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Risks and consequences of childhood and adolescent obesity

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This report reviews the risks and consequences associated with childhood and adolescent obesity. Although no consensus definition of childhood obesity exists, the various measures encountered in the literature are moderately well correlated. The paper is organized in three parts. The first section reviews childhood obesity sequelae that occur during childhood. These short-term risks, for orthopedic, neurological, pulmonary, gasteroenterological, and endocrine conditions, although largely limited to severely overweight children, are becoming more common as the prevalence of severe overweight rises. The social burden of pediatric obesity, especially during middle childhood and adolescence, may have lasting effects on self-esteem, body image and economic mobility. The second section examines the intermediate consequences, such as the development of cardiovascular risk factors and persistence of obesity into adulthood. These mid-range effects of early obesity presage later adult disease and premature mortality. In the final section, the small body of research on the long-term morbidity and mortality associated with childhood obesity is reviewed. These studies suggest that risk of cardiovascular disease and all-cause mortality is elevated among those who were overweight during childhood. The high prevalence and dramatic secular trend toward increasing childhood obesity suggest that without aggressive approaches to prevention and treatment, the attendant health and social consequences will be both substantial and long-lasting.

Keywords: childhood obesity; adolescent obesity; risk factors; persistence; morbidity

Introduction

With the recently documented increases in prevalence, pediatric obesity now represents one of the most pressing nutritional problems facing children in the United States today.[1] International population studies report comparable rates of increase, so that if current trends remain unchecked, childhood obesity is likely to challenge worldwide public health.[2] Substantial consequences to physical and mental health, both short and long term, must be anticipated. In this paper we review the many health consequences of pediatric obesity. Organized by time frame, we will consider 1) immediate physical and social sequelae associated with pediatric obesity, 2) intermediate consequences, including cardiovascular risk factor levels and persistence of childhood obesity into adulthood, and 3) long term consequences of pediatric obesity.

Unfortunately, there is no uniform definition of childhood obesity. Childhood obesity has been variously described by absolute weight, triceps skinfolds, weight-for-height percentiles, percent of ideal body weight and, most recently, by body mass index (BMI = weight in kg/height in meters^2). Although these measures reflect slightly different aspects of body composition or body size, they are moderately well correlated with body fat, even in growing children.[3] In addition to the lack of consistency in the anthropometric measure, the degree of obesity varies from study to study; however, for the majority of studies, a BMI greater than either the 85th or 95th percentile, or a weight-for-height greater than 120% of ideal was considered obese.

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Immediate consequences

Although for most children, complications of childhood obesity do not become apparent for decades, the metabolic consequences of obesity may be already evident in young children. Even a young child, if severely obese, can suffer serious morbidity. Prior to adulthood, the obese child may develop gallstones, hepatitis, sleep apnea and increased intracranial pressure. In fact, there are few organ systems that obesity does not affect in childhood. Of equal concern are the teasing, discrimination and vitimization of obese children.

Many of the studies reviewed below are based on samples drawn from pediatric specialty clinics. These children may be more likely to have health problems than overweight children who are not attending these clinics. Therefore, the risk estimates reported may not

reflect the risk that one would observe with a population-based approach. In addition, many of the studies are small, with risk estimates based on a relatively small number of cases. Nonetheless, collectively, these studies indicate a substantial burden of disease associated with early obesity.

Orthopedic

The presence of unfused growth plates and softer, cartilagenous bones of children, contributes to the occurrence of orthopedic abnormalities in obese children. Permanent damage to the femoral head may occur when dislocation occurs at the femoral growth plate. The incidence of slipped capital epiphyses is approximately 3.4 per 100 000 children.[4] Between 50% and 70% of patients with slipped capital epiphyses are obese,[5,6] and approximately two-thirds of patients with bilateral slipped capital epiphyses are obese.[7] Furthermore, slipped capital ephiphyses occur at significantly younger ages among obese children than among non-obese children.[7] Blount's disease (*tibia vara*) involves bowing of the legs and tibial tortion in response to unequal or early excess weight bearing. It is often progressive and recurrent requiring multiple osteotomies. Severe infantile Blount's disease appears associated with obesity in children. Approximately 80% of the children reported by Dietz *et al*[8] with Blout's disease were obese.

