PEDIATRIC REVIEW

Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review

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Background and objective: The last systematic review on the health consequences of child and adolescent obesity found little evidence on consequences for adult health. The present study aimed to summarize evidence on the long-term impact of child and adolescent obesity for premature mortality and physical morbidity in adulthood.

Methods: Systematic review with evidence searched from January 2002 to June 2010. Studies were included if they contained a measure of overweight and/or obesity between birth and 18 years (exposure measure) and premature mortality and physical morbidity (outcome) in adulthood.

Results: Five eligible studies examined associations between overweight and/or obesity, and premature mortality: 4/5 found significantly increased risk of premature mortality with child and adolescent overweight or obesity. All 11 studies with cardiometabolic morbidity as outcomes reported that overweight and obesity were associated with significantly increased risk of later cardiometabolic morbidity (diabetes, hypertension, ischaemic heart disease, and stroke) in adult life, with hazard ratios ranging from 1.1–5.1. Nine studies examined associations of child or adolescent overweight and obesity with other adult morbidity: studies of cancer morbidity were inconsistent; child and adolescent overweight and obesity were associated with significantly increased risk of later disability pension, asthma, and polycystic ovary syndrome symptoms.

Conclusions: A relatively large and fairly consistent body of evidence now demonstrates that overweight and obesity in childhood and adolescence have adverse consequences on premature mortality and physical morbidity in adulthood.

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Keywords: overweight; child; BMI; morbidity

Introduction

Child and adolescent obesity remain prevalent across the developed world, and prevalence is still increasing in most developing countries.1,2 Childhood and adolescent obesity have a number of adverse consequences for physical and mental health, in both the short term (for the obese child or adolescent) and long term (for the adult obese as a child).3

The short- and long-term physical and psychosocial comorbidities of child and adolescent obesity have been reviewed systematically,3 but the last systematic review of this topic was published in 2003 and it found very little evidence on the long-term health impact of obesity during childhood and adolescence, other than strong evidence of a tendency for obesity to persist.3 After the publication of this last systematic review,3 which searched the literature to the end of 2001, the amount of evidence on relationships between child and adolescent obesity and morbidity and premature mortality in adulthood has increased substantially.4 The aim of the present study was, therefore, to carry out a systematic review of the recent evidence (from 2002 to present) on associations between childhood and adolescent obesity and risk of both premature mortality and physical morbidity in adulthood.

Methods

Search strategy, inclusion and exclusion criteria
The present review aimed to include studies of associations between child and adolescent overweight and obesity (measured as an exposure between birth and 18 years) and...
adult physical morbidity and risk of premature mortality (outcomes). Studies that included only adult overweight and/or obesity as an outcome were excluded, as this topic was the subject of at least three earlier systematic reviews. Studies that were published before 2002 were also excluded because they had been considered in the previous systematic review of this topic.

The search strategy is described below:

1. ((child$ or adolescen$ or you$) adj3 (obes$ or overweight$ or bmi or adiposity$ or fat$ or weight$)).tw
2. limit/ to yr = ‘2002–2010’
3. (mortal$ or morbid$).tw
4. adult$.tw.
5. (heart$ or stroke$ or cancer or cardiovas$ or hypertens$).tw
6. or/3–5
7. 2 and 6

The search began in January 2002 and ended in mid June 2010, and the databases searched were Medline, Embase, and the Cochrane Register of Controlled Trials. All abstracts identified were considered for relevance by one author (JK), and full papers retrieved to consider eligibility. All eligible papers were searched manually for other potentially eligible papers. The eligible studies fell into three logical categories: studies of associations between child/adolescent overweight and obesity with premature mortality in adulthood; cardiometabolic morbidity in adulthood; other forms of morbidity in adulthood.

Studies that measured exposures other than child or adolescent overweight and obesity were excluded: a number of studies examined associations between child/adolescent BMI trajectory and adult premature mortality or physical morbidity. Studies that measured only adult cardiometabolic risk factors as outcomes—rather than cardiovascular morbidity or mortality—were also excluded. This meant excluding a number of studies on associations between child or adolescent obesity and variables, which are relevant to later morbidity and premature mortality, such as adult carotid intima media thickness11,12 and components of the metabolic syndrome in adult life.13–18 Studies that modelled the impact of child or adolescent obesity on later morbidity or mortality,19,20 or that examined healthcare expenditure as an outcome21 were of relevance, but were excluded as they went beyond the scope of the present review.

