Does Overweight in Childhood Have an Impact on Adult Health?

The consequences of overweight in childhood, including persistence into adulthood and as a risk factor for adverse health consequences, are of substantial concern given the recent upward trend in prevalence. A recent report on an historic cohort—a British 1947 birth cohort—is largely consistent with previous estimates of persistence of overweight. Long-term health consequences of obesity were not demonstrable in this study, likely owing to the small numbers of subjects who were overweight during post–World War II Britain.

Key Words:

The health consequences of adult obesity are substantial, rivaling smoking as the modifiable risk factor with the most adverse health effects. Childhood obesity is considered an important disease because of its link to long-term health consequences. Specifically, studies have demonstrated associations between elevated weight status in childhood and adolescence and all-cause mortality, heart disease morbidity and mortality, ovulatory dysfunction, metabolic syndrome, arthritis, gout, and other diseases.[1–5] A large body of prior research also indicated that overweight children become overweight adults.[6] Against this backdrop, the results of a recent report[7] and the accompanying commentary[8] on the health outcomes of adults who were initially studied as children in Newcastle, England, were unexpected.

Wright et al. conducted a long-term follow-up of the Newcastle Thousand Families Study, a prospective cohort study of all children born during two months in 1947 in Newcastle, England.[7] The authors used body mass index (BMI, kg/m^2) z-scores (standard deviation scores calculated relative to 1990 height and weight data from the United Kingdom) as the measure of weight status in childhood; they used percentage body fat by bioelectrical impedance and BMI (absolute or as a z-score referenced to age 20) as the measure of weight status in adults. The report indicated that BMI z-scores at age 9 and age 13 correlated significantly ($P < 0.001$) with BMI z-score at age 50 (i.e., at age 9, $r = 0.24$; at age 13, $r = 0.39$). BMI at age 13, but not at age 9, also correlated marginally with percentage body fat at age 50. Overweight in childhood was defined as a BMI z-score greater than the 90th percentile; overweight and obesity in adulthood were defined as a BMI of 24 and 30, respectively. Although most overweight and obese adults were not overweight as children, overweight at age 9 or 13 was associated with a five- to ninefold increase in risk of adult obesity. Associations between percentage body fat in childhood and adulthood were weaker, and suggested less evidence of tracking.

Analyses by Wright et al. also showed either absent or weak inverse association between childhood overweight and a large complement of classic cardiovascular disease risk factors, including carotid artery intima-media thickness, blood pressure, total cholesterol, triglycerides, 2-hour glucose, and serum insulin. For virtually all factors, BMI z-score in childhood was unrelated to adult BMI according to a simple bivariate analysis. After statistical control for adult BMI z-score or percentage body fat, associations were weak, significant, and inverse. By contrast, Wright et al. found that the subjects who were thinnest as children and fattest as adults had the highest adult health risk. The 20 subjects who met all criteria for metabolic syndrome (based on criteria for the presence of dyslipidemia, abnormal glucose metabolism and hypertension) and the 47 subjects who died in adulthood were not more likely to have been in the top quartile for BMI at ages 9 or 13 than the other study participants.

Wright et al. deduced that their study results suggested little tracking of childhood overweight into adulthood and no excess health risks associated with BMI at age 9 or 13. In their summary, the editors[8] asserted that the Newcastle data had "thrown doubt on the assumption that fat children become fat adults." Based on the discussion in their report,[7] and the editor's summary accompanying the article,[8] these findings would seem to conflict with previous studies.[9–12] To evaluate whether this interpretation is valid, one must review the methodology used and assess whether the conclusions drawn are consistent with the findings.