Neurological

Idiopathic increased intracranial hypertension (for example, *pseudotumor cerebri*) usually presents with headaches, vomiting, blurred vision or diploplia. It has been postulated that increased intra-abdominal pressure, due to obesity, causes increased pleural pressure and cardiac filling pressure which, in turn, results in increased resistance to venous return from the brain.[9] *Pseudotumor cerebri* most often occurs in females in the third decade. However, a review of 57 patients with pseudotumor revealed that approximately 15% presented before the age of 20 y, and approximately 90% of patients were obese.[10] Epidemiological studies indicate a 14-fold increase in the presence of *pseudotumor cerebri* in patients with weights $>10\%$ above the ideal, and a 20-fold increase in prevalence in people with weights 20% greater than ideal.[11] Conversely, obesity occurs in $30-80\%$ of children with pseudotumor, and it also accounts for the vast majority of cases not associated with infection, medication or underlying disease.[12]

Pulmonary

Among enrollees in a hospital-based weight control program, approximately 30% of the obese children suffered from asthma.[13] However, other studies suggest that the relationship between obesity and asthma is much weaker.[14] In one study of both obese and non-obese children $>80\%$ of obese children had a decrease of at least 15% in performance with exercise on one of the standard pulmonary function tests, compared to only 40% of the non-obese children.[15] Increased bronchial hyperactivity may contribute to both higher rates of reactive airway disease and decreased exercise tolerance in obese children.

Sleep disorders represent another pulmonary consequence of childhood obesity. A study of 41 children with severe obesity revealed that one-third of patients reported symptoms consistent with sleep apnea, and approximately one-third of patients had abnormal sleep studies. Two patients (5%) had evidence of severe sleep apnea.[16] Similar findings have been reported by Marcus *et al*.[17] In contrast, Silvesti *et al*[18] reported abnormal sleep patterns for 94% of the obese children they studied. Children whose weight was $>200\%$ above the ideal, had severely abnormal sleep patterns; oxygen saturation was $<90\%$ for approximately half of the total sleep time and 40% of the severely obese children demonstrated central hypoventilation. Of particular concern, are preliminary results reported by Rhodes *et al*,[19] which indicates that obese children with obstructive sleep apnea, demonstrate clinically significant decrements in learning and memory function compared to obese children without obstructive sleep apnea.

The Pickwickian syndrome refers to severe obesity associated with hypoventilation, somnolence, polycythemia and right ventricular hypertrophy and failure. Large amounts of abdominal fat leads to a rapid, shallow breathing pattern with subsequent increase in dead space ventilation. Central hypoventilation and obstruction from a lax laryngeal wall during sleep also result in severe hypercarbia and hypoxia. The prevalence of the Pickwickian syndrome in children is unknown; however, the obesity-hypoventilation syndrome is associated with pulmonary embolism and sudden death in children.[20]

Gastroenterological

In obese individuals, biliary excretion of cholesterol is increased, relative to bile acid and phospholipid secretion, resulting in increased likelihood of gallstone formation.[21] The development of gallstones and lithogenic bile has been studied prospectively in female Pima Indians living in the southwestern US, a population with almost uniform obesity. After the age of 13 y, bile becomes supersaturated with cholesterol, particularly in females.[22] The prevalence of gallstones in this population rises sharply beginning at the age of 17 y to approximately 70% at the age of 30 y.[23] In adult women, obesity is associated with a two-fold risk of symptomatic and asymptomatic gallstones.[24] In children, underlying medical conditions such as hemolytic disease, congenital heart disease, or prolonged nutritional support are most often associated with cholelithiasis. Nonetheless, obesity accounts for $8-33\%$ of the gallstones observed in children.[25,26] Furthermore, childhood obesity accounts for the majority of gallstones in children without underlying

medical conditions. Honore[27] has estimated that the relative risk of gallstones in adolescent girls with obesity (weight > 110% of the ideal) compared to those of normal weight, has been estimated at approximately 4.2.

