**Objective:** To investigate whether measures of obesity are associated with periodontitis.

**Design:** A case-control study.

**Setting:** A nationally representative sample using data from the Third National Health and Nutrition Examination Survey.

**Participants:** The sample included 2452 nonsmokers, aged 13 to 21 years, who received a periodontal examination and had complete information for age, sex, and smoking habits.

**Main Exposures:** Skinfold thickness, weight, and waist circumference were examined as independent variables in logistic regression models. Final models were adjusted for sex, race/ethnicity, poverty index ratio, last dental visit, and self-reported calcium intake.

**Main Outcome Measures:** Cases were subjects with the presence of 1 or more periodontal sites with both a loss of tissue attachment of 3 mm and a probing depth of 3 mm (n=111). Subjects who did not meet these criteria were classified as controls (n=2341).

**Results:** Total body weight and waist circumference were associated with periodontitis, but the association varied by age. Adolescents aged 13 to 16 years were not at increased risk of chronic periodontitis, while adolescents aged 17 to 21 years had an increased risk per 1-kg increase in body weight (adjusted odds ratio, 1.06 [95% confidence interval, 1.01-1.09]). Similarly, adolescents aged 13 to 16 years were not at increased risk for periodontal disease, while adolescents aged 17 to 21 years were at an increased risk of periodontal disease per 1-cm increase in waist circumference (adjusted odds ratio, 1.05 [95% confidence interval, 1.01-1.08]).

**Conclusion:** Periodontitis may follow patterns similar to other chronic conditions that originate early in life and are related to central adiposity.

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fold thickness and body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) were significant predictors of periodontal disease. However, to our knowledge, no study has investigated how early the relationship between adiposity and periodontitis arises among adolescents. We examined whether periodontitis, following traditionally age-dependent, chronic diseases patterns, was associated with overweight in adolescents.

STUDY DESIGN AND SAMPLE

We used a case-control design to test whether adiposity was associated with periodontitis among US adolescents aged 13 to 21 years. All subjects received a periodontal examination and had complete information on sex, age, and smoking habits. Since smoking is strongly related to both systemic health and periodontal disease, control for confounding can best be achieved by restricting subjects to nonsmokers. Accordingly, this study was restricted to self-reported nonsmokers. Data were collected on 3466 subjects aged 13 to 21 years; 2452 (71%) met the criteria.

DATA SOURCE

Adolescent body measurements and oral health status were taken from data collected in the Third National Health and Nutrition Examination Survey (NHANES III), a cross-sectional examination conducted by the National Center for Health Statistics and Centers for Disease Control and Prevention from 1988 to 1994. The NHANES III was designed to obtain nationally representative data on health and nutritional status in the United States through surveys, as well as medical and dental examinations in a mobile clinic. Nutritional data were collected by a trained interviewer administering a 24-hour recall. The US civilian, noninstitutionalized population 2 months or older is represented in NHANES III. Data were collected from 33,994 individuals. As part of the stratified, multistage probability design, African American and Mexican American subjects were oversampled. Details about the survey and its methods have been previously published.

INDEPENDENT VARIABLES: ANTHROPOMETRIC INDEXES

Adiposity was assessed by triceps, subscapular, suprailiac, thigh, and sum of skinfold thickness (triceps + subscapular) measures. Skinfold thickness measures were log transformed in the event of a skewed distribution. Weight, height, and waist circumference were examined as continuous independent variables. We did not use BMI as an independent variable. Although BMI is a useful screening tool, it may be a poor proxy for fat distribution and central adiposity in children and adolescents. Moreover, use of BMI in statistical models can lead to incorrect inferences, inadequately adjusting for each variable in the ratio. In this study, BMI was initially examined as an interaction term, with weight and height (1/m²) included as main effects. No significant relationship was found between BMI and periodontitis.

