 (4.0)	155	—	—
Obese as adults (% [#])	11 (27)*	235	19 (30)*	155	15 (57)	390
Predictor variables						
Age at AR (years)	5.4 (1.7)†	235	5.8 (1.9)†	155	5.5 (1.8)	390
BMI *z* score at AR	−0.23 (0.71)	228	−0.18 (0.69)	151	−0.21 (0.70)	379
Maternal BMI‡	22.8 (3.7)	200	23.0 (3.6)	119	22.9 (3.7)	319
Mother obese (% [#])‡	11 (22)	200	13 (15)	119	12 (37)	319
Paternal BMI‡	25.5 (3.4)	169	25.7 (3.3)	117	25.6 (3.4)	286
Father obese (% [#])‡	16 (27)	169	21 (24)	117	18 (51)	286
At least one parent obese (% [#])‡	24 (36)	147	31 (30)	96	27 (66)	243

* $P = .03$ for difference between males and females, all other *P* values are ≥.10 unless indicated.
† $P = .06$ for difference between males and females.
‡ At the time the child is 18 months old.

TABLE 2. Mean (SD) BMI *z* Score and Obesity Prevalence in Young Adulthood (Age 21–29 Years) by Tertile of Age at AR, Tertile of BMI *z* Score at AR, and Tertile of Parent BMI

	Female	Male	Total
Age at AR			
Early (age <4.8 years)			
BMI *z* score	0.55 (1.35)	0.63 (0.94)	0.58 (1.23)
% Obese (# obese/total)	20 (17/87)	37 (15/41)	25 (32/128)
Middle (4.8 years ≤ age <6.2 years)			
BMI *z* score	0.00 (0.64)	0.23 (1.25)	0.09 (0.94)
% Obese (# obese/total)	10 (8/79)	20 (11/54)	14 (19/133)
Late (age ≥6.2 years)			
BMI *z* score	−0.32 (0.63)	−0.19 (0.77)	−0.26 (0.70)
% Obese (# obese/total)	3 (2/69)	7 (4/60)	5 (6/129)
BMI *z* score at AR			
Lean (BMI *z* score <−0.54)			
BMI *z* score	−0.36 (0.73)	−0.34 (0.66)	−0.35 (0.70)
% Obese (# obese/total)	3 (2/71)	6 (3/54)	4 (5/125)
Medium (−0.54 ≤ BMI *z* score <0.05)			
BMI *z* score	0.04 (0.82)	0.10 (0.91)	0.06 (0.85)
% Obese (# obese/total)	10 (9/87)	15 (6/41)	12 (15/128)
Heavy (BMI *z* score ≥0.05)			
BMI *z* score	0.46 (0.89)	0.62 (1.20)	0.53 (1.04)
% Obese (# obese/total)	19 (13/70)	30 (17/56)	24 (30/126)
Maternal BMI			
Lean (BMI <21.0)			
BMI *z* score	−0.29 (0.52)	0.20 (.082)	−0.26 (0.65)
% Obese (# obese/total)	0 (0/66)	12 (5/40)	5 (5/106)
Medium (21.0 ≤ BMI <23.2)			
BMI *z* score	−0.05 (0.60)	0.20 (1.06)	0.04 (0.80)
% Obese (# obese/total)	9 (6/69)	18 (7/38)	12 (13/107)
Heavy (BMI ≥23.2)			
BMI *z* score	0.62 (1.52)	0.32 (1.15)	0.51 (1.39)
% Obese (# obese/total)	23 (15/65)	27 (11/41)	25 (26/106)
Paternal BMI			
Lean (BMI <24.1)			
BMI *z* score	−0.07 (0.88)	−0.47 (0.63)	−0.21 (0.82)
% Obese (# obese/total)	7 (4/61)	3 (1/34)	5 (5/95)
Medium (24.1 ≤ BMI <26.4)			
BMI *z* score	0.13 (0.88)	0.16 (0.82)	0.14 (0.85)
% Obese (# obese/total)	12 (7/56)	15 (6/40)	14 (13/96)
Heavy (BMI ≥26.4)			
BMI *z* score	0.31 (1.36)	0.27 (0.98)	0.29 (1.19)
% Obese (# obese/total)	15 (8/52)	28 (12/43)	21 (20/95)

