Hindawi Publishing Corporation International Journal of Pediatric Endocrinology Volume 2009, Article ID 108187, 4 pages doi:10.1155/2009/108187

Review Article

Relation between Childhood Obesity and Adult Cardiovascular Risk

Darren M. Allcock, Michael J. Gardner, and James R. Sowers

Division of Endocrinology, Department of Internal Medicine, University of Missouri, Columbia, MO 65212, USA

Correspondence should be addressed to Darren M. Allcock, allcockd@health.missouri.edu

Received 11 May 2009; Accepted 18 August 2009

Recommended by Paul L. Hofman

The incidence of overweight and obesity is rising at an alarming pace in the pediatric population, just as in the adult population. The adult comorbidities associated with this risk factor are well-recognized and are being further elucidated continually. Additionally, we are gradually developing a better understanding of the risks of overweight and obesity among children while they are still young. However, there is now a growing body of evidence showing that childhood obesity not only leads all too frequently to adult obesity, but is in itself a risk factor for cardiometabolic syndrome and resultant cardiovascular risk in adulthood. If current trends continue, the problem of pediatric overweight and obesity will become of unmanageable proportions once these individuals reach adulthood. Future research efforts toward understanding this complex problem will need to focus on those overweight and obese children who later went on to change their metabolic course and become normal-weight adults.

Copyright © 2009 Darren M. Allcock et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

1. Introduction

The prevalence of overweight and obesity in the United States and much of the world is increasing dramatically. Following in kind is the increased incidence of all of obesity's related comorbidities, including the cardiometabolic syndrome (CMS), type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, nonalcoholic fatty liver disease (NAFLD), and even evidence of cardiovascular disease (CVD). Of major concern is the escalating prevalence of overweight and obesity in children, and the impact that this premature onset of obesity may have in the future as these children grow up.

2. Definitions

A task force formed by the Endocrine Society has issued guidelines stating that overweight in the pediatric population should be defined as those individuals having a body mass index (BMI) greater than the 85th percentile for age and gender but less than the 95th percentile, and obesity should be defined as those individuals having a BMI greater than the 95th percentile [1]. Recent statistics from the Centers for Disease Control and Prevention (CDC) show that 11.3% of

children and adolescents aged 2 to 19 are at or above the 97th percentile of the 2000 BMI-for-age growth charts, 16.3% are at or above the 95th percentile, and 31.9% are at or above the 85th percentile [2]. By these definitions, nearly one third of individuals between the age of 2 and 19 years is either overweight or obese.

3. Pediatric Morbidity Associated with Obesity

Numerous complications are associated with children being overweight or obese, even at a very young age. These include all of the components of the CMS: elevated blood pressure, high serum triglycerides, low serum HDL cholesterol, high fasting glucose, and central obesity [3]. Looking at 1988–1994 NHANES data and applying the current definition of CMS, corrected by the use of age appropriate percentile cutoffs, the prevalence of CMS among 12–19 year olds was 4.2% (95% CI 2.9%–5.4%) [4]. A subsequent investigation using the same criteria and the 1999-2000 NHANES data found that the incidence had increased to 6.4% (*P*-value for the increase <.001). Additionally, at this time, the authors examined the relationship between obesity and prevalence of the cardiometabolic syndrome. Using the active CDC

criteria, 32.1% of overweight adolescents (BMI >95% for age) met the criteria for the cardiometabolic syndrome versus 7.1% of adolescents at risk of overweight (BMI 85th to <95th percentile). Using this data, they estimated that more than 2 million US adolescents have the cardiometabolic syndrome [5]. One estimate from the CDC suggests that, if the current overweight and obesity rates continue, one in three newborns born in the United States in 2000 will eventually develop diabetes [6]. The first step in the development of type 2 diabetes mellitus is generally that of insulin resistance and impaired glucose tolerance. One study detected impaired glucose tolerance in 25% of obese children and 21% of obese adolescents [7]. Combined with this problem, some have hypothesized that there is actually an accelerated process of conversion from a state of impaired glucose tolerance to over type 2 diabetes mellitus in children, when compared to adults [8]. In addition to impaired glucose tolerance and type 2 diabetes, the obese pediatric population is also at risk for nonalcoholic fatty liver disease (NAFLD), with one Japanese series showing that >10% of all obese children had at least modest increases in serum transaminases, if not overt NAFLD or steatohepatitis [9]. Other complications of overweight and obesity include cholelithiasis, pseudotumor cerebri, obstructive sleep apnea, polycystic ovary syndrome, psychological trauma, and orthopedic conditions such as slipped capital femoral epiphysis.

