



Child obesity

Last update: September 2018

Topic Editor:

Jennifer Orlet Fisher, PhD, Temple University, Center for Obesity Research and Education, USA

Table of content

Synthesis	5
<hr/>	
Obesity at an Early Age and Its Impact on Child Development	9
¹ JEAN-PHILIPPE CHAPUT, PHD, ² ANGELO, TREMBLAY, PHD, SEPTEMBER 2018	
<hr/>	
Obesity Prevention in the Preschool Years	18
¹ ANDREA DESILVA-SANIGORSKI, PHD, ² KAREN CAMPBELL, PHD, APRIL 2012	
<hr/>	
Appetitive Traits and Weight in Children	22
LUCY COOKE, PHD, APRIL 2012	
<hr/>	
Environmental/Family/Psychosocial Influences on Physical Activity in Young Children	28
TRINA HINKLEY, PHD, APRIL 2012	
<hr/>	
The Prenatal Origins of Obesity: Evidence and Opportunities for Prevention	36
VÉRONIQUE GINGRAS, RD, PHD, EMILY OKEN, MD, MPH, JULY 2019	
<hr/>	
Early Onset Obesity in Infants and Children: Nature, Nurture or Both? Commentary on Cooke, Hinkley, Chaput & Tremblay, Oken, Paul, Savage, Anzman-Frasca, Birch, and de Silva-Sanigorski & Campbell	44
MICHAEL I. GORAN, PHD, KELLY A. DUMKE, MS, JULY 2012	
<hr/>	
Obesity Prevention During Infancy: A Change of Focus	51
¹ IAN M. PAUL, MD, MSC, ² JENNIFER S. SAVAGE, PHD, ² STEPHANIE ANZMAN-FRASCA, PHD, ² LEANN L. BIRCH, PHD, APRIL 2012	
<hr/>	

Topic funded by:

LAWSON
FOUNDATION

Synthesis

How important is it?

Obesity is characterized by an excessive accumulation of body fat resulting from an imbalance between energy intake and spending. The reduced need for being physically active in the contemporary way of life, combined with the availability of affordable high-calorie food, contributes to the creation of an environment that increases the risk of obesity (“obesogenic” environment) in developed countries and, increasingly, in the developing world. The prevalence of obesity rose in the last decades among all age groups, including young children. In 2010, about 42 million children under the age of five were estimated overweight or obese, of whom almost 35 million were from developing countries. Once thought naturally physically active, young children spend in fact 80% or more of their time being sedentary. Children are considered overweight when their body mass index (BMI: the weight in kilograms divided by the height in meters) falls between the 85th and 94th percentile in established charts, and obese when their BMI falls at the 95th percentile or more, adjusted for age and sex. It is estimated that almost 10% of children younger than two years are obese and that over 25% of children aged between 2 and 5 years are overweight in the U.S. Yet, these prevalence rates should be interpreted with caution due to the variance across the U.S. populations. In low-income populations (e.g., Hispanic populations), the emergence of childhood obesity is highest and emerges most rapidly in the first few years of life.

Childhood obesity is associated with adult obesity, itself associated with hypertension, coronary artery disease, and diabetes. Obesity is very difficult to treat once developed and puts affected children at risk for lifelong health problems and reduced quality of life as well as social stigma and exclusion. Thus, the individual and social cost (e.g., health care) of obesity is a heavy one. The focus of childhood obesity research is on the causes and consequences of obesity, successful intervention programs, and effective prevention strategies.

What do we know?

If obesity is the direct result of insufficient energy expenditure relative to energy intake, the causes of this imbalance are complex and vary between individuals and social contexts. The family is a first and fundamental context for understanding children’s eating and activity behaviours. Parents provide children with genes as well as the environments in which behavioural

habits develop. Obese mothers tend to have obese children, but disentangling the genetic vs. environmental contributions remains a challenge.

While genetic contributions to body size are widely acknowledged, recent research has also revealed that eating behaviours are partially under genetic control. Individual differences in appetitive traits (eating speed, sensitivity to fullness, enjoyment of food, etc.) and eating behaviours (e.g., eating snack food when not hungry) may be a causal factor in the development of obesity. These differences could explain in part why the features of the environment that increase the risk of obesity do not affect all individuals equally; some people are more at risk than others because of their appetitive traits and eating behaviours.

Where the environment is concerned, children are thus growing in a world that gives them many opportunities for passive entertainment and provides an abundance of food. Studies of the activity level of very young children are few but their results indicate that infants and toddlers follow the trend toward lower levels of physical activity and higher level of screen-based entertainment. Moreover, changing social norms have resulted in a diminution of the time children devote to outdoor active physical play in favour of indoor play and/or academic activities in kindergarten and school.

Parental practices can reduce the risk of developing obesity in young children when encouraging physical activity, limiting the time allowed to passive activities such as television watching and other screen-based activities, and by being role models. Family practices also influence children's eating behaviour, notably because parents decide the kind of food, the amount served to children, and the timing of meals. Some studies suggest that when parents restrict children's access to palatable but unhealthy food they increase children's desire for it. However, other studies suggest an interactive relationship instead of a causal one: parents' feeding practices would be responsive to, rather than causal of, the eating behaviour of their children. The control that parents exert on the family environment influences the children's exposure to the environmental risks that foster overweight and obesity development.

Exposition to an environment that increases the risk of obesity begins before birth. Some studies suggest that features of the intrauterine environment (including exposure to maternal obesity, smoking and diabetes) can increase the child's long-term risk of obesity. A rapid weight gain during infancy could also predispose to obesity in later life. However, there is evidence that today's health professionals still emphasize weight gain in infants, even though nutritional

deficiency is rarely diagnosed in developed countries whereas obesity diagnoses keep rising.

Sleep has also been associated with obesity. Children sleep duration has declined in the past decades and there is growing evidence that short sleep duration is a determinant of obesity, possibly through its effect on hormone regulation and because less sleep means longer exposure to the features of the environment associated with obesity, like high-calorie food and passive entertainment. A study showed that an intervention directed at new mothers that addressed solid food introduction and soothing/sleeping behaviours resulted in lower weight-for-length in infants, though interventions focusing solely on feeding or sleeping had no effect.

What can be done?

Intervention studies aim to find strategies that would mitigate the risks and reduce the impact of obesity on children's health. It is worth noting that so far, no intervention strategies has ever proven to reduce body weight on a long-term basis for a significant number of individuals. An emphasis on weight loss can create more problems, including repeating cycles of weight loss and gain, eating disorders, and reduced self-esteem, whereas inducing healthy behaviours may improve health regardless of weight loss. Therefore, even if treatment is important, prevention of overweight and obesity should be the primary target of intervention programs.

Prevention

Prevention of obesity should begin before birth and account for genetic and prenatal conditions. Intervention with overweight women before and during pregnancy aiming at diet and behaviour change could reduce exposition of the foetus to smoking, obesity and diabetes. Current policy recommendations should be examined regarding the monitoring of weight gain by pregnant women and infants. The focus of this monitoring should shift from ensuring adequate weight gain to prevention of obesity. For example, although it is recommended to wake up newborns for feeding every three to four hours, this practice should be stopped when the baby has regained his or her birth weight and is growing steadily. Training new mothers in the introduction of solid food and appropriate soothing/sleeping behaviours could also help control infants' weight gain.

Prevention programs can be extended throughout the preschool years by targeting the activity level and dietary habits of children. Some studies showed that short-term reduction in saturated fat intake can be achieved, although the effect disappeared after two years and effectiveness seemed to vary among ethnic groups. The combined results of studies showed that intervention

directed at young children can result in a decrease of weight-for-length as measured by the Body Mass Index (BMI). Interventions aimed at preventing obesity should also promote sufficient sleep.

Prevention programs can also target the aspects of children's environment that increase the risk of developing obesity, such as the availability of passive entertainment and high-calorie food. Children should be encouraged to play outside, but introducing new play equipment is not enough to induce more physical activity. Parents can play an important role and improve their family environment by setting rules, modelling healthy behaviours and limiting the use of screen-based entertainment. Schools could also help reduce the risk of developing obesity by leaving children more time for active physical play.

In short, the current epidemic of obesity must be addressed by the following multifaceted interventions:

- Changing the medical monitoring of infants' weight gain in a way that emphasizes obesity prevention;
- Encouraging physical activity in young children;
- Improving children's sleep habits and eating behaviours;
- Emphasizing health improvement rather than weight loss when treating obesity.
- Taking into consideration genetic conditions that render children more or less vulnerable to obesity.

Obesity at an Early Age and Its Impact on Child Development

¹Jean-Philippe Chaput, PhD, ²Angelo, Tremblay, PhD

¹University of Ottawa, Canada, ²Université Laval, Canada

September 2018, 2e éd. rév.

Introduction

Obesity is characterized by the accumulation of excess body fat and can be conceptualized as the physical manifestation of chronic energy excess. Since 1980, the prevalence of obesity has doubled in more than 70 countries and has continuously increased in most other countries.¹ In 2016, 50 million girls and 74 million boys worldwide had obesity.² Although the rising trends in childhood obesity have plateaued in many high-income countries, albeit at high levels, they have accelerated in parts of Asia.² The current and future health burden of obesity is considerable, and experts recommend that prevention and intervention strategies should begin at the earliest age possible.³

Subject

A complex and interacting system of factors contributes to increasing rates of overweight and obesity – biological, behavioural, social, psychological, technological, environmental, economic and cultural – operating at all levels from the individual to the family to society as a whole. Public, private, non-profit and community sectors, parents, school boards and municipal governments all have a role to play, and their collective efforts will be required to start the significant society-wide shift needed to reverse the trend of childhood obesity. Attenuating these high rates of child obesity is a high priority in many countries not only from a population health perspective, but from a health care system's economic perspective. It is well known that obesity persists from childhood to adolescence to adulthood.⁴ Unfortunately, it is very difficult to treat once it has developed. This emphasizes the importance of prevention early in life and mathematical modeling suggests that targeted obesity interventions for young children (0-6 years) could yield considerable cost savings and important improvements in well-being.⁵

Problems

Obesity results in much suffering to individuals affected by this condition. Evidence from many studies indicates that childhood obesity contributes to the early development of a number of conditions, such as type 2 diabetes, dyslipidemia, hypertension, sleep-disordered breathing, nonalcoholic fatty liver disease, and polycystic ovarian syndrome.⁶⁻⁸ The greater risk of health complications associated with early morbidity affects normal childhood development and quality of life and thus the long-term health care burden is extraordinary if we include the obesity associated chronic co-morbid conditions. It has been projected that the current generation of children will be the first in modern history to see a shorter life-expectancy than their parents.⁹ Obesity is also associated with stigma, discrimination and reduced psychological well-being.¹⁰ Consequences of obesity stigma, such as isolation or social withdrawal, could contribute to the exacerbation of obesity through psychological vulnerabilities that increase the likelihood of over-eating and sedentary activity.

Research Context

There is no doubt that obesity is an important public health issue and strategies to address obesity and *obesogenic environments* will require a multifaceted, long-term approach involving interventions that operate at multiple levels and in complementary ways. Despite an impressive amount of research over the past decades, no methods have proven to reduce body weight over the long term for a significant number of individuals. Our failure to reverse the trend in obesity prevalence has helped us in realizing that a focus on weight loss as an indicator of success is not only ineffective at producing thinner, healthier bodies, but also damaging, contributing to food and body preoccupation, repeated cycles of weight loss and regain, reduced self-esteem, eating disorders, and weight stigmatization and discrimination.¹¹ There is an accumulating body of evidence showing that most health outcomes can be improved through changing health behaviours, regardless of whether weight is lost.^{12,13} However, an important challenge of today's world is that our so-called "obesogenic environment" encourages the consumption of energy and discourages the expenditure of energy. Modern, computer-dependent, sleep-deprived, physically-inactive humans live chronically stressed in a society of food abundance.^{14,15} The excess weight gain observed in prone individuals should be perceived as a normal physiological adaptation to a changed environment, rather than a pathological process.¹⁶ In other words, weight gain is a sign of the modern way of living or a "collateral damage" in the struggle for modernity. Accordingly, prevention and treatment strategies for obesity should ideally focus on modifying the environment and lifestyle in order to make the healthy choice the easy one.

Key Research Questions

There is accumulating evidence showing the negative effects of obesity at an early age on many health-related indicators later in life and on the development of the child. Additionally, there is increased recognition of the importance of active play for the motor, cognitive, language, social and emotional development of young children.¹⁷ However, many critical questions will need to be addressed, including:

- Are the benefits of weight loss interventions attributed to weight loss per se or to the changes in lifestyle habits?
- What are the root causes of weight gain in children? Are over-eating and lack of physical activity symptoms of something else? If yes, can we really impact body weight over the long term by simply targeting energy intake and energy expenditure?
- What are the main barriers faced by children that preclude them from spontaneously adopting a healthy lifestyle?
- Can we address childhood obesity without first addressing adult obesity?

Despite all the research on childhood obesity, we have not been able to reverse the epidemic. One explanation may be that obesity, like all health conditions that are primarily socially determined, resists durable solutions until there is a change in societal norms and the values underlying those norms.¹⁸ The childhood obesity epidemic is just one symptom of our modern way of living.

Reversing the trend may require that we apply a new approach to improving child health in the 21st century. One approach is to make societal changes to enhance human well-being rather than to prevent a particular symptom, such as childhood obesity. In the process, we may address obesity and other socially-determined health conditions while preventing new ones from emerging.

Recent Research Results

An impressive body of evidence on the association between childhood obesity and its impact on child development has been published over the past years. Optimal child development involves many dimensions, from physical to emotional development, and our intention is not to cover the literature on this issue here but rather to highlight two topics of interest: active play and short sleep duration.

Active play

Decades of research have shown that play is an important mediator in the physical, social, cognitive, and language development of young children.¹⁷ In spite of this, play faces many threats in America. The growing emphasis on standards, assessment and accountability in schools has led to a reduction in outdoor and active physical play. In many schools and centres, play has been eliminated to make room for quieter academic learning. Preschools and kindergartens in public school settings have become particularly regimented and adult-directed, with teachers feeling compelled to increase literacy and numeracy instruction at the expense of play time.¹⁷ Passive television viewing and use of other media also are replacing active play and have even been found to interrupt the play of young infants.¹⁷ Unfortunately, indoor active video gaming seems to replace outdoor active play in today's society. Given that physical play is important in physical development, learning, emotional and social development, we should embrace and defend play as a crucial part of children's daily lives, in and out of school. Active play should be promoted as part of a healthy and balanced lifestyle, not for body weight stability.