Results

Search results

The initial search produced 8535 ‘hits’, which were reduced to 200 potentially eligible abstracts by screening carried out by one author (JK). The study flow diagram is shown in Figure 1. A total of 8 papers, based on 5 cohort studies, of associations with premature mortality in adulthood were included; a total of 11 papers describing 11 studies of associations with cardiometabolic comorbidities of obesity in adulthood were included; a total of 9 papers described studies of associations with a wide variety of other outcomes (award of disability pension, cancers, asthma and atopy, polycystic ovary syndrome).

Childhood and adolescent overweight and obesity and premature mortality in adulthood

Evidence of associations between child or adolescent overweight and obesity, and premature mortality in adult life is summarized in Table 1. In four of the five cohorts (and 7/8 studies), obesity and/or overweight in childhood–adolescence were associated significantly with increased risk of premature mortality, with hazards ratios ranging from 1.4–2.9 (Table 1). These studies were all from the USA or Western Europe.

The cohort studies ranged in size from a sample of 4857 to 1 024 000, with median n 45 920. Seven of the eight studies (in four of the five cohorts) used definitions of overweight and/or obesity (exposure measures), which corresponded closely to the standard and widely-accepted definitions of overweight and obesity in use now, based on BMI for age. Two of the studies considered obesity as the exposure, two studies considered overweight, and the remaining two studies considered both overweight and obesity, and did so separately.
Engeland et al. measured. In the remaining study, the exposure measure based on BMI for age and sex, and in most studies, the disease, and hypertension. Hazard ratios ranged from 1.1 to increased risk of later diabetes, stroke, coronary heart overweight and obesity were associated significantly with Table 1. A total of 11 eligible studies were identified, and all reported that child/adolescent overweight and obesity and adult cardiometabolic morbidity

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample and setting</th>
<th>Exposure measure(s)</th>
<th>Outcome(s)</th>
<th>Main results</th>
</tr>
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<tbody>
<tr>
<td>Franks et al.</td>
<td>4857; USA; Born 1945–1984</td>
<td>≥ 95th percentile for BMI; vs &lt;95th percentile. Mean age 11 years</td>
<td>All-cause mortality; external mortality; endogenous mortality</td>
<td>Incidence rate ratio for premature mortality from endogenous causes 1.90 (95% CI 1.37–2.65) for BMI &gt;95th percentile at baseline</td>
</tr>
<tr>
<td>Neovius et al.</td>
<td>45 920; Sweden; Born 1949–1951</td>
<td>BMI &gt; 25 and &gt; 30 kg m⁻² vs 15.8–24.9</td>
<td>All-cause mortality</td>
<td>Hazard ratio 2.10 (95% CI 1.61–2.85) for obesity at age 18 years</td>
</tr>
<tr>
<td>Bjorge et al.</td>
<td>227 000; Norway; Born 1940–1960</td>
<td>BMI &gt; 58.5th centile vs 25–75th centiles; ages 14–19 years</td>
<td>Cause-specific mortality</td>
<td>Overweight associated with significant increase in variety of causes of premature mortality (RR 2.2–2.9)</td>
</tr>
<tr>
<td>Bjorge et al.</td>
<td>226 678; Norway; Born 1940–1960</td>
<td>BMI &gt; 58.5th centile vs 25–75th centiles at age 14–19 years</td>
<td>Cause-specific mortality</td>
<td>Overweight associated with significant increase in mortality from ischaemic heart disease, metabolic disease, respiratory disease, colon cancer</td>
</tr>
<tr>
<td>Van Dam et al.</td>
<td>524 000 women; USA; Born 1945–1965</td>
<td>Self-reported weight and height at age 18 years; BMI ≥ 25 and &gt; 30 kg m⁻² vs 18.5–21.9</td>
<td>All-cause mortality</td>
<td>Overweight and obesity both significantly associated with all-cause mortality Hazard ratio for BMI &gt; 30 was 2.79 (95% CI 3.04–3.81)</td>
</tr>
<tr>
<td>Engeland et al.</td>
<td>128 121; Norway; Born 1948–1968</td>
<td>BMI &gt; 58.5th centile vs 25–75th centiles; at age 14–19 years; mean 9.7 years follow-up</td>
<td>All-cause mortality</td>
<td>Significant increase in all-cause mortality with overweight, RR 1.4 (95% CI 1.0–1.8)</td>
</tr>
<tr>
<td>Engeland et al.</td>
<td>227 003; Norway; Born 1948–1968</td>
<td>BMI &gt; 58.5th centile at age 14–19 years vs 25th–75th centiles mean 31.5 years follow-up</td>
<td>All-cause mortality</td>
<td>Significant increase in all-cause mortality with obesity, RR 1.82 (95% CI 1.48–2.43) in men; RR 2.03 (95% CI 1.51–2.72) in women</td>
</tr>
<tr>
<td>Ferraro et al.</td>
<td>6497; USA; Born 1935–1945</td>
<td>Self-reported perceived ‘overweight’ vs normal weight at age 12 years</td>
<td>All-cause mortality</td>
<td>Significant reduction in all-cause mortality with ‘overweight’ at 12y, RR 0.68 (95% CI 0.59–0.80)</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; CI, confidence interval; RR, relative risk. *Several studies of cohort reported in Bjorge et al.*, with all-cause mortality and cause-specific mortality as the outcome and with varying durations of follow-up.