4. Adult Morbidity Associated with Obesity

The myriad morbidities associated with overweight and obesity in the adult population have been more thoroughly researched, and thus are perhaps better understood. In the adult population, overweight and obesity occur in two-thirds of the adult population, and are linked closely with the CMS and all of its components, as iterated earlier. NAFLD, obstructive sleep apnea, and orthopedic pathology are common also. In addition, the adult population begins to present more commonly the long-term consequences of obesity. Among these sequelae are coronary heart disease (CHD), congestive heart failure, obstructive respiratory disease, and chronic renal disease, with associated increases in their in prevalence, mortality, and costs to our health care system.

5. Relation between Pediatric Obesity and Adult Morbidity

It has been shown that overweight and obese children and adolescents are more likely to become obese adults [10]. In seeking to define this relationship, Guo et al. compiled data from four longitudinal studies started between 1929 and 1960 that compared BMI at age 1–18 years to BMI at a target age of 35 years. They found BMI at age 13 was a good predictor of BMI at 35 and that BMI at 18 was an excellent predictor [11]. A longitudinal prospective study completed in 1987 followed 164 subjects from one month of age until 21 years of age. The authors defined lean as BMI under the 25th percentile for age, medium weight was defined as a BMI >25th percentile but less than or equal to the 75th percentile

for age, and fat was defined as BMI greater than the 75th percentile for age. At age 21 years, 41% of the individuals that were lean at one year were still in the lean category and 41% of the fat infants were still in the fat category. Infants who began in the top tertile at one year of age had twice the relative risk of being in the top tertile at age 21 as compared to being in any of the lower tertiles at one year of age [12]. Another recent study has re-enforced that the roots of adolescent obesity may be found in early childhood [13]. Other studies have estimated that up to 81% of overweight (BMI >85th percentile) adolescents will become obese young adults [14]. With the evidence accumulating that childhood overweight and obesity is linked to obesity in adulthood, the question arises as to whether their risk of CVD is increased by their pediatric obesity or simply similar to weight-matched peers. A number of cross-sectional and longitudinal studies have sought to address this issue.

It has been reported that in a Finnish adult population, the risk of the cardiometabolic syndrome was lower among obese adults who had not been obese as children than among the obese adults who had also been obese as children [15]. These researchers followed 712 individuals from the age of 7 into adulthood. For the purposes of their study, they defined obesity, in both children and adults, as the sexspecific highest third of the body mass index. They defined the cardiometabolic syndrome as a cluster of hypertension (systolic blood pressure ≥140 mm Hg, a diastolic blood pressure ≥90, or treatment with an antihypertensive agent); dyslipidemia (triglycerides ≥1.70 mmol/L, or HDL cholesterol of <1.00 mmol/L in men and <1.20 mmol/L in women, or the presence of both dyslipidemia and hypertriglyceridemia); insulin resistance or hyperinsulinemia (≥78 pmol/L), or both. They demonstrated a strong continuum of disease. Half of the obese children in their study went on to become obese adults. Nearly all of the cases of cardiometabolic syndrome were diagnosed in obese adults, and the majority of these occurred among that group that was obese as

In a Dutch study an independent association was observed between a higher BMI at age 18 and premature mortality from all causes. The demographics of a population of over 78 000 Dutch men at the age of 18 were studied, and this population was then followed over a 32-year period. They found a higher incidence of all-cause deaths in those individuals with a BMI >25 (or <18), when compared to individuals with a BMI between 18 and 25 [16]. This supports the position that there is a certain optimal BMI range in young adults that provides for increased longevity.

The Princeton Followup Study provided an even more age-related, specific link between metabolic abnormalities in youth and the development of cardiovascular disease. Indeed, the study group having the CMS in childhood had a significantly higher risk of developing cardiovascular disease 25 years later, when compared to the study group that did not have the cardiometabolic syndrome in childhood. The presence of the CMS in childhood was a stronger predictor of future cardiovascular disease in adulthood than were gender or even parental history of cardiovascular disease. Only age had a similar relationship with the development