Short sleep duration

Sleep curtailment has become an endemic condition in modern societies, with population statistics revealing that sleep duration has decreased by more than an hour over the past few decades in children.¹⁹ A growing body of evidence shows that short sleep duration is associated with mental distress, depression, anxiety, weight gain, hypertension, diabetes, high cholesterol levels, premature death, and adverse health behaviours such as physical inactivity and poor eating habits.^{20,21} The evidence that short sleep duration is a determinant of obesity is accumulating.²²⁻²⁴ A number of mechanisms have been invoked to account for this association, including an up-regulation of appetite-stimulating hormones, a longer exposure to an obesogenic environment as well as a decrease in spontaneous physical activity.^{23,24} Thus, insufficient sleep is an under-recognized public health problem that has a cumulative effect on physical and mental health. Sleep is not a waste of time – its beneficial effects far exceed the restoration and maintenance of tissue structure and function. It is important to remember that a good night's sleep is the “normal” biological condition. No one can effectively argue that lack of sleep is healthy, and therefore there is minimal risk in taking a pragmatic approach and encouraging a good night's sleep as an adjunct to other health promotion measures.

Conclusions

Prevention of obesity in children should be the first line of treatment. Given the complex and multi-factorial nature of obesity, preventive interventions should target to root causes of the

problem in order to be successful. The Institute of Medicine published a report titled Early Childhood Obesity Prevention Policies that outlines several policy recommendations and potential actions for implementation designed to prevent obesity in infancy and early childhood by promoting healthy environments for young children.²⁵ The recommendations of this report are shown in the table below.

Implications

The modest effects of past health education interventions have increased interest in environmental and policy approaches to increase physical activity, decrease sedentary behaviour and/or reduce dietary energy intake to prevent obesity. These approaches attempt to alter the social, regulatory or physical environments resulting in individuals adopting more healthful behaviours, whether or not they are aware of their decisions to adopt those behaviours. Environmental and policy approaches may be particularly attractive for helping to shape child behaviours because (1) children spend a large part of their days in a relatively small number of settings that are susceptible to environmental and policy changes (e.g., home, school, transportation to/from school, child care and after-school programs); (2) children are often considered unable to make responsible behavioural decisions for themselves; and (3) presumed child vulnerabilities justify both pre-emptive and remedial protective actions by parents, institutions and policy-makers. Environmental and policy solutions designed to prevent obesity at an early age are enticing to many policy-makers at all levels of society, from parents to international agencies. However, implementing new strategies and policies without evidence of efficacy or effectiveness may lead to large investments of resources, effort and time that may or may not result in any benefits.

Table. Goals and recommendations from the 2011 Institute of Medicine’s Report on Early Childhood Obesity Prevention Policies.

Goals

Recommandations

- Healthcare providers should measure weight and length or height in a standardized way, plotted on World Health Organization growth charts (ages 0-23 months) or Centers for Disease Control and Prevention growth charts (ages 24-59 months), as part of every well-child visit.
1. Assess, monitor, and track growth from birth to age 5

Healthcare professionals should consider 1) children’s attained weight-for-length or BMI \geq 85th percentile, 2) children’s rate of weight gain, and 3) parental weight status as risk factors in assessing which young children are at highest risk of later obesity and its adverse consequences.
 2. Increase physical activity in young children

Child care regulatory agencies should require child care providers and early childhood educators to provide infants, toddlers, and preschool children with opportunities to be physically active throughout the day.

The community and its built environment should promote physical activity for children from birth to age 5.
 3. Decrease sedentary behaviour in young children.

Child care regulatory agencies should require child care providers and early childhood educators to allow infants, toddlers, and preschoolers to move freely by limiting the use of equipment that restricts infants’ movement and by implementing appropriate strategies to ensure that the amount of time toddlers and preschoolers spend sitting or standing still is limited.
 4. Help adults increase physical activity and decrease sedentary behaviour in young children.

Health and education professionals providing guidance to parents of young children and those working with young children should be trained in ways to increase children’s physical activity and decrease their sedentary behaviour, and in how to counsel parents about their children’s physical activity.

Adults who work with infants and their families should promote and support exclusive breastfeeding for 6 months and continuation of breastfeeding in conjunction with complementary foods for 1 year or more.

5. Promote the consumption of a variety of nutritious food, and encourage and support breastfeeding during infancy.

To ensure that child care facilities provide a variety of healthy foods and age-appropriate portion sizes in an environment that encourages children and staff to consume a healthy diet, child care regulatory agencies should require that all meals, snacks, and beverages served by early childhood programs be consistent with the Child and Adult Care Food Program meal patterns and safe drinking water be available and accessible to the children.

The Department of Health and Human Services and the U.S. Department of Agriculture should establish dietary guidelines for children from birth to age 2 years in future releases of the Dietary Guidelines for Americans.

6. Create a healthful eating environment that is responsive to children's hunger and fullness cues.

State child care regulatory agencies should require that child care providers and early childhood educators practice responsive feeding.

7. Ensure access to affordable healthy foods for all children.

Government agencies should promote access to affordable healthy foods for infants and young children from birth to age 5 in all neighbourhoods, including those in low-income areas, by maximizing participation in federal nutrition assistance programs and increasing access to healthy foods at the community level.

8. Help adults to support children's healthy eating.

Health and education professionals providing guidance to parents of young children and those working with young children should be trained and educated and have the right tools to increase children's healthy eating and counsel parents about their children's diet.

9. Limit young children’s screen time and exposure to food and beverage marketing. Adults working with children should limit screen time, including television, cell phone, or digital media, to less than two hours per day for children aged 2-5. Healthcare providers should counsel parents and children’s caregivers not to permit televisions, computers, or other digital media devices in children’s bedrooms or other sleeping areas.
10. Promote age-appropriate sleep durations among children. Child care regulatory agencies should require child care providers to adopt practices that promote age-appropriate sleep durations. Health and education professionals should be trained in how to counsel parents about their children’s age-appropriate sleep durations.

Adapted from the 2011 Institute of Medicine Report.²⁵

References

1. Bovet P, Chiolero A, Gedeon J. Health effects of overweight and obesity in 195 countries. *N Engl J Med* 2017;377:1495-1496.
2. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2016 population-based measurement studies in 28.9 million children, adolescents, and adults. *Lancet* 2017; 390: 2627-2642.
3. Gurnani M, Birken C, Hamilton J. Childhood obesity: causes, consequences, and management? *Pediatr Clin N Am* 2015;62:821-840.
4. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obes Rev* 2008;9:474-488.
5. Ma S, Frick KD. A simulation of affordability and effectiveness of childhood obesity interventions. *Acad Pediatr* 2011;11:342-350.
6. Lumeng JC, Taveras EM, Birch L, Yanovski SZ. Prevention of obesity in infancy and early childhood: a National Institutes of Health workshop. *JAMA Pediatr* 2015;169:484-490.
7. Han JC, Lawlor DA, Kimm SY. Childhood obesity. *Lancet* 2010;375:1737-1748.
8. Morrison K, Chanoine JP. Clinical evaluation of obese children and adolescents. *CMAJ* 2007;176(Suppl 8):45-49.
9. Daniels SR. The consequences of childhood overweight and obesity. *Future Child* 2006;16:47-49.
10. Puhl RM, Heuer CA. Obesity stigma: important considerations for public health. *Am J Public Health* 2010;100:1019-1028.
11. Bacon L, Aphramor L. Weight science: evaluating the evidence for a paradigm shift. *Nutr J* 2011;10:9.
12. Bacon L, Stern J, Van Loan M, Keim N. Size acceptance and intuitive eating improve health for obese, female chronic dieters. *J Am Diet Assoc* 2005;105:929-936.

13. Gaesser GA. Exercise for prevention and treatment of cardiovascular disease, type 2 diabetes, and metabolic syndrome. *Curr Diab Rep* 2007;7:14-19.
14. Chaput JP, Klingenberg L, Astrup A, Sjödín AM. Modern sedentary activities promote overconsumption of food in our current obesogenic environment. *Obes Rev* 2011;12:e12-20.
15. Siervo M, Wells JC, Cizza G. The contribution of psychological stress to the obesity epidemic: an evolutionary approach. *Horm Metab Res* 2009;41:261-270.
16. Chaput JP, Doucet E, Tremblay A. Obesity: a disease or a biological adaptation? An update. *Obes Rev* 2012;13:681-691.
17. Tremblay MS, Gray C, Babcock S, Barnes J, Bradstreet CC, Carr D, Chabot G, Choquette L, Chorney D, Collyer C, Herrington S, Janson K, Janssen I, Larouche R, Pickett W, Power M, Sandseter EB, Simon B, Brussoni M. Position statement on active outdoor play. *Int J Environ Res Public Health* 2015;12:6475-6505.
18. Whitaker R. The childhood obesity epidemic: lessons for preventing socially determined health conditions. *Arch Pediatr Adolesc Med* 2011;165:973-975.
19. Matricciani L, Olds T, Petkov J. In search of lost sleep: secular trends in the sleep time of school-aged children and adolescents. *Sleep Med Rev* 2012;16:203-211.
20. Chaput JP, Klingenberg L, Sjödín A. Do all sedentary activities lead to weight gain: sleep does not. *Curr Opin Clin Metab Care* 2010;13:601-607.
21. Chaput JP, Gray CE, Poitras VJ, Carson V, Gruber R, Olds T, Weiss SK, Connor Gorber S, Kho ME, Sampson M, Belanger K, Eryuslu S, Callender L, Tremblay MS. Systematic review. Am J Prev Med 2010; 38: 349. of the relationships between sleep duration and health indicators in school-aged children and youth. *Appl Physiol Nutr Metab* 2016; 41(6 Suppl 3):S266-282.
22. Li L, Zhang S, Huang Y, Chen K. Sleep duration and obesity in children: A systematic review and meta-analysis of prospective cohort studies. *J Paediatr Child Health* 2017;53:378-385.
23. Chaput JP. Is sleep deprivation a contributor to obesity in children? *Eat Weight Disord* 2016;21:5-11.
24. Chaput JP, Dutil C. Lack of sleep as a contributor to obesity in adolescents: impacts on eating and activity behaviors. *Int J Behav Nutr Phys Act* 2016;13:103.
25. Institute of Medicine. *Early Childhood Obesity Prevention Policies*. Birch LL, Parker L, Burns A, eds. Committee on Obesity Prevention Policies for Young Children. Washington, DC: The National Academies Press; 2011. <https://doi.org/10.17226/13124>.

Obesity Prevention in the Preschool Years

¹Andrea DeSilva-Sanigorski, PhD, ²Karen Campbell, PhD

¹University of Melbourne, Australia, ²Centre for Physical Activity and Nutrition Research, Deakin University, Australia

April 2012

Introduction and Subject

The preschool years are critically important for child health, developmental, learning and social outcomes. From about the age of two, many children spend increasing amounts of time away from their primary caregivers in early childhood settings such as day care, kindergartens, nurseries and early learning centres.^{1,2} These settings, along with the family/home environment influence children's dietary intake, physical activity and in turn energy balance, and as can help promote healthy behaviours and healthy weight, and reduce the risk of developing childhood obesity.¹⁻³

Recent Research Results

The results from our recent systematic review of the obesity prevention evidence⁴ and studies emerging in the literature more recently, suggest that obesity prevention in the early years can be effective. The recent Cochrane systematic review⁴ included controlled studies with a duration of at least 12 months, and a minimum of six clusters (if a *RCT [randomized controlled trial]*). Eight studies were included which targeted young children (0-5 years). Analysis showed that children in the intervention group had a smaller increase or a larger decrease in *body mass index* (BMI) from pre- to post- intervention compared to the children in the control group. Overall, the children in the intervention group had a change in BMI that was 0.26 units less than the control group. For a preschool child aged 3.7 years with a BMI of 16.3, this represents a difference in BMI of 1.6%. At a population-level this result is very encouraging, however there was a lot of variability in the studies, with some interventions based in homes, and others in education and healthcare settings. When we looked at these separately we found those that were either home-based or involved a healthcare setting^{5,6} produced bigger effects than the interventions conducted in an education-setting, although the number of studies is small.

When examining the individual studies, only two studies reported significant differences in measures of *adiposity* between groups immediately post-intervention;^{6,7} these were also the

longest-running studies in the group. Although based on only a few studies, and not all individual studies showed significant differences, these results suggest that for children aged 0-5 years, interventions set outside education settings are possibly more effective, which may relate to a number of factors including the level of parent engagement. This finding is consistent with Hesketh and Campbell's previous review.⁸ Further detailed exploration of these studies is needed to identify effective program components.⁹

Five of the eight studies incorporated both diet and physical activity intervention strategies; three were physical activity interventions only. All but one of the studies had short intervention periods (< one year; six studies \leq 24 weeks), with only Jouret⁷ having a longer intervention period (two years). Of the eight study designs, theoretical basis was explicitly reported in one¹⁰ however, we can surmise that behaviour change theory informed the design of five of the studies and environmental change models seemed to inform the design of two of the studies.⁴

On balance, quite modest behavioural impacts were achieved from the interventions in this age group. Dietary changes were reported in only two studies.^{6,11} Fitzgibbon reported that the children who received interventions had a significantly lower intake of saturated fat at one-year follow-up ($P = 0.002$), but not post-intervention or at two-year follow-up.¹¹ When the same intervention was conducted with a Latino community these same results were not observed.¹² The intervention by Keller resulted in significantly lower energy intake and percentage protein intake in the intervention group post-intervention.⁶ Of the three interventions targeting physical activity-related behaviours, the only positive impact was observed in the study by Reilly, and was specific to improved fundamental movement skills.¹³

Other evidence

The Cochrane review includes childhood obesity prevention studies published until early 2010. Since that time other controlled trials in preschool aged children have emerged in the literature. One such study is Romp & Chomp.¹⁴ This was a community-wide Australian intervention with a focus on environmental determinants of childhood obesity. It targeted children aged 0-5 years and the intervention program was delivered through child care (centre-based and family-based day care), education (preschools) and health (universal child health checks) services. The outcome evaluation showed an increase in the prevalence of overweight/obesity from baseline to follow up in all the groups, but this increase was significantly lower in both the 2-year-old and 3.5-year-old intervention groups (1.8 and 2.7 percentage points lower than the comparison groups, respectively). In addition, overall dietary patterns were improved as a result of the intervention.¹⁴

When exploring potential mediating pathways we observed that in at least one of the children's settings, family day care, there were positive improvements in the environment which significantly promoted active play and reduced screen-based sedentary activities. These included the implementation of rules and guidelines, more care provider practices supporting children's positive meal experiences, fewer unhealthy food items allowed, increased staff training in nutrition and physical activity and less unhealthy caregiver practices such as rewarding children with food.¹⁵ When changes in the kindergarten settings were examined, similar environmental changes were demonstrated, although there was stronger parent engagement in this setting, compared to the child care setting.

