## **Guest Editorial**

## International trends in pediatric obesity: A public health concern

## L. Preston Mercer\* and Rita DeBate

Department of Community and Family Health, College of Public Health, University of South Florida, Tampa, FL, USA

Since 1978, the number of overweight children (BMI above the 85<sup>th</sup> percentile) has tripled [1]. Moreover, 2009–2010 U.S. data reveals 12% of 2–5 year olds, 18% of 6–11, and 18.4% of adolescents 12–19 were observed as obese (BMI at or above the 95<sup>th</sup> percentile) [2]. Due to the evidence that indicates that 70% of obese adolescents will grow up to be obese adults [3], it is likely that all Americans will be considered overweight by the year 2030! [4].

Furthermore, this public health issue is quickly becoming an issue across the globe. Studies from Brazil and China indicate a shift in the occurrence of obesity and overweight from the middle classes to the poor – this trend is being replicated across many countries in Asia, Africa and Latin America [5]. Since 1975, obesity in 2–5 year olds has increased from 5% to over 15%. Early increases in BMI are associated with concurrent increases of up to 50% obesity in adults.

The public health significance of childhood overweight and obesity is noted with the associated rates of morbidity and mortality. For example, elevated BMI is closely associated with morbidity and mortality, since fat tissue can have a deleterious effect on nearly every organ system, leading to: glucose intolerance, insulin resistance, type 2 diabetes, hypertension (HBP), high cholesterol, hepatic steatosis (FLD), cholelithiasis (gallstones), sleep apnea, asthma, skin conditions, menstrual abnormalities, impaired balance, orthopedic problems, etc. Especially common is the appearance of metabolic syndrome: Raised triglycerides: > 150 mg/dL, reduced HDL cholesterol: < 40 mg/dL in males, < 50 mg/dL in females, raised blood pressure (BP): systolic BP > 130 or diastolic BP > 85 mm Hg, and raised fasting plasma glucose (FPG): > 100 mg/dL These problems lead to 300,000 premature, preventable deaths annually in United States, 2nd only to tobacco-related deaths. In developing countries, the burden of obesity and its complications is shifting rapidly towards the poor. Simultaneous malnutrition and overweight exist with obesity now 4 times more common than malnutrition in some developing countries.

1

In addition to physical health issues, overweight and obesity affect children's mental health. It is noted that overweight and obese children are observed with higher rates of depression, anxiety [5,6]; low self-esteem [1,8], suicidal ideation, and increased likelihood to engage in substance abuse [8]. Furthermore, obese children are 5x more likely to avoid participation in school activities and sports [8]. In addition, social exclusion and stigmatization become part of a cycle including school avoidance [9] which results in lower emotional, social and school functioning [8].

Subsequently, the public health significance of the rising rate of childhood overweight and obesity requires reinvestigation of the regulation of metabolism, food intake and weight gain, particularly with emphasis on new environmental factors. The neuroregulation of food intake is complex, hierarchical and redundant, including internal and external factors, psychological factors, molecular signals, brain cen-

<sup>\*</sup>Correspondind author: L. Preston Mercer, Department of Community and Family Health, College of Public Health, University of South Florida, Tampa, FL 33612, USA. Tel.: +1 813 974 8888; Fax: +1 813 974 8889; E-mail: pmercer@usf.edu. The Lawton and Rhea Chiles Center for Healthy Mothers and Babies, 3111 E. Fletcher Avenue Tampa, FL 33613, USA.

ters, cultural cues, gut, nervous system interactions, genetic makeup and the availability/palatability of food [9]. Biological phenomena are influenced by multiple etiologies, interacting in complex fashion. However, the generational increases in weight would indicate influential, recently appearing environmental factors, since most researchers feel that the human genome is more stable on the short term.

Some factors, characterized by the phrase "healthy mothers – healthy babies', include the recognition that the child inherits a gene pool, a lifestyle and a developmental trajectory from the mother. It is estimated that approximately 25 to 40% of BMI is heritable [10]. Thus, a child's weight outcome may be influenced when he/she is in utero, whereby lack of sleep, the intrauterine environment, and a decrease in variability of ambient temperatures in combination with excess calories and a decrease in hard labor, may have influenced these increases in adiposity [11]. Being an infant born to a mother with diabetes, even if not large for gestational age, conveys an additional risk [12-16] for obesity. In addition to intrauterine factors, formula feeding with solid foods introduced before 4 months predisposes the child to a four-fold increase in obesity while breast feeding with solid foods after six months produces better outcomes. Sugary drinks and inactivity are implicated in development of obesity.