Increased rates of lipolysis in obese individuals, as well as insulin resistance, combine to lead to liver steatosis.[28] Between 20–25% of obese children demonstrate evidence of steatohepatitis, based on either ultrasound or on increased transaminases.[29–31] Between 40–50% of children with severe obesity show laboratory evidence of steatohepatitis.[32] In the most severe cases, steatohepatitis in children is associated with liver fibrosis and cirrhosis.[30,32] Degree of obesity, duration and male gender are associated with the progression of fatty liver to fibrosis.[30,33]

Endocrine

Childhood obesity is associated with insulin resistance and hyperandrogenemia, particularly among adolescent females.[33] However, significant insulin resistance has been noted in children aged < 10 y. Euglycemic clamp studies indicate a 20–45% reduction of insulin-stimulated glucose uptake in prepubertal overweight children.[34] Insulin resistance is associated with higher levels of total cholesterol, low density lipoprotein (LDL) cholesterol, and triglycerides in obese children.[35–37] The development of severe insulin resistance is also associated with the increased prevalence of non-insulin dependent diabetes mellitus (NIDDM) in obese children. In the Bogalusa Heart Study[38] population, 2.4% of the overweight adolescents (BMI > 75th percentile) developed NIDDM by the age of 30 y compared to none of the lean adolescents. In the greater Cincinnati area, the prevalence of NIDDM among children increased 10-fold between 1982 and 1994 (from 0.7/100 000 to 7.2/100 000); with black youth experiencing a disproportionate number of cases.[39] In this study, one-third (19 of 58) of new diabetics in 1994 between the ages of 10–19 y had NIDDM. Furthermore, over 90% of new patients with NIDDM had a BMI > the 90th percentile, and approximately 40% had a BMI > 40 kg/m^2 (severe obesity). Symptoms at the time of diagnosis only rarely (20%) included the classic triad of polyuria, polydipsia and weight loss. Instead, the majority of patients were either asymptomatic or presented with unrelated symptoms such as vaginal monilial infection.

Menstrual abnormalities in obese children are also common. One hypothesis suggests that the levels of body weight and fatness are the critical physiologic triggers of menarche.[40] And in fact, obese girls are observed to experience earlier menarche, typically before the age of 10 y.[41] Late or absent menstruation is also associated with obesity. Oligomnorrhea or amenorrhea associated with obesity, insulin resistance, hirsuitism, acne and acanthosis nigricans constitutes the 'polycystic ovary syndrome.' Approximately 40–60% of adult women with polysystic ovary syndrome are overweight or obese.[42–44] The prevalence of polycystic ovary syndrome in adolescents is unknown, probably because children rarely have the characteristic laparoscopic or ultrasonic ovarian morphology and because altered menstrual patterns or late menstruation may go unrecognized. However, hormonal patterns typical of polycystic ovary syndrome are increasingly described in obese children.[33,45]

Social and economic consequences of childhood obesity

Few problems in childhood may have as significant an impact on childhood emotional development as obesity. Studies of children as young as six years of age suggest that overweight individuals are most likely to be described as lazy, lying, cheating, sloppy, dirty, ugly and stupid.[46,47] Landmark studies conducted in the 1960s also demonstrate that obese children are uniformly ranked by other children as the least desired friends.[48] A study reported 30 years ago, found that even physicians describe obese individuals as weak willed, ugly and awkward.[49]

Middle childhood is a critical period for the development of body image and self-esteem. Monello and Mayer[50] have observed that obese girls often have obsessive concern with body image as well as expectation of rejection and progressive withdrawal. Studies by Stunkard *et al*[51] suggest that women who became obese as children are likely to have persistent severe disturbances in body image. In contrast, adult women who became obese as adults demonstrate only minimal alterations in body image.

Although some obese children appear to have low self-esteem, the actual prevalence of this problem is controversial. Studies by Allon,[52] Sallade[53] and Strauss *et al*,[54] indicate decreased levels of self-esteem in obese children as a group. However, other studies report normal levels of self-esteem.[55,56] Differences in age, race and socioeconomic status (SES) of the subjects studied, may account for the discrepant findings. For instance, low self-esteem in childhood is not a characteristic of obese minorities.[57] In the only longitudinal study of self-esteem among obese children, Klesges *et al*[58] demonstrated that over a two-year period, self-esteem decreased as body fat increased.