Conclusion and Implications

Obesity prevention efforts in preschool children have shown promising results, although the number of studies is small and lower quality study designs limit our ability to make far-reaching recommendations. It is clear that further high-quality research is needed to enhance our knowledge of which interventions (and specific intervention components) are the most effective, cost-effective, safe and equitable for this age group, and how best to then embed them into current practice and systems so that all children benefit.

References

1. Mo-Suwan L, de Silva-Sanigorski AM. Obesity prevention interventions in early childhood. In: Waters S, Uuay, Seidell, editor. *Preventing childhood obesity: evidence, policy and practice*. Wiley Blackwell; 2010.
2. de Silva-Sanigorski AM, Corvalan, Uauy R. Obesity Prevention and control in early childhood and the practicalities of working in early childhood settings. In: Waters S, Uuay, Seidell editor. *Preventing childhood obesity: evidence, policy and practice*. Wiley Blackwell; 2010.
3. Hawkins SS LC. A review of risk factors for overweight in preschool children: a policy perspective. *Int J Pediatr Obes*. 2006;1(4):195-209.
4. Waters E, de Silva-Sanigorski AM, Hall BJ, Brown T, Campbell KJ, Gao Y, et al. Interventions for preventing obesity in children. *Cochrane Database of Systematic Reviews* 2011(12. Art. No.:CD001871).
5. Harvey-Berino J, Rourke J. Obesity prevention in preschool native-american children: a pilot study using home visiting. *Obesity Research*. 2003;11(5):606-11.
6. Keller A, Klossek A, Gausche R, Hoepffner W, Kiess W, Keller E. Prevention for obesity in childhood. *Deutsche Medizinische Wochenschrift*. 2009;134(1/2):13-8.
7. Jouret B, Ahluwalia N, Dupuy M, Cristini C, Nãgre-Pages L, Grandjean H, et al. Prevention of overweight in preschool children: results of kindergarten-based interventions. *International Journal Of Obesity* (2005). 2009;33(10):1075-83.
8. Hesketh KD, Campbell KJ. Interventions to prevent obesity in 0-5 year olds: an updated systematic review of the literature. *Obesity* (Silver Spring). 2010 Feb;18 Suppl 1:S27-35.
9. Waters E, Hall BJ, Armstrong R, Doyle J, Pettman TL, de Silva-Sanigorski A. Essential components of public health evidence reviews: capturing intervention complexity, implementation, economics and equity. *Journal of public health* (Oxford,

England). 2011 Sep;33(3):462-5.

10. Fitzgibbon ML, Stolley M. Promoting health in an unhealthful environment: lifestyle challenges for children and adolescents. *J Am Diet Assoc.* 2006;106(4):518-22.
11. Fitzgibbon ML, Stolley MR, Schiffer L, Van Horn L, KauferChristoffel K, Dyer A. Two-year follow-up results for Hip-Hop to Health Jr.: a randomized controlled trial for overweight prevention in preschool minority children. *J Pediatr.* 2005 May;146(5):618-25.
12. Fitzgibbon ML, Stolley MR, Schiffer L, Van Horn L, KauferChristoffel K, Dyer A. Hip-Hop to Health Jr. for Latino preschool children. *Obesity (Silver Spring).* 2006 Sep;14(9):1616-25.
13. Reilly JJ, Kelly L, Montgomery C, Williamson A, Fisher A, McColl JH, et al. Physical activity to prevent obesity in young children: cluster randomised controlled trial. *BMJ.* 2006;333(7577):1041-3.
14. de Silva-Sanigorski AM, Bell AC, Kremer P, Nichols M, Crellin M, Smith M, et al. Reducing obesity in early childhood: results from Romp & Chomp, an Australian community-wide intervention program. *Am J Clin Nutr.* 2010 April 1, 2010;91(4):831-40.
15. de Silva-Sanigorski A, Elea D, Bell C, Kremer P, Carpenter L, Nichols M, et al. Obesity prevention in the family day care setting: impact of the Romp & Chomp intervention on opportunities for children's physical activity and healthy eating. *Child Care Health Dev.* 2011 May;37(3):385-93.

Appetitive Traits and Weight in Children

Lucy Cooke, PhD

Health Behaviour Research Centre, University College London, United Kingdom

April 2012

Introduction

Global prevalence of obesity is increasing rapidly, with rates in low- and middle-income countries catching up with those in richer nations. In 2010, roughly 42 million children under five were estimated to be overweight or obese, of whom almost 35 million were from developing countries.¹ The simple explanation for the obesity epidemic is that there is less need to be physically active and more availability of low-cost, high-calorie food, creating an “*obesogenic*” environment.

Subject

There is a growing variation in *adiposity* in the population, with the lean staying lean while the fat get fatter. What is driving these differences? One explanation is that specific appetitive traits or eating behaviours may render some individuals more susceptible to environmental pressures – the behavioural susceptibility hypothesis.

Key Research Questions

- Do individual differences in appetite contribute to variation in weight gain?
- Are the determinants of appetite genetic or environmental?
- Can we devise interventions to modify ‘risky’ appetitive traits?

Recent Research Results

Research with children indicates that certain eating styles may be more pronounced in higher weight groups. For example, overweight children ate more snack foods than their lighter peers when not hungry,^{2,3} and were less able to regulate their food intake after consuming an appetizing preload than their leaner peers in two other studies.^{4,5} Overweight children appear to find food more reinforcing than lean children.⁶ Finally, research has also documented a faster eating rate in obese 6 year olds⁷ and 11 year olds,⁸ while the eating rate of 9-12 year olds increased linearly

with *body mass index (BMI)*.⁹ These observations suggest that the following appetitive traits increase the risk of weight gain: responsiveness to food, lack of sensitivity to fullness, a tendency to find food particularly reinforcing and higher speed of eating.

In addition to behavioural tests, recent research has investigated associations between appetitive traits and adiposity using psychometric measures. The Child Eating Behavior Questionnaire (CEBQ¹⁰) is a widely used parent-report instrument that includes both “food approach” behaviours (e.g., enjoyment of food, food responsiveness, and emotional over-eating) and “food avoidant” behaviours (e.g., speed of eating, satiety responsiveness, and food fussiness). Consistent associations between CEBQ subscale scores and weight have been observed in both normal and clinical populations of children. For example, higher food cue responsiveness and lower satiety responsiveness were associated with progressively higher adiposity in 8-11 and 3-5 year olds in one study.¹¹ Another study of 7-9 and 9-12 year-olds found that all food approach behaviours measured using the CEBQ showed a graded positive association with weight, while food avoidant behaviours showed the opposite pattern.¹² Similar findings have been reported in samples of 3 to 13-year-old Portuguese children¹³ and 7 to 12 year-old British children¹⁴ both of which included clinical groups seeking treatment. Longitudinal studies are adding weight to the argument that the relationship is causal.^{15,16}

If appetite is a determinant of weight gain, two questions arise: Firstly, how do eating behaviours develop and secondly, are they modifiable? In attempts to discern at what stage of development these appetitive traits emerge, two cohort studies have investigated whether variation in appetite can be identified early in life, and whether they are associated with later differences in weight.^{9,17} In a large twin study, parents completed the Baby Eating Behavior Questionnaire which measures enjoyment of food, food responsiveness, slowness in eating and satiety responsiveness during the period of milk-only feeding. Infants were weighed at 3, 9 and 15 months of age. Longitudinal analyses showed clear associations between appetitive traits and subsequent weight gain; stronger than between weight and subsequent appetite, supporting of the idea that differences in appetite influence weight gain in infancy, rather than weight gain driving appetite.¹⁷ That differences in appetite avidity emerge so early in life suggest a genetic component. In support, data from this and another twin cohort have revealed substantial heritability of eating behaviour traits in children^{9,18} and more recently in very young infants¹⁹ as well as an association with *FTO genotype*.^{20,21}

Familial influences on eating behaviour and adiposity are also of great importance. For very young children, parents determine their children's food environment by deciding what, when and how much food is offered. In addition, parents use specific feeding strategies to promote or restrict the type or amount of food that their child eats. There has therefore been considerable research interest in the extent to which these parental feeding practices might mitigate or exacerbate the influence of the wider environment, in terms of their impact on children's eating behaviour and ultimately, weight. One proposal is that when parents restrict their child's access to highly palatable unhealthy foods, children's liking for these foods is increased via a 'forbidden fruit' effect, thereby creating greater desire to eat more of them when they do become available. This is posited to teach children to eat when food is available regardless of whether they are hungry or not (food responsiveness). There is considerable support for the association between parental restriction and children's eating and weight (see reference 22 for a review) although findings are not entirely unequivocal.²³ Cross-sectional studies are the norm and the direction of causality remains unclear as a result. Rare longitudinal investigations have suggested interactional relationships²⁴ between parental feeding strategies and appetitive traits and weight. The suggestion is that parents adjust their feeding practices in response to the appetitive and anthropometric characteristics of their children. In other words, parents' feeding practices are responsive to, rather than causal of, the eating behaviour of their children. In support of this there is evidence, both qualitative and quantitative, of parents using different feeding strategies with different siblings.^{25,26} A full understanding of the complex interplay of factors that shape children's eating behaviours and weight remains out of reach at present.

Research Gaps

- Identification of specific genes affecting eating behaviours would elucidate the mechanisms through which individuals are rendered susceptible.
- Measurement of biological indicators of food-cue responsiveness indicating different salivary and hormonal responses and different patterns of brain activation in obese individuals.
- More prospective and longitudinal studies of the relationships between children's eating behaviours and weight, and parental feeding practices.
- Exploration of the impact on children's weight of behaviour change interventions targeting 'risky' appetitive traits.

Conclusions

The increase in childhood obesity since the 1980s has not been the result of genetic changes in the population, but is attributable to environmental changes favoring sedentary lifestyles and food over-consumption. However, obesity risk is not equal across the population – there are wide individual differences in body weight and these appear to be at least partly explained by differences in inherited appetitive traits. Broadly speaking, obese individuals appear to be less satiety responsive, more food cue responsive, to find food more reinforcing, and to eat more quickly than their leaner counterparts. To some extent these eating behaviours are heritable, but the degree to which they are expressed is determined by environmental conditions and for the youngest children, parents dictate those conditions in the family home. Early research suggested that maladaptive parental feeding styles might be to blame for children’s obesogenic eating behaviour. More recent research suggests a complex and dynamic bi-directional process by which parents respond to children’s eating style and weight status, which in turn are influenced by the behaviour of parents and the characteristics of the home and wider environment.

Implications for Parents, Services and Policy

Childhood obesity has reached epidemic proportions with enormous attendant costs, both in terms of health and in financial resources. Recognizing that children at high risk of obesity (with one or more overweight parents) frequently display risky eating behaviours offers the opportunity to intervene early in the home setting as well as in childcare facilities before problematic weight gain occurs. The effectiveness of interventions (both preventative and therapeutic) could be increased by incorporating strategies to modify these appetitive traits and therefore help children to better manage their own eating. Few intervention studies have been conducted, but there is some evidence to support the targeting of food responsiveness and satiety responsiveness/slowness in eating. If successful, such interventions could inform better child-feeding guidance to parents and health professionals so that inherited obesogenic eating styles are not exacerbated and exaggerated by counter-productive feeding practices. In terms of wider food environment, it is clear that children who are poor at regulating their food consumption would benefit from changes in terms of reduced portion sizes and energy density of foods offered, increased availability and promotion of healthy foods and opportunities for physical activity.

From government downwards, better understanding of the interaction between genes and environmental factors affecting appetite and weight would be of enormous benefit.

References

1. Childhood overweight and obesity. Available at <http://www.who.int/dietphysicalactivity/childhood/en/>. Accessed April 23, 2012.
2. Birch LL, Fisher JO. Mothers' child-feeding practices influence daughters' eating and weight. *Am J Clin Nutr* 2000 May;71(5):1054-61.
3. Hill C, Llewellyn CH, Saxton J et al. Adiposity and 'eating in the absence of hunger' in children. *Int J Obes (Lond)* 2008 October;32(10):1499-505.
4. Jansen A, Theunissen N, Slechten K et al. Overweight children overeat after exposure to food cues. *Eat Behav* 2003 August;4(2):197-209.
5. Johnson SL, Birch LL. Parents' and children's adiposity and eating style. *Pediatrics* 1994 November;94(5):653-61.
6. Temple JL, Legierski CM, Giacomelli AM, Salvy SJ, Epstein LH. Overweight children find food more reinforcing and consume more energy than do nonoverweight children. *Am J Clin Nutr* 2008 May;87(5):1121-7.
7. Drabman RS, Cordua GD, Hammer D, Jarvie GJ, Horton W. Developmental trends in eating rates of normal and overweight preschool children. *Child Dev* 1979 March;50(1):211-6.
8. Barkeling B, Ekman S, Rossner S. Eating behaviour in obese and normal weight 11-year-old children. *Int J Obes Relat Metab Disord* 1992 May;16(5):355-60.
9. Llewellyn CH, Van Jaarsveld CH, Boniface D, Carnell S, Wardle J. Eating rate is a heritable phenotype related to weight in children. *Am J Clin Nutr* 2008 December;88(6):1560-6.
10. Wardle J, Guthrie CA, Sanderson S, Rapoport L. Development of the Children's Eating Behaviour Questionnaire. *J Child Psychol Psychiatry* 2001 October;42(7):963-70.
11. Carnell S, Wardle J. Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. *Am J Clin Nutr* 2008 July;88(1):22-9.
12. Webber L, Hill C, Saxton J, Van Jaarsveld CH, Wardle J. Eating behaviour and weight in children. *Int J Obes (Lond)* 2009 January;33(1):21-8.
13. Viana V, Sinde S, Saxton JC. Children's Eating Behaviour Questionnaire: associations with BMI in Portuguese children. *Br J Nutr* 2008 August;100(2):445-50.
14. Croker H, Cooke L, Wardle J. Appetitive behaviours of children attending obesity treatment. *Appetite* 2011 October;57(2):525-9.
15. Agras WS, Kraemer HC, Berkowitz RI, Hammer LD. Influence of early feeding style on adiposity at 6 years of age. *J Pediatr* 1990 May;116(5):805-9.
16. Stunkard AJ, Berkowitz RI, Schoeller D, Maislin G, Stallings VA. Predictors of body size in the first 2 y of life: a high-risk study of human obesity. *Int J Obes Relat Metab Disord* 2004 April;28(4):503-13.
17. Van Jaarsveld CH, Llewellyn CH, Johnson L, Wardle J. Prospective associations between appetitive traits and weight gain in infancy. *Am J Clin Nutr* 2011 December;94(6):1562-7.
18. Carnell S, Haworth CM, Plomin R, Wardle J. Genetic influence on appetite in children. *Int J Obes (Lond)* 2008 October;32(10):1468-73.
19. Llewellyn CH, Van Jaarsveld CH, Johnson L, Carnell S, Wardle J. Nature and nurture in infant appetite: analysis of the Gemini twin birth cohort. *Am J Clin Nutr* 2010 May;91(5):1172-9.
20. Wardle J, Carnell S, Haworth CM, Farooqi IS, O'Rahilly S, Plomin R. Obesity associated genetic variation in FTO is associated with diminished satiety. *J Clin Endocrinol Metab* 2008 September;93(9):3640-3.
21. Carnell S, Wardle J. Appetitive traits in children. New evidence for associations with weight and a common, obesity-associated genetic variant. *Appetite* 2009 October;53(2):260-3.