OUTCOME VARIABLE: PERIODONTITIS

Oral health data from 1 randomly assigned upper and lower quadrant per mouth were collected on each subject in NHANES III. Measures of periodontal loss of tissue attachment and probing depth from mesiofacial and midfacial sites (28 sites per individual) were recorded to determine periodontal status. Third molars, partially erupted teeth, and retained roots were excluded. Cases of periodontitis were defined as the presence of 1 or more periodontal sites with both a loss of tissue attachment of 3 mm or more and a probing depth of 3 mm or more. Subjects not meeting these criteria were classified as controls.

STATISTICAL METHODS

Statistical software Stata 8.0 (StataCorp, College Station, Tex) was used for all analyses, allowing inclusion of population weights, calculation of the standard errors, and adjustment for the complex sampling design. Following the National Center for Health Statistics and Centers for Disease Control and Prevention guidelines, statistical sample weights were used to account for both oversampling and nonresponse. The final sample weight variable from the mobile clinic was used to obtain nationally representative estimates.

Demographic, dental, and nutritional variables of cases and controls were compared. Possible associations of skinfold thickness, weight, and waist circumference with periodontitis were examined in a series of logistic regression models. Crude odds ratios (ORs) and 95% confidence intervals (CIs) were estimated using logistic regression, with sociodemographic variables and periodontitis risk factors as potential confounding factors. Covariates included in the final models a priori were age, sex, race/ethnicity, poverty index ratio, last dental visit, and self-reported calcium intake. Ethnicity (non-Hispanic white, non-Hispanic black, Mexican American, and others) was obtained from the NHANES III race/ethnicity variable. The poverty index in NHANES III (family income divided by the poverty threshold) was categorized as high (>3.5), medium (1.851-3.499), and low (0-1.850) based on federal eligibility for free and reduced-price lunch services. History of dental care was categorized as less than 1 year, 2 to 3 years, more than 3 years, and never visited. Self-reported calcium intakes were divided into quartiles.

Selection of additional potential confounding variables was based on forward selection with the decision to include or delete based on changes in ORs of the exposure effects. Potential confounders examined using this method were insulin use (yes/no), nonfasting serum glucose level, and self-reported vitamin C intake. A glucose level of 200 mg/dL (11.1 mmol/L) or higher was considered elevated regardless of length of fast. Vitamin C intakes were divided into quartiles. Exposure coefficients did not change when insulin use, serum glucose level, and vitamin C intake were added to the respective models. Final models included sex, race/ethnicity, poverty index ratio, last dental visit, and calcium intake as confounding factors.

RESULTS

National estimates based on weighted data and the unweighted sample size on which these estimates were based are presented in Table 1. Of 2452 adolescents examined, 111 were classified as having periodontitis, yielding an overall weighted prevalence of 3.3%. Adolescents with periodontitis were more likely to be 17 to 21 years of age, male, African American or other race, in the lowest socioeconomic group, and without a dental visit for 3 years or more. Those with periodontitis were more likely to have self-reported calcium and vitamin C intakes in the lowest quartiles and less likely to have upper-quartile calcium intake. Subjects aged 17 to 21 years with

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periodontitis were, on average, 7 kg heavier, with a waist circumference 8 cm larger than controls of the same age (data not shown). Few of the 2452 adolescents reported taking insulin or had elevated serum glucose levels.

Interaction between age and weight, as well as age and waist circumference, was tested for significance using the Wald test with P<.05. Interaction between age and weight (P<.009) and age and waist circumference (P<.02) was significant. Therefore, final models evaluating weight and waist circumference were stratified by age. There were no significant interactions between race/ethnicity and weight, race/ethnicity and waist circumference, poverty index and weight, or poverty index ratio and waist circumference.