'Fear of fatness' has become a common condition that affects both normal weight and overweight girls. Girls as young as 5 y express fear of gaining weight.[59] Nearly 70% of adolescent girls have attempted to lose weight[60] and up to 50% of third grade girls have already attempted dieting.[61,62] A study of 270 adolescents showed that almost half of the girls believed they were too fat, when in fact, 83% of those who thought they were too fat were of normal weight-for-height.[63] A recent study has found that dieting and weight concerns are influenced more by body frame and body structure than the degree of fatness.[64]

Fear of fatness among adolescent girls can also be associated with growth failure and delayed puberty resulting from severe caloric restriction.[65] A prospective study of London schoolchildren, indicates that dieters are eight times more likely to develop an eating disorder than non-dieter.[66] Data from the Oxford Community Study, indicate that 40% of bulimic adults had a history of childhood obesith in comparison to 15% of the normal controls.[67] Of substantial concern, is the finding that approximately 20% of girls report smoking as means of controlling their weight.[68]

Finally, Goldblatt *et al* [69] have documented that obese women are twice as likely to experience downward social and economic mobility as upward mobilit. Discrimination against overweight adolescents, as well as low self-esteem and low confidence, may contribute to the lower academic achievement observed among obese adolescents. Uncontrolled studies have also documented lower rates of high school performance in overweight adolescents.[70] In addition, overweight adolescents have approximately one-half of college acceptance rates to elite universities as normal weight adolescents with similar achievement scores.[71] Gortmaker *et al*[72] analyzed data from the National Longitudinal Survey of Youth of 10 039 nationally representative adolescents and young adults (age 16–24 y) who were prospectively followed over a seven year period. Women who were initially overweight (>95th percentile BMI) completed 0.3 y less schooling, had lower household incomes ($6710 less annually), and had higher rates poverty (10% higher) as those who were initially of normal weight. Similar findings were observed among males, but the impact of overweight was smaller. These differentials remained after adjusting for baseline family income, education, ethinicity and self-esteem.

Intermediate consequences

In the following section, we review the evidence for the association of childhood obesity with cardiovascular disease (CVD) risk factor levels and with persistence into adulthood. CVD risk factor levels are considered mid-term consequences, because they do not represent morbidity *per se*, but rather the increased likelihood of the development of CVD. Similarly, the association of childhood obesity with adult obesity, foretells an increased likelihood of obesity-related morbidity in adulthood.

Cardiovascular disease risk factors

Approximately 20–30% of obese children (weight >120% ideal) between the ages of 5–11 y have elevated systolic or diastolic blood pressure.[73] Prospective data from the Muscatine Study[75] show that obese boys and obese girls (BMI > 90th percentile) are 9–10-fold more likely to develop high blood pressure as young adults than non-obese children. Similarly, data from the Bogalusa Heart Study[76] indicate that overweight adolescents (BMI > 75th percentile) are 8.5-fold more likely to have hypertension as adults than lean adolescents. In addition, hypertensive children who continue to have high blood pressure into adulthood are more likely to have greater body weight, BMI, skinfold thickness and substantially greater waist and hip circumferences.[77] Hanis *et al* [78] demonstrated that familial aggregation of hypertension can largely be explained by the similarities in body weight between family members. The mechanism by which obesity contributes to hypertension appears to be *via* hyperinsulinaemia, activation of the sympathetic nervous system and activation of the renin-angiotension system. All of these metabolic responses result in enhanced renal absorption of sodium and decreased natriuresis.[79] Therefore, blood pressure in obese adolescents appears to be particularly sensitive to sodium intake. When both obese and non-obese adolescents were shifted from a high salt to a low salt diet, a significantly larger decreased in blood pressure was observed among obese (mean: -12 ± 1 mm Hg) compared to a non-significant change among non-obese (mean: $+1 \pm 2$ mm Hg) adolescents.[80]

Adolescent obesity, particularly in males, is associated with deleterious effects upon total cholesterol and LDL-cholesterol in adulthood.[81] The association of high cholesterol and abnormal lipoprotein levels, with the degree of childhood obesity, is consistently stronger among males.[82,83] Longitudinal follow-up of 1598 children adtged 12 y reveals that increased triceps skinfold thickness during adolescence, was highly correlated with increased levels of LDL-cholesterol and triglycerides, and significantly decreased concentrations of serum high density lipoprotein (HDL).[84] In the Bogalusa Heart Study, overweight during adolescence (BMI > 75th percentile) was associated with a 2.4-fold increase in the prevalence of total cholesterol values above 240 mg/dl, a 3-fold increase in LDL values above 160 mg/dl, and an 8-fold increase in HDL levels below 35 mg/dl in adults aged 27–31 y.[38]