The Newcastle Thousand Families Study was initiated in 1947 and included 1142 newborn infants. At ages 9 and 13, 688 and 628 children were measured, respectively. For the adult follow-up, participants were traced through the National Health Service registrar; causes of death were coded for 47 decedents who died after age 18, and living participants were queried by a self-administered questionnaire ($n = 529$). Clinical assessment of cardiovascular risk factors and anthropometric measures of height, weight, waist circumference, and percent body fat were obtained for 412 participants. Adult BMI was

This review was prepared by Aviva Must, Ph.D., Department of Family Medicine and Community Health, Tufts University School of Medicine, Boston, MA 02111, USA.

based on a combination of measured and self-reported heights and weights. The clinical and anthropometric data were therefore available for 36% and 46% of the original cohort, respectively. Consequently, the sample studied for this report was quite small, which may account for why males and females were not evaluated separately. Based on the information provided, it is difficult to judge if the subgroup followed into adulthood was representative of the initial birth cohort. Heights and weights used to establish BMI z-scores were based on 1990 United Kingdom reference standards.[13] The authors also expressed height and weight z-scores based on the older Tanner Whitehouse reference.[14] Although not directly ascertainable from the data presented, it appears that the children studied, who were born in post–World War II Britain, were very short and somewhat lean.

An issue that typically arises when looking to historic cohorts for information relevant to the present is generalizability. The children studied in the Newcastle study were born in 1946 and thus experienced an environment very different in both food intake and activity patterns from the present environment. The thin children Wright et al. studied did not grow up in an environment dominated by sedentary behavior (e.g., television viewing) and consumption of calorie-dense foods.[15] Given the striking differences between the children of today and the children of post–World War II Britain, it is surprising that any persistence of weight status was demonstrable.

Persistence of relative weight status from childhood into adulthood has been consistently demonstrated in all populations.[9–12,16–19] These studies indicate that the likelihood of persistence is related to both severity of obesity and the age at which it is present. In general, the likelihood that overweight persists from childhood to adulthood is moderate. Viewed from the perspective of childhood, 20 to 50% of individuals who are obese in adolescence remain obese in adulthood.[10,17,19] Although fewer than half the children who were obese during childhood went on to become obese adults, therefore, the risk of adult obesity was two to 11 times higher for obese compared with non-obese children. From the adult vantage point, only 17 to 18% of 33-year-old obese adults had been obese in childhood.[18]

Although the simple observation that obesity persists from childhood into adulthood is consistent among the aforementioned studies, obesity criteria (i.e., both the indicator used and the definition applied) vary, as do the statistics used to evaluate the degree of persistence. For example, the Wright study applied a BMI reference standard from the 1990s to a population that appears to have been stunted. This clouds the interpretation of the childhood BMI z-score measures, and thus may explain the absence of an observed relationship between childhood BMI z-score and percentage body fat in adulthood. Interpretation and summarization of studies in this area is further complicated by the large variability in the time intervals between childhood and adult measurements. Intervals over which persistence has been examined range widely, with childhood ages ranging from 1 to 16 years and adult ages ranging from 18 to 47 years. In most studies the interval between childhood and adulthood is only approximately 10 years. In the Wright study, the follow-up interval was far longer: 37 and 41 years. In the Fels Longitudinal Study, a prospective study with up to 32 years of follow-up, persistence of overweight, based on BMI percentiles from the 2000 CDC growth charts, was similar in females and males; consistent with other studies in this area, the likelihood of persistent overweight increased with childhood age and with severity of childhood obesity.[19] Using data from a pooled sample from the Fels and other longitudinal studies, Guo reported correlations between BMI in children aged 9 and BMI at age 35 of approximately 0.28 for males and 0.60 females.[11] At age 13, correlations with BMI at age 35 were approximately 0.50 for males and 0.62 for females.[11] These reported correlations with adult measures for children born between 1929 and 1960 are substantially higher than those observed in the Newcastle families study.

Sex differences in the degree of obesity persistence were not evaluated in the Newcastle study. In previous studies of persistence, sex differences were apparent in several,[9,10] but not all,[12,18] of the larger studies. In studies that found differences by sex, females seemed to have greater likelihood of persistence. Differences in the degree of tracking between males and females appear to depend on childhood ages. For example, in the longitudinal sample studied by Guo,[11] the correlations between childhood BMI and BMI at age 35 were similar until age 6 (i.e., differences in correlations of approximately 0.05 or less), quite different from age 7 to age 11 (i.e., differences in correlations of approximately 0.35), and somewhat different through adolescence (i.e., differences in correlations of approximately 0.10). Using an internally referenced definition of overweight (>130% of ideal body weight), a follow-up study of females who were overweight in adolescence according to the National Survey of Health and Development (the British 1946 birth cohort) showed that these females were more likely to have overweight track into adulthood than males.[9] Among individuals who were obese at age 36, 11% of males and 26% of females were obese at age 20, and 14% of males and 32% of females were obese at age 14. In a report from the later 1958 British birth cohort, however, persistence was similar for males and females.