**RESULTS AND COMPARISONS WITH PREVIOUS REPORTS**

In a representative sample of US adolescents, weight and waist circumference were associated with increased risk of periodontitis among those aged 17 to 21 years. Skinfold thickness measures were not associated with periodontitis (data not shown). Adolescents aged 13 to 16 years were not at increased risk of periodontitis with each unit increase in weight (OR, 0.99 [95% CI, 0.97-1.01]), while adolescents aged 17 to 21 years were at a 6% increased risk of periodontitis for each 1-kg weight increase (adjusted OR, 1.06 [95% CI, 1.01-1.09]) (Table 2). Similarly, adolescents aged 13 to 16 years were not at increased risk of periodontitis (OR, 1.0 [95% CI, 0.98-1.04]) with each unit increase in waist circumference, while adolescents aged 17 to 21 years were at a 5% increased risk of disease for each 1-cm increase in waist circumference (adjusted OR, 1.05 [95% CI, 1.01-1.08]) (Table 3). Of 432 smokers excluded, 25 had periodontitis. Results were unchanged with smokers included in the analysis and smoking controlled in the model (data not shown).

Triceps, subcapular, suprailiac, and thigh skinfolds; sum of skinfold thickness; and log sum of skinfold thickness were not associated with periodontitis (data not shown). Adolescents aged 13 to 16 years were not at increased risk of periodontitis with each 1-cm increase in waist circumference (adjusted OR, 1.0 

### Table 1. Characteristics of Adolescents Aged 13 to 21 Years Sampled in the National Health and Nutrition Examination Survey III* (1988-1994) by Chronic Periodontitis Status

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Sample Size (N = 2452)†</th>
<th>Cases (n = 111)</th>
<th>Controls (n = 2341)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13-16</td>
<td>1022</td>
<td>28 (24.5)</td>
<td>994 (41.1)</td>
</tr>
<tr>
<td>17-21</td>
<td>1430</td>
<td>83 (57.6)</td>
<td>1347 (56.0)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1093</td>
<td>51 (46.7)</td>
<td>1042 (47.1)</td>
</tr>
<tr>
<td>Female</td>
<td>1359</td>
<td>60 (36.2)</td>
<td>1299 (52.8)</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic white</td>
<td>532</td>
<td>9 (37.1)</td>
<td>523 (62.0)</td>
</tr>
<tr>
<td>Non-Hispanic black</td>
<td>922</td>
<td>55 (28.2)</td>
<td>867 (17.4)</td>
</tr>
<tr>
<td>Mexican American</td>
<td>857</td>
<td>41 (11.8)</td>
<td>816 (9.2)</td>
</tr>
<tr>
<td>Other</td>
<td>141</td>
<td>6 (23.0)</td>
<td>135 (11.4)</td>
</tr>
<tr>
<td>Poverty index ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-1.850</td>
<td>1369</td>
<td>69 (66.2)</td>
<td>1300 (41.8)</td>
</tr>
<tr>
<td>1.851-3.499</td>
<td>581</td>
<td>23 (26.9)</td>
<td>558 (34.6)</td>
</tr>
<tr>
<td>≥3.5</td>
<td>256</td>
<td>2 (6.3)</td>
<td>254 (23.6)</td>
</tr>
<tr>
<td>Last dental visit, y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>1494</td>
<td>52 (54.1)</td>
<td>1442 (72.6)</td>
</tr>
<tr>
<td>1-3</td>
<td>449</td>
<td>23 (23.6)</td>
<td>426 (15.0)</td>
</tr>
<tr>
<td>&gt;3</td>
<td>252</td>
<td>14 (13.1)</td>
<td>238 (7.6)</td>
</tr>
<tr>
<td>Never</td>
<td>234</td>
<td>20 (9.3)</td>
<td>214 (5.0)</td>
</tr>
<tr>
<td>Quartile calcium intake‡</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-24</td>
<td>565</td>
<td>29 (28.1)</td>
<td>565 (20.3)</td>
</tr>
<tr>
<td>25-49</td>
<td>597</td>
<td>30 (25.0)</td>
<td>567 (23.5)</td>
</tr>
<tr>
<td>50-74</td>
<td>603</td>
<td>32 (32.7)</td>
<td>571 (25.6)</td>
</tr>
<tr>
<td>75-100</td>
<td>593</td>
<td>19 (14.1)</td>
<td>574 (30.6)</td>
</tr>
<tr>
<td>Quartile vitamin C intake‡</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-24</td>
<td>577</td>
<td>29 (31.1)</td>
<td>548 (24.9)</td>
</tr>
<tr>
<td>25-49</td>
<td>589</td>
<td>28 (29.0)</td>
<td>561 (26.2)</td>
</tr>
<tr>
<td>50-74</td>
<td>606</td>
<td>20 (11.8)</td>
<td>586 (25.8)</td>
</tr>
<tr>
<td>75-100</td>
<td>615</td>
<td>33 (28.1)</td>
<td>582 (23.1)</td>
</tr>
<tr>
<td>Insulin use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6</td>
<td>1 (0.02)</td>
<td>5 (0.03)</td>
</tr>
<tr>
<td>No</td>
<td>2359</td>
<td>106 (99.8)</td>
<td>2253 (99.6)</td>
</tr>
<tr>
<td>Nonfasting glucose level, mg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;200</td>
<td>2218</td>
<td>103 (100)</td>
<td>2115 (99.8)</td>
</tr>
<tr>
<td>≥200</td>
<td>3</td>
<td>0</td>
<td>3 (0.02)</td>
</tr>
</tbody>
</table>