Persistence

The persistance of obesity present in childhood or adolescence into adulthood represents another significant consequence of early obesity, inasmuch as adult obesity is an established independent risk factor for CVD, NIDDM, hyperlipidaemia, gall bladder disease, osteoarthritis and certain cancers.[85] Persistance has been consistently demonstrated in all populations in which it has been studied.[86–92] These studies indicate that the likelihood of persistance is related both to severity of obesity and to the age during childhood at

which it is present. In general, the likelihood of persistence from childhood to adulthood is moderate. Viewed from the perspective of childhood, 25–50% of individuals who are obese in adolescence remain obese in adulthood.[89,91] That means that less than half of children who were obese during childhood went on to become obese adults, even though the risk of adult obesity was 2–11 times higher for obese compared to non-obese children. From the adult vantage point, only 17–18% of 33-year old obese adults had been obese in childhood.[92]

Although the simple observation that obesity persists from childhood into adulthood is consistent among the studies referenced, obesity criteria (both the indicator used and the definition applied) vary, as do the statistics used to evaluate the degree of persistence. The interpretation and summarization of these studies is further complicated by the large variability in the time intervals between the childhood and adult measurements. Intervals over which persistence is examined range from 1–16 y for the childhood measures to 18–47 y for the adult measures. In most of the studies, the interval from childhood to adulthood is only about 10 y. A prospective study with long follow-up, found that children aged 18 y who were $\geq$ the 95th percentile BMI, were at substantially increased risk of overweight at the age of 35 y. Probabilities of adult overweight were estimated at 78% and 66%, respectively, for male and female adolescents.[88]

Gender differences in the degree of persistence were apparent in several[87,89] but not all[88,90,92] of the larger studies. In studies that found differences by gender, females evidenced a greater likelihood of persistence. Using an internally referenced definition of overweight ($>$130%), a follow-up of the National Survey of Health and Development (the British 1946 birth cohort), females who were overweight in adolescence were more likely to have the condition continue into adulthood than were males. Among individuals who were obese at the age of 36 y, 11% males and 26% females were obese at the age of 20 y, and 14% of males and 32% of females were obese at the age of 14 y. However, in a report from the later 1958 British birth cohort, persistence was similar for males and females. Of obese men and women aged 33 y, 17% and 18% respectively, had been $>$95th percentile BMI at the age of 7 y.[90] Whether this difference in findings from the two British cohorts represents a true difference or results from the differing overweight criterion applied, can not be determined. Among 181 Harvard Growth Study participants we studied over 55 years from adolescence to old age, of those who were overweight as adolescents ($>$75th percentile BMI for at least two years), 46% of females and 52% of males were overweight at mid-life (mean age 52 y), and 53% of females and 41% of males were overweight at mean age 73 y (Must, unpublished observations).

The later into adolescence overweight persists and the more severe the obesity, the greater the likelihood of persistance.[87,89,91] The disturbing secular trends seen in the United States and globally, have important implications for the likelihood of persistence. The weight distribution has shifted to the right and is increasingly right-skewed. Thus, there are not only more obese children and adolescent, but more very obese children. This observation suggests that the likelihood of persistence from childhood into adulthood will increase in the future.

Long-term consequences

The prevalence trends in pediatric overweight have appropriately focused attention on the associated long-term consequence to adult morbidity and mortality. The study of remote aftereffects is difficult. Ideally, such investigation requires longitudinally measures of adiposity and continues follow-up to obtain information on incident morbidity and, ultimately, on cause of death. To study any but the most common outcomes requires large sample sizes. The expense of such studies, in terms of both time and personnel, has made the ideal study design prohibitive. Instead this sparse literature is largely characterized by retrospective cohort studies that take advantage of existing records collected for other purposes, such as military registration, college physicals, or as part of school-based or national growth studies. We will consider the long-term consequences to morbidity and mortality separately.

Adult morbidity

Only three studies have considered the long-term impact of childhood obesity on adult morbidity. They seem to indicate that, risk of morbidity is elevated in relation to overweight in childhood, although results are far from conclusive. In the next few decades, with adequate research funding, this literature may expand substantially, as some of the large childhood and adolescent cohort studies established in the last two decades reach middle-age.

School-based growth records collected in Washington County, Maryland between 1923–1928 were used to classify the weight status of boys aged 9–13 y. Of those traced 42% were examined by county health departments clinicians after 37 y. Concentrations of serum cholesterol, blood pressure and fasting blood sugar, did not differ by childhood weight status. Morbidity from hypertensive vascular disease and cardiovascular renal disease, was elevated for males who became overweight in adulthood, but not in relation to childhood weight status.[93] Unfortunately, the data were not anlayzed with multivariate approaches which would permit the evaluation of the relative importance of weight status during childhood and adulthood. Additionally, the small pro-

portion of subjects for whom outcome data were available, may limit the generalizability of these findings.

