Appetite 93 (2015) 3–12

Contents lists available at ScienceDirect

Appetite

journal homepage: www.elsevier.com/locate/appet

Research report

Pediatric obesity. An introduction ☆

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ARTICLE INFO

Article history:
Received 3 February 2015
Received in revised form 23 March 2015
Accepted 25 March 2015
Available online 30 March 2015

Keywords:
Children
Adolescents
Hyperphagia
Overeating
Etiology

ABSTRACT

The prevalence of child and adolescent obesity in the United States increased dramatically between 1970 and 2000, and there are few indications that the rates of childhood obesity are decreasing. Obesity is associated with myriad medical, psychological, and neurocognitive abnormalities that impact children's health and quality of life. Genotypic variation is important in determining the susceptibility of individual children to undue gains in adiposity; however, the rapid increase in pediatric obesity prevalence suggests that changes to children's environments and/or to their learned behaviors may dramatically affect body weight regulation. This paper presents an overview of the epidemiology, consequences, and etiopathogenesis of pediatric obesity, serving as a general introduction to the subsequent papers in this Special Issue that address aspects of childhood obesity and cognition in detail.

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Introduction

Among children in the US, the percentage of children classified as obese, according to the U.S. Centers for Disease Control growth standards, has more than tripled since the 1970s (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010; Ogden et al., 2006; Ogden, Carroll, Kit, & Flegal, 2014; Ogden, Flegal, Carroll, & Johnson, 2002). Obesity-related diseases rarely seen in children previously, including obesity-associated sleep apnea (Muzumdar & Rao, 2006), non-alcoholic fatty liver disease with resultant cirrhosis (Molleston, White, Teckman, & Fitzgerald, 2002), and type 2 diabetes (Dabelea et al., 2014; Pettitt et al., 2014), are increasingly diagnosed in children and adolescents. Because childhood onset obesity frequently persists into adulthood, it is also associated with increased long-term morbidity and mortality (Must, Jacques, Dallal, Bajema, & Dietz, 1992). For all these reasons, it is crucial to understand the causes and consequences of childhood obesity. This paper serves as a general introduction to the subsequent papers in this Special Issue that more specifically address childhood obesity and cognition by presenting an overview of the epidemiology, consequences, and etiopathogenesis of pediatric obesity.

Body mass index-based definitions and the epidemiology of pediatric obesity

"Obesity" is defined as the accumulation and storage of excess body fat, while "overweight" is weight in excess of a weight reference standard (Ogden & Flegal, 2010). Because there are no consensus criteria defining childhood obesity on the basis of excessive body adipose tissue, weight-based classification based on body mass index (BMI, kg/m^2) has been routinely used for both epidemiological and clinical purposes.

The BMI typically decreases immediately after birth, increases through the first 6–8 months (WHO Multicentre Growth Reference Study Group, 2006), decreases until age 5–7 years, with corresponding decreases in percentage body fat (Garn & Clark, 1976) and then increases for the reminder of childhood up to adult levels. Because the distribution of BMI changes dramatically with age and differs by sex in children and adolescents, age- and sex-specific BMI percentiles rather than raw BMI values are used for BMI-based classification (Rolland-Cachera et al., 1982). The reference standards most commonly used in the United States for evaluating children's BMI are the 2000 Centers for Disease Control and Prevention (CDC 2000) growth charts that provide age- and sex-specific standards for ages 2 to 18 (Kuczmarski et al., 2000, 2002). These charts supply smoothed percentiles for BMI that were constructed using a modified LMS (lambda, mu, and sigma) estimation procedure (Kuczmarski et al., 2002) from data obtained in nationally representative U.S. surveys conducted between 1963 and 1980 (Kuczmarski et al., 2000). More recent data were not included because of the marked increases in BMI that were seen in subsequent U.S. surveys (Flegal, Ogden, Wei, Kuczmarski, & Johnson, 2001;

☆ *Funding:* NIH Intramural Research Program Grant 1ZIAHD000641 (J. Yanovski) from the National Institute of Child Health and Human Development with supplemental funding from the Office of Behavioral and Social Sciences Research. *Disclaimers:* The opinions and assertions expressed herein are those of the author and are not to be construed as reflecting the views of the DHHS or the U.S. Public Health Service.
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http://dx.doi.org/10.1016/j.appet.2015.03.028
0195-6663/Published by Elsevier Ltd.