SI conversion factor: To convert nonfasting glucose level to micromoles per liter, multiply by 0.555.†Weighted to produce nationally representative estimates.†Total differs in some cells because of missing data.‡Nutrient intakes were self-reported by the 24-hour recall method.

### Table 2. Crude and Adjusted ORs for Weight and Chronic Periodontitis in 2109 Adolescents

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Crude OR (95% CI)</th>
<th>Adjusted OR* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13-16</td>
<td>1.0 (0.98-1.0)</td>
<td>0.99 (0.97-1.0)</td>
</tr>
<tr>
<td>17-21</td>
<td>1.03 (1.01-1.05)</td>
<td>1.06 (1.01-1.09)</td>
</tr>
</tbody>
</table>

### Table 3. Crude and Adjusted ORs for Waist Circumference and Chronic Periodontitis in 2079 Adolescents

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Crude OR (95% CI)</th>
<th>Adjusted OR* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13-16</td>
<td>1.0 (0.98-1.02)</td>
<td>1.0 (0.98-1.01)</td>
</tr>
<tr>
<td>17-21</td>
<td>1.04 (1.01-1.07)</td>
<td>1.05 (1.01-1.08)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; OR, odds ratio.†Adjusted for height, age, sex, race/ethnicity, poverty index ratio, last dental visit, and calcium intake.‡Adjusted for sex, race/ethnicity, poverty index ratio, last dental visit, and calcium intake.
weight and risk of periodontitis for adolescents aged 13 to 16 years. These results extend previous findings of a relationship between central adiposity and periodontal disease to include adolescents aged 17 to 21 years. This suggests that periodontitis could be an age-dependent, chronic condition clinically recognizable after the age of 16 years and associated with excess fat accumulated by adolescence.

In a previous analysis, young adults aged 18 to 34 years with a BMI greater than 30 had a 76% increased risk for periodontal disease, and those with a large waist circumference had a 127% increased risk of disease. Others found obesity and central adiposity predictive of periodontal disease in Japanese adults. Reportedly, the relationship between obesity and periodontal disease was even greater for adults who had insulin resistance.

In the United Kingdom, trends of waist-circumference increases have surpassed trends of BMI increases among adolescents aged 11 to 16 years. In the United States, increasing central adiposity rates from adolescence to adulthood are greatest among children of low socioeconomic status. Adiposity and related disease risk also differ between ethnicities. Low-income and minority communities generally lack access to oral health education and services and may be disproportionately affected by the possible relationship between excess central adiposity and periodontal disease.

Unlike adults, skinfold thickness and periodontitis were not associated among adolescents in this study. Skinfold measures may not appropriately assess body composition among a heterogeneous population undergoing growth and development. The thickness of subcutaneous adipose tissue may not reflect a constant proportion of total body fat, since the relationship between subcutaneous fat and fat stores around organs is nonlinear and varies by body weight and age. Subcutaneous fat distribution also varies by sex, race, and age.