22. Faith MS, Scanlon KS, Birch LL, Francis LA, Sherry B. Parent-child feeding strategies and their relationships to child eating and weight status. *Obes Res* 2004 November;12(11):1711-22.
23. Carnell S, Wardle J. Associations between multiple measures of parental feeding and children's adiposity in United Kingdom preschoolers. *Obesity* (Silver Spring) 2007 January;15(1):137-44.
24. Webber L, Cooke L, Hill C, Wardle J. Child adiposity and maternal feeding practices: a longitudinal analysis. *Am J Clin Nutr* 2010 September 29.
25. Webber L, Cooke L, Wardle J. Maternal perception of the causes and consequences of sibling differences in eating behaviour. *Eur J Clin Nutr* 2010 November;64(11):1316-22.
26. Farrow CV, Galloway AT, Fraser K. Sibling eating behaviours and differential child feeding practices reported by parents. *Appetite* 2009 April;52(2):307-12.

Environmental/Family/Psychosocial Influences on Physical Activity in Young Children

Trina Hinkley, PhD

Faculty of Education, University of Wollongong, Australia

April 2012

Introduction

For many years, parents and researchers believed that “young children are naturally physically active.”¹ Recent evidence suggests that this is not the case with studies showing that young children spend around 80-85% of their time being sedentary, very little time being active²⁻³ and between two and four hours a day watching television.^{2, 4-6} Recommendations for physical activity vary between countries. The U.S. recommends that young children participate in at least two hours of physical activity each day,⁷ while the more recent Australian and U.K. recommendations suggest three or more hours per day.⁸⁻⁹ Recent estimates of compliance with the physical activity recommendations suggest that the majority of children (up to 95%) fail to achieve the recommended amount.^{2,10} Recommendations for screen-based entertainment (television, computer, electronic games) vary between age groups and countries but consistently suggest that children younger than two years should participate in no screen-based entertainment.^{8,11} In Australia, children aged 2-5 years are recommended to participate in no more than one hour per day,⁸ while in the U.S. no more than two hours is recommended for that age group.¹² Recent estimates of children meeting these guidelines vary between about 22%² and 34%.⁴ Studies in children younger than three years of age are sparse however these suggest that low levels of physical activity¹³ and high levels of screen-based entertainment⁵ might also be evident in infants and toddlers. These behaviours are important to investigate and understand as evidence shows that they are associated with health and other outcomes, including obesity,¹⁴⁻¹⁵ bone mineral content¹⁶ and cognitive and behavioural outcomes.¹⁷⁻¹⁸

Subject

Given the small number of young children achieving the recommended amount of physical activity and screen-based entertainment, it is important to investigate and identify the factors, or correlates, that might support or constrain those behaviours. In so doing, it may be possible to

identify targets for intervention to support healthy levels of physical activity during early childhood.

Problems and Research Context

There exist several challenges to measuring physical activity and screen-based entertainment and their correlates during the early childhood period. Young children cannot self-report how much time they spend in such behaviours and it may also be difficult for parents to estimate as their children may spend many hours each day in non-parental care, for instance in preschool or childcare. Objective measures of physical activity, such as activity monitors (*accelerometers*) are available and commonly used, however, objective measures of screen-based entertainment, although available, are seldom used.

Many studies investigating potential correlates of physical activity and screen-based entertainment during the early childhood period have focused on the preschool years, that is, roughly three to five years of age. Subsequently, there is very little understanding of children's behaviours during the period from birth to two years of age. Importantly, knowledge of behaviours and their correlates during the preschool period may not be transferrable to younger children.

Studies investigating correlates of physical activity and screen-based entertainment have also typically measured only a few correlates, such as parental factors¹⁹⁻²⁰ or variables in the preschool/childcare²¹ or home²² environments. Because of this, there is little understanding of how the correlates might be associated with each of the behaviours when considered in their broader context. That is: how might parental factors such as self efficacy be associated with young children's physical activity or screen-based entertainment when environmental factors such as availability of equipment in the home or access to parks in the neighbourhood are also considered?

Key Research Questions

Key questions in this field include identifying the domains of correlates of behaviour during early childhood. That is, are there correlates at the individual, social and physical environment levels that might be associated with young children's behaviours? It is also important to identify what the key correlates in each of those domains might be as these can then be used as targets in programs to support healthy behaviours in young children.

Recent Research Results

Correlates of young children's physical activity and screen-based entertainment, mostly in three- to five-year-olds, have previously been reported across several studies. Correlates are evident in different domains – that is, individual (sex, age, individual preferences), social (parental factors) and physical environment (home, preschool, neighbourhood factors) correlates appear to be associated with young children's physical activity²³ and screen-based entertainment.²⁴ For instance, across many studies, boys are consistently reported to be more active than girls, even in this young age group^{2,21,23} however there appears to be no difference in time in screen-based entertainment between boys and girls.²⁴ Associations between age with physical activity or screen-based entertainment are less clear. Some studies report that older children are less active than young children in this age group^{2,25-26} and participate in more screen-based entertainment,²⁷ while others report that there is no difference in behaviours between older and younger children during the early childhood period.^{22,28-30} Differences in findings may be attributable to different sample characteristics, such as social or geographical considerations, or sizes, or methods of measuring behaviours or correlates. There appears to be no association between socioeconomic position and physical activity or screen-based entertainment during early childhood.²³⁻²⁴ Virtually nothing is known about children's innate preferences to be active or engage in other behaviours and these may also be important considerations.

Social correlates such as parental physical activity^{19,23,26} and supporting their child to be active³¹ have been shown to be associated with physical activity in young children. For screen-based entertainment, parental rules restricting such entertainment are associated with children spending less time in those behaviours.^{20,22,32} Parents who spend more time in screen-based entertainment themselves appear to also have children with higher levels of participation.³³ However little is known about many other social factors that may be potential correlates of these behaviours (e.g. sibling influence or interaction, and interaction with other people such as grandparents who increasingly play more prominent roles in young children's lives).

While many potential correlates of physical activity and screen-based entertainment have been investigated in the physical environment, most have only been explored in one or two studies, so it is difficult to draw overall conclusions.²³⁻²⁴ However more time spent outdoors²³ appears to be consistently associated with higher levels of physical activity in young children. Conversely, the amount of play equipment in the home has been shown to have no association with how active young children are³⁴⁻³⁵ and an intervention designed to increase physical activity by introducing

new play equipment into the preschool environment found no change in physical activity.³⁶ For screen-based entertainment, having the television constantly on in the home appears to be associated with higher levels of television viewing in young children.^{20,32} Other factors in the physical environment, such as access to facilities, require further investigation.

Research Gaps

Many gaps exist in the current knowledge base around the correlates of physical activity and screen-based entertainment in young children. Specifically these include very limited knowledge about the level of physical activity and screen-based entertainment in children younger than about three years¹³ or of any potential correlates that might be associated with those behaviours during that period.

While many correlates have been investigated, studies have primarily looked at only a small number of potential correlates or have investigated only correlates in one domain, such as individual or social level correlates.²³⁻²⁴ Research therefore needs to investigate a larger number of correlates, across multiple domains simultaneously, to gain a more comprehensive understanding of how specific factors may be associated with physical activity and screen-based entertainment in their broader contexts. One study that did this for physical activity found that correlates were mainly at an individual level but varied between boys and girls.³⁵ However, the number of correlates that the study investigated was quite small and further investigation is required. Evidence supporting differences or similarities in correlates between boys and girls is also required.

Correlates in the physical environment have typically shown little association with physical activity or screen-based entertainment during early childhood and are generally measured by parent report. Objective measures of such correlates, including availability and accessibility of facilities, aesthetics in the neighbourhood environment, and safety, may provide further insight into factors which may support healthy behaviours during the early childhood period. Additionally, interventions aiming to change correlates and observe subsequent change in physical activity or screen-based entertainment during this period are sparse, and as they provide stronger evidence than the cross-sectional studies typically undertaken to date, would be a valuable addition to the literature.

Conclusions

Recommendations for participation in physical activity and screen-based entertainment during the early childhood period (birth to five years) are now available in several countries, including the U.S. and Australia.^{7-8,11-12} However, young children still largely fail to achieve the recommended amount of physical activity and the majority of children exceed recommendations for screen-based entertainment. Identifying the correlates of these behaviours is therefore crucial for the development of programs to support healthy levels of physical activity and screen-based entertainment during the early childhood period, which, in turn, support healthy outcomes for children. Although few individual correlates have shown consistent associations with young children's behaviours, parental correlates appear to be important across studies. Given the amount of time and input parents have in their young children's lives, this is hardly surprising. Supporting parents is therefore important in achieving the ultimate goal of providing healthy environments for children's development.

Implications for Parents, Services and Policy

From a policy perspective, it is important to disseminate information about health behaviours, including recommended levels of physical activity and screen-based entertainment. Such dissemination enables parents and service providers to be aware of how much time young children should spend in these behaviours. Additionally, dissemination of information about the health outcomes of excessive screen-based entertainment or inadequate physical activity would provide parents and service providers with further evidence to support decisions regarding children's opportunities to be active or engage in screen-based entertainment.

Service providers such as preschools, childcare centres, and health providers, should be required to undertake training in provision of physical activity during the early childhood period. Ensuring that children have several hours outside during the day may also be important to support adequate opportunities to be active. Given young children spend so much time in screen-based entertainment, removal of such opportunities from preschools and childcare centres may be warranted. Service providers are also often a point of reference and information for parents of young children, and as such, have a responsibility to be able to provide evidence-based information to those parents for the care of their children.

Parents require information, advice and support about what the optimal levels of a range of behaviours, including physical activity and screen-based entertainment, are for their young children, and this role falls primarily with policy makers and service providers. However, parents

also have an important role to play in modelling healthy behaviours to their children, ensuring their children have adequate opportunities for outdoor play and minimising opportunities for screen-based entertainment.

References

1. American Academy of Pediatrics: Committee on Sports Medicine and Fitness. Fitness in the Preschool Child. *Pediatrics* 1976;58(1):88-89.
2. Hinkley T, Salmon J, Okely AD, Crawford DA, Hesketh KD. Preschoolers' physical activity, screen time and compliance with recommendations. *Medicine and Science in Sports and Exercise*. In press.
3. Cardon G, De Bourdeaudhuij I. Are preschool children active enough? Objectively measured physical activity levels. *Research Quarterly for Exercise and Sport* 2008;79(3):326-32.
4. Tandon PS, Zhou C, Lozano P, Christakis DA. Preschoolers' total daily screen time at home and by type of child care. *Journal of Pediatrics* 2011;158(2):297-300.
5. Zimmerman FJ, Christakis DA, Meltzoff AN. Television and DVD/video viewing in children younger than 2 years. *Archives of Pediatrics and Adolescent Medicine* 2007;161(5):473-479.
6. Stolley MR, Fitzgibbon ML, Dyer A, Horn LV, KauferChristoffel K, Schiffer L. Hip-Hop to Health Jr., an obesity prevention program for minority preschool children: baseline characteristics of participants. *Preventive Medicine* 2003;36(3):320.
7. American Alliance for Health PE, Recreation and Dance (AAHPERD). NASPE releases first ever physical activity guidelines for infants and toddlers. *Illinois Journal for Health, Physical Education, Recreation, and Dance* 2002;50:31-32.
8. Department of Health and Aging (DoHA). Get Up and Grow: Healthy Eating and Physical Activity for Early Childhood. Available at:
[http://www.health.gov.au/internet/main/publishing.nsf/Content/EDFEDB588460BCE3CA25762B00232A13/\\$File/gug-directorscoord-2.pdf](http://www.health.gov.au/internet/main/publishing.nsf/Content/EDFEDB588460BCE3CA25762B00232A13/$File/gug-directorscoord-2.pdf). Accessed April 24, 2012.
9. Department of Health Physical Activity Health Improvement and Protection. Start Active, Stay Active: A report on physical activity from the four home countries' Chief Medical Officers. In. *London: Department of Health, Physical Activity, Health Improvement and Protection*; 2011:1-62.
10. Beets MW, Bornstein D, Dowda M, Pate RR. Compliance with national guidelines for physical activity in U.S. preschoolers: measurement and interpretation. *Pediatrics* 2011;127(4):658-64.
11. American Academy of Pediatrics. Policy statement: media use by children younger than 2 years. *Pediatrics* 2011;128(5):1040-1045.
12. American Academy of Pediatrics Committee on Public Education. Children, adolescents, and television. *Pediatrics* 2001;107(2):423-426.
13. Cardon G, van Cauwenberghe E, de Bourdeaudhuij I. Physical activity in infants and toddlers. In: Tremblay RE, Barr RG, Peters RDeV, Boivin M, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development; 2011:1-6. Available at: http://www.child-encyclopedia.com/documents/Cardon-van_Cauwenberghe-de_BourdeaudhuijANGxp1.pdf. Accessed April 24, 2012.
14. Jiménez-Pavón D, Kelly J, Reilly JJ. Associations between objectively measured habitual physical activity and adiposity in children and adolescents: Systematic review. *International Journal of Pediatric Obesity* 2010;5(1):3-18.
15. Hancox RJ, Poulton R. Watching television is associated with childhood obesity: but is it clinically important? *International Journal of Obesity* 2006;30:171-175.