In the only study that did not find that childhood overweight and obesity was related to increased adult mortality, the exposure measure was recalled perceived overweight at age 12–13 years, rather than objectively measured overweight or obesity. In most of the studies participants were born between the 1940 and 1960 (Table 1). Two studies that were ineligible because the exposure measure of overweight/obesity was made in early adulthood, when participants were (median) age 22 years, found that early adult overweight and obesity increased risk of subsequent cardiovascular and cancer mortality.

**Overweight and obesity in childhood and adolescence and risk of adult cardiometabolic morbidity**

Evidence of associations between child and adolescent overweight and obesity and adult cardiometabolic morbidity is summarized in Table 2. A total of 11 eligible studies were identified, and all reported that child/adolescent overweight and obesity were associated significantly with increased risk of later diabetes, stroke, coronary heart disease, and hypertension. Hazard ratios ranged from 1.1 to 5.1 (Table 2). Exposure variables in 10 of the 11 eligible studies were based on BMI for age and sex, and in most studies, the exposures were definitions of either overweight or obesity, which are widely used and accepted today, though in one study, weights and heights were recalled rather than measured. In the remaining study, the exposure measure was perceived/self-reported ‘overweight’ in childhood. All studies were from the USA or Western Europe. The studies ranged in size from an n of 130 to 780 694, with a median of 6253 (Table 2). Year of birth of the study participants in the eligible studies ranged from the 1940’s to 1980’s.

**Overweight and obesity in childhood and adolescence and risk of subsequent miscellaneous morbidity**

Nine studies were found (Table 3) with a variety of outcome measures of physical morbidity (risk of cancer in five studies, disability pension in two, asthma and atopy in one, polycystic ovary syndrome in one). Both studies that used disability pension as the outcome found that late adolescent obesity was associated with significantly increased risk of disability pension being awarded in adult life. The single study of asthma and atopy, and the study of polycystic ovary syndrome found that obesity at age 14 years was associated with significantly increased risk of asthma and polycystic ovary syndrome symptoms in adult life.