Seventeen percent and 18% of obese 33-year-old men and women, respectively, had a BMI that was greater than the 95th percentile at age seven.[12] Whether the findings from the two British cohorts reflect a true difference or the application of different overweight criteria is unclear.

Of 181 Harvard Growth Study participants studied over a 55-year interval from adolescence to old age who were overweight as adolescents (>75th percentile BMI for at least two years), 46% of females and 52% of males were overweight at midlife (mean age 52 years), and 53% of females and 41% of males were overweight at mean age 73 years (Must, unpublished observations, 1994). The degree of tracking of BMI z-score by sex was not reported in the Newcastle study.

The degree of tracking of overweight from childhood into adulthood reported in the Newcastle study is, in fact, wholly consistent with previous studies. Although the children classified as overweight (>90th percentile BMI by 1990 British standards) would be considered in-between "at risk for overweight" (85th percentile) and "overweight" (95th percentile) by contemporary U.S. terminology,[20] they were five to nine times more likely to be obese at age 50 compared with children in the lowest quartile of BMI percentile scores. Thirty percent of those who were obese at age 50 were overweight at age 13. These observations underscore the strength of childhood overweight as a risk factor for adult overweight, even for this historic cohort. The authors are certainly accurate in their assertion that the proportion of adult obesity attributable to childhood overweight is only moderate; nonetheless, childhood obesity is a potent risk factor for adult obesity, consistent with observations from other studies.[9,11,17,19] Unfortunately, owing to the leanness of the Newcastle cohort and the small number of overweight subjects, Wright et al. could not assess whether childhood overweight that persists into adulthood is associated with more severe obesity, as has been demonstrated in other studies.[21] Furthermore, the weak correlations between childhood and adult BMI z-scores reported by Wright et al. should not be interpreted as weak effects. A linear correlation coefficient is a poor measure of the degree to which overweight tracks because it reflects tracking across all levels of BMI (not just overweight). The correlations will be attenuated by the narrow range of BMI z-scores, and will account only for the linear correlation.

How far into adolescence overweight persists and how severe the obesity is determines the likelihood of persistence.[9–11,17] The disturbing secular trends in childhood obesity seen in the United States[22] and the world[23] have important implications for the likelihood of persistence. The weight distribution has shifted to the right and is increasingly skewed.[24] Thus, not only are there more obese children and adolescents, but there are more very obese children. This suggests that the likelihood of persistence of overweight from childhood into adulthood will increase in the future.

Overall, the sample studied was quite small. In childhood, only 24 9-year-old children and 38 13-year-old children (many of the same children) were overweight by the definition used at that time. At age 50, only 20 of the study subjects met the criteria for metabolic syndrome, and 48 had died. These small numbers preclude analyses stratified by sex. In our retrospective follow-up of the Third Harvard Growth Study, the associations of adolescent overweight (defined as overweight after age 13) with morbidity and mortality was largely limited to males.[3] Most of the other long-term studies of health consequences of childhood overweight have been conducted in males[25–27] with mortality as an endpoint. These studies generally find an approximate doubling of risk for all-cause mortality and mortality from heart disease for males who were overweight children. Adult follow-up (to age 34, for the oldest children studied initially) of the Bogalusa Heart Study cohort suggests that adult levels of triglycerides, low-density lipoprotein cholesterol, insulin, systolic blood pressure, and diastolic blood pressure are all higher in children who are >95th percentile for BMI compared with those below the median.[28] In Bogalusa, only for triglyceride levels did there appear to be an effect of childhood weight status and it was independent of adult obesity. In a study of more than 400 Finns with periodically collected data from birth through mid-life, subjects in the highest BMI quartile at age 7 were four times more likely to have metabolic syndrome as adults (defined as the cluster of hypertension, dyslipidemia, and insulin resistance) than were the remaining subjects.[4] Data were not reported by sex. In the face of the limited power of the study by Wright et al., any conclusions about the absence of effects seem unwarranted.