16. Janz KF, Burns TL, Torner JC, et al. Physical activity and bone measures in young children: the Iowa Bone Development Study. *Pediatrics* 2001;107(6):1387.
17. Mistry KM, Minkovitz CS, Strobino DM, Borzekowski DLG. Children's television exposure and behavioural and social outcomes at 5.5 years: Does timing of exposure matter? *Pediatrics* 2007;120:762-769.
18. Hancox RJ, Milne BJ, Poulton R. Association of television viewing during childhood with poor educational achievement. *Archives of Pediatrics and Adolescent Medicine* 2005;159(7):614-618.
19. Oliver M, Schofield GM, Schluter PJ. Parent influences on preschoolers' objectively assessed physical activity. *Journal of Science and Medicine in Sport* 2010;13(4):403-9.
20. Vandewater EA, Rideout VJ, Wartella EA, Huang X, Lee JH, Shim M. Digital childhood: electronic media and technology use among infants, toddlers, and preschoolers. *Pediatrics* 2007;119:e1006-e1015.
21. Cardon G, Van Cauwenberghe E, Labarque V, Haerens L, De Bourdeaudhuij I. The contribution of preschool playground factors in explaining children's physical activity during recess. *International Journal of Behavioral Nutrition and Physical Activity* 2008;5:11.
22. Kuepper-Nybelen J, Lamerz A, Bruning N, Hebebrand J, Herpertz-Dahlmann B, Brenner H. Major differences in prevalence of overweight according to nationality in preschool children living in Germany: determinants and public health implications. *Archives of Disease in Childhood* 2005;90(4 (Electronic)):359-363.
23. Hinkley T, Crawford D, Salmon J, Okely AD, Hesketh K. Preschool children and physical activity: a review of correlates. *American Journal of Preventive Medicine* 2008;34(5):435-441.
24. Hinkley T, Salmon J, Okely AD, Trost SG. Correlates of sedentary behaviours in preschool children: a review. *International Journal of Behavioral Nutrition and Physical Activity* 2010;7:66.
25. Pate RR, McIver K, Dowda M, Brown WH, Addy C. Directly observed physical activity levels in preschool children. *Journal of School Health* 2008;78(8):438-444.
26. Taylor RW, Murdoch L, Carter P, Gerrard DF, Williams SM, Taylor BJ. Longitudinal study of physical activity and inactivity in preschoolers: the FLAME study. *Medicine and Science in Sports and Exercise* 2009;41(1):96-102.
27. Dennison BA, Erb TA, Jenkins PL. Television viewing and television in bedroom associated with overweight risk among low-income preschool children. *Pediatrics* 2002;109(6):1028-1035.
28. Kelly LA, Reilly JJ, Fisher A, et al. Effect of socioeconomic status on objectively measured physical activity. *Archives of Disease in Childhood* 2006;91(1):35-38.
29. Pate RR, Pfeiffer KA, Trost SG, Ziegler P, Dowda M. Physical activity among children attending preschools. *Pediatrics* 2004;114(5):1258-1263.
30. Miller SA, Taveras EM, Rifas-Shiman SL, Gillman MW. Association between television viewing and poor diet quality in young children. *International Journal of Pediatric Obesity* 2008;1(9).
31. Zecevic CA, Tremblay L, Lovsin T, Michel L. Parental influence on young children's physical activity. *International Journal of Pediatrics* 2010;2010:468526.
32. Spurrier NJ, Magarey AA, Golley R, Curnow F, Sawyer MG. Relationships between the home environment and physical activity and dietary patterns of preschool children: a cross-sectional study. *International Journal of Behavioral Nutrition and Physical Activity* 2008;5:31.
33. Kourlaba G, Kondaki K, Liarigkiovinos T, Manios Y. Factors associated with television viewing time in toddlers and preschoolers in Greece: the GENESIS study. *Journal of Public Health (Oxford)* 2009;31(2):222-30.
34. Sallis JF, Nader PR, Broyles SL, et al. Correlates of physical activity at home in Mexican-American and Anglo-American preschool children. *Health Psychology* 1993;12(5):390-398.

35. Pfeiffer KA, Dowda M, McIver KL, Pate RR. Factors related to objectively measured physical activity in preschool children. *Pediatric Exercise Science* 2009;21(2):196-208.
36. Cardon G, Labarque V, Smits D, De Bourdeaudhuij I. Promoting physical activity at the pre-school playground: The effects of providing markings and play equipment. *Preventive Medicine* 2009;48(4):335-40.

The Prenatal Origins of Obesity: Evidence and Opportunities for Prevention

Véronique Gingras, RD, PhD, Emily Oken, MD, MPH

Harvard Medical School and Harvard Pilgrim Health Care Institute, USA

July 2019, Éd. rév.

Introduction

The obesity epidemic has spared no segment of the population, even infants and young children.¹ In 2015-16, almost 14% of U.S. toddlers aged 2 to 5 years and over 18% of children between 6 and 11 years had obesity.² Over the past decades, researchers have looked to events that occur in very early life, even before birth, to understand the causes of childhood obesity and identify factors that may be targeted for prevention.³ In this section, we outline parameters for normal growth in infancy, review prenatal factors that have been found to be associated with later obesity, and identify areas for intervention.

Subject

During well-child visits, pediatric clinicians use growth charts to document serial measures of weight and length, and screen for abnormalities in weight status.⁴ In the U.S., among children over the age of two years, obesity is defined as a body mass index (BMI, weight in kg divided by height in m squared)² at or above the 95th percentile for age and sex, compared with a reference population – typically the Centers for Disease Control and Prevention (CDC) 2000 growth charts.⁵ Overweight is a BMI between the 85th and 94th percentile.⁵

In infants below 24 months, excess weight has traditionally been defined using weight for length percentiles based on the CDC reference data. However, weight for length percentiles do not reflect age-dependent variations in weight or length. The World Health Organization (WHO) 2006 Growth Standard, which includes BMI percentiles for infants under age 2 years, has therefore been recommended by the American Academy of Pediatrics and other groups as a better reference for healthy growth in infancy.⁶ Another advantage of this standard is that WHO included only healthy term infants who were breastfed for at least 12 months, followed them longitudinally, and excluded data for children with excess adiposity and growth failure. Using the WHO Growth Standard, fewer children are diagnosed with poor weight gain, and more with excess adiposity,

than when using the CDC Growth Reference.^{7,8} A BMI above the 97th percentile for sex and age indicates excess adiposity.⁹ Since BMI reflects both lean and fat mass, however, BMI screening may result in misdiagnosis of individuals with higher or lower lean body mass than expected. In a recent study using two large prospective birth cohorts in the United States (Project Viva) and Belarus (Promotion of Breastfeeding Intervention Trial), Aris et al. showed that using either weight for length or BMI to define overweight during the first 2 years of life both strongly predict adiposity and cardio-metabolic outcomes in early adolescence.¹⁰

In addition to an infant's weight at any given time, a rapid weight gain trajectory predicts later risk of obesity as well as later prevalence of several health outcomes including cardio-metabolic diseases and asthma.¹¹⁻¹³ Rapid weight gain in infancy, often defined as a change in weight z-score > 0.67 (equivalent to the upward crossing of at least one percentile band on growth charts), has been associated with increased odds of obesity in adulthood.¹² In a study of 44,622 children aged 1 month to 10 years with 122,214 length/height and weight measurements, Taveras et al. found that upward crossing of ≥ 2 major weight-for-length percentiles (i.e., the 5th, 10th, 25th, 50th, 75th, 90th, and 95th percentile lines on the growth chart) in the first 6 months of life was not only common but was also associated with the highest risk of obesity 5 and 10 years later.¹⁴ Growth and weight trajectories have been increasingly studied over the past years and could provide useful indicators of risks for later obesity and associated cardio-metabolic risk.¹⁵⁻¹⁸ Typically, a child's BMI will peak around one year of age, followed by a decrease through 4 to 6 years of age, called the "BMI rebound".¹⁶ An infancy BMI peak at a later age and of greater magnitude, and an earlier adiposity rebound, each predict later risk for obesity and related cardio-metabolic disorders.¹⁷⁻¹⁸

Problems

Obesity in infancy predicts obesity and related cardio-metabolic risk in later life. Also serious morbidity associated with obesity may occur even within childhood, including asthma, orthopedic problems, psychosocial adversity, high hospital admission rates, and increasingly, type 2 diabetes.¹⁹⁻²⁴ The biggest babies are not necessarily the fattest – babies born small for gestational age have reduced lean body mass, and thus are relatively fatter compared with appropriate-for-gestational-age babies, both at birth and in later life.²⁵ Since the combination of low weight at birth and rapid postnatal weight gain is a strong predictor of later metabolic disease risk, it is especially important to avoid 'fattening up' these small babies.²⁶

Research Context

Numerous animal experiments dating back decades show that perturbations before birth can have lifelong effects on health.^{27,28} Whereas early studies focused on under-nutrition in early life as a risk factor for cardiovascular disease, more recently research has focused on a wider range of early life exposures that may predispose to later obesity and cardiometabolic diseases. In humans, accumulating research demonstrates that maternal prenatal obesity, excess weight gain during pregnancy, nutritional inadequacies, gestational diabetes, maternal prenatal environmental exposures, and smoking during pregnancy predict later obesity and its adverse sequelae.²⁹⁻³⁶

Key Research Questions

Current research into the developmental origins of obesity clusters around the following questions: 1) What factors in early life predict later obesity risk, and how much influence do these early life factors have compared to risk factors that occur later? 2) What are the pathways and mechanisms by which early life exposures influence later health? 3) How can we intervene in these early life exposures to prevent or ameliorate risks for obesity and its adverse health effects?

Recent Research Results

Mothers with obesity tend to have overweight children.³⁷⁻³⁹ Initially these associations were mostly studied as evidence for a genetic underpinning of obesity risk.⁴⁰ Studies have now provided strong evidence that the obese intrauterine environment itself programs body weight.⁴¹ Similarly, a number of epidemiologic studies have found that higher maternal gestational weight gain, especially in early pregnancy, is associated with higher child weight in childhood and adolescence, and consequent risk for obesity and elevated blood pressure.^{32,34,42-44} Pre-conception obesity and excessive gestational weight gain are both associated with increased odds of gestational diabetes, which itself has been associated with child's obesity risk. Infants born to mothers with diabetes during pregnancy are heavier at birth, but then grow slower after birth and are often no larger during the preschool years. However, even in early life they are likely to have more body fat,⁴⁵ and beginning in mid-childhood these children are heavier than their peers who were not born to mothers with diabetes during pregnancy.²⁹ Finally, although infants born to mothers who smoked during pregnancy are small at birth, they grow faster and have a higher risk for obesity in childhood and adulthood.³³

The question remains whether these intrauterine experiences actually program long-term weight regulation and disease risk, or whether they are solely markers for other, shared causes of both maternal weight and child weight. Shared genes and extra-uterine environment certainly account for some of the similarity in maternal and offspring weight.^{46,47} For example, parents and children tend to have similar diet quality and physical activity habits.⁴⁸ Also, mothers who smoke, are obese, had gestational diabetes, or gained excessive weight during pregnancy are less likely to breastfeed, which itself predicts later overweight.⁴⁹⁻⁵²

However, associations of these prenatal factors with child weight persist even after statistical adjustment for factors such as socioeconomic status, infant feeding, and child diet and physical activity.⁴² Furthermore, studies that compare siblings with discordant prenatal exposures but presumably otherwise similar genetic and extra-uterine experiences, provide additional evidence that the prenatal is a critical period for obesity risk.⁵³⁻⁵⁵ Also, these human findings are supported by abundant evidence from animal studies among rodents, sheep and primates.⁵⁶⁻⁵⁸

Research Gaps

An optimal approach to understanding the role of intrauterine exposures for later health would be to conduct a well-powered randomized clinical trial, in which women are randomized to usual care or to an effective weight change intervention before and/or during pregnancy, and follow children longitudinally. Evidence of the effectiveness of preconception interventions for the mother and child's health is scarce; yet, a few trials are underway and might provide new insights in the near future.⁵⁹ Trials to improve diet or other behaviours during pregnancy have been conducted, but few have followed infants after delivery.⁶⁰ To date, the limited data available suggest that dietary and lifestyle interventions during pregnancy are modestly effective at reducing weight gain and may reduce the prevalence of gestational diabetes in overweight and obese mothers, but they have not been successful in reducing rates of large for gestational age births and macrosomia.⁶¹ Interventions before conception might be more effective compared to those beginning in pregnancy,⁶²⁻⁶³ and ongoing studies with larger sample sizes are expected to provide additional information in the coming years. Another area of future research is the paternal contribution to his child's later risk of obesity and disease beginning in early life. Recent evidence suggests that paternal preconception obesity and diabetes could be associated with later child outcomes.⁶⁴

Conclusions

Childhood overweight is common and an important predictor of later health. Numerous observational studies among humans and abundant experimental data from animals suggest that experiences before birth including intrauterine exposure to maternal smoking, obesity, excess gestational weight gain or diabetes can “program” trajectories of adiposity and metabolic health throughout life. Clear guidelines exist for each of these factors, including recommendations for optimal maternal BMI before pregnancy, gestational weight gain guidelines, advice against smoking during pregnancy, and recommendations for universal gestational diabetes screening.⁶⁵⁻⁶⁶ What is less clear, however, is how to help women achieve these optimal behaviours.

Implications for Parents, Service Providers, and Policy Makers

All young women should be encouraged to maintain a healthy weight and abstain from smoking prior to pregnancy. During pregnancy, provider advice is an important predictor of healthful behaviours and of weight gain concordant with guidelines. Women should be counseled regarding the implications of their own weight and health status for their child’s health.

Pediatricians should identify and document prenatal and familial factors that are likely to increase a child’s obesity risk, including parental obesity and maternal smoking, excess gestational weight gain and gestational diabetes. All infants and children should be routinely screened for overweight and for rapid weight gain using standard measurement techniques and the appropriate growth charts. The postpartum period is an opportunity to promote healthful behaviors that may not only improve the mother’s long-term health and provide a role model for the infant’s future behaviours, but also may optimize the mother’s health entering subsequent pregnancies.