MECHANISMS

A variety of potential mechanisms could account for an association between obesity and periodontitis, including noncausal mechanisms related to health risk behaviors. Overweight adolescents may have unhealthy dietary patterns with insufficient micronutrient and excess sugar and fat content increasing the risk for periodontal disease. Other possible mechanisms include changes in host immunity and/or increased stress levels, often related to gaining excess fat early in life.

Poor dietary patterns may affect oral tissues and the immune response. Calcium and vitamin C are important cofactors for maintaining these systems in animals and humans. Obesity and periodontal disease were strongly related in young but not in middle-age or older adults. We propose that a cohort effect whereby less healthy dietary patterns among young adults (compared with patterns among older generations) may explain the increased periodontal disease risk. If this is true, the total population oral disease burden could be significantly affected as adolescents continue to consume unhealthy diets into adulthood, deposit fat earlier in life, and maintain excess weight as they age.

It has been proposed that obesity triggers the immune response, generating a state of chronic subclinical systemic inflammation. Adipocytes have been identified as active producers of cytokines, including tumor necrosis factor α, a powerful producer of IL-6, and to some degree IL-1. Increases in cytokines stimulate a variety of acute immune responses by the liver such as C-reactive protein and fibrinogen production. In overweight children, elevated C-reactive protein levels have been documented, suggesting systemic inflammation may occur early in life. In children as young as 11 years, obesity has been related to impaired microvascular functioning. Periodontal blood vessels and tissues may be affected by alterations in the inflammatory response as well as decreased blood flow; both can affect cell signaling and host immunity.

Insulin resistance may mediate the relationship between obesity and periodontal disease. Dietary free fatty acids may cause insulin resistance by increasing apoptosis of pancreatic β cells. In turn, the immune response may interfere with glucose transporters, inhibiting insulin signaling and causing the body to develop an inflammatory state. An increased inflammatory state "sets the stage for increased levels of periodontal disease triggered by oral pathogens."

Psychosocial stress associated with adolescent overweight may affect periodontal health through physiological and behavioral pathways. Physiological responses to stress may alter blood and salivary flow, decreasing the immune response to oral pathogens. Psychosocial stress related to teasing about body weight may affect oral health behaviors such as regular brushing, flossing, fluoride use, and and/or seeking preventive dental services. Overweight adolescents report engaging in significantly more unhealthy behaviors compared with their nonoverweight peers. However, little is known about these relationships among teens. Among adults, oral health behaviors and general health characteristics are correlated; a lack of daily flossing is a strong marker of obesity. Thus, the observed association between obesity and periodontitis may be due to factors associated with health awareness rather than a biological mechanism.

LIMITATIONS

This study had several limitations. First, the cross-sectional design precluded determining the temporal sequence. Whether adolescents with periodontitis had accumulated excess fat during childhood is unknown. Second, the role of genetics, oral health behaviors (eg, brushing or flossing), or "local factors" (eg, gingival inflammation, active caries, dental plaque, and calculus) were not considered in periodontitis etiology. Therefore, results may have been confounded by uncontrolled factors. Nonresponse and missing values for variables may have led to bias, though several authors have indicated little bias due to nonresponse to HANES and NHANES III surveys.

Despite its limitations, the current study raises questions about the role of early excess central adiposity in oral disease etiology. Sample representativeness and weighting provide generalizability of the results to the
US adolescent population. Many covariates that could have confounded the relationship between weight, central adiposity, and periodontal disease were controlled. Since smokers were excluded, we can be confident that our results were not due to residual confounding by smoking, a strong causal factor for periodontitis.