Troiano & Flegal, 1998). Because of the paucity of data for children at the greatest BMIs in the data sets used, the top percentile defined by the CDC 2000 growth charts is the 97th percentile.

Before 2010, CDC 2000 growth charts for ages 2 to 18 years demarcated the 85th to 94.99th percentiles for BMI as "at risk for overweight" and ≥95th BMI percentile as "overweight" (Himes & Dietz, 1994). These cut points were subsequently renamed "overweight" for the 85th to 94.99th BMI percentiles and "obese" for ≥95th BMI percentile, to be consistent with recommendations by other groups (Barlow, 2007; Barlow & Dietz, 1998; Himes & Dietz, 1994; Koplan, Liverman, & Kraak, 2005; Krebs et al., 2007; Obesity: preventing and managing the global epidemic, report of a WHO consultation, 2000; Ogden & Flegal, 2010; Physical status: The use and interpretation of anthropometry, 1995; Koplan, Liverman, & Kraak, 2004).

There are limitations to the use of BMI-based standards to define obesity (Barlow & Dietz, 1998; Barlow, 2007; Flegal, Ogden et al., 2010; Himes & Dietz, 1994; Krebs et al., 2007) because BMI cannot discriminate between lean and fat mass (Wellens et al., 1996) and thus excess body fatness cannot be measured directly from weight and height (Himes & Dietz, 1994). However, there is a high correlation between fat mass and BMI among children (Field et al., 2003; Mei et al., 2002), and the majority of children with BMI ≥95th percentile have high adiposity (Freedman, Mei, Srinivasan, Berenson, & Dietz, 2007). Nevertheless, about 25% of U.S. children with BMI ≥95th percentile do not appear to have particularly high amounts of body fat (Flegal, Ogden et al., 2010); thus BMI is a first screening tool to identify children who may be overfat. BMI percentiles may be particularly inaccurate for children belonging to some racial and ethnic minorities (Flegal, Ogden et al., 2010).

An expert committee convened by the American Medical Association proposed recognition of the 99th BMI percentile as a cut point to classify children with severe obesity who are likely to be at increased risk for cardiovascular risk factors (Barlow, 2007). However, estimates of the cut points for the 99th percentile for age and sex are considered unstable. Extreme percentiles extrapolated from the CDC-supplied LMS parameters do not match well to the empirical data for the 99th percentile. A statistically defensible cut point for severe (sometimes called "extreme") obesity based on available U.S. data is 120% of the smoothed 95th percentile (Flegal et al., 2009). Many investigators also include all adolescents with BMI ≥35 kg/m^2 in the severe/extreme obesity group (Kelly et al., 2013; Koebnick et al., 2010). Children with extreme/severe obesity are at even higher risk for the complications of obesity detailed below (Kelly et al., 2013).

Over the past 50 years, global trends suggest that the prevalence of obesity among children (using BMI-based criteria) has increased significantly (Lobstein, 2010; Wang & Lobstein, 2006). Since the 1960s, prevalence rates have quadrupled in many countries (Lobstein, Baur, & Uauy, 2004). Based on the CDC 2000 BMI standards, among those ages 2–19 years, in 2012, 31.8% had BMI ≥85th percentile, 16.9% (approximately 12.7 million children) had BMI ≥95th percentile (Ogden et al., 2014) and in 2010, 12.3% had BMI ≥97th percentile (Ogden, Carroll, Kit, & Flegal, 2012). Certain racial and ethnic minority populations, especially African Americans, Hispanics, and American Indians, are at particular risk for obesity, while Asian children appear to have lower BMI-based risk of obesity (Flegal, Carroll, Ogden, & Curtin, 2010; Ogden et al., 2014; Spiegel & Alving, 2005). Although some recent data suggest obesity rates have stabilized in children and may even have decreased in those ages 2–5 years (Flegal, Carroll, Kit, & Ogden, 2012; Flegal, Carroll et al., 2010; Ogden et al., 2010, 2012, 2014; Yanovski & Yanovski, 2011), the obesity prevalence among children and adolescents remains alarmingly high.