References

1. Kim J, Peterson KE, Scanlon KS, et al. Trends in Overweight from 1980 through 2001 among Preschool-Aged Children Enrolled in a Health Maintenance Organization[ast]. *Obesity*. 2006;14(7):1107-1112.
2. Hales CM, Fryar CD, Carroll MD, Freedman DS, Ogden CL. Trends in Obesity and Severe Obesity Prevalence in US Youth and Adults by Sex and Age, 2007-2008 to 2015-2016. *JAMA*. 2018;319(16):1723-1725.
3. Oken E, Gillman MW. Fetal origins of obesity. *Obes Res*. 2003;11(4):496-506.
4. Story M, Holt K, Sofka D, eds. *Bright futures in practice (Nutrition)*. 2nd ed. Arlington, VA: National Center for Education in Maternal and Child Health; 2002.
5. Centers for Disease Control and Prevention, National Center for Health Statistics. CDC Growth Charts: United States. 2000.
6. World Health Organization. *WHO child growth standards: length/height-for-age, weight-for-age, weight-for-height and body mass index-for-age: Methods and development*. Geneva, Switzerland: World Health Organization; 2006.
7. Maalouf-Manasseh Z, Metallinos-Katsaras E, Dewey KG. Obesity in preschool children is more prevalent and identified at a younger age when WHO growth charts are used compared with CDC charts. *J Nutr*. 2011;141(6):1154-1158.

8. Parsons HG, George MA, Innis SM. Growth assessment in clinical practice: whose growth curve? *Curr Gastroenterol Rep.* 2011;13(3):286-292.
9. Grummer-Strawn LM, Reinold C, Krebs NF. Use of World Health Organization and CDC growth charts for children aged 0-59 months in the United States. *MMWR Recomm Rep.* 2010;59(RR-9):1-15.
10. Aris IM, Rifas-Shiman SL, Li LJ, Yang S, Belfort MB, Thompson J, Hivert MF, Patel R, Martin RM, Kramer MS, Oken E. Association of weight for length vs body mass index during the first 2 years of life with cardiometabolic risk in early adolescence. *JAMA Netw Open.* 2018;1(5):e182460.
11. Taveras EM, Camargo CA, Jr., Rifas-Shiman SL, et al. Association of birth weight with asthma-related outcomes at age 2 years. *Pediatr Pulmonol.* 2006;41(7):643-648.
12. Gillman MW. Early infancy as a critical period for development of obesity and related conditions. *Nestle Nutr Workshop Ser Pediatr Program.* 2010;65:13-20.
13. Ong KK, Loos RJ. Rapid infancy weight gain and subsequent obesity: Systematic reviews and hopeful suggestions. *Acta Paediatr.* 2006;95(8):904-8.
14. Taveras EM, Rifas-Shiman SL, Sherry B, et al. Crossing growth percentiles in infancy and risk of obesity in childhood. *Arch Pediatr Adolesc Med.* Nov 2011;165(11):993-998.
15. Marinkovic T, Toemen L, Kruithof CJ, Reiss I, van Osch-Gevers L, Hofman A et al. Early Infant Growth Velocity Patterns and Cardiovascular and Metabolic Outcomes in Childhood. *J Pediatr.* 2017;186:57-63.e4.
16. Wen X, Kleinman K, Gillman MW, Rifas-Shiman SL, Taveras EM. Childhood body mass index trajectories: modeling, characterizing, pairwise correlations and socio-demographic predictors of trajectory characteristics. *BMC Med Res Methodol.* 2012;12:38.
17. Hughes AR, Sherriff A, Ness AR, Reilly JJ. Timing of adiposity rebound and adiposity in adolescence. *Pediatrics.* 2014;134(5):e1354-61.
18. Aris IM, Bernard JY, Chen LW, Tint MT, Pang WW, Lim WY et al. Infant body mass index peak and early childhood cardio-metabolic risk markers in a multi-ethnic Asian birth cohort. *Int J Epidemiol.* 2017;46(2):513-525.
19. Charney E, Goodman HC, McBride M, Lyon B, Pratt R. Childhood antecedents of adult obesity. Do chubby infants become obese adults? *N Engl J Med.* 1976;295(1):6-9.
20. Franks PW, Hanson RL, Knowler WC, Sievers ML, Bennett PH, Looker HC. Childhood obesity, other cardiovascular risk factors, and premature death. *N Engl J Med.* 2010;362(6):485-493.
21. Biro FM, Wien M. Childhood obesity and adult morbidities. *Am J Clin Nutr.* 2010;91(5):1499S-1505S.
22. Sinha R, Fisch G, Teague B, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *N Engl J Med.* 2002;346(11):802-810.
23. Noal RB, Menezes AM, Macedo SE, Dumith SC. Childhood body mass index and risk of asthma in adolescence: a systematic review. *Obes Rev.* 2011;12(2):93-104.
24. Shibli R, Rubin L, Akons H, Shaoul R. Morbidity of overweight (\geq 85th percentile) in the first 2 years of life. *Pediatrics.* 2008;122(2):267-272.
25. Hediger ML, Overpeck MD, Kuczmarski RJ, McGlynn A, Maurer KR, Davis WW. Muscularity and fatness of infants and young children born small- or large-for-gestational-age. *Pediatrics.* 1998;102(5):E60.
26. Barker D. Mothers, babies, and health in later life. Second ed. Edinburgh: Harcourt Brace and Company; 1998.
27. McCance RA, Widdowson EM. The determinants of growth and form. *Proc R Soc Lond.* 1974;185:1-17.
28. Plagemann A, Heidrich I, Gotz F, Rohde W, Dorner G. Obesity and enhanced diabetes and cardiovascular risk in adult rats due to early postnatal overfeeding. *Exp Clin Endocrinol.* 1992;99(3):154-158.

29. Gillman MW, Rifas-Shiman S, Berkey CS, Field AE, Colditz GA. Maternal gestational diabetes, birth weight, and adolescent obesity. *Pediatrics*. 2003;111(3):e221-226.
30. Moore TR. Fetal exposure to gestational diabetes contributes to subsequent adult metabolic syndrome. *Am J Obstet Gynecol*. 2010;202(6):643-649.
31. Oken E, Huh SY, Taveras EM, Rich-Edwards JW, Gillman MW. Associations of maternal prenatal smoking with child adiposity and blood pressure. *Obes Res*. Nov 2005;13(11):2021-2028.
32. Oken E, Kleinman KP, Belfort MB, Hammitt JK, Gillman MW. Associations of gestational weight gain with short- and longer-term maternal and child health outcomes. *Am J Epidemiol*. 2009;170(2):173-180.
33. Oken E, Levitan EB, Gillman MW. Maternal smoking during pregnancy and child overweight: systematic review and meta-analysis. *Int J Obes (Lond)*. 2008;32(2):201-210.
34. Oken E, Rifas-Shiman SL, Field AE, Frazier AL, Gillman MW. Maternal gestational weight gain and offspring weight in adolescence. *Obstet Gynecol*. 2008;112(5):999-1006.
35. Wu G, Bazer FW, Cudd TA, Meininger CJ, Spencer TE. Maternal nutrition and fetal development. *J Nutr*. 2004;134(9):2169-72.
36. La Merrill M, Birnbaum LS. Childhood obesity and environmental chemicals. *Mt Sinai J Med*. 2011;78(1):22-48.
37. Guillaume M, Lapidus L, Beckers F, Lambert A, Bjorntorp P. Familial trends of obesity through three generations. *Int J Obes Relat Metab Disord*. 1995;19 Suppl 3:S5-9.
38. Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. *Arch Dis Child*. 1997;77(5):376-381.
39. Fisch RO, Bilek MK, Ulstrom R. Obesity and leanness at birth and their relationship to body habitus in later childhood. *Pediatrics*. 1975;56(4):521-528.
40. Stunkard AJ, Sorensen TI, Hanis C, et al. An adoption study of human obesity. *N Engl J Med*. 1986;314(4):193-198.
41. Oken E. Maternal and child obesity: the causal link. *Obstet Gynecol Clin North Am*. 2009;36(2):361-377, ix-x.
42. Oken E, Taveras EM, Kleinman KP, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. *Am J Obstet Gynecol*. Apr 2007;196(4):322 e321-328.
43. Wrotniak BH, Shults J, Butts S, Stettler N. Gestational weight gain and risk of overweight in the offspring at age 7 y in a multicenter, multiethnic cohort study. *Am J Clin Nutr*. 2008;87(6):1818-1824.
44. Mamun AA, Mannan M, Doi SA. Gestational weight gain in relation to offspring obesity over the life course: a systematic review and bias-adjusted meta-analysis. *Obes Rev*. 2014;15(4):338-47.
45. Wright CS, Rifas-Shiman SL, Rich-Edwards JW, Taveras EM, Gillman MW, Oken E. Intrauterine exposure to gestational diabetes, child adiposity, and blood pressure. *Am J Hypertens*. 2009;22(2):215-220.
46. Rankinen T, Zuberi A, Chagnon YC, et al. The human obesity gene map: the 2005 update. *Obesity (Silver Spring)*. 2006;14(4):529-644.
47. Nelson MC, Gordon-Larsen P, North KE, Adair LS. Body mass index gain, fast food, and physical activity: effects of shared environments over time. *Obesity (Silver Spring)*. 2006;14(4):701-709.
48. Oliveria SA, Ellison RC, Moore LL, Gillman MW, Garrahe EJ, Singer MR. Parent-child relationships in nutrient intake: the Framingham Children's Study. *Am J Clin Nutr*. 1992;56(3):593-598.
49. Gunderson EP. Breastfeeding after gestational diabetes pregnancy: subsequent obesity and type 2 diabetes in women and their offspring. *Diabetes Care*. 2007;30 Suppl 2:S161-168.
50. Li R, Jewell S, Grummer-Strawn L. Maternal obesity and breast-feeding practices. *Am J Clin Nutr*. 2003;77(4):931-936.

51. Hilson JA, Rasmussen KM, Kjolhede CL. Excessive weight gain during pregnancy is associated with earlier termination of breast-feeding among White women. *J Nutr.* 2006;136(1):140-146.
52. Hilson JA, Rasmussen KM, Kjolhede CL. High pre-pregnant body mass index is associated with poor lactation outcomes among white, rural women independent of psychosocial and demographic correlates. *J Hum Lact.* 2004;20(1):18-29.
53. Dabelea D, Hanson RL, Lindsay RS, et al. Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships. *Diabetes.* 2000;49(12):2208-2211.
54. Ludwig DS, Currie J. The association between pregnancy weight gain and birthweight: a within-family comparison. *Lancet.* 2010;376(9745):984-990.
55. Smith J, Cianflone K, Biron S, et al. Effects of maternal surgical weight loss in mothers on intergenerational transmission of obesity. *J Clin Endocrinol Metab.* 2009;94(11):4275-4283.
56. Howie GJ, Sloboda DM, Kamal T, Vickers MH. Maternal nutritional history predicts obesity in adult offspring independent of postnatal diet. *J Physiol.* 2009;587(Pt 4):905-915.
57. Wu Q, Suzuki M. Parental obesity and overweight affect the body-fat accumulation in the offspring: the possible effect of a high-fat diet through epigenetic inheritance. *Obes Rev.* 2006;7(2):201-208.
58. Robinson S, Marriott L, Poole J, et al. Dietary patterns in infancy: the importance of maternal and family influences on feeding practice. *Br J Nutr.* 2007;98(5):1029-1037.
59. Barker M, Dombrowski SU, Colbourn T, Fall CHD, Kriznik NM, Lawrence WT, Norris SA, Ngaiza G, Patel D, Skordis-Worrall J, Sniehotta FF, Steegers-Theunissen R, Vogel C, Woods-Townsend K, Stephenson J. Intervention strategies to improve nutrition and health behaviours before conception. *Lancet.* 2018;391(10132):1853-1864.
60. Oken E, Gillman MW. Intervention strategies to improve outcome in obese pregnancy I: Focus on Gestational Weight gain. In: Gillman MW, Poston L, eds. *Maternal Obesity.* Cambridge, UK: Cambridge University Press; 2012.
61. Oteng-Ntim E, Varma R, Croker H, Poston L, Doyle P. Lifestyle interventions for overweight and obese pregnant women to improve pregnancy outcome: systematic review and meta-analysis. *BMC Med.* 2012;10:47.
62. Stephenson J, Heslehurst N, Hall J, Schoenaker D, Hutchinson J, Cade J, Poston L, Barrett G, Crozier SR, Barker M, Kumaran K, Yajnik CS, Baird J, Mishra GD. Before the beginning: nutrition and lifestyle in the preconception period and its importance for future health. *Lancet.* 2018;391(10132):1830-41.
63. Gingras V, Hivert MF, Oken E. Early-Life Exposures and Risk of Diabetes Mellitus and Obesity. *Curr Diab Rep.* 2018;18(10):89.
64. Fleming TP, Watkins AJ, Velazquez MA, Mathers JC, Prentice AM, Stephenson J, Barker M, Saffery R, Yajnik CS, Eckert JJ, Hanson MA, Forrester T, Gluckman PD, Godfrey KM. Origins of lifetime health around the time of conception: causes and consequences. *Lancet.* 2018;391(10132):1842-1852.
65. Institute of Medicine and National Research Council of the National Academies. *Weight Gain During Pregnancy: Reexamining the Guidelines.* Washington, DC: National Academies Press; 2009.
66. American Diabetes Association. Management of Diabetes in Pregnancy: Standards of Medical Care in Diabetes—2019. *Diabetes Care.* 2019;42 Suppl 1:S165-72.

Early Onset Obesity in Infants and Children: Nature, Nurture or Both? Commentary on Cooke, Hinkley, Chaput & Tremblay, Oken, Paul, Savage, Anzman-Frasca, Birch, and de Silva-Sanigorski & Campbell

Michael I. Goran, PhD, Kelly A. Dumke, MS

Childhood Obesity Research Center, Keck School of Medicine, University of Southern California, USA

July 2012

Introduction

By the year 2030, obesity rates are predicted to rise from about 30% to nearly 42% of the population of the United States.¹ Childhood obesity rates alone have tripled in the last 30 years with 1 out of 3 U.S. children considered obese or overweight, with the latest prevalence of obesity in the nation being 16.9% with higher levels among some minority groups.² Over 10% of U.S. children under the age of two are already obese and 20% of children ages 2 to 5 are overweight.² From a public health perspective, this rise in obesity is associated with earlier onset of associated problems like type 2 diabetes, fatty liver disease, high blood pressure, and heart disease, with exacerbated complications in children including disruption of normal development, psychosocial distress, and long-term health care burdens. Beyond individual co-morbid conditions associated with obesity, the Centre for Disease Control and Prevention (CDC) predicts healthcare costs will top \$550 billion by 2030 if rates continue to grow.¹

This series of six papers³⁻⁸ addresses factors contributing to the development of obesity in early life. While each paper has a distinct approach to childhood obesity prevention, in the end, each gets at the age-old debate: nature versus nurture. This commentary organizes the discussion by examining nature versus nurture approaches to childhood obesity prevention and synthesizes conclusions into a series of policy implications.