CONCLUSIONS

The results of this study have implications for the care of overweight adolescents, an increasing proportion of youth. Periodontitis appears to be yet another disease associated with overweight and obesity. Our research suggests periodontal disease follows risk patterns similar to other age-dependent, chronic conditions originating early in life and is related to lifestyles associated with central adiposity. Increasing adiposity during childhood may potentially affect adolescent periodontal health through a variety of mechanisms including unhealthy dietary habits, alterations in host immunity, and increased psychosocial stress. Alternatively, adiposity may be a marker for unhealthy lifestyles that cause periodontitis.

The results described earlier and related research indicate that obesity is a potential marker for periodontal disease among youth. Medical and dental professionals should develop interdisciplinary approaches for identifying and treating early signs of oral disease among children and adolescents. Since oral and general health behaviors are related, it is important to query parents, children, and teens about their oral hygiene practices when assessing health/risk behaviors and offer oral health education and care when necessary.

Like other health conditions, periodontal disease and overweight disproportionately affect low-socioeconomic and minority youth and/or those lacking access to health care. Primary care and other medical and dental professionals can potentially promote adolescent oral health by screening overweight teens, especially those with central adiposity, for early signs of gum disease and referring them for appropriate care. Since waist circumference is a simple, accurate measure for identifying youth at risk for chronic disease, health care professionals can use it to screen adolescents with or without a high BMI, especially in high-risk populations. To appropriately screen, professionals serving adolescents should be well informed about the connections between oral and systemic health, which historically have been believed to have little overlap.6

From a public health perspective, longitudinal studies can further explore associations between overweight and periodontitis in youth, such as whether hormonal changes related to physical maturation influence periodontal disease. Ethnic differences in periodontal disease risk can also be investigated. If associations between obesity, periodontal disease, and maturation are confirmed, the fat distribution patterns among low-income and minority adolescents could be recognized as increasing the oral disease burden in African American, Hispanic, and Native American communities. Since it is known that dietary, oral hygiene, and other behavioral practices during this period are likely to influence long-term health, public health planners should consider including oral health education and care in childhood and adolescent health programs.

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REFERENCES


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Who's Got Cooties?

“Cooties are ‘imaginary germs’ or ‘contaminated bugs.’ All a child does to start a game is touch someone and say, ‘You’ve got cooties!’ Or he may specify the source of the infection: ‘You’ve got Larry’s cooties!’ He may add, ‘No tag backs.’ Children mimic immunization shots with ballpoint pens or mark a magic ‘X’ on their hands and write with ballpoint pens or mark a magic ‘X’ on their hands. ‘C. P.’ on their tennis shoes—for ‘Cootie Protection.’”

—From One Potato, Two Potato: The Secret Education of American Children by Mary and Herbert Knapp, 1976

---

20. Cruz ML, Bergman RN, Goran MI. Unique effect of visceral fat on insulin sensiti-


15. Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a mea-

17. Tanner JM. Fallacy of per-weight and per-surface area standards, and their re-


12. Fagot-Campagna A, Saaddine JB, Flegal KM, Beckles GL. Diabetes, impaired fast-


20. Cruz ML, Bergman RN, Goran MI. Unique effect of visceral fat on insulin sensiti-

21. Goran MI, Ball GD, Cruz ML. Obesity and risk of type 2 diabetes and cardiovas-


mmon carotid artery and endothelial dysfunction in severely obese children: a pro-


30. da Silva AM, Newman LN, Oakley DA. Psychosocial factors in inflammatory peri-

31. Eisenberg ME. Neumark-Sztainer D, Story M. Associations of weight-based teas-

32. Ackard DM. Neumark-Sztainer D, Story M, Perry C. Overeating among adoles-
cents: prevalence and associations with weight-related characteristics and psy-

33. Mellin AE. Neumark-Sztainer D, Story M, Ireland M, Resnick MD. Unhealthy be-
haviors and psychosocial difficulties among overweight adolescents: the poten-

34. Hujel PP, Cunha-Cruz J, Kressin NR. Spurious associations in oral epidemiolo-


36. Troiano RN, Flegal KM. Overweight children and adolescents: description, epi-

37. Winkleby MA, Robinson TN, Sundquist J, Kraemer HC. Ethnic variation in car-


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