Morbid consequences of adolescent overweight were studied in a second school-based retrospective study, the Third Harvard Growth Study of 1922–1935.[94] To form two weight status groups 508 subjects were selected: those who had been overweight during adolescence and those who were lean. Medical histories from interviews conducted among 181 of the original study participants who survived to the age of 73 y indicated that morbidity was increased for those who had been overweight during adolescence. Specifically, risk of heart disease and atherosclerosis was elevated for males and females, risk of colon cancer and gout was elevated for males, and risk of arthritis, hip fracture and for difficulty with activities of daily living (reflective of functional capacity) was elevated for females. Furthermore, the elevation in the risk of morbidity in relation to adolescence overweight was independent of adult weight status. It is noteworthy that the apparent elevation in risk of diabetes mellitus in relation to adolescent overweight was accounted for entirely, by adult weight status.

The impact of extremes of body weight in relation to female reproductive health was evaluated prospectively in the 1958 British birth cohort study.[95] Menarche before the age of 12 y was associated with mild and severe menstrual problems at the age of 16 y. Obesity at the age of 7 y and at the age of 23 y was associated with menstrual problems and gestational hypertension by the age of 33 y. Obesity at the age of 23 y (but not at the age of 7 y) was also associated with subfertility. Unfortunately, reproductive health outcomes were not evaluated in relation to overweight during adolescence.

Adult mortality

As a measure of disease risk, mortality represents a useful endpoint for long-term study, because death certificates provide a practical way to obtain mortality data on a large number of subjects. However, mortality is an imperfect measure of disease risk for non-fatal diseases or conditions (such as arthritis or reproductive outcomes). Some additional limitations include cause of death misclassification from ICD codes[96] and regional or temporal variability in disease classification. Nonetheless, the studies of mortality in relation to early overweight show a consistent elevation in risk, despite the wide range of populations studied, both in geography and in the populations selected for study.

Hoffmans *et al* [97] examined all-cause and coronary heart disease mortality among Dutch men followed for 32 years. Elevated adolescent BMI ($>25\,kg/m^2$ compared to $19\,kg/m^2$) based on height and weight recorded at military registration, was associated with a relative risk 1.5 for all-cause and 2.5 for coronary heart disease mortality. Cancer mortality was not related to increased BMI at the age of 18 y. The study emphasizes the importance of adequate follow-up: the increased mortality was not evident until follow-up was of at least 20 years duration.

Paffenbarger's studies of college alumni presented an opportunity to examine coronary heart disease mortality among men whose heights and weights were recorded as part of college physicals. Using an older height-weight ratio to define obesity (ponderal index <12.9), a relative risk of 1.3 was observed for coronary heart disease (CHD) mortality. Important effect modification by age group was evident: the relative risks were highest (1.7) for the men aged 30–44 y, and dropped to a relative risk of 1.0 (no elevation in risk) for men aged 55–69 y. Furthermore, the effects of obesity were also potentiated by elevated systolic blood pressure and by cigarette smoking.[98]

As part of a nation-wide screening for tuberculosis in 1963, the entire population of Norway (except for two counties) over the age of 15 y were studied and subsequently their 10-year mortality experience was observed. Ten-year mortality from all-causes was elevated for both males and females initially aged 20–24 y.[99] Unpublished observations for 15–19 year olds show an elevation of 10-year mortality for males with BMI >27, but not for females (H. Waaler, personal communication, 1991).

Childhood growth data from the Carnegie (Boyd Orr) Survey of Diet and Health in Pre-War Britain (1937–1939) were used to evaluate the relationship between overweight during childhood (2–14 y) and subsequent mortality from all causes, CVD, CHD and stroke among 2399 cohort members.[100] This investigation was novel in that the data collected during childhood included several SES measures. The childhood cohort was young: the median age was 7 y, with fewer than 9% of subjects aged 13 y or 14 y. The relative risk for all-cause mortality and for mortality from coronary heart disease was 1.5 and 2.0, respectively, when male and female subjects with childhood BMIs between the 25–29th percentile were compared to those with BMIs greater than the 75th percentile. There was a suggestion of increased risk for CVD, but no evidence of any elevation in the risk of death from stroke. Remarkably, the inclusion of measures of SES during childhood had virtually no effect on the estimates.