TABLE 3. Mean (SD) BMI and Obesity Prevalence in Young Adulthood (Age 21–29 Years) by Tertile of Age at AR and Tertile of BMI *z* Score at AR

Age at AR	BMI *z* Score at AR					
	Female			Male		
	Lean	Medium	Heavy	Lean	Medium	Heavy
Early						
BMI	23.8 (5.3)	24.2 (4.9)	26.5 (5.0)	25.3 (3.0)	25.7 (4.1)	27.3 (3.2)
% Obese (# obese/total)	11 (2/18)	17 (5/30)	22 (7/32)	10 (1/10)	27 (3/11)	44 (7/16)
Middle						
BMI	21.1 (2.1)	23.0 (2.6)	24.6 (3.2)	22.7 (2.3)	24.2 (3.3)	27.4 (5.8)
% Obese (# obese/total)	0 (0/20)	9 (3/32)	19 (5/27)	7 (1/14)	12 (2/17)	35 (8/23)
Late						
BMI	20.3 (1.8)	22.6 (3.7)	23.3 (2.5)	22.4 (2.0)	24.5 (3.3)	25.0 (3.3)
% Obese (# obese/total)	0 (0/33)	4 (1/25)	9 (1/11)	3 (1/30)	8 (1/13)	12 (2/17)

values, we used a mathematical model to estimate both the age and the BMI at AR. The statistical effect of estimation error is to attenuate associations between predictors and outcomes. The actual associations, therefore, may be even greater than those we estimated.

Analyzing prospective data from the Fels Longitudinal Study, Siervogel and colleagues used the same method as our own to determine the age and BMI at AR.[9] In the Fels cohort, 496 subjects (also non-Hispanic whites) were measured semiannually from birth to 18 years of age, and the mean age at AR was 5.1 years in boys and 5.3 years in girls, values extremely close to our own. In the Fels cohort, there was a significant inverse correlation between the age at AR and BMI at 18 years of age ($r = -0.46$ in boys and $r = -0.54$ in girls; $P = .01$), and these correlations were similar in magnitude to those in our own study ($r = -0.31$ in boys and $r = -0.43$ in girls; $P < .01$). In the two other longitudinal cohorts in which

TABLE 4. Logistic Regression Model for Adult Obesity as a Function Age at AR, BMI *z* Score at AR, and Sex (n = 379)

	Odds Ratio	95% Confidence Interval	*P* Value*
Age at AR			<.01
Middle versus late	2.8	(1.0–7.5)	
Early versus late	4.9	(1.9–12.9)	
BMI *z* score at AR			<.01
Medium versus low	2.8	(1.0–8.3)	
High versus low	5.9	(2.1–16.2)	
Sex			
Male versus female	2.1	(1.1–4.0)	.02

* *P* value is for significance of factor in the model.

TABLE 5. Mean (SD) BMI *z* Score and Obesity Prevalence in Young Adulthood (Age 21–29 Years) by Tertile of Age at AR and Parental Obesity

	At Least One Parent Obese	Neither Parent Obese
Age at AR		
Early		
BMI *z* score	1.4 (1.8)	0.2 (0.7)
% Obese (# obese/total)	43 (10/23)	13 (8/60)
Middle		
BMI *z* score	0.2 (0.8)	−0.2 (0.7)
% Obese (# obese/total)	27 (6/22)	7 (4/57)
Late		
BMI *z* score	0.0 (0.7)	−0.4 (0.7)
% Obese (# obese/total)	5 (1/21)	3 (2/60)

TABLE 6. Logistic Regression Model for Adult Obesity as a Function of Parent Obesity, Age at AR, BMI at AR, and Gender (n = 234)

	Odds Ratio	95% Confidence Interval	*P* Value*
Paternal obesity			.007
Obese versus nonobese	4.1	(1.5–11.4)	
Maternal obesity			.04
Obese versus nonobese	3.2	(1.1–9.5)	
Age at AR			.06
Middle versus late	3.8	(0.9–16.2)	
Early versus late	6.0	(1.3–26.6)	
BMI *z* score at AR			.16
Medium versus low	2.5	(0.6–10.0)	
High versus low	3.6	(1.0–13.7)	
Sex			.11
Male versus female	2.2	(0.8–5.5)	

* *P* value is for significance of factor in the model.