Consequences of pediatric obesity

Pediatric overweight and obesity are of concern because of both immediate and later onset health consequences (Daniels, 2009). Children at the highest levels of BMI are usually at the greatest risk of obesity-associated adverse health outcomes (Koplan et al., 2004). Obesity in childhood is more likely to lead to adult obesity (Freedman et al., 2007) and to the tracking of poor health throughout adulthood; thus obesity appears to be a major contributor to many preventable causes of morbidity. The risk of adult obesity appears higher for older obese children, for those with more severe obesity, and for those with obese parents. (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). Some data suggest that those with extremely high BMI percentiles (significantly above the 97th percentile) are even more likely to have tracking of obesity into adulthood (Freedman et al., 2007). Although there is variation in the estimates among studies examining the question of persistence, it appears that approximately 40% of obese children become obese adults (Freedman et al., 2004; Must & Strauss, 1999; Power, Lake, & Cole, 1997). The appearance of obesity-associated conditions in childhood has been shown to lead to an earlier onset of related medical complications (Pavkov et al., 2006). Some (Jeffreys, McCarron, Gunnell, McEwen, & Smith, 2003; Must et al., 1992) but not all (Gray, Lee, Sesso, & Batty, 2011; Juonala et al., 2011) studies suggest that pediatric obesity itself has a unique impact on later health independent of adult weight; regardless, there is unanimity that pediatric obesity is a strong risk factor for adult obesity and its complications. The current U.S. childhood obesity epidemic thus has the potential to reverse the improvements in life-expectancy that occurred during the 20th century in the U.S. (Olshansky et al., 2005) and to cause more functional disability in those who survive to old age (Alley & Chang, 2007).

Cardiovascular disease

Obese and overweight youth are more likely to have cardiovascular risk factors resulting in cardiac structural and hemodynamic alterations (Freedman et al., 2007) including hypertension (Speiser et al., 2005), increases in ventricular mass (Daniels, 2009) endothelial dysfunction, with carotid artery intimal medial thickening, and early coronary and aortic fatty streaks and fibrous plaque (Freedman et al., 2004; Tounian et al., 2001), as well as atherosclerosis (Berenson et al., 1998; Daniels, 2009; McGill et al., 2002). Analyses have suggested, however, that there is little evidence that childhood BMI is an independent risk factor for adult cardiovascular risk once adult BMI is taken into consideration (Lloyd, Langley-Evans, & McMullen, 2010).

Dyslipidemia

Childhood obesity is associated with dyslipidemia, with the most common abnormality being elevated triglycerides and decreased high-density lipoprotein (HDL) cholesterol (Daniels, 2009). Elevated low-density lipoprotein (LDL)-cholesterol is also seen in obese children; however, the association between adiposity and LDL-cholesterol is weaker than that of adiposity with triglycerides and HDL-cholesterol (Daniels, 2011). BMI is also positively associated with likelihood for LDL particle size <25.5 nm (Shimabukuro, Sunagawa, & Ohta, 2004). Childhood dyslipidemia has been shown to persist and to be a predictor of adult dyslipidemia, adult carotid intimal media thickness, and other cardiovascular disease risks (Nadeau, Maahs, Daniels, & Eckel, 2011) (Lauer, Lee, & Clarke, 1988).

Impaired glucose homeostasis

Obesity is commonly accompanied by insulin resistance and hyperinsulinemia, which precede and play a major role in the development of type 2 diabetes mellitus (T2DM) (Shulman, 2000). In children, total body fat and visceral fat are positively associated with fasting insulin (Caprio et al., 1995; Freedman et al., 1987; Gutin et al., 1994), and impaired insulin sensitivity may worsen with duration