Baer et al. found a significant decrease in pre-menopausal breast cancer risk associated with higher recalled/perceived weight status in childhood and adolescence, and suggested that this was biologically plausible. Swerdlow et al. found that lower (recalled) weight status at age 11 years was associated with significantly increased risk of pre-menopausal breast cancer, and Bardia et al. reported that recalled/perceived high-weight status at age 12 years was associated significantly with lower risk of post-menopausal breast
Table 2  Associations between child and adolescent overweight and obesity and cardiometabolic morbidity in adulthood

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample and setting</th>
<th>Exposure measure(s)</th>
<th>Outcome(s)</th>
<th>Main results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mamun et al. 35</td>
<td>2639; Australia; born 1981–1983</td>
<td>BMI at age 5 years, International obesity task force defined overweight and obese vs non-overweight non-obese</td>
<td>Self-reported diabetes at age 21 years</td>
<td>Significant increase in diabetes risk with overweight and obesity at age 5 years; AOR 2.6 (95% CI 1.3–5.2)</td>
</tr>
<tr>
<td>Narayan et al. 36</td>
<td>780 694; USA; cross-sectional data in 2004</td>
<td>BMI at age 18 years, defined as overweight (&gt; 25) and obese (&gt; 30) vs normal weight (BMI 18.5–25) BMI z-score quintiles at age 5 years,</td>
<td>Lifetime risk of self-reported diabetes at age 18 years</td>
<td>Significantly increased lifetime risk of diabetes associated with obesity at age 18 years (37% increased risk)</td>
</tr>
<tr>
<td>Lawlor et al. 37</td>
<td>5 793; Scotland; born 1950–1956</td>
<td>BMI z-score at age 7–13 years, treated as a continuous variable</td>
<td>CHD</td>
<td>Significant increase in risk of adult diabetes with higher-BMI z-score at age 5 years</td>
</tr>
<tr>
<td>Baker et al. 38</td>
<td>276 825; Denmark; born 1930–1976</td>
<td>BMI ≥ 30 vs 18.5–20.99 kg m⁻²; at age 18 years</td>
<td>CHD and stroke hospitalisation</td>
<td>Significant increase in CHD and stroke risk with obesity. Hazard ratio for CHD 4.3 (95% CI 3.1–5.9)</td>
</tr>
<tr>
<td>Falkstedt et al. 39</td>
<td>49 321; Sweden; men born 1949–1951</td>
<td>Self-reported BMI at age 15 years; International obesity task force defined overweight and obese vs non-overweight plus non-obese</td>
<td>Self-reported hypertension in early adulthood</td>
<td>Significant increase in later hypertension with overweight and obesity at age 15 years; AOR 2.04 (95% CI 1.57–2.65)</td>
</tr>
<tr>
<td>Ford et al. 40</td>
<td>14,322; USA; born 1976–1981</td>
<td>BMI at ages 7, 11 and 16 years; International obesity task force defined overweight and obesity vs those non-overweight, non-obese</td>
<td>Hypertension at age 45 years</td>
<td>Significant increase in adult hypertension with overweight and obesity during childhood and adolescence; at age 16 years; AOR 1.96 (95% CI 1.64–2.35)</td>
</tr>
<tr>
<td>Li et al. 41</td>
<td>6764–7502; UK; born 1958</td>
<td>BMI at ≥ 5th vs ≤ 75th centiles; at age 8–15 years</td>
<td>Hypertension</td>
<td>Significant increase in risk of hypertension with overweight; OR 5.1 (95% CI 1.4–18.1)</td>
</tr>
<tr>
<td>Field et al. 42</td>
<td>130; USA; boys born 1963–1976</td>
<td>Self-perceived and -reported childhood ‘obesity’</td>
<td>Gestational hypertension</td>
<td>Significant increase in gestational hypertension for perceived childhood overweight vs healthy weight; AOR 1.46 (95% CI 1.01–2.12)</td>
</tr>
<tr>
<td>Leeners et al. 43</td>
<td>766 hypertensive female cases vs 951 controls; Germany</td>
<td>Top 2.5% of BMI distribution at age 4–6 years vs all others</td>
<td>CHD and Stroke</td>
<td>CHD not significant; Hazards ratio for stroke 2.41 (95% CI 1.00–5.86)</td>
</tr>
<tr>
<td>Lawlor et al. 44</td>
<td>5685; Scotland; born 1950–1956</td>
<td>For those under 18 years, BMI defined using Cole–IOTF method and expressed per s.d. of BMI</td>
<td>Ischaemic heart disease and stroke</td>
<td>Pooled hazard ratio for IHD per 1 s.d. of BMI 1.09 (1.01–1.10); Not significant for stroke</td>
</tr>
</tbody>
</table>