Table 1

Study	Population	Outcome measured	Significant result
Vanhala et al. [15]	1008 Finnish people born in the years 1947, 1952, and 1957	Relationship between obesity and cardiometabolic syndrome in childhood and adulthood	Risk of cardiometabolic syndrome was lower among obese adults who had not been obese as children
Hofmans et al. [16]	78,612 18 year old Dutch men	Impact of BMI on all-cause mortality	Higher incidence of all-cause deaths in those with BMI >25 or <18
Morrison et al. [17]	771 children from Cincinnati, followed into adulthood	Relationship between cardiometabolic syndrome in childhood and CHD in adulthood	Adults more likely to develop CHD if they had cardiometabolic syndrome in childhood
Li et al. [18]	486 adults age 25–37 from Bogalusa, La. with at least 3 cardiovascular risk factors	Association of carotid IMT with cardiovascular risk factors	Childhood measures of LDL-C and BMI predict carotid IMT in young adults

of cardiovascular disease. The youth in their initial study exhibited an incidence of cardiometabolic syndrome of 4%. In the followup study, this had risen to 27.2%. Another dramatic result of the Princeton Followup Study was in the correlation between change (increases) in BMI and change in risk of cardiovascular disease. This study demonstrated an increased risk of cardiovascular disease of 24% for each increase in BMI of 10 percent [17]. This correlation showed not only a link between cardiometabolic syndrome and cardiovascular disease but also a linear correlation between adiposity and cardiovascular morbidity.

More concrete evidence for increased cardiovascular risk in adulthood following a history of childhood obesity was provided by the Bogalusa Heart Study. This study examined 486 adults between the ages of 25 and 37 years who were found to have at least 3 traditional cardiovascular risk factors during childhood. Carotid artery intima-media thickness (IMT) was measured by ultrasound. Those individuals with cardiovascular risk factors persisting from childhood into adulthood had a significantly higher carotid artery IMT when compared to others examined. As carotid artery IMT has been shown to be a reliable predictor of cardiovascular disease, this study demonstrates a direct relationship between the presence of risk factors in childhood and cardiovascular pathology later in early adulthood [18].

Finally, another investigative group has recently published a computer projection of the effect of adolescent overweight and obesity on future CVD. The projections are quite disturbing. Results suggest that 30%–37% of 35-year-old men and 40%–44% of 35-year-old women will be obese by the year 2020, up from 16.7% and 15.4%, respectively, in the year 2000 when these people were teenagers. Even with this model of modest projection of future obesity, they estimate significant morbidity and mortality beginning in young adulthood, with more than 100 000 excess cases of CHD by 2035 [19]. The authors of this study go on further to suggest that this projection could actually underestimate the future impact of childhood obesity on future CHD risk (see Table 1).

6. Summary and Conclusions

Overweight and obesity rates continue to increase among the pediatric population, as well as in adults. This rise leads invariably to increased incidence of morbidity among those children affected, and also translates into increased cardio-vascular risk later in life. This effect has been demonstrated through numerous studies, showing a direct correlation between BMI in childhood and cardiovascular morbidity later in adulthood. More tangible evidence is even seen through measurements of increased carotid artery IMT in young adults who had increased cardiovascular risk factors in childhood. If unabated, the problem stands to proceed at an accelerated pace, potentially reaching an unmanageable and unaffordable level in the very near future.

7. Future Directions

Up to this point, most research attention has been directed toward treating specific disease states and determining correlations that increase risk of disease. Given the magnitude of the childhood (and adulthood) obesity problem, this is certainly welldeserved. Further efforts have been focused on trying to treat obesity itself and, therefore, prevent progression to overt sequelae, either in childhood or later in adult life. Again, this will remain imperative. However, it is important to note that not all obese children become obese adults. There is a significant percentage of overweight or obese children and adolescents who go on to become normal weight adults and thereby escape this major risk factor. One of these studies discussed earlier noted that half of obese children became obese adults. While this is a very concerning statistic, it also means that half of the obese children did not go on to become obese adults. Future research must focus on obtaining a better understanding of the root causes of the recent increases in childhood obesity and how it is that these individuals changed their metabolic course. What was their environment when they were obese or overweight children, and did it change? Did they begin an exercise program or drastically change their diet? What was the health profile of their parents, and did this change in some way? When we begin to probe these and many other unanswered questions about such individuals, then we will begin to have a better understanding of effective prevention and treatment strategies.