Currently, prevention and treatment efforts have emphasized interventions which are focused on changing behaviors pertaining to physical activity and healthy eating. For example, pre-diabetic mothers can improve outcomes by going into pregnancies with lower weight – even seven to ten pounds lower is significant. Breast feeding and monitoring the addition of solid foods is helpful. Avoiding sugary drinks – juices and soda pop – can be important. Lifestyle, such as TV time and video games could be "bought" by equal times of physical activities. Many suggestions are available [17].

However, emerging evidence has increased the emphasis of pre-emptive public health. New areas of research into the rise in obesity include nutritional genomics, early exposure to excessive antibiotics changing the microbiome, epigenetics and environmental obesogens [18] — environmental pollutants, which cause obesity by altering metabolic and homeostatic regulations, causing weight increase. They also disrupt appetite controls, cause adipocyte or fat cell enlargement and stimulate the increase of adipocyte cells in the body. Interestingly, it has been re-

cently observed that cesarean deliveries can double the chance of obesity in an infant. Dietary advice based on the human genome will become more prevalent and new pharmacological interventions may be developed. Many researchers view this challenge as of highest importance to avoid raising the first generation that will live a shorter time than their parents.

In this edition, we present discussions of relevant areas: an overview of recent data, gestational diabetes, environmental obesogens, neurochemical signaling, classroom influences and cultural influences: all have been shown to be influential and offer insights into areas which combine to answer a serious health issue with long-term ramifications for world public health

## References

- [1] Chou SY, Rashad I, Grossman M. Fast-food restaurant advertising on television and its influence on childhood obesity. National Bureau of Economic Research, Working Paper Retrieved from: http://www.nber.org/papers/w11879.
- [2] Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among us children and adolescents 1999-2010. JAMA 2012; 307(5): 483-490.
- [3] Dehghan MN, Akhtar-Danesh AT, Merchant. Childhood obesity, prevalence and prevention. Nutr J 2005; 4(1): 24.
- [4] Wang Y, Beydoun MA, Liang L, Caballero LB, Shiriki SK, Kumanyika K, Will All Americans Become Overweight or Obese? Obesity 2008 16: 2323-30.
- [5] Popkin B, The World Is Fat: new dynamics of global obesity, Department of Nutrition, School of Public Health and Medicine, Department of Economics, The University of North Carolina at Chapel Hill, Retrieved from: http://www. yaleruddcenter.org/resources/upload/docs/seminar/2009-spri ng/Slides/Popkin.pdf.
- [6] Gundersen C, Mahatmya D, Garasky S, Lohman B. Linking psychosocial stressors and childhood obesity. Obesity Rev 2011; 12(5): e54-e63.
- [7] Wyatt, SB, Winters KP, Dubbert PM. Overweight and obesity: prevalence, consequences, and causes of a growing public health problem. Am J Med Sci 2006; 331(4): 166-74.
- [8] Reilly JJ. Obesity in childhood and adolescence: evidence based clinical and public health perspectives. Postgrad med J 2006; 82(969): 429-37.
- [9] Mercer LP. Preston Histamine and the neuroregulation of food intake. Nutrition 1997; 13: 581-2.
- [10] Wieting JM. Cause and effect in childhood obesity: solutions for a national epidemic. J Am Osteopath Assoc 2008; 108(10): 545-52.
- [11] Huang JS, Lee TA, Lu MC. Prenatal programming of child-hood overweight and obesity. Matern Child Health J 2007; 11(5): 461-73.
- [12] Reilly JJ, Childhood obesity: An overview. Children & Society 2007; 21(5): 390-6.
- [13] Anderson PM, Butcher KF. Childhood obesity: trends and potential causes. Future Child 2006; 16(1): 19-45.
- [14] Apovian CM. The causes, prevalence, and treatment of obesity revisited in 2009: what have we learned so far? Am J Clin Nutr 2010; 91(1): 277S-79S.

- [15] Biro FM, Wien M. Childhood obesity and adult morbidities. Am J Clin Nutr 2010; 91(5): 1499S-1505S.
- [16] Han JC, Lawlor DA, Kimm S. Childhood obesity. Lancet 2010; 375(9727): 1737-48.
- [17] Kumanyika SK. Environmental influences on childhood
- obesity: ethnic and cultural influences in context. Physiol Behav 2008; 94(1): 61-70.
- [18] Gr ün F, Blumberg B. Endocrine disrupters as obesogens. Mol Cell Endocrinol 2009; 304(1-2): 19-29.