Abbreviations: AOR, adjusted odds ratio; BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; IHD, ischaemic heart disease; OR, odds ratio.

cancer. Sanderson et al.49 found no significant relationships between recalled weight status at age 15 years and subsequent breast cancer risk. Jeffreys et al.46 found that higher-measured BMI for age and sex in childhood was associated with significantly increased risk of later cancers, which were not smoking related.

One ineligible study54 (ineligible because the exposure measure was made in early adulthood) found that higher-measured BMI in early adulthood was associated with significantly increased risk of mortality from prostate cancer and breast cancer. The study by Bjorge et al.25—included in the mortality analyses above (Table 1)—also found that adolescent overweight and obesity were related to significantly increased risk of mortality from colon cancer (but not breast cancer).

Discussion

The present review found a relatively large and highly consistent body of evidence, which reported that measured—and conventionally defined—overweight and obesity in childhood and adolescence were associated with increased risk of both premature mortality and adult morbidity, particularly cardiometabolic morbidity. Evidence from recalled weight status in childhood and adolescence was much less consistent, and this presumably relates to difficulties associated with recall of childhood and adolescent weight status in adult life. There has been a marked increase in the availability of evidence on this topic since the last systematic review.3 The evidence reviewed here was also broadly consistent with the smaller body of evidence from older studies of this topic,55–58 and with a recent systematic review on associations between weight status (but not specifically overweight and obesity) and ages 2–30 years and subsequent coronary heart disease risk.58

Short-term consequences of childhood and adolescent obesity for physical and mental health are important, and this issue was reviewed systematically some time ago.1 Child and adolescent obesity generally has adverse effects on these constructs. However, the present short review was focused on the long-term impact of child and adolescent obesity. In the experience of the authors, clinical service providers and policy makers tend to perceive commonly that the
short-term impacts of child and adolescent obesity are relatively unimportant. Clinical service providers and policy makers may be more readily persuaded to consider childhood obesity prevention and treatment interventions if these might mitigate the long-term (adult) consequences of childhood obesity, which they consider of greater importance. The present review may, therefore, be helpful in making the case for greater emphasis on prevention and treatment interventions in childhood. Moreover, the estimates of strength of associations between child or adolescent obesity and later outcome may also be helpful to health economic assessments of the long-term impact of childhood obesity prevention and treatment interventions.

The present review, and the studies summarized in it, had a number of limitations. First, the high degree of consistency of the eligible evidence might in part reflect a degree of publication bias. We note that the larger studies tended to support the hypothesis that childhood obesity has adverse long-term impact at least as strongly as the smaller studies, and most of the studies included in the review were large. Furthermore, most of the associations observed are biologically plausible as discussed below. Secondly, the issue of generalisability of the evidence reviewed. Most, but not all, of the available evidence was from cohorts studied well before the obesity epidemic and from the developed world. The generalisability of the evidence to contemporary populations in general, and to low–middle income countries in particular, is unclear. Generalisability of the findings of the present review may be enhanced by the fact that most of the eligible studies defined child and adolescent overweight and obesity using contemporary and established definitions of overweight and obesity based on BMI.

For a number of reasons, the present review probably provides a highly conservative view of the consequences of child and adolescent overweight and obesity for life-long health and well-being. First, a good deal of evidence was beyond the scope of the present review and so was excluded. This involved excluding the large body of evidence on adverse long-term impacts of childhood or adolescent obesity on adult cardiometabolic risk factors rather than adverse long-term impacts; studies that modelled long-term impacts; studies that defined child and adolescent overweight or obesity7–10) and that found that excessive weight gain during childhood or adolescence as the exposure measure (rather than overweight or obesity7–10) and that found that excessive weight gain during childhood or adolescence had adverse long-term impacts; studies that modelled long-term impacts rather than studied these empirically and that had health economic outcomes rather than morbidity outcomes;19–21 evidence on the long-term associations of obesity with mental health and educational outcomes;3,59,60 the exclusion of evidence on the short-term consequences of child and adolescent obesity (for child and adolescent health).5,61 The fact that so many of the eligible studies considered overweight rather than obesity as the exposure