References

- [1] T. Hampton, "Pediatric obesity guidelines released," *The Journal of the American Medical Association*, vol. 300, no. 19, p. 2238, 2008.
- [2] C. L. Ogden, M. D. Carroll, and K. M. Flegal, "High body mass index for age among US children and adolescents, 2003–2006," *The Journal of the American Medical Association*, vol. 299, no. 20, pp. 2401–2405, 2008.
- [3] "Third report of the national cholesterol education program expert panel on detection, evaluation and treatment of high blood cholesterol in adults (adult treatment panel III): executive Summary," Tech. Rep. 01-3670, National Institutes of Health, Bethesda, Md, USA, 2001.
- [4] S. Cook, M. Weitzman, P. Auinger, M. Nguyen, and W. H. Dietz, "Prevalence of a metabolic syndrome phenotype in adolescents: findings from the Third National Health and Nutrition Examination Survey, 1988–1994," Archives of Pediatrics and Adolescent Medicine, vol. 157, no. 8, pp. 821–827, 2003.
- [5] G. E. Duncan, S. M. Li, and X. H. Zhou, "Prevalence and trends of a metabolic syndrome phenotype among U.S. adolescents, 1999-2000," *Diabetes Care*, vol. 27, pp. 2438– 2443, 2004.
- [6] K. M. V. Narayan, J. P. Boyle, T. J. Thompson, et al., "Lifetime risk for diabetes mellitus in the United States," *The Journal of* the American Medical Association, vol. 290, no. 14, pp. 1884– 1890, 2003.
- [7] R. Sinha, G. Fisch, B. Teague, et al., "Prevalence of impaired glucose tolerance among children and adolescents with marked obesity," *The New England Journal of Medicine*, vol. 346, no. 11, pp. 802–810, 2002.
- [8] R. Weiss and S. Caprio, "The metabolic consequences of childhood obesity," Best Practice and Research Clinical Endocrinology and Metabolism, vol. 19, no. 4, pp. 405–419, 2005.
- [9] A. Kinugasa, K. Tsunamoto, N. Furukawa, et al., "Fatty liver and its fibrous changes found in simple obesity of children," *Journal of Pediatric Gastroenterology and Nutrition*, vol. 3, pp. 408–414, 1984.
- [10] D. S. Freedman, L. K. Khan, M. K. Serdula, et al., "The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study," *Pediatrics*, vol. 115, no. 1, pp. 22–27, 2005.
- [11] S. S. Guo, A. F. Roche, W. C. Chumlea, J. D. Gardner, and R. M. Siervogel, "The predictive value of childhood body mass index values for overweight at age 35 y," *The American Journal of Clinical Nutrition*, vol. 59, no. 4, pp. 810–819, 1994.
- [12] C. Rolland and M. F. Rolland-Cachera, "Tracking the development of adiposity from one month of age to adulthood," Annals of Human Biology, vol. 14, pp. 219–229, 1987.
- [13] L. A. Francis and E. J. Susman, "Self-regulation and rapid weight gain in children from age 3 to 12 years," *Archives of Pediatrics and Adolescent Medicine*, vol. 163, no. 4, pp. 297–302, 2009.
- [14] R. C. Whitaker, J. A. Wright, M. S. Pepe, K. D. Seidel, and W. H. Dietz, "Predicting obesity in young adulthood from childhood

- and parental obesity," *The New England Journal of Medicine*, vol. 337, no. 13, pp. 869–873, 1997.
- [15] M. Vanhala, P. Vanhala, E. Kumpusalo, P. Halonen, and J. Takala, "Relationship between obesity from childhood to adulthood and the metabolic syndrome: population based study," *British Medical Journal*, vol. 317, p. 319, 1998.
- [16] M. D. Hoffmans, D. Kromhout, and C. de Lezenne Coulander, "The impact of body mass index of 78,612 18-year old Dutch men on 32-year mortality from all causes," *Journal of Clinical Epidemiology*, vol. 41, pp. 749–756, 1988.
- [17] J. A. Morrison, L. A. Friedman, and C. Gray-McGuire, "Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: the Princeton Lipid Research Clinics Follow-up Study," *Pediatrics*, vol. 120, no. 2, pp. 340– 345, 2007.
- [18] S. Li, W. Chen, S. R. Srinivasan, et al., "Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study," *The Journal of the American Medical Association*, vol. 290, no. 17, pp. 2271–2276, 2003.
- [19] K. Bibbins-Domingo, P. Coxson, M. J. Pletcher, J. Lightwood, and L. Goldman, "Adolescent overweight and future adult coronary heart disease," *The New England Journal of Medicine*, vol. 357, no. 23, pp. 2371–2379, 2007.