AR has been studied, one from France[10] and one from the Czech Republic,[8] the mean age at AR was not reported. In both of those studies, the point of AR was determined by visual inspection of each subject's plotted BMI values rather than derived from a polynomial function fitted to each subject's available BMI values. In the French cohort (n = 164), subjects were classified as having an early (≤5.5 years), average (6 to 6.5 years), or late (≥7 years) age at AR. Mean BMI at 21 years of age was highest in both males and females who were in the early AR group. In the Czech cohort (n = 300), those heaviest at 18 years of age (BMI > cohort 90th percentile) had a significantly earlier AR than those in the leanest group (BMI < cohort 10th percentile)—4.6 versus 7.8 years of age in males and 5.3 versus 7.4 years of age in females.

Rolland-Cachera and colleagues suggested that early AR might constitute a marker for generalized growth acceleration and cell hyperplasia.[6] They showed that early AR was associated with advanced bone age, and they suggested that this acceleration in skeletal maturation was, perhaps, also a marker for fat cell hyperplasia. No study has yet shown that the adipose tissue of children with early AR is more hyperplastic than the adipose tissue of children with late AR. Despite the suggestion from all studies that early AR is associated with later BMI, a shared limitation of these studies is that BMI reflects both lean and fat mass. There have been no studies that measured body composition directly at AR and again in adolescence or adulthood. Even if the hypothesis posed by Rolland-Cachera and colleagues was supported by new data, there is conflicting evidence[17,18] with regard to the theory that fat cell hyperplasia early in childhood increases the risk for later obesity.[19]

The age at AR may be genetically programmed, relatively difficult to alter, and merely a reflection of an inherited susceptibility to obesity. Alternatively, the age at AR also could reflect important environmental influences that are modifiable.[10] For example, parent feeding practices, particularly the practices of obese parents whose children are genetically more susceptible to obesity, may promote the expression of both early obesity and early AR. In 3- to 5-year-old children, Johnson and Birch have shown that a high degree of maternal control over feeding is associated with poorer ability of these children to regulate energy intake, which, in turn, is associated with higher childhood BMI.[20] Strategies used by parents to encourage toddlers to eat, such as contingencies (eg, If you eat this, then . . .), may teach the child at a critical developmental period to attend to external rather than to internal satiety cues.[21] These parent strategies may be a well intentioned response to the natural decline in adiposity that occurs between 1 and 5 years of age, a period also marked by increased levels of both physical activity and picky eating. Thus, environmental factors in early childhood, signaled by early AR, may have an enduring effect on the proper regulation of energy intake and on later obesity risk.

The increasing prevalence of childhood[22] and adult obesity,[23] the risk of childhood obesity for adult obesity,[11] and the challenge of treating obesity after it has developed[24] all suggest that future research should examine the biological and behavioral determinants of AR. In addition, obesity prevention and treatment trials in children should examine whether the age at AR is a modifiable risk factor for adult obesity, especially in children born to obese parents.

ACKNOWLEDGMENTS

This work was supported by the Generalist Physician Faculty Scholars Award from the Robert Wood Johnson Foundation (R.C.W.).

We thank the following individuals: Evan Charney, MD, and Heidi J. Kalkwarf, PhD, for their careful review of this manuscript; Edward H. Wagner, MD, MPH, for facilitating this research at the

Center for Health Studies at Group Health Cooperative of Puget Sound, Seattle, WA; Richard L. Furman for detailed abstraction of all the medical records for this study; and Vicki L. Livengood for her assistance with formatting this manuscript.

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