Research and Conclusions

Nurture

Chaput and Tremblay³ hypothesize that obesity may be socially determined, viewing the epidemic in children as a symptom of modern living (computer-dependent, sleep-deprived, physically-inactive, stressed), rather than a genetic or pathological process. Focusing on a systems approach to modifying the environment and lifestyle in order to make the “healthy choice the easy one,” the authors focus on interventions targeting key areas of child development: active play and short sleep duration. Similarly, Hinkley⁴ targeted interventions at increasing physical activity and reducing screen-time, and sought to determine the specific individual, social and environmental factors (or “correlates”) that can be the target of interventions. Both studies limit their intervention scope to two specific activities, and recognize the need for more objective measures of success beyond weight and self-reports of activity, screen-time or sleep.

Researchers in Australia have taken system approaches to obesity prevention a step further by examining where specifically obesity prevention efforts will be most effective in a child’s day. Examining preschool-age environments (home, school, healthcare, etc.), de Silva-Sanigorski and Campbell⁵ found home-based or healthcare-based interventions to have a larger effect on *body mass index (BMI)* reductions compared to interventions in educational settings. However, the study lacked longitudinal-data, needed guidance from behavioural theories, and failed to control for parental involvement, which has been shown as a key contributor to excessive weight-gain in preschool years.⁹ In contrast, Paul et al.⁶ focused on parenting during infancy as a means to prevent early obesity by shifting dated clinician recommendations from the prevention of “failure to thrive” (i.e., when the rate of weight gain is significantly lower than that of other children of similar age and gender) to preventing early obesity.

The articles discussed above, not only target key activities, but seek to determine in which part of a child’s day an intervention can be most impactful and which social correlates, particularly parents, can be most influential in obesity prevention. However, while researchers focusing on “nurture-based” or socio-environmental approaches agree that prevention of childhood obesity should be the first line of treatment and incorporate multi-factor environmental approaches, it is important not to discount “nature” and be open to using multi-factor interventions that also account for genetic or prenatal conditions.

Nature

While “nurture” certainly plays a role in obesity and offers more tangible potential solutions, “nature” also likely plays an influential role. In contrast to the previous papers, Cooke⁷

hypothesizes that eating behaviours have a genetic component that may render certain individuals susceptible to environmental food cues. This paper reviews studies that suggest that responsiveness to food, lack of sensitivity to fullness, tendency to find food reinforcing, and higher speeds of eating all have a heritable components. However, the degree to which each eating behaviour is expressed is determined by environmental conditions, emphasizing the role of parents in the development of eating behaviour in young children. The author notes that much research blames parent feeding styles for a child's obesogenic eating behaviour; but new research suggests a bi-directional process in which parents respond to a child's genetic eating style and weight, which are in turn influenced by the environment.

Oken⁸ also suggests a link among body weight, genes and the intrauterine environment but focuses on maternal behaviours during pregnancy. Specifically, this paper reviews data on maternal prenatal obesity, excess weight gain, gestational diabetes, and smoking as predictors of later obesity and adverse complications in infants. Previous research suggests a genetic component for obese mothers having obese children; however, new evidence suggests that the obese intrauterine environment also programs body weight. Further research is needed to clarify if it is the intrauterine experiences that program long-term weight gain or if they are just markers for shared genetic traits.

What's missing?

Several other key issues not mentioned in these papers should be highlighted. Childhood obesity in the U.S. is highest in lower-income populations, highlighting the economic contributions to obesity development. In addition, new studies show that early-life critical periods may be more impactful among certain sub-groups of the population, including low-income Hispanic populations where the emergence of childhood obesity is most rapid in the first few years of life.² For example, Hispanic three-year-old children are twice as likely to be overweight or obese compared to blacks or whites.¹⁰ It is important to understand the nature and nurture factors at play in specific sub-groups of the population at greater risk.

In addition, emerging evidence suggests that beyond maternal obesity and smoking, other intra-uterine exposures to certain environmental pollutants or contaminants, termed obesogens or endocrine disruptors, also play a role in the programming of fat cells and life-long susceptibility to obesity.^{11,12} In addition to this new theory, we need to understand more about how genetic variants influence early-onset obesity. While obesity genes have been identified, they only explain a small

portion of the variance, although new genes are being identified that are specific to early development of obesity.¹³

Breastfeeding is another important developmental factor to consider. In developed countries, there is a robust association between breastfeeding, especially extended breastfeeding >12 months and a lower risk of becoming overweight during childhood and adolescence, even after accounting for maternal obesity and family lifestyle behaviours.^{14,15} Finally, there is likely to be important interactions between nature and nurture. For example, evidence shows that breastfeeding protects against the negative influence of the peroxisome proliferator-activated receptor gamma 2 (PPARγ2) Pro 12Ala *polymorphism* on weight gain in the first 18 months of life and BMI in adolescents.^{16,17} Other examples of gene-environment interactions are likely to play a critical role in the early development of obesity.

Another issue that needs to be considered is whether today's food environment of highly processed foods is applicable to infants and children. One particular aspect is the increase in dietary fructose due to the use of high fructose corn syrup as an added sweetener.¹⁸ This high level of fructose consumption may be more problematic for infants and children. Studies show a strong link between high sugar consumption and obesity beginning in infancy.¹⁹ Why? Because from an evolutionary perspective babies and infants are not programmed to handle fructose,²⁰ which is not present in breast milk. Lactose, the principal sugar in mother's milk, is made from glucose and galactose. The metabolic process required to handle fructose only emerges later in development, so this is another reason why promoting breastfeeding may be particularly important to prevent childhood obesity.

Development and Policy Implications

Childhood obesity prevention efforts have generated momentum in the last several years with initiatives spanning across academia, non-profits, local and state governments, and even the White House. The previously-mentioned papers target obesity prevention from diverse perspectives offering a set of policy implications that target systems change, focus on modifiable risk factors, change social norms, improve evaluation measures of success, and reexamine long-held public policies that shape the U.S. food supply.

A systems approach works at different levels of society – individual, interpersonal, organizational, community and public policy – and focuses on modifying environments and lifestyles in order to

make the healthy choice, the easy choice. The policy implications of the majority of the papers targeted the interpersonal and organizational levels. More specifically, at an interpersonal level, researchers believe parents are key to preventing of childhood obesity. Policies are needed to educate parents and help limit sedentary activity, boost sleep and increase physical activity. In addition, at an organizational level, researchers emphasize policies that target healthcare providers and key institutions where children spend time (childcare settings, schools, etc.) and using them to disseminate health education to make parents aware of the increased health risks of prenatal obesity, smoking, gestational diabetes, etc. Utilizing existing infrastructure as a cost-effective means for delivering preventive interventions helps focus efforts on vulnerable age groups at various school, childcare, and home-based settings. In addition, policies targeting lifestyle and eating behaviour modification need to be grounded in evidence-based practice and sound behavioural theory models.

In addition to changing dated social norms like preventing “failure to thrive,” policies need to further address laws for food marketing to children, portion control, and overall public policy towards food subsidies that have created an environment of excess and cheap overeating. Several of the researchers point to the IOM’s recently published “Early Childhood Obesity Prevention Policies” as a key resource for policy design and implementation.²¹ But do these go far enough? The U.S. food supply needs a massive overhaul requiring a paradigm shift to re-align the balance between making healthy fresh foods more available and less expensive, and cheap processed foods more expensive. For example, Farm Bill subsidies and legislation should be reexamined to reduce political and economic incentives that promote items like cheap, nutrient-poor high fructose corn syrup (HFCS). Even a simple change to corn subsidies alone can potentially remove HFCS from the food supply, lessening the obesogenic environment of cheap, super-sized, sugar-sweetened beverages, which are the largest source of calories and added sugar in the American diet.²² Individuals can force industry to make changes by rejecting foods made with high fructose corn syrup. This will also force food and beverage manufacturers to re-consider using HFCS and turn to using natural sugar instead. This might increase the cost of foods and beverages, which will in turn force the food industry to re-consider portion sizes and force consumers to purchase more fresh foods instead of highly processed items. Actions such as this are needed to share the burden of effort for obesity prevention between individuals, federal and state governments, and the ever-expanding food and beverage industry.

In conclusion, childhood obesity prevention requires a multi-faceted approach targeting “nature,” “nurture” and nature-nurture aspects. Policy recommendations need to focus on systems change, modifiable risk factors, social norms, in addition to a much-needed reexamination of food and agricultural policies that shape our nation’s food environment.

References

1. Finkelstein EA, Khavjou OA, Thompson H, et al. Obesity and severe obesity forecasts through 2030. *American journal of preventive medicine* 2012;42:563-70.
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 : the journal of the American Medical Association* 2012;307:483-90.
3. Chaput J-P, Tremblay A. Obesity at an early age and its impact on child development. Rev ed. Fisher JO, topic ed. In: Tremblay RE, Boivin M, Peters RDeV, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development; 2012:1-8. Available at: <http://www.child-encyclopedia.com/documents/Chaput-TremblayANGxp2.pdf>. Accessed July 16, 2012.
4. Hinkley T. Environmental/family/psychosocial influences on physical activity in young children. Fisher JO, topic ed. In: Tremblay RE, Boivin M, Peters RDeV, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development; 2012:1-8. Available at: <http://www.child-encyclopedia.com/documents/HinkleyANGxp1.pdf>. Accessed July 16, 2012.
5. de Silva-Sanigorski A, Campbell K. Obesity prevention in the preschool years. Fisher JO, topic ed. In: Tremblay RE, Boivin M, Peters RDeV, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development; 2012:1-4. Available at: <http://www.child-encyclopedia.com/documents/deSilva-Sanigorski-CampbellANGxp1.pdf>. Accessed July 16, 2012.
6. Paul IM, Savage JS, Anzman-Frasca S, Birch LL. Obesity prevention during infancy: a change of focus. Fisher JO, topic ed. In: Tremblay RE, Boivin M, Peters RDeV, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development; 2012:1-7. Available at: <http://www.child-encyclopedia.com/documents/Paul-Savage-Anzman-Frasca-BirchANGxp1.pdf>. Accessed July 16, 2012.
7. Cooke L. Appetitive traits and weight in children. Fisher JO, topic ed. In: Tremblay RE, Boivin M, Peters RDeV, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development; 2012:1-6. Available at: <http://www.child-encyclopedia.com/documents/CookeANGxp1.pdf>. Accessed July 16, 2012.
8. Oken E. Prenatal origins of obesity: Evidence and opportunities for prevention. Fisher JO, topic ed. In: Tremblay RE, Boivin M, Peters RDeV, eds. *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development; 2012:1-8. Available at: <http://www.child-encyclopedia.com/documents/OkenANGxp1.pdf>. Accessed July 16, 2012.
9. Cromley T, Neumark-Sztainer D, Story M, Boutelle KN. Parent and family associations with weight-related behaviors and cognitions among overweight adolescents. *Journal of Adolescent Health* 2010;47:263-9.
10. Kimbro RT, Brooks-Gunn J, McLanahan S. Racial and ethnic differentials in overweight and obesity among 3-year-old children. *American Journal of Public Health* 2007;97:298-305.
11. Blumberg B, Iguchi T, Odermatt A. Endocrine disrupting chemicals. *Journal of Steroid Biochemistry and Molecular Biology* 2011;127:1-3.

12. Janesick A, Blumberg B. Obesogens, stem cells and the developmental programming of obesity. *International Journal of Andrology* 2012;35:437-48.
13. Bradfield JP, Taal HR, Timpson NJ, et al. A genome-wide association meta-analysis identifies new childhood obesity loci. *Nature Genetics* 2012;44:526-531.
14. Harder T, Bergmann R, Kallischnigg G, Plagemann A. Duration of breastfeeding and risk of overweight: a meta-analysis. *American Journal of Epidemiology* 2005;162:397-403.
15. Arenz S, Ruckerl R, Koletzko B, von Kries R. Breast-feeding and childhood obesity--a systematic review. *Journal of Obesity and Related Metabolic Disorders* 2004;28:1247-56.
16. Mook-Kanamori DO, Steegers EA, Uitterlinden AG, et al. Breast-feeding modifies the association of PPARgamma2 polymorphism Pro12Ala with growth in early life: the Generation R Study. *Diabetes* 2009;58:992-8.
17. Verier C, Meirhaeghe A, Bokor S, et al. Breast-feeding modulates the influence of the peroxisome proliferator-activated receptor-gamma (PPARG2) Pro12Ala polymorphism on adiposity in adolescents: The Healthy Lifestyle in Europe by Nutrition in Adolescence (HELENA) cross-sectional study. *Diabetes care* 2010;33:190-6.
18. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *American Journal of Clinical Nutrition* 2004;79:537-43.
19. Davis JN, Whalley S, Goran MI. Effects of breastfeeding and low sugar sweetened beverage intake on obesity prevalence in Hispanic toddlers. *American Journal of Clinical Nutrition* 2012;95:3-8.
20. Davidson NO, Hausman AM, Ifkovits CA, et al. Human intestinal glucose transporter expression and localization of GLUT5. *American Journal of Physiology* 1992;262:C795-800.
21. Institute of Medicine: Early Childhood Obesity Prevention Policies. 2011. Available at: <http://www.iom.edu/Reports/2011/Early-Childhood-Obesity-Prevention-Policies.aspx>. Accessed July 16, 2012.
22. Institute of Medicine. Accelerating progress in obesity prevention: solving the weight of the nation. (2012) Available at: <http://www.iom.edu/>. Accessed July 16, 2012.