Retrospective follow-up of a school-based study of childhood growth in Washington County, MD conducted between 1933–1945 evaluted prepubertal and postpubertal overweight in relation to mortality from all causes.[101] The index of obesity used was an internally derived relative weight z-scores. For children in the top quintile of relative weight compared to the bottom quintile, the relative risk of mortality was 1.5 for children prepubertally and 1.6 postpubertally. Results were quite consistent for males and females, with the exception of postpubertal males, where there did not appear to be a significant elevation in risk associated with overweight. Unfortunately, the degree of overweight evaluated in this study is unclear, due to the use of an internally derived z-score.

Growth records from the Third Harvard Growth Study of 1922–1934 were also used to assess the impact of adolescent obesity on all-cause and cause specific mortality.[94] The longitudinal data were used to define adolescent obesity as BMI in excess of the 75th percentile for at least two years between the ages of 13–17 y. The elevated mortality risk observed was limited to males. The relative risk of all-cause mortaility and CHD mortality were 1.8 and 2.3, respectively, for males overweight during adolescence compared to males who were lean. Examination of the survival curves suggests that the differences in risk are due to differential survival during the fifth decade of life. The relative risk of colon cancer and of atherosclerotic cerebrovascular disease were also significantly elevated, but the estimates are very unstable. The investigation is unique in its ability to assess the influence of adult weight status on these estimates. Adjustment for all adult BMI on the subset for which adult values were available did not appreciably change the observed risks. The relative risks of all-cause and coronary heart disease mortality remained elevated in relation to adolescent weight status, even after statistical control for mid-life weight. Furthermore, accounting for smoking during adulthood did not alter the relative estimates.

The six studies reviewed, represent the body of research relating childhood obesity to mortality. Despite varying definitions of obesity and childhood ages studied, the results of these studies are remarkably consistent, Furthermore, the findings emphasize the importance of adequate length of follow-up. It appears that all-cause mortaility and CHD mortality are significantly elevated in relation to overweight during childhood. Relative risk estimates of about 1.5 for all-cause mortaility and 2.0 for CHD mortality, coupled with an increasingly high prevalence of obesity in the pediatric population, suggest that premature mortality from these causes can be expected to increase in the future. Moreover, risk appears to be elevated independently of some of the factors thought to mediate weight/mortality relationships, such as adult weight status, smoking and SES. Much additional work in this nascent area is needed to corroborate these early findings.

Conclusion

As indicated by the preceding review, the health and social consequences of childhood obesity are substantial. The short-term risks are largely confined to more severely overweight children and adolescents. However, from about 1980–1990, the number of children with BMI in excess of the 95th percentile rose substantially, from approximately 7.5–10.7% for children aged 6–11 y and from 5.7–10.8% for teens aged 12–17 y.[1] Therefore, we can anticipate that the once rare orthopedic, endocrinal, gastroenterological, pulmonary and neurological consequences will become far more commonplace. The findings that one-third of the newly diagnosed diabetics aged between 10–19 y, were type II diabetics, is particularly ominous. This condition, once coined 'adult-onset diabetes' may become a prevalent chronic disease of adolescence. The long-term consequences are also of significant public health importance. Relative risks of 2.0 for relatively common outcomes, such as CHD morbidity, or CHD or all-cause mortality, are of great concern.

Thus, treatment and prevention of obesity in childhood are each essential. Unfortunately, both remain problematic. The long-term success reported by Epstein *et al* [102,103] is promising, but has not yet been duplicated. Such treatment approaches, if confirmed, require intensive treatment and close patient follow-up. In addition, population-based programs which have focused on school-based interventions in older children have produced only minimal changes in weight or BMI. In particular, the Child and Adolescent Trial for Cardiovascular Health (CATCH), a recently completed multi-center study involving approximately 5000 students aged 8 y and 9 y and their parents, failed to demonstrate significant differences in students' weight, skinfold thicknesses, BMI, cholesterol or blood pressure, between the intervention and control schools at the three-year follow-up.[105,106] As reviewed elsewhere in this issue,[107] school-based approaches offer several theoretical advantages compared to other primary and secondary prevention approaches. Clearly, in the face of increasing numbers of overweight children and mounting evidence of the substantial health consequences, both near and long-term, further research in design and implementation strategies for successful prevention and treatment of childhood obesity is essential.

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