The current global epidemic of obesity across sex, age, and racial-ethnic groups represents a serious public health threat. Evidence suggests that successful efforts to reduce the incidence of obesity in childhood would reduce adult obesity and its associated diseases. The findings in the Wright study largely consistent with previous research.

1. Rich-Edwards JW, Goldman MB, Willett WC, et al. Adolescent body mass index and infertility caused by ovulatory disorder. *Am J Obstet Gynecol.* 1994; 171:171–177.
2. Lake JK, Power C, Cole TJ. Women's reproductive health: the role of body mass index in early and adult life. *Int J Obes.* 1997;21:432–438.
3. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz

WH. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med.* 1992;327:1350–1355.

4. Vanhala MJ, Vanhala PT, Keinanen-Kiukaanniemi SM, Kumpusalo EA, Takala JK. Relative weight gain and obesity as a child predict metabolic syndrome as an adult. *Int J Obes.* 1999;23:656–659.

5. Nieto FJ, Szklo M, Comstock G. Childhood weight and growth rate as predictors of adult mortality. *Am J Epidemiol.* 1992;136:201–213.

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

7. 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.

8. Childhood overweight does not lead to adult fatness. *BMJ.* 2001;323.

9. Braddon FEM, Rodgers B, Wadworth MEJ. Onset of obesity in a 36 year birth cohort study. *BMJ.* 1986;293:299–303.

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

11. Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35 years. *Am J Clin Nutr.* 1994;59:810–819.

12. Power C, Lake JK, Cole TJ. Body mass index and height from childhood to adulthood in the 1959 British birth cohort. *Am J Clin Nutr.* 1997;66:1094–1101.

13. Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child.* 1995;73:25–29.

14. Tanner JM, Whitehouse RH, Takaishi M. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. I. *Arch Dis Child.* 1966;41:454–471.

15. Vitetta L, Sall A, Mrazek L. A delay in the onset of obesity? (Electronic rapid response). *BMJ.* 2001;323:1280–1284.

16. Freedman DS, Shear CL, Burke GL, et al. Persistence of juvenile-onset obesity over eight years: the Bogalusa Heart Study. *Am J Public Health.* 1987;77:588–592.

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

18. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med.* 1997;337:869–873.

19. Guo S, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr.* 2002;76:653–658.

20. Barlow SE, Dietz WH, Klish WJ, Trowbridge FL. Medical evaluation of overweight children and adolescents: reports from pediatricians, pediatric nurse practitioners, and registered dietitians. *Pediatrics.* 2002;110:222–228.

21. Rimm IJ, Rimm AA. Association between juvenile onset obesity and severe adult obesity in 73, 532 women. *Am J Public Health.* 1976;66:479–481.

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

23. WHO. Obesity: preventing and managing the global epidemic. Geneva, Switzerland: World Health Organization, Division of Noncommunicable Diseases; 1998.

24. Flegal KM, Troiano RP. Changes in the distribution of body mass index of adults and children in the US population. *Int J Obes.* 2000;24:807–818.

25. Hoffmans MDAF, Kromhout D, Coulander CD. Body mass index at the age of 18 and its effects on 32 year mortality from coronary heart disease and cancer. *J Clin Epidemiol.* 1989;42:513–520.

26. Paffenbarger RS, Wolf PA, Notkin J, Thorne MC. Chronic disease in former college students: early precursors of fatal coronary heart disease. *Am J Epidemiol.* 1966;83:314–328.

27. Sorensen TIA, Sonne-Holm S. Mortality in extremely overweight young men. *J Chronic Dis.* 1977;30:359–367.

28. Freedman DS, Serdula MK, Kettel Khan L. The adult health consequences of childhood obesity. In: Chen C, Dietz WH, eds. *Obesity in Childhood and Adolescence*. Philadelphia: Lippincott, Williams & Wilkins; 2002:63–82.