Obesity Prevention During Infancy: A Change of Focus

¹Ian M. Paul, MD, MSc, ²Jennifer S. Savage, PhD, ²Stephanie Anzman-Frasca, PhD, ²Leann L. Birch, PhD

¹Penn State College of Medicine, USA, ²Center for Childhood Obesity Research, Penn State University, USA

April 2012

Introduction

Over the course of human history, feeding practices for newborns and infants have focused on ensuring adequate caloric intake to support adequate growth. These parenting practices evolved due to the possibility that famine was a common risk, and have persisted despite the unlikely occurrence of food scarcity in most of the developed world. Healthcare providers also have focused on ensuring adequate newborn and infant growth, and more specifically avoiding insufficient growth during the neonatal period and infancy, consistently guarding against the possibility of the diagnosis of “failure to thrive.” This however is problematic given that today, in the developed world, food is plentiful, and it is easy to obtain inexpensive, palatable, high-calorie foods even for very young children. Failure to thrive due to too few calories is uncommon, but parenting practices such as encouraging infants to finish bottles, “topping a child off” with extra calories before bedtime, pressuring infants to eat large servings of solid foods, and using food to soothe non-hunger related infant distress have persisted¹ and can result in excessive weight gain during infancy. Healthcare providers also contribute to this problem from the first days of a baby’s life. While giving appropriate guidance to wake newborns every 3-4 hours to feed in the week following birth to prevent excessive neonatal weight loss, clinicians commonly neglect to inform parents to stop this practice once a baby returns to their birth weight and has established a healthy pattern of weight gain. Although most infants return to birth weight by age 2 weeks, a popular reference from the American Academy of Pediatrics recommends waking a baby that sleeps longer than 4-5 hours for a feeding during the first month.² Further, clinicians continue to guard against failure to thrive but despite increasing rates of obesity, are very unlikely to diagnose excessive weight gain or obesity during infancy.³

Subject

In recent decades the prevalence of obesity has increased dramatically among all age groups, including infants and young children.^{4,5} Almost 10% of U.S. children less than 2 years are already obese and over 25% of children between ages 2 and 5 years are overweight.⁵ Across the globe, similar trends exist.⁶ Both overweight and rapid growth during infancy have significant long-term consequences, and are associated with adult obesity and its co-morbidities including hypertension, coronary artery disease and *type 2 diabetes mellitus*.⁷⁻¹⁰

Problems

Because infancy is a critical period of developmental plasticity with long-lasting metabolic and behavioural consequences,^{10,11} interventions developed for delivery during this period may alter long-term risk for obesity and associated co-morbidities. However, while modifiable factors promoting overweight and rapid growth during infancy have been identified,¹²⁻¹⁴ preventive interventions addressing these factors are scant.¹⁵ Further, both parents and healthcare providers must be educated to change their traditional focus from ensuring sufficient growth to recognizing and intervening upon early life obesity and its risk-factors.^{16,17}

Research Context

Although it is a major public health problem with long-term consequences, parents and healthcare providers typically do not see early life obesity or rapid infant weight gain as problematic and instead often consider a chubby baby to be a healthy baby.¹⁸

Key Research Questions

Can the increasing prevalence of early life obesity be reversed?

Given the complex biological, behavioural and socioeconomic factors that interact to result in childhood obesity, what time periods in development should be targeted on both individual and societal levels and what are the most effective intervention components?

1. What model is best for delivering obesity prevention interventions for infants (e.g., primary care-based, home visitation, other)?
2. Can early life metabolic and behavioural influences on obesity be permanently changed via early life interventions?

3. Is pregnancy or infancy the best time to intervene to prevent childhood obesity and its co-morbidities? Could interventions during both periods be synergistic?

Recent Research Results

Prior to 2010, there were no intervention studies demonstrating beneficial effects on weight status of children younger than 2 years.¹⁹ Since then, two pilot studies showed positive effects on weight status of early intervention programs,²⁰ and other trials are in progress.²¹⁻²⁴ In one pilot, nurses assisted mothers in developing parenting skills related to three areas of infant behaviour believed to affect early obesity risk: sleeping, crying and feeding. First-time mothers who intended to breastfeed were randomly assigned to receive a soothe/sleep intervention, an introduction of solids intervention, both interventions, or no interventions. Interventions were delivered via home visits at ages 2-3 weeks and 4-6 months. One hundred and ten mother-infant pairs completed the one-year study. The soothe/sleep intervention focused on strategies to lengthen infant sleep and taught parents soothing strategies to use rather than indiscriminately feeding in response to infant fussing. At age 2-3 weeks, dyads randomized to this intervention were instructed on alternate soothing responses, including swaddling, side or stomach position, shushing, swinging, and (non-nutritive) sucking. Parents were also taught to emphasize day/night differences. The introduction of solids intervention focused on “when,” “how,” and “which” foods to introduce to infants and provided systematic experiences with new foods between ages 4-6 months. Infants receiving both interventions had significantly lower weight-for-length percentiles at 1 year compared to other groups. This result is depicted in Figure 1, where the y-axis depicts weight-for-length percentiles using the World Health Organization (WHO) growth charts.²⁵

The second pilot study assessed a primary care based intervention to promote healthful behaviours among 0-6 month old infants and their mothers including motivational counseling and group parenting workshops.²⁶ Eighty infants completed the 6-month trial with fewer intervention infants being in the highest quartile of weight-for-length *z-score* at 6 months of age (22% vs. 42%).

Research Gaps

While these pilot intervention results are promising, there are additional possible targets for the primary prevention of early life obesity²⁷ including improving dietary content, increasing physical activity, reducing sedentary behaviours, improving sleep hygiene, improving parental understanding of normal infant growth, and improving parental responsiveness to infant cues and infant distress. For most candidate interventions, however, there are insufficient data on their

effectiveness or data to support their reproducibility in and generalizability to different setting, cultures, or populations. Further, there is no evidence that early life prevention of overweight or obesity will lead to long-term prevention of obesity or its co-morbidities.

Conclusions

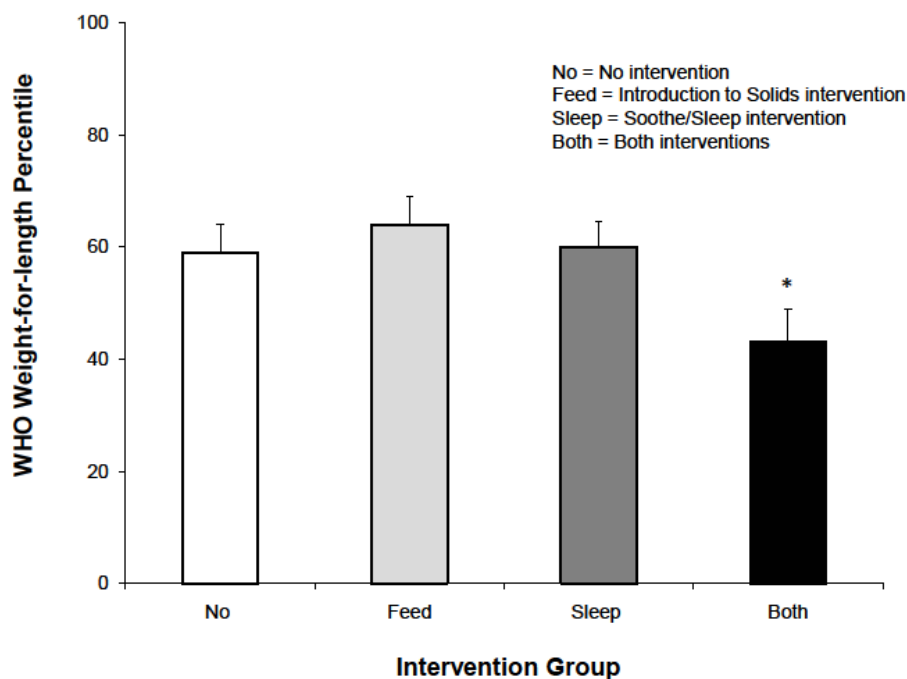
With obesity rates climbing for infants and toddlers globally, interventions are needed for this age group, given the lifelong consequences of early life obesity. Despite the limited evidence to date supporting interventions for obesity prevention during infancy, there has been increasing interest in this topic because infancy is a time of exceptional metabolic and behavioural plasticity. While there are numerous opportunities for intervention, a culture change among parents and pediatric healthcare providers is needed in order to change the primary focus of weight status in infancy from prevention of failure to thrive to the prevention of over-thriving. Societal acknowledgement of this problem and a change in focus by clinicians and parents will be timely as solutions appear to be forthcoming. In the meantime, those interested in the complexity of this topic plus potential solutions can review the Institute of Medicine’s recent publication, *Early Childhood Obesity Prevention Policies*, which focuses on the period from birth to age 5 and includes brief reviews of the evidence for some of these factors influencing early growth in infancy and early childhood.¹⁸

Implications for Parents, Services and Policy

In our current *obesogenic environment*, it has become easier for parents, healthcare providers, and policy makers to view being overweight as normal. Unfortunately, obesity and its co-morbidities may have devastating health effects for individuals and economic effects on a societal level. Failure to recognize overweight infants is common and its consequences can be dire. Fortunately, research into promising interventions is increasing, and policy makers are drawing attention to the problem. First Lady Michelle Obama’s program, “Let’s Move,” aims to solve the childhood obesity epidemic within a generation and includes messages that begin during pregnancy and infancy, such as the promotion of breastfeeding as the preferred source for infant nutrition.²⁸ In addition, the recent Institute of Medicine report on *Early Childhood Obesity Prevention Policies* focuses on preventing obesity during infancy and early childhood.¹⁸ Policy recommendations include promoting growth monitoring and early screening by healthcare professionals to identify infants and young children who may be at risk based on early growth. The report also includes policies designed to impact developing lifestyle behaviours associated with increased obesity risk, including sleep, eating patterns, physical activity and media exposure. The

recommendations are based on current evidence and the evidence-based belief that changing policy can change obesity. The report also highlights the need for research to evaluate the impact of policy recommendations and to develop more effective early interventions. All of these programs and research demonstrate clearly that our society must shift its traditional primary focus from ensuring adequate growth to the preventing obesity.

Figure 1. Infants who received both interventions had lower weight-for-length percentiles at age 1 year (n=110), relative to the WHO growth standards. (Adapted from Paul et al. Preventing Obesity during Infancy: A Pilot Study. Obesity 2011; 19: 353-6120).



References

1. Black MM, Siegel EH, Abel Y, Bentley ME. Home and videotape intervention delays early complementary feeding among adolescent mothers. *Pediatrics* 2001;107:E67.
2. Shelov SP, Hannemann RE, eds. *Caring for your baby and young child: Birth to age 5*. 5th ed: Bantam Books; 2009.
3. McCormick DP, Sarpong K, Jordan L, Ray LA, Jain S. Infant obesity: are we ready to make this diagnosis? *J Pediatr* 2010;157:15-9.
4. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA* 2002;288:1728-32.
5. 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;307:483-90.
6. de Onis M, Blossner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. *Am J Clin Nutr*;92:1257-64.

7. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med* 1993;22:167-77.
8. Stettler N, Zemel BS, Kumanyika S, Stallings VA. Infant weight gain and childhood overweight status in a multicenter, cohort study. *Pediatrics* 2002;109:194-9.
9. Leunissen RW, Kerkhof GF, Stijnen T, Hokken-Koelega A. Timing and tempo of first-year rapid growth in relation to cardiovascular and metabolic risk profile in early adulthood. *JAMA* 2009;301:2234-42.
10. Gluckman PD, Hanson MA, Cooper C, Thornburg KL. Effect of in utero and early-life conditions on adult health and disease. *N Engl J Med* 2008;359:61-73.
11. Gluckman PD, Hanson MA. Developmental and epigenetic pathways to obesity: an evolutionary-developmental perspective. *Int J Obes (Lond)* 2008;32 Suppl 7:S62-71.
12. Paul IM, Bartok CJ, Downs DS, Stifter CA, Ventura AK, Birch LL. Opportunities for the Primary Prevention of Obesity during Infancy. *Adv Pediatr* 2009;56:107-33.
13. Gillman MW, Rifas-Shiman SL, Kleinman K, Oken E, Rich-Edwards JW, Taveras EM. Developmental origins of childhood overweight: potential public health impact. *Obesity (Silver Spring)* 2008;16:1651-6.
14. Anderson SE, Whitaker RC. Household Routines and Obesity in US Preschool-Aged Children. *Pediatrics* 2010;125:420-8.
15. Hesketh KD, Campbell KJ. Interventions to Prevent Obesity in 0-5 Year Olds: An Updated Systematic Review of the Literature. *Obesity (Silver Spring)* 2010;18:S27-S35.
16. Laraway KA, Birch LL, Shaffer ML, Paul IM. Parent perception of healthy infant and toddler growth. *Clin Pediatr (Phila)* 2010;49:343-9.
17. Sullivan SA, Leite KR, Shaffer ML, Birch LL, Paul IM. Urban parents' perceptions of healthy infant growth. *Clin Pediatr (Phila)*;50:698-703.
18. Birch LL, Parker L, Burns A, eds. *Early Childhood Obesity Prevention*. Washington, D.C.: Institute of Medicine; 2011.
19. Ciampa PJ, Kumar D, Barkin SL, et al. Interventions aimed at decreasing obesity in children younger than 2 years: a systematic review. *Arch Pediatr Adolesc Med*;164:1098-104.
20. Paul IM, Savage JS, Anzman SL, et al. Preventing obesity during infancy: a pilot study. *Obesity (Silver Spring)* 2011;19:353-61.
21. Wen LM, Baur LA, Rissel C, Wardle K, Alperstein G, Simpson JM. Early intervention of multiple home visits to prevent childhood obesity in a disadvantaged population: a home-based randomised controlled trial (Healthy Beginnings Trial). *BMC Public Health* 2007;7:76.
22. Campbell K, Hesketh K, Crawford D, Salmon J, Ball K, McCallum Z. The Infant Feeding Activity and Nutrition Trial (INFANT) an early intervention to prevent childhood obesity: cluster-randomised controlled trial. *BMC Public Health* 2008;8:103.
23. Wen LM, Baur LA, Rissel C, et al. Healthy Beginnings Trial Phase 2 study: Follow-up and cost-effectiveness analysis. *Contemp Clin Trials* 2011.
24. Daniels LA, Magarey A, Battistutta D, et al. The NOURISH randomised control trial: positive feeding practices and food preferences in early childhood - a primary prevention program for childhood obesity. *BMC Public Health* 2009;9:387.
25. Grummer-Strawn LM, Reinold C, Krebs NF. Use of World Health Organization and CDC growth charts for children aged 0-59 months in the United States. *MMWR Recomm Rep* 2010;59:1-15.
26. Taveras EM, Blackburn K, Gillman MW, et al. First steps for mommy and me: a pilot intervention to improve nutrition and physical activity behaviors of postpartum mothers and their infants. *Matern Child Health J* 2010;15:1217-27.
27. Paul IM, Bartok CJ, Downs DS, Stifter CA, Ventura AK, Birch LL. Opportunities for the primary prevention of obesity during infancy. *Adv Pediatr* 2009;56:107-33.

28. Wojcicki JM, Heyman MB. Let's Move--childhood obesity prevention from pregnancy and infancy onward. *N Engl J Med* 2010;362:1457-9.