<table>
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<th>Table 3</th>
<th>Associations between childhood and adolescent overweight, and obesity and miscellaneous morbidity in adulthood</th>
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<tr>
<td>Study</td>
<td>Sample and setting</td>
</tr>
<tr>
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<td>---------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Jeffreys et al.46</td>
<td>2997; UK; born 1927–1937</td>
</tr>
<tr>
<td>Baer et al.47</td>
<td>109 266 women; USA; born 1947–1964</td>
</tr>
<tr>
<td>Swedlow et al.48</td>
<td>400 cases vs 400 controls; Denmark, Sweden, UK; born 1896–1964</td>
</tr>
<tr>
<td>Sanderson et al.49</td>
<td>288 cases vs 350 controls; China; born 1942–1964</td>
</tr>
<tr>
<td>Bardia et al.50</td>
<td>35 941; USA; born 1917–1931</td>
</tr>
<tr>
<td>Neovius et al.51</td>
<td>1 191 027; Sweden; born 1951–1976</td>
</tr>
<tr>
<td>Karnehed et al.52</td>
<td>366 929; Sweden; born 1952–1959</td>
</tr>
<tr>
<td>Xu et al.53</td>
<td>4719; Finland; born 1966</td>
</tr>
<tr>
<td>Laitinen et al.54</td>
<td>2007; Finland; born 1966</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; CI, confidence interval; RR, regression ratio.
probably also underestimates the long-term impact of child and adolescent obesity.

Additional limitations in the evidence base are likely to mean that the conclusions of the present study must be very conservative. The studies included in the present review tended to focus on older cohorts, born well before the obesity epidemic in most cases. Earlier onset obesity is much more common in contemporary populations than in the recent past, and a more prolonged duration of obesity may increase the probability of later comorbidity. In the present review, most of the eligible studies examined the exposure of overweight and obesity during adolescence: only 3/28 of the eligible papers examined obesity in children under 5 years old as an exposure, and in some of these studies younger participants were not considered separately. More extreme forms of childhood obesity are also much more common now than in the recent past, and are associated even more strongly with cardiometabolic risk factors. For example, Freedman et al. suggested a BMI at or above the 99th percentile as a definition of more extreme obesity in the USA, showed that it was not rare (at around 4% of US children), and demonstrated that being above the 99th percentile for BMI denoted dramatically increased cardiometabolic risk. More extreme forms of obesity are likely to have even greater impact on later risk of premature mortality and physical morbidity, but could not be considered adequately by the present review as they were not generally considered as an exposure in the eligible studies.

The issue of mechanisms by which child and adolescent overweight and obesity lead to increased risk of later morbidity and mortality was beyond the scope of the present review. Some authors have considered it important that associations between child and adolescent obesity are adjusted for adult obesity in an attempt to quantify the contribution of child/adolescent obesity to later outcome, distinct from the contribution made by the well-established tendency for child and adolescent obesity to persist into adult life. Many studies in the present review attempted to consider this issue by adjustment for adult (current) weight status, and some found that associations between child and adolescent obesity, and adult outcomes attenuated markedly with adjustment, but other studies found negligible attenuation after such adjustments. The mechanisms that link childhood obesity to subsequent impairments in cardiometabolic health are increasingly well understood, and there is a good deal of evidence that obesity in childhood and adolescence represents an insult to the cardiovascular system, which results in increased risk of later cardiovascular morbidity and mortality.

Conclusion

In conclusion, there is now a relatively large and consistent body of evidence, which shows that overweight and obesity in childhood and adolescence have substantial and adverse long-term consequences for physical health.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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14 Sachdev HP, Osmond C. Predicting adult metabolic syndrome from childhood BMI. *Arch Dis Child* 2009; 94: 768–774.

Long term consequences of childhood obesity

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